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Scientific Understandings of Postpartum Illness: Improving Health Law and Policy?

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SCIENTIFIC UNDERSTANDINGS OF POSTPARTUM ILLNESS: IMPROVING HEALTH LAW AND POLICY?

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In its broadest sense, this Article examines the relationship between science and the law in the context of postpartum illness.[1] From classical antiquity to the present day, physicians and scientists have investigated the causes, correlates, and consequences of the depressions and psychoses that develop in some women following their transition to motherhood.[2] The scientific investigation of postpartum illness has been characterized by an open-ended search for knowledge with the recognition that scientific findings published one day are subject to revision the next.[3] Legislators and judges also have sought to understand postpartum illness as necessary to make laws that affect and adjudicate disputes involving new mothers,[4] although legal inquiries regarding postpartum illness have yielded factual findings that are fixed in time.[5] When the results of scientific research and legal research are compared in the context of postpartum illness, several differences emerge,[6] perhaps because science and law share neither a common process nor common goals.[7] This Article resolves these differences and develops a new organizing principle for understanding illness; that is, a unified health law framework that limits distinctions between physical and mental illness.

This Article has three Parts. Part I examines the scientific understanding of postpartum illness from 400 B.C. to the present day. From classical antiquity to the late nineteenth century, scientific understandings of postpartum illness were based primarily on observed temporal relationships between childbirth or lactation and the symptoms of mental illness.[8] At the turn of the twentieth century, psychoanalytic theories of postpartum illness, including theories that explained causality in terms of female intra-psychic

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[1] As discussed in more detail at text accompanying note 27, *infra*, postpartum depression is characterized by sadness, crying, self-blame, loss of control, irritability, anxiety, tension, and sleep difficulties that may develop in new mothers between two weeks and six months postpartum and that may last as long as six to twelve months. Postpartum psychosis is characterized by extreme emotional liability, mania, disorganization, and/or the experience of hallucinations and delusions with an onset of three to fourteen days postpartum. *See infra* text accompanying note 28.

[2] *See infra* Part I.

[3] *See infra* Part I; *see also* Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 590 (1993) ("Science is not an encyclopedic body of knowledge about the universe. Instead, it represents a *process* for proposing and refining theoretical explanations about the world that are subject to further testing and refinement.") (emphasis in original) (internal references and citations omitted); NAT'L RESEARCH COUNCIL, A CONVERGENCE OF SCIENCE AND LAW: A SUMMARY REPORT OF THE FIRST MEETING OF THE SCIENCE, TECHNOLOGY, AND LAW PANEL 1 (2001) [hereinafter NRC] (discussing the process of fact finding in science).

[4] *See infra* Part II.

[5] *See infra* Part II; *see also* Vern R. Walker, *Epistemic and Non-epistemic Aspects of the Factfinding Process in Law*, 5 J. PHIL., SCI. & L., 1–2 (2005), http://www.psljournal.com/archives/all/walkerpaper.cfm (examining the process of fact finding in law).

[6] *See infra* Part II.

[7] *See, e.g.*, David Goodstein, *How Science Works*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 67, 80–81 (2nd ed. 2000).

[8] *See infra* Part I(A).

conflict surrounding gender and interpersonal relationships and roles, gained popularity.[9] In the second half of the twentieth century, unicausal hormone-based theories[10] were followed by theories explaining causality in terms of a reduction in neurotransmitters[11] and the influence of psychosocial and sociocultural factors.[12] In the first decade of the twenty-first century, a number of neuroanatomical and neurofunctional findings[13] as well as genetic findings[14] and evolutionary theories[15] contributed to the scientific understanding of postpartum illness. Part I concludes by summarizing postpartum illness's current (and still incomplete) scientific understanding and outlining a contemporary multifactorial model for postpartum illness that gives weight to hormonal, neurochemical, neuroanatomical, genetic, evolutionary, psychosocial, sociocultural, and other biological and environmental factors.[16] Because Part I provides scientific support for the legal arguments presented in Parts II and III, Part I is necessarily technical.

Part II examines the legal understanding, incorporation, and utilization of postpartum illness. Part II finds that a range of past, current, and proposed legal authorities, including criminal infanticide laws, judicial opinions interpreting health insurance policy provisions, and postpartum awareness laws expressly reference postpartum illness, its probable causes, and its observed effects.[17] Part II discovers that the science lawmakers use to support and interpret postpartum law and policy is not always accurate,[18] and suggests that lawmakers who rely on outdated scientific findings risk developing inappropriate postpartum laws and policies, encouraging the introduction of expert testimony that will not meet evidentiary standards for use in litigation, establishing conflicts between different health laws and policies, and supporting the public misunderstanding of postpartum illness.[19] Part II concludes by arguing that lawmakers, judges, and other stakeholders need to recognize the complexity of illness etiology, including the etiology of postpartum illness.[20] To assist with these efforts, Part II proposes a contemporary legal explanation of postpartum illness that emphasizes the illness's incomplete understanding and likely multifactorial etiology.[21] Part II also proposes the repeal or reformulation of outdated common law tests designed to distinguish physical and mental illness.

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[9] *See infra* Part I(B).
[10] *See infra* Part I(C).
[11] *See infra* Part I(D).
[12] *See infra* Part I(H).
[13] *See infra* Part I(E).
[14] *See infra* Part I(F).
[15] *See infra* Part I(G).
[16] *See infra* Part I(I).
[17] *See infra* Parts II(A), II(B), and II(C).
[18] *See infra* Parts II(A), II(B), and II(C).
[19] *See infra* Part II(D).
[20] *See infra* Part II(D).
[21] *See infra* Part II(D).

Part III demonstrates that advances in the scientific understanding of postpartum illness combined with recent developments in mental health parity and disability discrimination law may impact future litigation in favor of some women with postpartum illness.[22] Using mental health parity and disability discrimination law as a platform, Part III also questions the appropriateness of health law frameworks that are built on binary understandings of illness as well as the legal consequences that attach to judicial findings of mental versus physical illness.[23] Part III concludes by proposing a limited, legal merger of physical and mental illness.[24]

A note regarding the diagnostic terms used in this Article: health professionals currently recognize three types of postpartum conditions including the postpartum blues, postpartum depression, and postpartum psychosis. The postpartum blues affect a reported 30% to 80% of new mothers and may be described as a transitory, natural reaction to the sequelae of childbirth that is characterized by increased emotionality, irritability, anxiety, and tearfulness.[25] The postpartum blues typically last one to two weeks following childbirth and may peak at postpartum days three, four, and five.[26] The scientific and legal understanding of the postpartum blues is not at issue in this Article.

Postpartum depression, the second most common postpartum illness, affects a reported 3% to 30% of new mothers and is characterized by sadness, crying, self-blame, loss of control, irritability, anxiety, tension, and sleep difficulties that develop between two weeks and six months postpartum and may last as long as six to twelve months after childbirth.[27] Postpar-

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[22] *See infra* Parts III(A) and III(B).

[23] *See infra* Part III(C).

[24] *See infra* Part III(C).

[25] The characteristics, time of onset, length of illness, and percentage of women affected by the postpartum blues vary depending on the source of information. *See* AM. PSYCHIATRIC ASS'N, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS 423 (4th ed., text rev. 2000) [hereinafter DSM-IV-TR] (citing a 70% prevalence); IRIS F. LITT, TAKING OUR PULSE: THE HEALTH OF AMERICA'S WOMEN 182 (1997) (citing a 50–80% prevalence); Joan C. Chrisler & Ingrid Johnston-Robledo, *Raging Hormones? Feminist Perspectives on Premenstrual Syndrome and Postpartum Depression*, *in* RETHINKING MENTAL HEALTH AND DISORDER: FEMINIST PERSPECTIVES 174, 180 (Mary Ballou & Laura S. Brown eds., 2002) (citing a 50–80% prevalence); Maureen J. Giovannini, *The Relevance of Gender in Postpartum Emotional Disorders*, *in* GENDER CONSTRUCTS AND SOCIAL ISSUES 209, 211 (Tony L. Whitehead & Barbara V. Reid eds., 1992) (citing a 60–80% prevalence); Lucy Martinez-Schallmoser, *Postpartum Depression in Hispanic Women*, *in* ANNUAL REVIEW OF WOMEN'S HEALTH VOLUME II 243, 245 (Beverly J. McElmurry & Randy Spreen Parker eds., 1995) (citing a 50–80% prevalence); Jo Ann Rosenfeld, *Depression and Premenstrual Syndrome*, *in* HANDBOOK OF WOMEN'S HEALTH: AN EVIDENCE-BASED APPROACH 437, 452 (Jo Ann Rosenfeld ed., 2001) (citing a 30–75% prevalence).

[26] *See* DSM-IV-TR, *supra* note 25, at 423; LITT, *supra* note 25, at 182; Chrisler, *supra* note 25, at 180; Giovannini, *supra* note 25, at 211; Martinez-Schallmoser, *supra* note 25, at 245; Rosenfeld, *supra* note 25, at 452.

[27] Chrisler, *supra* note 25, at 180. Although current diagnostic classification manuals classify depression as "postpartum" only when its onset occurs within the first four weeks following childbirth, many researchers and clinicians believe that the postpartum

tum psychosis, the third—and most serious and least common—postpartum illness, affects a reported 0.1% to 2.0% of new mothers and is characterized by extreme emotional lability, mania, disorganization, and/or the experience of hallucinations and delusions with an onset of three to fourteen days postpartum.[28] The scientific and legal understandings of postpartum depression and postpartum psychosis are at issue in this Article. Hereinafter this Article will refer to either postpartum depression or postpartum psychosis, as appropriate and as defined herein, unless the reference is to both postpartum depression and postpartum psychosis, in which case the generic phrase "postpartum illness" is used.

I. Scientific Understandings of Postpartum Illness

A. Early Medical Observations

For centuries, physicians have observed a temporal relation between the occurrence of female-specific reproductive events and the symptoms of illness that traditionally have been classified as "mental."[29] One of the earliest recorded medical observations of postpartum illness occurred in 400 B.C., when Greek physician Hippocrates described a severe case of insomnia and restlessness that began on the sixth day in a woman who bore twins.[30] In the eleventh century, Italian physician Trotula di Ruggiero documented a link between uterine functions and melancholia: "[I]f the womb is too moist, the brain is filled with water & the moisture running over to the eyes compels them involuntarily to shed tears."[31] In the eighteenth century, German phy-

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period can last up to six months or even a year following childbirth. *See* DSM-IV-TR, *supra* note 25, at 422; *see, e.g.*, Shaila Kulkarni Misri, Pregnancy Blues: What Every Woman Needs to Know about Depression During Pregnancy 191 (2005). For wide-ranging descriptions of the characteristics, time of onset, length of illness, and percentage of women affected by postpartum depression, see, for example, Litt, *supra* note 25, at 183; Giovannini, *supra* note 25, at 211; Martinez-Schallmoser, *supra* note 25, at 245; Rosenfeld, *supra* note 25, at 452–53.

[28] For various descriptions of the characteristics, time of onset, length of illness, and percentage of women affected by postpartum psychosis see, for example, Litt, *supra* note 25, at 183; Chrisler, *supra* note 25, at 179–80; Giovannini, *supra* note 25, at 211; Martinez-Schallmoser, *supra* note 25, at 245; Rosenfeld, *supra* note 25, at 453.

[29] *See, e.g.*, Ilza Veith, Hysteria: The History of a Disease 1 (1965) (examining the historical development of hysteria, including its nature, origins, symptoms, and management); s*ee also* Martinez-Schallmoser, *supra* note 25, at 247 (discussing early observations and documentation of mental illness in women experiencing reproductive events).

[30] *See, e.g.*, Michael W. O'Hara & Scott Stuart, *Pregnancy and Postpartum*, *in* Psychiatric Treatment of the Medically Ill 253, 253 (Robert G. Robertson & William R. Yates eds., 1999) (referencing the story); George Stein, *Perinatal Psychiatric Disorders*, *in* Seminars in General Adult Psychiatry 635, 635 (George Stein & Greg Wilkinson eds., 2d ed. 2007) (referencing the story); Margaret G. Spinelli, *Psychiatric Disorders During Pregnancy and Postpartum*, 53 J. Am. Med. Women's Ass'n. 165, 165 (1998) (referencing Hippocrates's documentation of postpartum illness).

[31] Meir Steiner, Kimberly A. Yonkers, & Elias Eriksson, *Preface to* Mood Disorders in Women, at xv (Meir Steiner, Kimberly A. Yonkers, & Elias Eriksson eds., 2000); Rudy

sician Berger published a thesis referring to a "lacteal irritation [that] sometimes falls upon the brain, either immediately after confinement, or at the period of the lacteal revolution."[32] According to Berger, "Some who have been confined . . . have a fixed pain in the head; others are in a stupor, have a dull expression, and reason falsely."[33]

The first comprehensive studies of postpartum illness were conducted in the nineteenth century and tended to categorize postpartum illness as pregnancy-related (if occurring during gestation), puerperal (if occurring within six weeks of childbirth), or lactational (if occurring after six weeks of childbirth).[34] In 1838, French psychiatrist Jean-Etienne-Dominique Esquirol published his famous volume *Des Maladies Mentales*, which included an essay examining the *Mental Alienation of Those Recently Confined and of Nursing Women*.[35] Based on his years of experience in private psychiatric practice and in treating women at Salpêtrière, the famous Parisian teaching hospital,[36] Esquirol believed that postpartum illness was caused by a woman's failure to nurse, or suppression of her milk: "[I]nsanity manifests itself most frequently, among women who do not nurse."[37] Esquirol concluded by recommending treatment, not punishment, for women with postpartum illness.[38]

Nydegger, *Postpartum Depression: More than the "Baby Blues"?, in* 3 MENTAL DISORDERS OF THE NEW MILLENNIUM 1, 2 (Thomas G. Plante ed., 2006).

[32] *See* E. ESQUIROL, DES MALADIES MENTALES 127 (E. K. Hunt trans., Philadelphia, Lea, and Blanchard 1845) (1838) (referencing Berger's thesis and findings).

[33] *Id.*

[34] *See, e.g.*, JOHN MACPHERSON, MENTAL AFFECTIONS: AN INTRODUCTION TO THE STUDY OF INSANITY 47 (1899) (categorizing postpartum illness by "pregnancy," "parturition," or "lactation"); Hugh F. Butts, *Post-Partum Psychiatric Problems: A Review of the Literature Dealing with Biological Theories*, 61 J. NAT'L MED. ASS'N 136, 136 (1969) (noting the nineteenth-century classification of postpartum illnesses as "puerperal" or "lactational").

[35] ESQUIROL, *supra* note 32, at 125–43.

[36] *Id.* at 125.

[37] *Id.* at 129–30.

> Here are very naturally presented, two interesting questions, which for a long time furnished matter for debate. 1. Is the suppression or diminution of the milk, the cause or effect of mental alienation? We may reply, that insanity manifests itself most frequently, among women who do not nurse. Of our ninety-two insane women, twenty-nine were single, and sixty-three married. Now single women rarely nurse. The greatest number of facts, prove, that the milk diminishes, is suppressed, or loses its distinctive qualities, before the explosion of delirium; but there are observations also, which furnish undeniable proof, that the suppression or diminution of milk, takes place only after the explosion of insanity.

Id.

[38] *Id.* at 132.

> It is unnecessary to remark, that women who have become insane in consequence of confinement and lactation, ought to be submitted to the same general principles of treatment, with other insane persons; that isolation, the aids of hygiene, and moral means, ought not to be neglected, and that these alone have sufficed to effect a cure, although more rarely, than in other forms of mental alienation.

Id.

George Fielding Blandford, a British physician and Fellow of the Royal College of Physicians in London, also spoke of connections between childbirth, lactation, and mental illness in a series of lectures given at St. George's Hospital during the second half of the nineteenth century.[39] In a lecture entitled "The Pathology of Insanity," Blandford formally recognized a "puerperal insanity," the symptoms of which "may first be noticed during labor or immediately after, within a week, within a month, two months, six months—in fact, at almost any period within a twelvemonth."[40] Blandford also recognized an "insanity of lactation" that appeared within a few months of childbirth, but disagreed with Esquirol's failure-to-nurse and suppression-of-milk theories, focusing instead on the negative effects of excessive nursing. According to Blandford, "insanity of lactation" was due to "anaemia brought about by prolonged suckling, or by the mother making undue efforts to nurse, and so overtaxing her strength."[41]

Perhaps confused by Esquirol's and Blandford's competing theories, physicians writing at the turn of the century attributed postpartum illness to both lactation as well as the cessation of lactation. John MacPherson, a British physician who gave several lectures on "Mental Diseases" at the Royal Colleges School of Medicine in Edinburgh, described his understanding of postpartum illness in 1899.[42] According to MacPherson, postpartum illness can be caused both by lactation, which produces a "physical exhaustion, accompanied by malnutrition and anemia," as well as the "sudden cessation, of any cause, from the milk secretion."[43]

Notwithstanding these early observations, today's physicians and scientists generally agree that neither lactation nor the cessation thereof causes postpartum illness,[44] although no consensus has emerged regarding the exact role or roles played by genetic, biological, and environmental factors.[45]

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[39] George Fielding Blandford, Insanity and its Treatment: Lectures on the Treatment, Medical and Legal, of Insane Patients v (3rd ed., New York, William Wood & Co. 1886).

[40] *Id.* at 39; *see also id.* at vii (noting listings of "insanity of pregnancy" and "puerperal insanity" within Lecture III).

[41] *Id.* at 39. In 1893, George H. Rohé, an American physician and insane asylum superintendent, agreed that prolonged or excessive lactation was probably the chief cause of insanity occurring during the extended nursing period (and that sepsis was probably the cause of insanity in the immediate postpartum). *See* George H. Rohé, *Lactational Insanity*, 21 JAMA 325, 325 (1893).

[42] *See* MacPherson, *supra* note 34, at v.

[43] *Id.* at 48.

[44] *See* Ann Dunnewold & Jeannette Crenshaw, *Breastfeeding and Postpartum Depression: Is There a Connection?,* 15 Breastfeeding Abstracts 25, 25 (May 1996) (exploring the connection between breastfeeding and postpartum illness and concluding that breastfeeding does not increase a woman's risk of postpartum depression or anxiety); J. C. Ingram, R. J. Greenwood & M. W. Woolridge, *Hormonal Predictors of Postnatal Depression at 6 Months in Breastfeeding Women*, 21 J. Reprod. & Infant Psychol. 61, 62 (2003) (referencing Dunnewold and Crenshaw's findings); text accompanying notes 182–183, *infra*.

[45] *See infra* Parts I(C)–(I).

B. Psychoanalytic Theories

During the first half of the twentieth century, psychoanalytic theories of postpartum illness, including those that explained causality in terms of female intra-psychic conflict surrounding gender and interpersonal relationships and roles, gained popularity.[46] Gregory Zilboorg, a Russian-born psychoanalyst and historian of psychiatry who eventually established a psychoanalytic practice in New York City, stated his belief in 1928 that postpartum psychosis was the result of an "unresolved positive Oedipus situation, or with its anal regressive resultant—father identification."[47] American psychiatrist Martin Orens described in 1955 a case of postpartum depression highlighting female castration conflicts.[48] American psychoanalyst Gilbert Rose described in 1962 a case of postpartum depression in which "depression was largely based on need for punishment for the unconscious oedipal gratification of the transference pregnancy and birth."[49] Robert Daniels and Herbert Lessow, two mid-twentieth century American psychiatrists, explained their understanding in 1964 that women with postpartum illness "anticipate being treated as dependent children rather than adults. They can accept only limited responsibility at home and find it difficult or impossible to function outside the home."[50] Dutch psychoanalyst Hendrika Halberstadt-Freud argued in 1993 that a "woman's unresolved *symbiotic illusion* with her mother plays a central role in postpartum depression."[51] These and other psychoanalytic theories tend to place fault on the mother for her unconscious failure to achieve a mature femininity.[52]

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[46] *See, e.g.*, Giovannini, *supra* note 25, at 213 (discussing psychoanalytic theories of postpartum illness).

[47] Gregory Zilboorg, *Post-Partum Schizophrenias*, 68 J. NERVOUS & MENTAL DISEASE 370, 383 (1928); *see also* Butts, *supra* note 34, at 137 (referencing Zilboorg's findings).

[48] Lawrence D. Blum, *Psychodynamics of Postpartum Depression*, 24 PSYCHOANALYTIC PSYCHOLOGY 45, 51 (2007) (citing Martin H. Orens, *Setting a Termination Date—An Impetus to Analysis*, 3 JOURNAL OF THE AMERICAN PSYCHOANALYTIC ASSOCIATION 651-665 (1955)).

[49] Blum, *supra* note 48, at 51 (quoting Gilbert J. Rose, *Unconscious Birth Fantasies in the Ninth Month of Treatment*, 10 JOURNAL OF THE AMERICAN PSYCHOANALYTIC ASSOCIATION 677–88 (1962)).

[50] Robert Daniels & Herbert Lessow, *Severe Postpartum Reactions*, 5 PSYCHOSOMATICS 21, 24 (1964). *See generally* Clifford K. Dorne, *Psychodynamics Versus Endocrinology in Postpartum Psychosis: A Critique of the Diminished Capacity Test in Infanticide Cases*, 16 AM. J. CRIM. JUST. 16, 23 (1991) (placing Daniels and Lessow's understanding of postpartum illness in historical context).

[51] Blum, *supra* note 48, at 51 (citing Hendrika C. Halberstadt-Freud, *Postpartum Depression and Symbiotic Illusion*, 10 PSYCHOANALYTIC PSYCHOLOGY 407–23 (1993)).

[52] Giovannini, *supra* note 25, at 213.

C. Hormone-Based Theories

Although psychoanalytic theories of postpartum illness gained popularity in the first half of the twentieth century, biological theories, especially unicausal hormone-based theories, began to emerge in the second half of the twentieth century.[53] Hormones are chemical messengers produced by the endocrine glands that are released into the bloodstream and transported to distant parts of the body, where they exert a specific effect for the benefit of the body as a whole.[54] The three main sex hormones (estrogen, progesterone, and testosterone) are derived from cholesterol through a series of chemical reactions.[55] Changes in the levels of sex hormones in the human body allow for conception, pregnancy, and childbirth.[56] During pregnancy, for example, a woman's estrogen and progesterone levels drastically increase to allow for the maintenance of pregnancy.[57] Within three days of childbirth, a woman's estrogen and progesterone levels return to pre-pregnancy levels.[58]

Historically, clinicians and scientists observed a temporal relation between the onset and recurrence of depression in women and the times at which hormone levels were known to fluctuate significantly, including during the reproductive events of puberty, pregnancy, childbirth, and menopause.[59] It is not surprising, then, that many clinicians, scientists, medical journalists, and scholars emphasize the centrality of hormones when describ-

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[53] *See, e.g.*, Gwen Stern & Laurence Kruckman, *Multi-Disciplinary Perspectives on Postpartum Depression: An Anthropological Critique*, 17 SOC. SCI. AND MED. 1027, 1030 (1983) (discussing the rise of hormone-based theories of postpartum illness in the middle of the twentieth century).

[54] *See, e.g.*, LAURALEE SHERWOOD, FUNDAMENTALS OF PHYSIOLOGY 94 (3rd ed. 2006).

[55] *See, e.g.*, MISRI, *supra* note 27, at 35; Idit Oren, Sarel J. Fleishman, Amit Kessel & Nir Ben-Tal, *Free Diffusion of Steroid Hormones Across Biomembranes: A Simplex Search with Implicit Solvent Model Calculations*, 87 BIOPHYSICAL J. 768, 768 (2004).

[56] *See, e.g.*, MISRI, *supra* note 27, at 35–37.

[57] *See, e.g.*, *id.*

[58] *See, e.g.*, Elka Serrano & Julia K. Warnock, *Depressive Disorders Related to Female Reproductive Transitions*, 20 J. PHARMACY PRACTICE 385, 388 (2007) ("Women in the postpartum period experience drastic changes in both progesterone and estrogen levels. Estradiol may increase 50-fold before it drops to follicular phase levels on the third day postpartum."); Claudio N. Soares & Brook Zitek, *Reproductive hormone sensitivity and risk for depression across the female life cycle: A continuum of vulnerability?*, 33 J. PSYCH. NEUROSCIENCE 331, 333 (2008) ("The postpartum period is a time of abrupt decreases in the amounts of circulating estrogen and progesterone."); Spinelli, *supra* note 30, at 165 ("Estrogen and progesterone levels rise gradually during gestation, and with the loss of placenta at delivery, plummet within 24 to 48 hours.").

[59] *See, e.g.*, Lorah D. Dorn & George P. Chrousos, *The Neurobiology of Stress: Understanding Regulation of Affect During Female Biological Transitions*, 15 SEMINARS IN REPROD. ENDOCRINOLOGY 19, 29 (1997) ("Certainly, studying hormone-behavior relations during the biological transition of pregnancy is congruent with the knowledge of the multiple endocrine changes of pregnancy and the incidence of postpartum mood disorders and depression."); Soares, *supra* note 58, at 331 ("It has been hypothesized that women presenting with episodes of depression associated with reproductive events (i.e., premenstrual, postpartum, menopausal transition) may be particularly prone to experiencing depression, in part because of a heightened sensitivity to intense hormonal fluctuations.").

ing the etiology of postpartum illness.[60] Perhaps the most popular theory is that the sharp drop of estrogen and progesterone to pre-pregnancy levels within three days of childbirth causes postpartum illness.[61] In one well-known study published in 2000, a group of American scientists affiliated with the National Institutes of Health investigated the effects of withdrawing estrogen and progesterone in women with and without a history of postpartum depression following an artificial eight-week treatment of the two hormones.[62] The scientists found that 62.5% of the women with a history of postpartum depression and none of the control subjects developed significant mood symptoms during the withdrawal period.[63] The scientists concluded that they had "provided direct evidence in support of the involvement of the reproductive hormones in the development of postpartum depression," and that women with a history of postpartum depression are "differentially sensitive to mood-destabilizing effects of gonadal steroids."[64]

Scientists continue to investigate the relationship between neuroendocrine changes and postpartum illness.[65] In 2009, for example, a

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[60] *See, e.g.*, Chrisler, *supra* note 25, at 181–82 (discussing the centrality of hormones in early biomedical models of postpartum illness); Giovannini, *supra* note 25, at 212.

[61] *See, e.g.*, CAROL DIX, THE NEW MOTHER SYNDROME: COPING WITH POSTPARTUM STRESS AND DEPRESSION 22 (1985).

> We cannot have a hormonal change without a change in biochemistry and without some mental change. In searching for the root cause of the bodily and psychic upheaval [of postpartum psychosis], we must turn to the relatively new science of endocrinology and ongoing studies attempting to associate hormonal changes and depression.

Id. See also, e.g., Elizabeth J. Corwin & Kathleen Pajer, *The Psychoneuroimmunology of Postpartum Depression*, 17 J. WOMEN'S HEALTH 1529, 1530 (2008) (noting that acute changes in reproductive hormone levels have been proposed as a risk factor for postpartum depression); Giovannini, *supra* note 25, at 212 (noting that the postpartum drop in progesterone has been linked to postpartum illness); Serrano, *supra* note 58, at 388 ("Women in the postpartum period experience drastic changes in both progesterone and estrogen levels. Estradiol may increase 50-fold before it drops to follicular phase levels on the third day postpartum. This neurobiologic stress likely plays a role in the vulnerability to postpartum psychosis, along with other psychosocial factors . . . ."); Spinelli, *supra* note 30, at 165 ("Estrogen and progesterone levels rise gradually during gestation, and with the loss of placenta at delivery, plummet within 24 to 48 hours. This precipitous drop is the first step in the sequence of biological events that may trigger psychiatric symptoms in vulnerable women."); VALLEY WOMEN'S HEALTH ACCESS PROGRAM, WOMEN'S HEALTH COORDINATION CENTER AT GRIFFIN HOSPITAL, POSTPARTUM DEPRESSION (PPD) 1 (2004), http://vwhcc.org/whcc_html/docs/postpartum.pdf ("One study concluded that women with the greatest drop in progesterone levels after delivery were more likely to rate themselves depressed within 10 days of delivery.").

[62] *See* Miki Bloch, Peter J. Schmidt, Merry Danaceau, Jean Murphy, Lynette Nieman & David R. Rubinow, *Effects of Gonadal Steroids in Women with a History of Postpartum Depression*, 157 AM. J. PSYCH. 924, 924 (2000).

[63] *Id.* at 926.

[64] *Id.* at 924; *see also id.* at 928.

[65] *See, e.g.*, Corwin, *supra* note 61, at 1530–31 (reviewing how pregnancy-related changes in the innate immune system and the hypothalamic-pituitary-adrenal (HPA) axis may contribute to the development of postpartum depression); Pietro Grussu, Maria T. Nasta & Rosa M. Quatraro, *Serum Cholesterol Concentrations and Distress in the Initial Days After Childbirth*, 151 PSYCH. RESEARCH 159, 159 (2007) (casting doubt on the hy-

group of American scientists published their study of a hypothesized link between placental corticotropin-releasing hormone ("pCRH") and postpartum depression.[66] The scientists assessed pCRH in blood samples taken at 15, 19, 25, 31, and 37 weeks gestational age in 100 adult women with a single pregnancy and then measured depressive symptoms using a standardized questionnaire at the last four pregnancy visits and postpartum.[67] The scientists found that pCRH was a strong predictor at 25 weeks gestational age of postpartum depression in the 16 women who developed symptoms of postpartum depression[68] and concluded that pCRH is a "sensitive and specific early diagnostic test for [postpartum depression] symptoms."[69] Given these findings, it is not surprising that several medical news articles have reported that a hormone screening test for postpartum depression, perhaps to be called the "pCRH PPD Screen," could be incorporated into standard prenatal care at 24 to 28 weeks gestation.[70]

Although hormone-based theories of postpartum illness continue to enjoy widespread acceptance in many medical circles,[71] contrary findings and models also have been reported and published,[72] including findings that the

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pothesis of a possible association between cholesterol and depression in the general population and in mothers who have just given birth); M. Kammerer, A. Taylor & V. Glover, *The HPA Axis and Perinatal Depression: A Hypothesis*, 9 ARCHIVES WOMEN'S MENTAL HEALTH 187, 187 (2006) (suggesting that the physiologic changes of the HPA axis in pregnancy may contribute to depression); Alfonso Troisi, Anna Moles, Lea Panepuccia, Domenica Lo Russo, Giampaolo Palla & Stefano Scucchi, *Serum Cholesterol Levels and Mood Symptoms in the Postpartum Period*, 109 PSYCH. RESEARCH 213, 213 (2002) (study concluding that the physiological fall in blood lipids in the postpartum period can be a useful model to test the relationship between serum cholesterol levels and mood).

[66] Ilona S. Yim, Laura M. Glynn, Christine Dunkel Schetter, Calvin J. Hobel, Aleksandra Chicz-DeMet & Curt A. Sandman, *Risk of Postpartum Depressive Symptoms with Elevated Corticotropin-Releasing Hormone in Human Pregnancy*, 66 ARCHIVES OF GEN. PSYCH. 162, 162 (2009).

[67] *Id.* at 163–64.

[68] *Id.* at 162, 165–66.

[69] *Id.* at 167.

> These data are, to our knowledge, the first to suggest a sensitive period in mid-pregnancy during which pCRH, as measured in maternal plasma, is a moderate and independent predictor of PPD symptoms. We propose that pCRH during this period may serve as a sensitive and specific early diagnostic test to identify women at high risk for developing PPD symptoms.

Id.

[70] *See, e.g.*, Rick Nauert, *Hormone Test to Predict Postpartum Depression,* PSYCHCENTRAL, Feb. 26, 2009, http://psychcentral.com/news/2009/02/04/hormone-test-to-predict-postpartum-depression/3909.html (reporting that women who have a higher level of a placenta-produced hormone midway through their pregnancies are more likely to develop postpartum depression and suggesting that a postpartum depression screening test could be performed mid-pregnancy).

[71] *See, e.g.*, Giovannini, *supra* note 25, at 212 (noting the widespread acceptance of hormone-based theories of postpartum illness and stating that, "[u]nfortunately, despite significant challenges from feminist scholars and researchers, the medical establishment still operates under the assumption that female biology is a woman's destiny.").

[72] *See, e.g.*, Chrisler, *supra* note 25, at 182 (referencing contrary scientific findings); Giovannini, *supra* note 25, at 212; Jessica Zonana & Jack M. Gorman, *The Neurobiology of Postpartum Depression*, 10 CNS SPECTRUMS 792, 792 (2005) ("Most studies of gona-

experimental manipulation of hormones triggers depression only in women with a history of postpartum illness, as well as models suggesting that it is behavior that influences hormones and not hormones that influence behavior.[73] As a result, some physicians and scientists believe that the current data regarding the hormone-based theory of postpartum illness are negative or equivocal at best.[74]

D. The Role of Neurotransmitters

Because all women experience an increase in estrogen and progesterone during pregnancy and a corresponding decrease following childbirth, but not all women experience postpartum illness, many scientists have turned their attention to the ways in which some women may be genetically predisposed to postpartum illness due to differences in neurochemistry and neuroanatomy, as well as the ways in which social, cultural, and environmental factors may interact with genetics to exacerbate the risk of postpartum illness.[75] To that end, recent scientific investigations of postpartum illness have focused on the specific relationship between changes in hormone levels and the disruption of chemicals in the brain called neurotransmitters, which are endogenous chemicals that relay, amplify, and modulate signals between a neuron and another cell.[76]

The human brain has more than one hundred neurotransmitters, including serotonin, norepinephrine, dopamine, acetylcholine, and gamma-aminobutyric acid ("GABA"),[77] and a number of recent studies have been designed to investigate neurotransmitter levels in women with postpartum illness. In 2006, for example, scientists at The University of British Columbia and Canadian Blood Services published a study of platelet serotonin levels in women with postpartum depression at two points in time and compared them with questionnaire outcomes in an attempt to determine whether a measurement of platelet serotonin could aid in the diagnosis of postpartum

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dal and pituitary hormones have shown conflicting evidence for consistent roles in the development of postpartum depression.").

[73] *See, e.g.*, Dorn, *supra* note 59, at 23–24 (identifying four models of hormone-behavior relations, including a model that acknowledges that behavior may influence hormones and a model that acknowledges the possible bi-directionality of hormones and behavior).

[74] *See*, *e.g.*, Chrisler, *supra* note 25, at 182; Giovannini, *supra* note 25, at 212; Zonana, *supra* note 72, at 798.

[75] *See, e.g.*, Misri, *supra* note 27, at 41–44; Press Release, Nat'l Insts. of Health, Mechanism for Postpartum Depression Found in Mice: Discovery May Lead to Better Treatments (July 31, 2008), http://nih.gov/news/health/jul2008/nimh-31.htm.

[76] *See, e.g.*, Misri, *supra* note 27, at 41 (noting that scientists have turned their attention from unicausal hormonal theories to theories that rely on the interplay of hormones and neurotransmitters).

[77] *See, e.g.*, *id.* at 32.

depression.[78] The scientists found that platelet serotonin levels in patients with depression were reduced 50% compared to normal levels and suggested that platelet serotonin levels obtained with a new immunocytochemical test might be useful as evidence-based support for postpartum illness questionnaires.[79]

In a second study published in 2008, scientists at The University of Pittsburgh, Emory University, and the National Institute of Mental Health used positron emission tomography ("PET") to investigate brain serotonin-1A ("5HT1A") receptor binding potential in seven healthy postpartum controls and nine postpartum depressed subjects.[80] The scientists found that age, time since delivery, and reproductive hormones did not differ between groups, but that postsynaptic 5HT1A receptor binding in the depressed subjects was reduced 20% to 28% relative to controls.[81] The scientists opined that their recognition of this "neurobiological deficit" in women with postpartum depression may be useful in developing treatment and prevention strategies.[82]

E. Neuroimaging Findings

Since 1895, when German physicist Wilhelm Conrad Röntgen discovered x-rays,[83] clinicians have used a range of imaging technologies, including x-ray, computed tomography ("CT"), positron emission tomography ("PET"), single-photon emission computed tomography ("SPECT"), and magnetic resonance imaging ("MRI"), in an attempt to provide neuroradiological proof of physical conditions such as broken bones, torn ligaments,

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[78] *See* Elisabeth Maurer-Spurej, Cheryl Pittendreigh & Shaila Misri, *Platelet Serotonin Levels Support Depression Scores for Women with Postpartum Depression*, 32 J. PSYCHIATRY NEUROSCIENCE 23, 23–24 (2007).

[79] *Id.* at 27–28 ("Our results support the usefulness of platelet serotonin as a peripheral marker for postpartum depression and treatment response. In this preliminary study, we showed that platelet serotonin levels are significantly lower in women with postpartum depression, compared with healthy control subjects.").

[80] *See* Eydie L. Moses-Kolko, Katherine L. Wisner, Julie C. Price, Sarah L. Berga, Wayne C. Drevets, Barbara H. Hanusa, Tammy L. Loucks & Carolyn C. Meltzer, *Serotonin 1A Receptor Reductions in Postpartum Depression: A Positron Emission Tomography Study*, 89 FERTILITY & STERILITY 685, 685 (2008).

[81] *Id.* at 685.

[82] *Id.* at 691. Other studies have focused on the role of the neurotransmitter GABA in postpartum illness. *See, e.g.*, C. Neill Epperson, Ralitza Gueorguieva, Kathryn A. Czarkowski, Stephanie Stiklus, Edward Sellers, John H. Krystal, Douglas L. Rothman & Graeme F. Mason, *Preliminary Evidence of Reduced Occipital GABA Concentrations in Puerperal Women: A 1H-MRS Study*, 186 PSYCHOPHARMACOLOGY 425, 425 (2006); Jamie Maguire & Istvan Mody, *$GABA_AR$ Plasticity During Pregnancy: Relevance to Postpartum Depression*, 59 NEURON 207, 207 (2008); Yale Sch. of Med., Women's Health Research at Yale, Clinical Trials: Finding Predictors of Depression and Response to Treatment, http://www.med.yale.edu/womenshealth/trials/index.html (last visited Nov. 15, 2009).

[83] *See, e.g.*, Stacey A. Tovino, *Imaging Body Structure and Mapping Brain Function: A Historical Approach*, 33 AM. J.L. & MED. 193, 207 (2007) (discussing the discovery and forensic use of Röntgen's x-rays).

and cancerous tumors for diagnostic, treatment, and forensic purposes.[84] Historically, health professionals have diagnosed mental illness through clinical observation of symptoms and behaviors (that is, through the principles of descriptive psychiatry) rather than through radiological identification of neurobiological processes or pathology.[85]

Over the last fifteen years, however, several groups of scientists have investigated the ability of neuroimaging technology to provide radiological evidence of a number of illnesses traditionally classified as "mental," including postpartum illness.[86] Specific areas of inquiry include whether the brain structure and function of women with postpartum illness differ from those of controls (including new mothers without postpartum illness as well as non-postpartum women with depression and psychosis), in addition to whether any observed differences are causes, correlates, or consequences of postpartum illness. Broader areas of inquiry relate to the extent to which new findings add to existing research investigating the neural processes associated with maternal responsiveness and parental attachments.[87]

In one illustrative study published in 1998, a group of German scientists used computed tomography ("CT") to quantify the ventricular and cisternal cerebrospinal fluid ("CSF") spaces in 14 women, 12 of whom had cycloid psychoses with postpartum onset.[88] The scientists found that certain CSF spaces were significantly larger in patients with postpartum psychosis when compared to age-matched female patients with non-postpartum cy-

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[84] *Id.* at 207–21 (examining the development of these imaging technologies and their ethical, legal, and social implications).

[85] *See generally* GERMAN E. BERRIOS, THE HISTORY OF MENTAL SYMPTOMS: DESCRIPTIVE PSYCHOPATHOLOGY SINCE THE NINETEENTH CENTURY 15–26 (1996) (exploring the roots of descriptive psychopathology in nineteenth-century medicine and philosophy); Paul Raeburn, *The Therapeutic Mind Scan*, N.Y. TIMES, Feb. 20, 2005, at 20 ("Psychiatrists are among the few doctors who don't look at the organs they treat. . . . Instead of looking at the brain, they rely on interviews, experiences, hunches, and trial and error. But that soon could change.").

[86] *See infra* text accompanying notes 88–98.

[87] *See, e.g.*, Anna J. Abramson, *The Postpartum Brain*, GREATER GOOD MAG., Spring 2008, at 36, *available at* http://greatergood.berkeley.edu/greatergood/2008spring/Abramson.pdf (describing new imaging research uncovering specific biological mechanisms associated with parental attachment); Sarah Squire & Alan Stein, *Functional MRI and Parental Responsiveness: A New Avenue into Parental Psychopathology and Early Parent-Child Interactions?*, 183 BRIT. J. PSYCH. 481, 481 (2003) ("[R]elatively straightforward functional neuroimaging studies could be conducted that would help to illuminate the neural processes underlying parental emotional responses to children."). While researching the neuroimaging studies discussed in the next four paragraphs, I was asked to give a brief talk about my preliminary findings at the University of Akron School of Law's Neuroscience, Law and Government Symposium, held on September 25 and 26, 2008, in Akron, Ohio. *See* Stacey A. Tovino, *Remarks: Neuroscience, Gender, and the Law*, 42 AKRON L. REV. 941 (2009). I reserved permission to reprint and expand upon those remarks within the context of this complete Article. The next four paragraphs are taken (with updates and technical changes) with copyright permission granted October 29, 2008, 20:19:00 CST, by Jason M. Fuller, Symposium Editor, Akron Law Review.

[88] *See* M. Lanczik, E. Hofmann, C. Schulz, M. Knoche, & T. Becker, *Ventricular Abnormality in Patients with Postpartum Psychoses*, 1 ARCHIVES WOMEN'S MENTAL HEALTH 45, 45–47 (1998).

cloid psychoses or bipolar affective disorders outside the puerperium.[89] The scientists concluded that their findings could reflect an unspecific neurostructural vulnerability marker in some patients with postpartum psychosis.[90]

In a second study, published in 2007, a group of New York scientists used fMRI to compare the brain function of women with postpartum depression compared to asymptomatic postpartum female control subjects.[91] The study, believed to be the first neuroimaging study specifically designed to identify neural activity changes in unmedicated postpartum depressed women,[92] suggested that the neural mechanisms related to postpartum depression appear somewhat different than those of non-postpartum depression.[93] Although the scientists stated that it would be premature to conclude that postpartum depression is a unique depression phenotype, they stated that functional neuroimaging may have the potential to identify an empirically-based neural characterization of postpartum depression.[94]

These are just two of the neuroimaging studies that have investigated the brain structure and function of women with postpartum illness. In other studies, scientists have concluded that future research may provide a method for diagnosing postpartum illness,[95] and may be used to predict maternal style, such as child neglect, as well as offspring temperament, including depression and anxiety.[96] Today's scientists continue to use neuroimaging technologies in an attempt to better understand the neuroanatomy and processes

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[89] *Id.* at 45.

[90] *Id.* at 47 ("The results underline evidence of subtle, unspecific brain structural abnormalities in patients with postpartum cycloid, and possibly other types of postpartum psychosis. Such abnormalities might constitute an unspecific vulnerability factor.").

[91] Michael E. Silverman, Holly Loudon, Michal Safier, Xenia Protopopescu, Gila Leiter, Xun Liu & Martin Goldstein, *Neural Dysfunction in Postpartum Depression: An fMRI Pilot Study*, 12 CNS Spectrums 853, 853–54 (2007).

[92] *Id.* at 859–60.

[93] *Id.* at 861.

[94] *Id.*

[95] Nat'l Alliance for Research on Schizophrenia and Depression, Pregnancy & Postpartum Depression: New Research Directions, http://www.narsad.org/?q=node/799 (last visited Nov. 15, 2009) (summarizing recent advances in postpartum depression research, including a neuroimaging study involving postpartum subjects conducted at the Medical University of South Carolina ("MUSC"); stating that the findings from the MUSC research "may provide a method for diagnosing postpartum depression").

[96] Jeffrey P. Lorberbaum, John D. Newman, Judy R. Dubno, Amy R. Horwitz, Ziad Nahas, Charlotte C. Teneback, Courtnay W. Bloomer, Daryl E. Bohning, Diana Vincent, Michael R. Johnson, Naresh Emmanuel, Olga Brawman-Mintzer, Sarah W. Book, Bruce Lydiard, James C. Ballenger & Mark S. George, *Feasibility of Using fMRI to Study Mothers Responding to Infant Cries*, 10 Depression & Anxiety 99, 99 (1999).

> While parenting is a universal human behavior, its neuroanatomic basis is currently unknown. . . . Future work in this area may help: (1) unravel the functional neuroanatomy of the parent-infant bond; and (2) examine whether markers of this bond, such as maternal brain response to infant crying, can predict maternal style (i.e., child neglect), offspring temperament, or offspring depression or anxiety.

Id.

that underlie postpartum illness,[97] and their findings lend support to the characterization of postpartum illness as a neurodegenerative disorder that disrupts the structure and function of brain cells and precipitates cognitive decline.[98]

F. Genetic Bases

Over the past forty years, scientists also have been considering the genetic bases of postpartum illness.[99] Initial genetic studies attempted to identify a link between mental illness, including major depression and bipolar disorder, and the X chromosome, although these studies have proved incon-

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[97] *See, e.g.*, Marcelo Febo, Michael Numan & Craig F. Ferris, *Functional Magnetic Resonance Imaging Shows Oxytocin Activates Brain Regions Associated with Mother-Pup Bonding During Suckling*, 25 J. NEUROSCIENCE 11637, 11637 (2005) (fMRI study investigating whether oxytocin released in the maternal brain during breastfeeding may help strengthen the mother-infant relationship); Jack B. Nitschke, Eric E. Nelson, Brett D. Rusch, Andrew S. Fox, Terrence R. Oakes & Richard J. Davidson, *Orbitofrontal Cortex Tracks Positive Mood in Mothers Viewing Pictures of Their Newborn Infants*, 21 NEUROIMAGE 583, 583 (2004) (fMRI study implicating the orbitofrontal cortex in a mother's affective responses to her infant and suggesting that individual variations in orbitofrontal activation to infant stimuli may reflect an important dimension of maternal attachment); Squire, *supra* note 87, at 481 (recommending that research investigating the neural processes underlying parental emotional responses to children be pursued); Yale Program for Women's Reprod. Behavioral Health, Research in the Service of Patient Care, http://www.med.yale.edu/psych/clinics/YBG.html (last visited Nov. 15, 2009) (providing information about the current research projects of the Yale Program for Women's Reproductive Behavioral Health, including a research study involving the diagnosis and treatment of postpartum depression using neuroimaging).

[98] *See, e.g.*, Hara Estroff Marano, *Depression: Beyond Serotonin*, PSYCHOL. TODAY, Mar. 1, 1999, at 30, 32.

> The newest evidence indicates that recurrent depression is in fact a neurodegenerative disorder, disrupting the structure and function of brain cells, destroying nerve cell connections, even killing certain brain cells, and precipitating cognitive decline. At the very least, depression sets up neural roadblocks to the processing of information and keeps us from responding to life's challenges.

Id. at 32.

[99] *See, e.g.*, John J. Medina, *Is There a Gene for Postpartum Depression?*, 25 PSYCH. TIMES, Dec. 1, 2008 at 44, 44–45 (exploring whether there is a molecular basis for postpartum depression); *Post-Natal Psychosis Genes Found*, BBC NEWS (July 5, 2007), http://news.bbc.co.uk/2/hi/uk_news/wales/6273182.stm.

> Researchers at Cardiff University have helped locate the genes which can make women more vulnerable to severe mental illness just after childbirth. . . . Finding these genes will allow us to better identify bipolar women at very high risk and will help them and their doctors make the difficult decisions about taking medications through pregnancy.

Id. See also, e.g., Graham Tibbetts, *Mothers' Acute Post-Natal Depression Could be Genetic*, TELEGRAPH.CO.UK, Nov. 2, 2008, http://www.telegraph.co.uk/health/3366667/Mothers-acute-post-natal-depression-could-be-genetic.html ("Work has begun to isolate the gene to enable doctors to identify and treat women at risk from postpartum psychosis before they are affected.").

clusive.[100] Although women continue to be statistically more likely to develop depression than men,[101] scientists have turned their attention in the last five years to specific genes that may influence susceptibility to postpartum illness.[102]

In 2007, scientists affiliated with Cardiff University, Birmingham University, and Trinity College conducted one of the first systematic genome scans designed to localize genes that influence susceptibility to bipolar affective puerperal psychosis.[103] The scientists selected families with bipolar disorder from a previous bipolar disorder genome scan in which there was at least one family member with a manic or psychotic episode with an onset within six weeks of delivery, and then the scientists coded the individuals as affected if they had been diagnosed with bipolar I disorder, bipolar II disorder, or schizoaffective disorder of bipolar type, according to the DSM-IV-TR classification system.[104] The cohort included 36 pedigrees contributing 54 affected sibling pairs, and the scientists' genome scan consisted of an analysis of 494 microsatellite markers using Genehunter and Mapmaker/Sibs statistical genetic analysis software.[105] The scientists observed a genome-wide significant linkage signal on chromosome 16p13 and a genome-wide suggestive linkage on chromosome 8q24, even though no significant or suggestive linkage was observed in these regions in the scientists' original bipo-

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[100] *See, e.g.*, Robert M.A. Hirschfeld & Myrna M. Weissman, *Risk Factors for Major Depression and Bipolar Disorder*, *in* NEUROPSYCHOPHARMACOLOGY: THE FIFTH GENERATION OF PROGRESS 1023 (Kenneth L. Davis, Dennis Charney, Joseph T. Coyle & Charles Nemeroff, eds., 5th ed. 2002), *available at* http://www.acnp.org/Docs/G5/CH70_1017-1026.pdf ("There have been a number of investigations aimed at determining the actual genes involved in bipolar illness. Attempts to demonstrate linkage to the X-chromosome . . . have not been conclusive.").

[101] *See, e.g.*, Harvard Med. Sch., *Women and Depression: How Biology and Society Make Women More Vulnerable to Mood Disorders*, 20 HARV. MENTAL HEALTH LETTER, May 2004, at 1, 1 ("All over the world, depression is much more common in women than in men. In the United States, the ratio is two to one, and depression is the main cause of disability in women."); Serrano, *supra* note 58, at 385 ("[E]pidemiologic studies note that the risk of depression in women is twice that of men, regardless of racial and socioeconomic variables.").

[102] *See, e.g.*, Tibbetts, *supra* note 99 (quoting Dr. Ian Jones, who stated that he had "identified chromosomal regions that are likely to harbour genes that predispose individuals to [postpartum psychosis]"); Amelia Hill, *Postnatal Depression 'in the Genes:' Fresh Medical Research Suggests the Serious Mental Illness Which Bedevils New Mothers May be Due to Nature, not Nurture*, THE OBSERVER, Nov. 2, 2008, at 18 ("The most severe form of postnatal depression, which affects one in 500 new mothers and has been linked to suicide and infanticide, could be genetic, according to new research.").

[103] *See* Ian Jones, Marian Hamshere, Jeanne-Marrie Nangle, Philip Bennett, Elaine Green, Jess Heron, Ricardo Segurado, David Lambert, Peter Holmans, Aiden Corvin, Mike Owen, Lisa Jones, Michael Gill & Nick Craddock, *Bipolar Affective Puerperal Psychosis: Genome-Wide Significant Evidence for Linkage to Chromosome 16*, 164 AM. J. PSYCH. 1099, 1103 (2007).

[104] *See id.* at 1099. The DSM-IV-TR classification system is introduced later in this article; *see infra* text accompanying notes 287–289 and 294–297.

[105] Jones, *supra* note 103, at 1099–1100.

lar scan.[106] The scientists concluded that they had identified the "chromosomal regions that are likely to harbor genes that predispose individuals to bipolar affective puerperal psychosis,"[107] and that their findings supported the hypothesis that bipolar affective puerperal psychosis is a "genetically meaningful subtype of bipolar disorder."[108]

In 2008, two scientists at the University of California, Los Angeles further investigated the genetic basis of postpartum depression, this time in mice.[109] In their research, the scientists studied mice that developed symptoms that in postpartum humans we would call postpartum depression, including anhedonia, despair, increased anxiety, and decreased maternal care.[110] The scientists found that the gene disrupted in these mice, delta subunit of gamma-aminobutyric acid A receptor ("$GABA_AR$"), encodes a subunit of a receptor that is located on the surface of neural cell,[111] and believe that their findings point to a genetic source for predisposition to postpartum depression.[112]

G. Evolutionary Theories

In addition to genetics-based research, several anthropologists and other evolutionary theorists are investigating whether postpartum illness serves an evolutionary function. The theory underlying many of these studies is that postpartum illness may be an adaptation that informs mothers that they are suffering a fitness cost, such as lack of paternal or social support, and that motivates them to reduce or eliminate investment in their offspring.[113] An-

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[106] *Id.* at 1101 ("We found one chromosome region (16p13) with a LOD [logarithm of the odds ratio] score (4.07) meeting the Lander and Kruglyak criteria (26) for genome-wide significance ($p=0.02$). . . . We also report an additional chromosomal region (8q24) that meets the Lander and Kruglyak (26) criteria for genome-wide suggestive linkage.").

[107] *Id.* at 1099.

[108] *Id.* at 1103 ("In summary, we report the first systematic genome scan aimed at localizing genes that influence susceptibility to bipolar affective puerperal psychosis and provide further support for the hypothesis that this is a genetically meaningful subtype of bipolar disorder.").

[109] *See* Maguire, *supra* note 82, at 207.

[110] *Id.* at 208–10.

[111] *Id.* at 211–12.

[112] *Id.*

[113] *See, e.g.*, Edward H. Hagen, *Depression as Bargaining: The Case Postpartum*, 23 Evolution and Human Behavior 323, 324 (2002); Edward H. Hagen, *The Functions of Postpartum Depression*, 20 Evolution and Human Behavior 325, 325–26 (1999) [hereinafter *Functions*]; Edward H. Hagen & H. Clark Barrett, *Perinatal Sadness among Shuar Women: Support for an Evolutionary Theory of Psychic Pain*, 21 Med. Anthropology Q. 22, 25–27 (2007); Randy Thornhill & F. Bryant Furlow, *Stress and Human Reproductive Behavior: Attractiveness, Women's Sexual Development, Postpartum Depression, and Baby's Cry*, *in* 27 Stress and Behavior 319, 341–52 (Anders Pape Møller, Manfred Milinski & Peter J.B. Slater eds., 1998); Stefanie L. Turner & Francis T. McAndrew, *A Laboratory Simulation of Parental Investment Decisions: The Role of Future Reproductive Opportunities and Quality of Offspring in Determining Levels of Parental Investment*, 4 Evolutionary Psychol. 197, 198 (2004); Margo Wilson & Martin Daly, *The Psychology of Parenting in Evolutionary Perspective and the Case of Human Fili-*

thropologist Edward Hagen proposed in 1999, for example, three related adaptive functions for postpartum depression.[114] Hagen first proposed that sad or depressed mood should be associated with social circumstances that are reproductively costly in ancestral environments, including situations in which mothers lack social support and in which the infant's health is poor.[115] Second, Hagen proposed that mothers will take actions to reduce their level of psychological pain in order to reduce their reproductive costs.[116] Third, Hagen proposed that major postpartum depression, characterized by marked loss of interest in virtually all activities, psychomotor retardation, significant weight loss, diminished ability to think or concentrate, and recurrent thoughts of death, may enable the mother to negotiate greater levels of social support.[117]

A second evolutionary theory relates to the development of obsessive-compulsive symptoms (including recurrent, intrusive thoughts of harm coming to the baby)[118] as a means of ensuring infant survival. For example, a group of American scientists who used fMRI in 2002 to investigate the brains of healthy, new parents as they heard their babies cry found that the parents' neural networks were similar to the networks known to be associated with obsessive-compulsive disorder ("OCD").[119] The scientists believe that listening to babies cry triggers a deeply anxious neural response even in healthy parents, and suggest that even the healthy parental brain may be "hardwired" for a period of transient, mild OCD that may have been adaptive in our ancestral environment, when new parents needed to be hypervigilant about protecting their young from dangerous predators.[120] According to the scientists, however, the persistence of these behaviors in the mod-

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cide, *in* INFANTICIDE AND PARENTAL CARE 73, 74–75 (Stefano Parmigiani & Frederick S. vom Saal eds., 1994); Australian Broadcasting Corporation National Radio Broadcast, *All in the Mind: The Evolution of Depression—Does It Have a Role?* (Apr. 3, 2004), http://www.abc.net.au/rn/science/mind/stories/s1077027.htm (discussing whether depression can serve an evolutionary function). *But see* Daniel Nettle, *Evolutionary Origins of Depression: A Review and Reformulation*, 81 J. AFFECTIVE DISORDERS 91, 91 (2004) (proposing an alternative formulation in which depression is seen not as an adaptation but nonetheless as the outcome of evolutionary processes).

[114] Hagen, *Functions*, *supra* note 113, at 326–27.

[115] *Id.* at 335–37.

[116] *Id.* at 342–45.

[117] *Id.* at 345–49.

[118] *See, e.g.*, Faruk Uguz, Cemal Akman, Nazmiye Kaya & Ali Savas Cilli, *Postpartum-Onset Obsessive-Compulsive Disorder: Incidence, Clinical Features, and Related Factors*, 68 J. CLINICAL PSYCHIATRY 132, 132 (2007) (investigating the incidence, rate, and symptomatology of postpartum-onset obsessive-compulsive disorder ("PPOCD"), the factors associated with PPOCD, and comparing clinical characteristics of obsessive-compulsive disorder with and without postpartum onset).

[119] James Swain, James Leckman, Linda Mayes, Ruth Feldman & Robert Schultz, *Functional Neuroimaging and Psychology of Parent-Infant Attachment in the Early Postpartum*, 5 ANNALS GEN. PSYCHIATRY S85 (2006); Abramson, *supra* note 87 at 37 (referencing Swain's study). The full publication of Swain's findings is forthcoming. *See* Dr. Edward James Swain, Profile, http://myprofile.cos.com/jameseswain (last visited Nov. 15, 2009) (listing Swain's submitted but as yet unpublished manuscripts).

[120] Abramson, *supra* note 87, at 37.

ern environment may not be useful, and an excess (or lack) of this mild vigilance may play a role in the obsessive (or neglectful) symptoms experienced by some mothers diagnosed with postpartum illness.[121]

H. Psychosocial and Sociocultural Models

In the last three decades, many socially-trained scientists have given less credence to straight biological theories of postpartum illness, labeling them reductionist and sexist, and have focused instead on socioenvironmental stressors and cultural factors as important etiological factors in postpartum illness.[122] Within psychosocial models, childbirth is viewed as a significant life event that requires the assumption of new social roles, skills, and behaviors, which can lead to uncertainty and stress in new mothers.[123] In order to minimize these stressors and assist women in successfully transitioning to their new maternal role, pregnant and postpartum women are believed to require emotional support, advice, information, and assistance.[124] If these forms of support are either not available or not accessible, new mothers may be more vulnerable to postpartum illness.[125]

A number of recent studies have investigated the psychosocial factors that may contribute to postpartum illness.[126] In a 2005 study of the narra-

> The researchers offer an evolutionary hypothesis for the neural signs of anxiety they saw in these parents. They believe that, after the birth of a child, a period of high alert may have helped parents protect their babies from environmental harm in times when this was a treacherous and all-consuming task. . . . The Yale researchers hypothesize that the healthy maternal brain is hardwired for a period of transient OCD. . . . But, . . . once mothers are endowed with this kind of neural machinery, there's a danger they could connect up OCD behaviors with irrational things not for survival.

Id. (internal quotation marks omitted). *See also* Swain, *supra* note 119, at S85.

[121] *See* Abramson, *supra* note 87, at 37.

[122] Giovannini, *supra* note 25, at 213–14.

[123] *See, e.g.*, Maigun Edhborg, Malin Friberg, Wendela Lundh & Ann-Marie Widström, *"Struggling with Life:" Narratives from Women with Signs of Postpartum Depression*, 33 SCANDINAVIAN J. PUB. HEALTH 261, 261 (2005) ("Social scientist studies are predominantly within a qualitative tradition and highlight women's perceptions of the depression. Feminist researchers link depression to women's inferior status in society and to structural conditions and constraints, e.g., current labour market structures and family policy in division of household labour."); Giovannini, *supra* note 25, at 213–14.

[124] Giovannini, *supra* note 25, at 214.

[125] *Id.*

[126] *See, e.g.*, Laura S. Abrams & Laura Curran, *Not Just a Middle-Class Affliction: Crafting a Social Work Research Agenda on Postpartum Depression*, 32 HEALTH & SOC. WORK 289, 290–94 (2007) (reviewing the current postpartum depression literature with specific attention to the sociocultural dimensions of the disease and barriers to treatment); Cecilia Benoit, Rachel Westfall, Adrienne E. B. Treloar, Rachel Phillips & S. Mikael Jansson, *Social Factors Linked to Postpartum Depression: A Mixed-Methods Longitudinal Study*, 16 J. MENTAL HEALTH 719, 719 (2007) (investigating the association between social factors, the organization of maternity care services, and the prevalence of depression among a sample of new mothers at three to four weeks and four to six months postpartum; finding links between income and postpartum depression and satisfaction with the birth experience and postpartum depression); Wendy A. Mason, Michael J. Rice

tives of women with postpartum depression, a group of Swedish scientists found that the women expressed feelings of loss of who they are, felt overwhelmed by the responsibility for the child, struggled with feelings of abandonment, worries, and breastfeeding problems, and struggled to keep their equality with their husbands and to get them involved in childcare.[127] The scientists concluded that the depressed feelings of these postpartum women may be explained in terms of losses and changes in the dimensions of self, child, and partner.[128]

In a 2007 review article, two American social scientists reported known risk factors for postpartum depression, including prior experiences with mental illness, complications or dissatisfaction with labor and delivery, and other concurrent life stressors such as divorce, loss, illness, or major transitions.[129] The scientists also reported that prevalence rates for postpartum depression are higher among single mothers, mothers who have lower educational attainment, women who have low levels of social support, and women who have low-income status or financial hardship.[130] Finally, the scientists reported feminist theories of postpartum depression, including those that focus on the physical trauma of and recovery from childbirth, sleep deprivation, new responsibilities, the need to quickly master new skill sets, postpartum losses of autonomy and time, and transformations in personal and occupational identity, physical appearance, sexuality, and relationships.[131]

Within sociocultural models, the symptoms associated with postpartum illness are viewed as culture-bound, meaning they may be categorized as a disorder or disease in some societies but not others, or that family and community support (including education, social support, shared child care, and social recognition of the importance and value of the motherhood role) may be more available in some cultures compared to others, making women outside those cultures more vulnerable to postpartum illness.[132] A number of recent studies have focused on sociocultural models of postpartum illness.

& Kathie Records, *The Lived Experience of Postpartum Depression in a Psychiatric Population*, 41 PERSP. IN PSYCHIATRIC CARE, Apr.–June 2005, at 52, 52 (using a psychophenomenological design to examine reports from women with postpartum depression regarding the role life experiences played in their labor, delivery, and postpartum periods); Verinder Sharma & Dwight Mazmanian, *Sleep Loss and Postpartum Psychosis*, 5 BIPOLAR DISORDERS 98, 98–103 (2003) (reviewing the literature on the relationship of sleep disruption and postpartum psychosis and noting that sleep loss resulting from the interaction of various putative causal factors may be the final common pathway in the development of psychosis in susceptible women).

[127] *See* Edhborg, *supra* note 123, at 261.

[128] *Id.*

[129] *See* Abrams, *supra* note 126, at 290.

[130] *Id.*

[131] *Id.* at 292.

[132] *See, e.g.*, Chrisler, *supra* note 25, at 184–87; Sophie Grigoriadis & Sarah Romans, *Postpartum Psychiatric Disorders: What Do We Know and Where Do We Go?*, 2 CURRENT PSYCHIATRIC REV. 151, 155–56 (2006) (noting that postpartum depression may represent a culture-bound syndrome).

In one study published in 2007, for example, a group of Chinese scientists investigated the relationship between fetal gender and postpartum depression in a small cohort of female Chinese subjects.[133] The scientists found that the rate of postpartum depression in women who gave birth to a female infant was 24.6% compared to 12.2% for women who gave birth to a male infant.[134] The relative risk for postpartum depression in women who gave birth to a female infant as compared with those who gave birth to a male was 2.89, after adjusting for potential confounding by maternal age, education level, family income, living conditions, gravidity, number of prenatal care visits, and mode of delivery.[135] The scientists concluded that Chinese women who give birth to female infants have an increased risk of postpartum depression, perhaps due to the negative reactions of family members towards the birth of female babies.[136]

Not all scientists who are interested in sociocultural models of postpartum illness completely discount the role of biology, however. In one study jointly authored by a scientist from Taiwan and a scientist from the UK investigating and comparing the etiology of postpartum illness in Taiwan and the UK, the scientists found that sociocultural factors were not the only factors associated with postpartum depression.[137] More specifically, the scientists found that the prevalence of postpartum depression was similar in both cultures despite large differences, at least historically, in cultural and postnatal care systems, suggesting that postpartum depression may have biological determinants as well as sociocultural ones.[138]

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[133] *See* Ri-hua Xie, Guoping He, Aizhong Liu, Jacques Bradwejn, Mark Walker & Shi Wu Wen, *Fetal Gender and Postpartum Depression in a Cohort of Chinese Women*, 65 Soc. Sci. & Med. 680, 680 (2007); *see also* Sandhya Ramashwar, *In China, Women Who Give Birth to Girls Face an Increased Risk of Postpartum Depression*, 33 Int'l Family Planning Persp. 191, 191–92 (2007) (summarizing the Chinese study investigating the relationship of fetal gender and postpartum depression).

[134] Xie, *supra* note 133, at 680, 682.

[135] *Id.* at 680.

[136] *Id.* at 683 ("We speculate that the negative reaction of family members towards the birth of a female baby may be influential in creating or exacerbating depression here. The preference for male children may be communicated to the mother and those who deliver females may receive less support and less positive feedback.").

[137] *See* Yu-Chu Huang & Nigel Mathers, *Postnataldepression—Biological or Cultural? A Comparative Study of Postnatal Women in the UK and Taiwan*, 33 J. Advanced Nursing 279, 279 (2001).

[138] *Id.* at 286.

> In conclusion, we have observed big differences in both postnatal rituals and the postnatal care systems between the UK and Taiwan. The findings of our study suggest that despite these differences, the prevalence of postnatal depression is similar in both cultures. This gives some support to a hypothesis that postnatal depression may have at least some important biological determinants as well as cultural and social ones.

Id.

I. Current Understandings

The most current understandings of postpartum illness are based on multifactorial models that give weight to hormonal, neurochemical, neuroanatomical, genetic, evolutionary, psychosocial, sociocultural, and environmental factors, and that recognize a subtle interplay between and among these factors depending on the patient.[139] Unicausal theories, especially unicausal hormone-based theories, receive little support in the current scientific literature.[140] In one current multifactorial model, socioenvironmental stressors experienced early in life are believed to alter nerve circuits that control emotion, and such alterations are believed to exaggerate later responses to stress, which may lead to the neurochemical and behavioral changes associated with postpartum illness.[141] Very recent studies also are showing that all forms of depression, including postpartum depression, can lead to heart disease and exacerbate existing cardiac problems, and that the hormone deficiencies that sometimes accompany depression, including increased levels of cortisol, can lead to reductions in calcium and eventually

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[139] *See, e.g.*, JAMES ALEXANDER HAMILTON, POSTPARTUM PSYCHIATRIC ILLNESS: A PICTURE PUZZLE xix (James A. Hamilton & Patricia N. Harberger, eds., 1992) ("[E]vidence from a variety of sources suggest[s] that postpartum psychiatric syndromes implicate a range of pathophysiology which is fairly extensive and extended in time. Looking at individual variables, a very complex phenomenon is suggested."); Butts, *supra* note 34, at 139 ("In all likelihood there are many factors responsible for decompensation in any post-partum patient. These factors are organic, biochemical, physiological and psychological. One should bear in mind the subtle interplay between these and other parameters in the production of post-partum illness."); Martinez-Schallmoser, *supra* note 25, at 246–47 ("It appears that the etiology of postpartum depression may be complex and multifactorial."). A multifactorial model was recognized as early as 1838 by French Psychiatrist Jean-Etienne-Dominique Esquirol, who stated in *Des Maladies Mentales* that, "[t]he causes which especially predispose the recently confined and nurses to this malady are, hereditary predisposition, an extreme susceptibility, attacks of insanity anterior to pregnancy, and attacks consequent upon preceding confinements, or during lactation." ESQUIROL, *supra* note 32, at 128.

[140] *See, e.g.*, Marano, *supra* note 98, at 30, 32.

> Much more than a chemical imbalance, depression is a disorder of mind, brain, and body . . . . New research is challenging the assumption that the world's most common mental ailment is just a chemical imbalance in the brain . . . . It is not possible to explain either the disease or its treatment based solely on levels of neurotransmitters.

Id.

[141] *See, e.g.*, *id.* at 32.

> Stress-related events may kick off 50 percent of all depression and early life stress can prime people for later depression. Ongoing research in animals and in people demonstrates that early strain can alter nerve circuits that control emotion, exaggerating later responses to stress and creating the neurochemical and behavioral changes of depression. In other words, the deeper researchers probe the brain, the more they validate the psychoanalytic view that early adverse life events can create adult psychopathology.

Id.

osteoporosis.[142] As a result, many physicians and scientists now classify depression in all its forms as a physical illness due to its probable physical causes and documented physical effects, and recognize depressive and psychotic episodes as secondary responses to primarily biologically-based illnesses.[143]

II. Legal Understandings of Postpartum Illness

Given these advances in the scientific understanding of postpartum illness, I now turn to its legal understanding, incorporation, and utilization. A range of past, current, and proposed legal authorities, including criminal infanticide laws, judicial opinions interpreting health insurance policy provisions, and postpartum awareness laws, expressly reference postpartum illness, its probable causes and its understood effects. In this Part II, I demonstrate that the science lawmakers use to support and interpret postpartum law and policy is not always accurate. A criminal infanticide bill introduced in one state's most recent legislative session continues to refer to lactation as a cause of postpartum impaired judgment, for example.[144] Judicial opinions interpreting health insurance policy provisions continue to classify postpartum illness as a non-physical, nervous disorder notwithstanding readily available scientific evidence to the contrary.[145] Many states' recent

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[142] *See, e.g.*, *id.*; Lana J. Williams, Julie A. Pasco, Felice N. Jacka, Margaret J. Henry, Seetal Dodd & Michael Berk, *Depression and Bone Metabolism*, 78 Psychotherapy & Psychosomatics 16–25 (2009) (reviewing the current evidence on the relationship between depression and bone mineral density and identifying potential mechanisms); Depression Increases Risk for Heart Disease More Than Genetics or Environment, ScienceDaily, Mar. 5, 2009, http://www.sciencedaily.com/releases/2009/03/090304182113.htm (referencing a study conducted by researchers at the Washington University School of Medicine and the St. Louis Veterans Affairs Medical Center reporting that a history of major depression increases the risk of heart disease over and above any genetic risks common to depression and heart disease); Marilyn Elias, Depression Can Break the Heart, USA Today, Mar. 4, 2009, http://www.usatoday.com/news/health/2009-03-04-depression-heart_N.htm (referencing the same study); Kerri Watchter, 19 Postmenopausal Women Studied: Cortisol May Mediate Effect of Depression on Osteoporosis, OB/GYN News, June 15, 2004, http://findarticles.com/p/articles/mi_m0CYD/is_12_39/ai_n6101590/ (referencing data presented at the 2004 American Association for Geriatric Psychiatry suggesting that women with a history of depression history appear to have increased stress-related cortisol levels, a finding that may help explain the link between depression and osteoporosis).

[143] *See, e.g.*, Marano, *supra* note 98, at 74 (suggesting that psychiatry is moving towards the view that the brain is a biological organ).

> The new corporeality of "mental" illness is perhaps most daringly embodied in the work of Bruce Charlton, a research psychiatrist in the department of psychology at the University of Newcastle in England. Depression, Charlton provocatively contends, doesn't just have physical concomitants; it is wholly a physical disorder, one that is misinterpreted by the brain. Sickness is read as sadness.

Id.

[144] *See infra* Part II(A).

[145] *See infra* Part II(B).

postpartum awareness laws are premised on outdated unicausal hormone-based theories of postpartum illness.[146] The reasons for these inaccuracies likely include the difficulties faced by non-scientifically trained lawmakers in understanding complex disease etiology, the reliance on common law tests and contractual provisions that contain outdated formulas for distinguishing physical and mental illness, the lack of time and resources necessary to update outdated law, the frequency with which lawmakers borrow language from old law to create new law without updating the science therein and, perhaps, the desire of lawmakers to rely on the perceived objectivity and allure of science to support socially desirable health laws and policies.[147]

Unfortunately, inaccurate understandings of illness and behavior are not only difficult to remove from the law once established, but they also tend to reappear within new statutes and judicial opinions notwithstanding readily accessible scientific findings to the contrary.[148] In this Part, I also demonstrate that lawmakers who rely on outdated science risk developing inappropriate health laws and policies, encouraging the introduction of expert testimony that will not meet evidentiary standards for use in litigation, establishing conflicts between different health laws and policies, and supporting the public misunderstanding of postpartum illness. Going forward, lawmakers, judges, and other stakeholders need to recognize the complexity of disease etiology, including the etiology of postpartum illness. To assist with these efforts, I propose a contemporary legal explanation of postpartum illness that emphasizes the illness's incomplete understanding and likely multifactorial etiology, as well as the elimination or reformulation of common law tests designed to distinguish physical and mental illness.

A. Criminal Infanticide Legislation

International criminal infanticide legislation provides one of the oldest express legal recognitions of a relationship between childbirth and mental illness.[149] The English Infanticide Act of 1922 reduced the maximum charge

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[146] *See infra* Part II(C).

[147] *See infra* Part II.

[148] *See, e.g.*, *infra* text accompanying notes 182–200.

[149] *See, e.g.*, GEOFFREY R. MCKEE, WHY MOTHERS KILL: A FORENSIC PSYCHOLOGIST'S CASEBOOK 42 (2006) ("Legislative confirmation of the influence of pregnancy and delivery on a woman's emotional status, for example, was provided in the English Infanticide Act of 1922."). Older infanticide laws, such as the English Infanticide Act of 1623, reduced the maximum charge a mother could face for concealing the death of a bastard child, were thought to result not from legal recognition of postpartum illness but from recognition of socio-economic difficulties, such as the inability of a mother to secure support for a child born out of wedlock from the child's biological father; *see* An Act to Prevent the Destroying and Murdering of Bastard Children, 1623, 20 Jac., c. 27 (Eng.). *See generally* Katherine O'Donovan, *The Medicalisation of Infanticide*, *reprinted in* SOURCEBOOK ON FEMINIST JURISPRUDENCE 518 (Hilaire Barnett, ed., 1997) (examining criminal law's treatment of infanticide from the seventeenth century to the present).

a mother could face for killing her child from the crime of murder to manslaughter if (i) the child was "newly-born"; and (ii) "at the time of the act or omission [the mother] had not fully recovered from the effect of giving birth to such child, but by reason thereof the balance of her mind was then disturbed."[150] Although the Infanticide Act of 1922 did not contain any within-statute legislative findings or preamble referencing particular scientific findings addressing the relationship between childbirth and mental illness, a literal reading of the Act suggests to the public and the legal community that a woman's mind can be "disturbed" during the period in which her child is "newly-born."[151]

The problem with the Infanticide Act of 1922 was that it did not define the phrase "newly-born."[152] Five years later, a case involving a woman named Mary O'Donoghue, who had killed her 35-day-old child, was brought to trial.[153] The trial court held that the child was not "newly-born" and, therefore, that Ms. O'Donoghue must be sentenced to death under the general murder statute, a sentence the Crown later commuted to life imprisonment.[154] On appeal, defense counsel argued that the Infanticide Act of 1922 was designed to protect women who suffered from "puerperal mental derangement," a condition that could last from two to up to six weeks after childbirth, and that Ms. O'Donoghue's criminal behavior occurred during this extended period.[155] The Court of Criminal Appeal disagreed, finding no error in the trial court's holding that the defendant's 35-day-old child was not "newly-born."[156]

Parliament responded eleven years later by amending the Infanticide Act of 1922 to apply to a broader range of killings, including those involving children up to 12 months of age, and to killings of such children due to a second reason; that is, due to lactation. The Infanticide Act of 1938 thus

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[150] Infanticide Act, 1922, 12 & 13 Geo. 5, c. 18, § 1(1) (Eng.).

> Where a woman by any wilful act or omission causes the death of her newly-born child, but at the time of the act or omission she had not fully recovered from the effect of giving birth to such child, and by reason thereof the balance of her mind was then disturbed, she shall, notwithstanding that the circumstances were such that but for this Act the offence would have amounted to murder, be guilty of felony, to wit of infanticide, and may for such offence be dealt with and punished as if she had been guilty of the offence of manslaughter of such child.

Id. *See also* McKee, *supra* note 149, at 42 (discussing the Infanticide Act of 1922).

[151] Infanticide Act of 1922, § 1(1).

[152] *Id.*

[153] R. v. Mary O'Donoghue, (1927) 20 Cr. App. R. 132 (Eng.).

[154] *Id.* at 132–33.

[155] *Id.* at 133 ("[T]here was between insanity and sanity a degree of mental derangement which the medical authorities called 'puerperal' which might appear physically for any period from two to six weeks after childbirth, to meet which condition the statute was designed.").

[156] *Id.* at 136 ("Mr. Justice Talbot made no error in law in holding, with reference to a child of more than a calendar month of age, that there was no evidence upon which he could invite or permit a jury to find that that child was newly-born within the meaning of the statute.").

reduced the maximum charge a mother could face from murder to infanticide if (i) the child was "under the age of twelve months"; and, (ii) "at the time of the act or omission the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child or by reason of the effect of lactation consequent upon the birth of the child."[157] Like the Infanticide Act of 1922, the Infanticide Act of 1938 did not contain any within-statute legislative findings or preamble referencing particular scientific studies finding a relationship between or among childbirth, lactation, and mental illness, although a literal reading of the Act suggests to the public and the legal community that a woman's mind can be "disturbed" during the twelve-month period following birth due to a failure to recover from birth or the effects of lactation.

The bulk of the law review scholarship referencing the Infanticide Acts of 1922 and 1938 focuses on the question of whether postpartum women who kill their children should benefit from separate, gender-specific criminal infanticide provisions.[158] My inquiry as a law-medicine scholar, however, is

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[157] Infanticide Act, 1938, 1 & 2 Geo. 6, c. 36, § 1(1) (Eng.).

> Where a woman by any willful act or omission causes the death of her child being a child under the age of twelve months, but at the time of the act or omission the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child or by reason of the effect of lactation consequent upon the birth of the child, then, notwithstanding that the circumstances were such that but for this Act the offence would have amounted to murder, she shall be guilty of felony, to wit of infanticide, and may for such offence be dealt with and punished as if she had been guilty of the offence of manslaughter of the child.

Id. See generally Kimberly Fisher, Note, *To Save Her Children's Souls: Theoretical Perspectives on Andrea Yates and Postpartum-Related Infanticide*, 25 T. JEFFERSON L. REV. 599, 615–18 (2003) (discussing the Infanticide Act of 1938).

[158] *See, e.g.*, Sheri L. Bienstock, *Mothers Who Kill Their Children and Postpartum Psychosis*, 32 SW. U. L. REV. 451, 453 (2003) (recommending recognition of a statutory "heat of passion" or "guilty except for insanity" scheme for women with postpartum psychosis); Fisher, *supra* note 157, at 600 (examining "whether postpartum psychosis should be recognized as a general defense to infanticide, or as a separate statutory defense."); Christine Ann Gardner, Note, *Postpartum Depression Defense: Are Mothers Getting Away with Murder?*, 24 NEW ENG. L. REV. 953, 955 (1990) (arguing that postpartum depression should not be viewed as an illness in need of special recognition in U.S. courts as postpartum depression is in other countries); Connie Huang, *It's a Hormonal Thing: Premenstrual Syndrome and Postpartum Psychosis as Criminal Defense*, 11 S. CAL. REV. L. & WOMEN'S STUD. 345, 345 (2002) ("[P]ostpartum psychosis should be allowed as a type of insanity defense, but not as a separate defense."); Jessie Manchester, *Beyond Accommodation: Reconstructing the Insanity Defense to Provide an Adequate Remedy for Postpartum Psychotic Women*, 93 J. CRIM. L. & CRIMINOLOGY 713, 717 (2003) ("[T]he best solution for ensuring that postpartum psychotic women can adequately present evidence of their mental illness is for states to return to a broader insanity test."); Cristie L. March, *The Conflicted Treatment of Postpartum Psychosis under Criminal Law*, 32 WM. MITCHELL L. REV. 243, 245–46 (2005) (exploring a variety of options regarding the criminal treatment of women with postpartum psychosis who commit infanticide); Laura E. Reece, Comment, *Mothers Who Kill: Postpartum Disorders and Criminal Infanticide*, 38 UCLA L. REV. 699, 717–54 (1991) (arguing that criminal law should accept postpartum disorder evidence in infanticide cases and proposing several methods therefore); Heather Leigh Stangle, *Murderous Madonna: Femininity, Violence,*

the Acts' allegedly scientific basis.[159] Were the 1922 and 1938 Acts recognizing known scientific links between and among childbirth, lactation, and mental illness? Or, were the Acts legislatively creating links where none existed, perhaps to bolster support for the more lenient treatment of postpartum women,[160] or out of a judicial desire not to prosecute postpartum women for murder unless the black-cap capital punishment would actually be carried out, which it rarely was?[161] Although other law-medicine scholars have concluded that the answers to these questions remain, to this day, unclear,[162] a careful review of the early medical observations presented in Part I(A) of this Article combined with a review of then-current newspaper reports and the legislative history of the Infanticide Act of 1938, presented below, reveals an early twentieth-century public and legal recognition of medical observations of the temporal relation between and among childbirth, lactation, and mental illness. Studies confirming a causal relationship between childbirth or lactation and mental illness that would meet today's standards for the scientific method[163] did not exist at the time of the Acts' enactment, however.

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and the Myth of Postpartum Mental Disorder in Cases of Maternal Infanticide and Filicide, 50 WM. & MARY L. REV. 699, 704 (2008) ("[T]he states have no need to enact an Infanticide Act or other gender-specific laws."); April J. Walker, *Application of the Insanity Defense to Postpartum Disorder-Driven Infanticide in the United States: A Look Toward the Enactment of an Infanticide Act*, 6 U. MD. L.J. RACE, RELIGION, GENDER & CLASS 197, 201–07 (2006) (examining the legal treatment of women with postpartum psychosis who commit infanticide under state and international law).

[159] *See, e.g.*, Michelle Oberman, *Mothers Who Kill: Coming to Terms with Modern American Infanticide*, 8 DEPAUL J. HEALTH CARE L. 3, 19–20 (2004) (also examining the quasi-scientific basis of early twentieth-century European infanticide acts).

[160] *See, e.g.*, *id.* at 20 (asking the question).

[161] *See, e.g.*, 108 PARL. DEB., H.L. (5th ser.) (1938) 303 (remarks of Lord Arnold) ("In the last ten years, or rather, in the ten years down to 1936, there have been fifteen cases which come under the category of what the Press call the 'black cap farce,' that is, the sentencing of a woman to death for the murder of her child when it is perfectly well known that the sentence will not be carried out."); O'Donovan, *supra* note 149, at 520 ("Even when there was a conviction capital punishment was rarely carried out. Despite 39 convictions for child murder between 1849 and 1864, no woman was executed, from 1905 to 1921, 60 women were sentenced to death but in 59 of these cases the sentence was commuted.").

[162] Oberman, *supra* note 159, at 19.

> Interestingly, the most fundamental criticism of the Infanticide Act is not the law's lenience, but rather its quasi-scientific basis. Professor Osborne echoed the sentiments of many when she concluded that the Act did not recognize the existence of a link between childbirth and infanticide, but created it. Even at the time of the Act's passage, it was unclear whether the Act was based on an actual belief that women who killed their children were mentally ill, or whether "a medical model was adopted to justify moderation in the imposition of punishments." In recent decades, as various English law reform groups have reconsidered the Infanticide Act, both supporters and critics of the Act agree that "there is little or no evidence for an association between lactation and mental disorder," and that mental disorder is "probably no longer a significant cause of infanticide."

Id.

[163] *See generally* A. F. CHALMERS, WHAT IS THIS THING CALLED SCIENCE? (1976) (discussing theories of the scientific method); James Woodward & David Goodstein,

At the time of the Acts' enactment, medical reports of a temporal relation between and among childbirth, lactation, and mental illness did exist. Recall the medical observations, dating back to 400 B.C., of the temporal relation between the occurrence of female-specific reproductive events and the symptoms of mental illness.[164] Further recall eighteenth century German physician reports of mental illness concurrent with lactation,[165] French psychiatrist Esquirol's observation in 1838 that mental illness in the postpartum period was caused by a woman's failure to nurse,[166] British physician Blandford's late nineteenth century lectures recognizing both a "puerperal insanity" as well as an "insanity of lactation," the latter of which was believed to be caused by excessive nursing,[167] and British physician MacPherson's turn-of-the-century lectures attributing mental illness in the postpartum to both lactation and the sudden cessation of lactation.[168]

Given these published medical observations and formal medical school lectures, Parliament's decision to include a medical justification for infanticide (that is, the failure of a woman who committed infanticide to "fully recover[ ] from the effect of giving birth to such child, and by reason thereof the balance of her mind was then disturbed")[169] in the Infanticide Act of 1922 may be characterized as grounded in the medical literature that was available at that time. If asked to re-write the 1922 provision today, I might add a clinical definition of postpartum psychosis and delete the old-fashioned "disturbance" and "balance of mind" language. Given the early medical observations of mental illness concurrent with or following childbirth, however, I would not characterize the association between childbirth and mental illness made by the Infanticide Act of 1922 as not based in then-current medical literature. Stated another way, I would not say that the Infanticide Act of 1922 legislatively created a link between childbirth and mental illness where none existed.[170]

Conduct, Misconduct and the Structure of Science, 84 AM. SCIENTIST 479, 479–90 (1996) (discussing the ethical implications of the various theories of the scientific method).

[164] *See supra* text accompanying note 30.

[165] *See supra* text accompanying notes 32–33.

[166] *See supra* text accompanying notes 35–38.

[167] *See supra* text accompanying notes 39–41.

[168] *See supra* text accompanying notes 42–43.

[169] Infanticide Act of 1922, § 1(1)

> Where a woman by any wilful act or omission causes the death of her newly-born child, but at the time of the act or omission she had *not fully recovered from the effect of giving birth to such child, and by reason thereof the balance of her mind was then disturbed*, she shall, notwithstanding that the circumstances were such that but for this act the offence would have amounted to murder, be guilty of . . . the offence of manslaughter of such child.

Id. (emphasis added); *see also* MCKEE, *supra* note 149, at 42 (discussing the Infanticide Act of 1922).

[170] *See, e.g.*, Oberman, *supra* note 159, at 19 (asking whether infanticide acts were recognizing known links between childbirth and mental illness or were legislatively creating them).

The next question is whether Parliament legislatively created additional links between and among childbirth, lactation, and mental illness where none existed when it reconsidered the provisions of the Infanticide Act of 1922 in 1938 and decided not only to maintain the first medical justification for infanticide (that is, the "effects of childbirth"), but to add a second medical justification relating to lactation (that is, "the balance of her mind was disturbed . . . by reason of the effect of lactation consequent upon the birth of the child").[171] The medical literature available in 1938 continued to support the concept of mental illness associated with childbirth as well as the concept of mental illness associated with lactation (although today's scientific literature would not support and actually refutes a causal relationship between lactation and mental illness). Consider, for example, a 1936 *London Times* report of a case involving a 24-year-old mother who was tried that year in England for murdering her second child three weeks after the child's birth.[172] According to the *Times*, Lord Dawson of Penn, President of the Royal College of Physicians, testified on behalf of the defendant mother that she was suffering from "puerperal insanity and did not know what she was doing."[173] Lord Dawson's testimony suggests that he, and perhaps other physicians associated with the prestigious Royal College of Physicians, believed in the existence of a form of insanity occurring during or following childbirth, and that the symptoms of individual women could lead to this or another form of insanity classification.

Medical reports such as these, as well as public beliefs based on these medical reports, did not go unnoticed by British lawmakers, including lawmakers involved in the passage of the Infanticide Act of 1938. While urging a second reading of the bill that would become the Act, Viscount Dawson of Penn testified that the purpose of the bill was to protect ill new mothers: "[T]he intention of this Bill is to secure recognition by Parliament that under certain circumstances the killing of infants is provoked by illness and not always by criminal intent, and to procure for such cases appropriate handling."[174] Viscount Dawson did not reference particular scientific studies or medical authorities, although he did refer to what might be called a public belief that some women who kill their children seem to do so as a result of the effects of childbirth: "The horse sense of the public detected that amongst these cases of child murder there were two groups. There was what we know as the child-birth group, and there was the group which constituted the murdering of unwanted children . . . ."[175] Viscount Dawson concluded his testimony by re-emphasizing that certain offenses, including some infan-

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[171] Infanticide Act of 1938, § 1(1).

[172] *Infanticide*, London Times, July 22, 1936, at 13.

[173] *Id.*

[174] 108 Parl. Deb., H.L. (5th ser.) (1938) 292. Viscount Dawson also stated that until the Infanticide Act of 1922, "there was no comprehension that there is a group of cases in which the cause is illness rather than criminal intent." *Id.* at 293.

[175] *Id.* at 292.

ticides, can be due to illness and that both humanity and present-day thinking require leniency to certain postpartum women:

> [C]ertain offences can be due to illness, and if and when that mental illness amounts to irresponsibility for the offence charged, I suggest that the patient should neither be punished nor dubbed a lunatic but bound over for appropriate treatment. I suggest that that would be a practice more in harmony with present-day thinking and with sound humanity, and I beg to move the Second Reading of this Bill.[176]

Other Lords present at the second reading of the bill that would become the Infanticide Act of 1938 also referenced an association between childbirth and mental illness. The Lord Archbishop of Canterbury testified:

> My Lords, I think there can be no question that the women with whom this Bill deals are entitled to our compassion; women, that is to say, who, owing to the effects of child-birth and the instability of mind which it sometimes occasions, have committed an act for which they either cannot be regarded as responsible or of which they could not, at the time, be supposed to be able to discern the wrong.[177]

Lord Snell agreed: "[I]t is frequently a cause of ill-health rather than of criminality."[178] Perhaps in response to the unstated concern that mentally healthy mothers who had given birth to unwanted children would take advantage of the leniency afforded by the 1938 Act, Lord Archbishop of Canterbury also testified that physicians were capable of distinguishing mentally disturbed women situated in the postpartum period from other women: "I am told that the evidences of this unhappy form of insanity are well known and can easily be recognised."[179]

The bill, the second reading of which was being urged by Lord Archbishop of Canterbury and his colleagues, ultimately became the Infanticide

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[176] *Id.* at 298.
[177] *Id.* at 301.
[178] *Id.* at 298.
[179] *Id.* at 302. This statement was preceded by:

> I wondered whether there would not be a risk under this Bill that the mother of such a very unwanted child might be tempted to avail herself, after the child was actually horn, of the merciful provisions of this Bill in order to get rid of it; but I am assured by my noble friend that by no possibility could the mere act of infanticide alone be regarded as evidence of disturbance of mind, but that in every case it would have to be clearly proved that there was, quite apart from the act itself, evidence of that natural illness.

Id.

Act of 1938. That Act represents the modern international trend.[180] Canada, Zimbabwe, New South Wales, and other jurisdictions have also enacted laws that permit women who kill their children within certain time periods following birth to be charged with a lesser crime if, at the time of the killing, the woman's mind is disturbed due to a failure to recover from birth and, in some jurisdictions, the effects of lactation.[181]

International infanticide laws remained on the books for a few decades with little medical controversy. In the last third of the twentieth century, however, scientists began investigating the purported scientific relationship between lactation and mental illness and reporting negative findings. In 1996, two Texas scientists published a lengthy article reviewing the available scientific literature and concluding that lactation does not increase a woman's risk of developing postpartum illness.[182] Throughout the first decade of the twenty-first century, scientists also published studies suggesting that oxytocin, a hormone produced during breastfeeding, may actually promote and reinforce maternal behavior, and that breastfed babies are less likely to be neglected.[183]

Shortly following the release of these new studies and review articles, a number of international law reform commissions began recommending the

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[180] *See, e.g.*, Gardner, *supra* note 158, at 958 (explaining that England, Canada, and other countries have female-specific infanticide laws); Fisher, *supra* note 157, at 615–16 (same).

[181] *See, e.g.*, Canada Infanticide Act (codified at R.S.C., ch. C-46, § 233 (1985)) ("A female person commits infanticide when by a willful act or omission she causes the death of her newly-born child, if at the time of the act or omission she is not fully recovered from the effects of giving birth to the child and by reason thereof or of the effect of lactation consequent on the birth of the child her mind is then disturbed."); Infanticide Act, 1990 ZWE-1990-L-57018, No. 27 of 1990 (codified at Criminal Law Act, Chapter 5, Part I, § 48 (Zimb.)) ("Any woman who, within six months of the birth of her child, causes its death . . . intentionally; or . . . by conduct which she realises involves a real risk to the child's life; at a time when the balance of her mind is disturbed as a result of giving birth to the child, shall be guilty of infanticide if the evidence establishes that she committed that crime."); New South Wales Crimes Act 1900, § 22A(1) (Austl.) ("Where a woman by any wilful act or omission causes the death of her child, being a child under the age of twelve months, but at the time of the act or omission the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child or by reason of the effect of lactation consequent upon the birth of the child, . . . she shall be guilty of infanticide . . . .").

[182] Dunnewold, *supra* note 44, at 25 (exploring the connection between breastfeeding and postpartum illness and concluding that breastfeeding does not increase the risk of developing postpartum depression or anxiety); *see also* Ingram, *supra* note 44, at 62 (referencing Dunnewold and Crenshaw's findings). Interestingly, several studies suggest that depressive symptoms early in the postpartum period may actually lower the prevalence of breastfeeding. *See, e.g.*, Daniel C. Hatton, Jennifer Harrison-Hohner, Sarah Coste, Veronica Dorato, Luis B. Curet & David A. McCarron, *Symptoms of Postpartum Depression and Breastfeeding*, 21 J. Hum. Lactation 444, 444 (2005).

[183] *See, e.g.*, Lane Strathearn, Abdullah A. Mamun, Jake M. Najman & Michael J. O'Callaghan, *Does Breastfeeding Protect Against Substantiated Child Abuse and Neglect? A 15-Year Cohort Study*, 123 Pediatrics 483, 483 (2009) ("Among other factors, breastfeeding may help to protect against maternally perpetrated child maltreatment, particularly child neglect.").

repeal of all or part of their infanticide acts, based in part on their lack of continuing medical and scientific support.[184] In 1997, for example, the New South Wales Law Reform Commission recommended the repeal of New South Wales' infanticide provision,[185] due to the lack of evidence supporting a causal relationship between lactation and mental illness.[186] According to the Commission, "it seems now to be generally doubted that there is any medical basis for the notion of 'lactational insanity.'"[187] Other international law reform commissions, including those of England, Wales, and Victoria also recommended omitting from their jurisdictions' infanticide provisions any reference to lactation as a ground for mental illness due to the relationship's dubious validity.[188]

Notwithstanding these efforts, the provisions of the English Infanticide Act of 1938, including the concept of lactational insanity, remain intact and represent the modern international trend, although not the law of the United States.[189] Assuming that an infanticide bill currently pending before the Texas Legislature does not become law,[190] American neonaticide (the killing of a baby during the first day of life), infanticide (the killing of a child less than one year of age), and filicide (the killing of a child older than one year of age)[191] generally are prosecuted under the law of murder.[192] The states generally do not have stand-alone acts creating manslaughter-like infanticide offenses or recognizing either postpartum depression or postpartum psychosis as independent defenses to homicide (complete or partial), although evidence of postpartum psychosis has been used to support the defenses of insanity and diminished capacity as well as the verdict of "guilty but mentally ill," where available.[193] The United States' state-by-state and case-by-

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[184] *See* text accompanying notes 186–188, *infra*.

[185] *See* New South Wales Crimes Act 1900, § 22A (Austl.) (infanticide provision); *id.* § 23A (providing a defense of diminished responsibility).

[186] Law Reform Commission, New South Wales, Report 83—Partial Defenses to Murder—Provocation and Infanticide, § 3.27 (1997) [hereinafter LRC] ("The offence/defence of infanticide requires the existence of mental disturbance resulting from the effects of lactation or the effects of giving birth. The validity of these medical principles has been widely questioned.").

[187] *Id.* at § 3.28.

[188] *See id.*; Criminal Law Revision Committee, Offences Against the Person, 1980, Cmnd. 7844, at 47 (Eng.); Law Commission of England and Wales, 1 Criminal Code for England and Wales (Law Com 177, 1989) c. 64(1); Law Reform Commission of Victoria, Mental Malfunction and Criminal Responsibility (Report No. 34, 1990), Rec. 28 at ¶ 166 (Austl.).

[189] *See, e.g.*, Connel, *supra* note 180, at 162–64 (recommending that jurisdictions in the United States adopt infanticide provisions); Fisher, *supra* note 157, at 615–16 (discussing international trends in infanticide legislation).

[190] H.B. 3318, 81st Reg. Sess. (Tex. 2009); *see infra* text accompanying notes 197–200.

[191] McKee, *supra* note 149, at 5 (distinguishing neonaticide, infanticide, and filicide).

[192] *See, e.g.*, Colleen Kelly, *The Legacy of Too Little, Too Late: The Inconsistent Treatment of Postpartum Psychosis as a Defense to Infanticide*, 19 J. Contemp. Health L. & Pol'y 247, 248–49 (2002) ("In the United States, infanticide falls under general homicide statutes, which vary from state to state.").

[193] *See, e.g.*, *id.* at 249 (summarizing state approaches to infanticide).

case approach has been criticized for its lack of uniformity, consistency, and justice.[194] Indeed, law professor Daniel Maier Katkin studied twenty-four American infanticide cases in which the mother asserted postpartum psychosis as a legal defense.[195] Of those cases, eight women were judged not guilty by reason of insanity, four were given probation, three were incarcerated for less than five years, seven were incarcerated between five and twenty years, and two were sentenced to life imprisonment.[196]

Some U.S. lawmakers have responded to the lack of infanticide-specific offense or defense provisions by introducing international-like infanticide bills. On March 11, 2009, Representative Jessica Farrar (D-Houston) introduced Texas House Bill 3318 ("H.B. 3318") to amend the Texas Penal Code to create the offense of infanticide, punishable as a state jail felony.[197] H.B. 3318 generally tracks the language of the English Infanticide Act of 1938 (including its dual medical bases of childbirth and lactation), and establishes the offense of infanticide defendants whose "judgment was impaired as a result of the effects of giving birth or the effects of lactation following the birth."[198] Texas Representative Farrar included the now-refuted medical justification of lactational insanity notwithstanding readily available scientific evidence—including scientific review articles authored by local Texas scientists—to the contrary.[199] Like its international predecessors, H.B. 3318 suggests to the public and the Texas legal community that a woman's mind can be "disturbed" during the twelve-month-period following birth due to the effects of lactation. The bill remains pending in the Texas House Criminal Jurisprudence Committee as of this writing.[200]

International and U.S. infanticide legislation thus provides one measure of the legal understanding of postpartum illness. When prominent physicians report associations between and among childbirth, lactation, and mental illness century after century, these reports become part of the public and legal understanding of postpartum behavior. Even when scientists later refute a purported scientific relationship, such as the alleged causal relationship between lactation and mental illness, the now incorrect relationship may remain in the law, as in the case of international infanticide laws that con-

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[194] *See, e.g.*, Connel, *supra* note 180, at 146–53, 161–62 (summarizing state approaches to infanticide and the criticisms thereof).

[195] Daniel Maier Katkin, *Postpartum Psychosis, Infanticide, and Criminal Justice*, *in* Postpartum Psychiatric Illness: A Picture Puzzle 275, 279 (James A. Hamilton & Patricia N. Harberger eds., 1992).

[196] *Id.*

[197] Tex. H.B. 3318, *supra* note 190; Texas Legislature Online, 81(R) Actions for HB 3318, http://www.legis.state.tx.us/BillLookup/Actions.aspx?LegSess=81R&Bill=HB3318 (last visited Nov. 15, 2009) (providing information about bill filing date and sponsorship).

[198] Tex. H.B. 3318, *supra* note 190, at § 1.

[199] *See* Dunnewold, *supra* note 44, at 25–26.

[200] Texas Legislature Online, 81(R) Actions for HB 3318, http://www.legis.state.tx.us/BillLookup/Actions.aspx?LegSess=81R&Bill=HB3318 (last visited Nov. 15, 2009) (showing that the Committee Report was sent to Calendars on April 28, 2009).

tinue to allow impaired judgment concurrent with lactation to serve as a ground for a lesser charge of infanticide. Even after scientists have conclusively refuted a purported scientific relationship, unwitting lawmakers borrow language from old laws referring to the outdated science to create new laws, as in the case of the 2009 Texas infanticide bill that refers to impaired judgment caused by lactation, thus perpetuating the law's incorrect understanding of postpartum illness.[201]

B. Judicial Interpretation of Health Insurance Policies

Criminal infanticide laws provide one measure of the legal understanding of postpartum illness.[202] Judicial opinions interpreting health insurance policies that distinguish physical illness from mental illness and provide fewer benefits for the treatment of mental illness provide a second measure. At least two federal courts, the United States District Court for the Southern District of Florida and, on appeal, the United States Court of Appeals for the Eleventh Circuit, appear open to recognizing postpartum illness as a physical illness, but would understandably require physiological proof, such as a test result showing an imbalance in serotonin or norepinephrine, before requiring an insurance company to provide comprehensive coverage of postpartum treatments in accordance with the company's physical illness policy provisions.[203] Because the plaintiff in the case failed to introduce physical evidence of her illness, the courts classified her illness as mental for purposes of applying the insurance policy's coverage limits.[204]

At issue in this case, *Blake v. UnionMutual Stock Life Insurance Company*, was the proper interpretation of a provision within Pam Blake's health insurance policy that limited insurance coverage of mental illnesses (defined as "any mental, nervous or emotional diseases or disorders") to 30 days of inpatient care and $1,000 worth of outpatient treatments.[205] Familiar with the hormone- and neurotransmitter-based theories of postpartum illness described in Parts I(C) and I(D) of this Article, Blake believed that her postpartum depression should be classified as a physical illness, defined in the policy as an "illness or disease . . . [including] pregnancy unless excluded elsewhere."[206] When Blake's insurance company classified her postpartum

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[201] *See supra* text accompanying notes 197–200.

[202] *See supra* Part II(A).

[203] *See infra* text accompanying notes 205–212.

[204] Blake v. UnionMutual Stock Life Ins. Co., 906 F.2d 1525, 1527 (11th Cir. 1990) (affirming district court's findings of fact and reasoning with regards to the insurance policy); Blake v. UnionMutual Stock Life Ins. Co., No. 87-0543-CIV, 1989 U.S. Dist. LEXIS 16331, at *4–*5 (S.D. Fla. Mar. 10, 1989) ("Because of Plaintiffs' failure to prove an organic causation for this illness, we find that . . . she suffered a mental illness within the terms of the policy.").

[205] *Blake*, 1989 U.S. Dist. LEXIS 16331 at *4.

[206] *Id.*

depression as a mental illness and refused to cover the entirety of its treatment, Blake sued to recover $33,279.55 in unpaid medical bills.[207]

At trial, the United States District Court for the Southern District of Florida reviewed the evidence provided about Blake's postpartum depression and was asked to decide whether Blake had a physical or a mental illness.[208] Although several expert and treating psychiatrists and psychologists testified that imbalances in serotonin and norepinephrine, as well as other hormonal imbalances, caused Blake's postpartum depression, the court focused on Blake's failure to introduce into evidence any hormonal or other tests or measurements that could prove that she had a physical illness: "[W]hile hormonal changes may be an important cause of postpartum depression, no proof has been offered in this case to show that the levels in Mrs. Blake were abnormal following childbirth."[209] The court thus held that Blake had a mental illness that was subject to the less comprehensive insurance coverage.[210] The Eleventh Circuit affirmed the district court's decision.[211]

It is tempting to classify the *Blake* opinions, now 19 and 20 years old, as examples of the courts' then-inability to understand postpartum depression as anything other than a mental illness, despite the emerging scientific literature documenting the role or roles likely played by sex hormones, neurotransmitters, brain structure, brain function, and genetics in the illness's etiology. The carefully worded holdings, however, show that the courts focused more on Blake's failure to provide evidence proving an organic cause of her postpartum depression than their desire to pigeonhole postpartum depression as a non-physical nervous disorder.[212] The opinions do not foreclose the possibility that future insureds can prove that their postpartum depressions are physical illnesses if they can provide test results showing chemical or hormonal imbalances or other physical pathology.[213]

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[207] *Id.*

[208] *Id.* ("Plaintiffs argue that the Defendant applied the wrong section [of the insurance policy].").

[209] *Id.* at *9–*10. The court also stated that:

> Neither Pam Blake's serotonin and neopinephrine levels nor her hormonal levels were ever measured so far as this Court is aware. While Dr. Moreno did state that testing the serotonin and neopinephrine levels is difficult, she did not state why Mrs. Blake's hormonal levels were never measured. Dr. Moreno's testimony simply failed to prove a physical illness caused Mrs. Blake's psychiatric hospitalization.

Id. at *8.

[210] *Id.* at *12 ("Because of Plaintiffs' failure to prove an organic causation for this illness, we find that the treatment Mrs. Blake received is only more convincing proof that she suffered a mental illness within the terms of the policy.").

[211] *Blake*, 906 F.2d at 1527.

[212] *Blake*, 1989 U.S. Dist. LEXIS 16331, at *13 ("[T]he Court concludes that the Plaintiffs have failed to demonstrate that Pam Blake suffered an organic mental illness under our de novo review.").

[213] *See, e.g.*, Phillips v. Lincoln Nat'l Life Ins. Co., 978 F.2d 302, 309 (7th Cir. 1992) ("[O]ur reading of *Blake* leads us to conclude that had the plaintiff demonstrated an

Notwithstanding the carefully-worded *Blake* opinions, some subsequent courts have referenced the opinions as support for a second proposition: that postpartum depression is a mental illness, not a physical illness, because postpartum depression is treated by a psychiatrist using well-recognized psychiatric treatments such as psychoactive drug therapy and electroconvulsive therapy.[214] Elsewhere I explored the different tests used by courts to determine whether an illness is physical or mental for purposes of interpreting a health or disability insurance policy, including tests that inquire as to the nature of the (1) treatment provided to the patient (tube feedings provided to eating-disordered patients leading to the classification of eating disorders as a physical illness, but psychotherapy, psychotropic medications, and electroconvulsive therapies leading to a finding of mental illness); (2) symptoms of the illness (symptoms of dehydration and malnourishment leading to the classification of eating disorders as a physical illness, with symptoms of high moods followed by low moods leading to the classification of bipolar disorder as mental illness); and (3) origin of the disease (expert testimony regarding the neurobiological origin of bipolar disorder leading to the classification of bipolar disorder as a physical illness).[215] Subsequent courts' understanding of Pam Blake's postpartum depression as a mental illness based on the types of treatments she received constitutes a judicial application of the first common law test, the "treatment provided to the patient" test.

Advances in the scientific understanding of a range of illnesses traditionally classified as "mental" beg for a reconsideration of this test. For example, does the test comport with the current scientific understanding of illness if the test results in the classification of an illness as "mental" just because the illness is treated by a psychiatrist? For the moment, refrain from the common law urge to view any treatment provided by a psychiatrist as a treatment for mental illness. Further, consider that psychiatrist-prescribed psychoactive drugs (that is, chemical substances that act primarily upon the central nervous system by altering brain function) and psychiatrist-ordered electroconvulsive therapy (that is, a procedure that delivers a brief electrical current to the brain through the placement of electrodes on one or both sides of the head for the purpose of inducing seizures) are treatments that rely on chemicals and electricity to produce physiological changes in the brain.

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organic basis for her illness, the Eleventh Circuit may well have held that the policy's mental illness limitation did not apply.").

[214] *See, e.g.*, Fitts v. Fed. Nat'l Mortgage Ass'n, 191 F.Supp. 2d 67, 74 (D.D.C. 2002) (citing Blake v. UnionMutual Stock Life Ins. Co., 906 F.2d 1525, 1530 (11th Cir. 1990)) (noting parenthetically that *Blake* "not[ed] that the plaintiff's postpartum depression was properly considered a mental illness because 'she was treated primarily by psychiatrists receiving well recognized psychiatric treatment, including individual psychotherapy, psychoactive drug therapy, electroconvulsive therapy and participation in group sessions.'").

[215] *See* Stacey A. Tovino, *Neuroscience and Health Law: An Integrative Approach*, 42 Akron L. Rev. 469, 478–83 (2009).

Now compare psychiatrist-prescribed psychoactive drugs and psychiatrist-ordered electroconvulsive therapy to the types of drugs and devices prescribed and ordered by non-psychiatrist physicians for a range of illnesses traditionally classified as "physical." For example, consider a neurologist who prescribes the drug Levodopa to help a patient control the symptoms of her Parkinson's disease, characterized by trembling of the arms and legs, stiffness and rigidity of the muscles, and slowness of movement.[216] Levodopa works by encouraging dopamine receptors in the brain to bypass degenerating brain cells.[217] Patients who have Parkinson's disease and are treated with Levodopa are considered by their health insurance companies to be individuals with physical illnesses, not mental illnesses, even though Levodopa has its primary physiological effect on the brain.[218]

Additionally, consider a neurologist who implants a device called a vagus nerve stimulator in a patient who has a treatment-resistant seizure disorder.[219] Vagus nerve stimulators work by sending small electrical pulses to the vagus nerve, which delivers the pulses to the brain and helps prevent the electrical irregularities in the brain that cause seizures.[220] Patients who have seizure disorders and are implanted with vagus nerve stimulators are considered by their health insurance companies to be individuals with physical illnesses, not mental illnesses, even though the implanted devices have their primary physiological effect on the brain.[221]

Psychoactive drugs used to treat psychiatric patients and dopamine-enhancing drugs used to treat patients with neurological disorders both produce a physiological effect in the brain. Electroconvulsive therapy used to treat psychiatric patients and vagus nerve stimulation used to treat patients with neurological (seizure) disorders both use electricity to produce a physiological effect in the brain. Yet application of the common law "treatment provided to the patient" test would result in the classification of patients treated

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[216] *See, e.g.*, Miho Murata, *Levodopa in the Early Treatment of Parkinson's Disease*, 15 PARKINSONISM & RELATED DISORDERS S17 (2009) (discussing the use of Levodopa for the treatment of Parkinson's disease).

[217] *Id.*

[218] *See, e.g.*, Michael J. Carroll, *The Mental Health Parity Act of 1996: Let It Sunset if Real Changes Are Not Made*, 52 DRAKE L. REV. 553, 579 & n.203 (2004) (noting that most health insurance plans cover Parkinson's disease under their physical illness provisions).

[219] *See, e.g.*, Cyberonics, VNS Therapy™ Basics, http://www.vnstherapy.com/epilepsy/patient/About_Basics.asp (last visited Nov. 15, 2009) (describing the cause of some seizure disorders and their treatment by vagus nerve stimulation).

[220] *Id.*

[221] *See, e.g.*, Centers for Medicare and Medicaid Services, Pub. 100-04*, Medicare Claims Processing Transmittal 1271*, § I(A) (June 22, 2007), *available at* http://www.cms.hhs.gov/Transmittals/Downloads/R1271CP.pdf (noting that the Medicare Program covers vagus nerve stimulation for medically refractory partial onset seizures when surgery is not recommended or has failed); *see also* Jones v. ING N. Am. Ins. Group, No. 8-087 / 07-1099, 2008 Iowa App. LEXIS 135, at *6 (Iowa Ct. App. Feb. 27, 2008) (noting that an insurance company determined that an insured's illness was physical due in part to her history of epilepsy).

with Levodopa and vagus nerve stimulation as patients with physical illnesses and the classification of patients treated with psychoactive drugs and electroconvulsive therapy as patients with mental illnesses simply because the latter set of patients are treated by psychiatrists. Stated another way, the "treatment provided to the patient" test can lead to illogical results when similar treatments that produce similar physiological effects are prescribed or ordered by both psychiatrist and non-psychiatrist physicians.

Judicial opinions interpreting health insurance policy provisions thus provide a second measure of the legal understanding of postpartum illness. When a claimant sues a health insurance company in an attempt to seek more comprehensive insurance coverage under a health insurance policy's physical illness provision, the claimant is essentially asking the court to make a medical—and thus contractual—determination that the claimant's condition is physical rather than mental in nature. A health insurance claimant is encouraged to request this determination by the health insurance policy itself, which distinguishes physical and mental illness even though the distinction may be characterized as reductionist and perpetuative of a mind-body dualism that is not necessarily supported by the current scientific literature.[222] Even in cases in which forward-thinking courts give substantial weight to expert testimony regarding the likely biological etiology of an insurance claimant's particular illness, procedural technicalities (such as the failure of the claimant to properly introduce evidence supporting such neurobiology), combined with subsequent courts' reinterpretation of these cases based on arguably outdated common law tests, can reinforce the traditional legal understanding of postpartum illness as a mental illness. As discussed in more detail in Part III, I anticipate that these legal understandings may change as new federal and state mental health parity laws are litigated and to the extent a physiological diagnostic test for postpartum illness, such as a placental corticotropin-releasing hormone test for postpartum depression[223] or a structural or functional neuroimaging test for postpartum illness[224] becomes publicly available.

C. Postpartum Awareness Legislation

Criminal infanticide legislation and judicial opinions interpreting health insurance policy provisions provide two measures of the legal understanding of postpartum illness. Laws that seek to improve postpartum illness aware-

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[222] *See, e.g.*, DSM IV-TR, *supra* note 25, at xxx ("Although this volume is titled the *Diagnostic and Statistical Manual of Mental Disorders*, the term *mental disorder* unfortunately implies a distinction between 'mental' disorders and 'physical' disorders that is a reductionistic anachronism of mind/body dualism. A compelling literature documents that there is much 'physical' in 'mental' disorders and much 'mental' in 'physical' disorders.").

[223] *See supra* text accompanying notes 66–70.

[224] *See supra* text accompanying notes 95–98.

ness, education, treatment, research funding, and health and disability insurance coverage ("postpartum awareness laws") provide a third measure. During the last decade, federal and state lawmakers have introduced a number of postpartum awareness laws that would, among other things (i) require the Secretary of the Department of Health and Human Services to make available grants that would provide education to new mothers and their families about postpartum illness, provide screening for postpartum illness during the first year of postnatal check-up visits, and provide treatment for mothers with postpartum conditions;[225] (ii) direct the heads of federal agencies to develop a research plan relating to postpartum illness;[226] (iii) require the compilation and synthesis of data relating to postpartum illness;[227] (iv) proclaim certain days and months in certain states as Postpartum Depression Awareness Day[228] or Month,[229] as appropriate; (v) prohibit the denial of disability insurance for a history of postpartum depression;[230] and (vi) require public and private health plans to provide insurance coverage for treatments for postpartum illness.[231]

A careful reading of the legislative findings and preambles in these statutes suggests that part of the impetus for these laws is the improved, although not necessarily correct, legal understanding of the etiology of postpartum illness. In a 1999 West Virginia resolution requesting the study of postpartum depression and encouraging the United States Surgeon General to place postpartum depression on the agenda of a forthcoming study of suicide, the West Virginia House found that, "[p]ostpartum depression is the result of a chemical imbalance triggered by a sudden, dramatic drop in hormonal production after the birth of a baby . . . ."[232] One year later, when

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[225] Mom's Opportunity to Access Health, Education, Research, and Support for Postpartum Depression (MOTHERS) Act, S. 1375, 110th Cong. (2007).

[226] *Id.*

[227] *See, e.g.*, H.B. 6567, 2001 Gen. Assem., Reg. Sess. (R.I. 2001) (proposed Rhode Island House resolution directing the Rhode Island Department of Health to establish a panel to compile and synthesize data relating to postpartum depression and psychosis).

[228] *See, e.g.*, S. Res. 164, 210th Leg., 2d Reg. Sess. (N.J. 2003) (New Jersey Senate Resolution declaring June 25, 2003, as Postpartum Depression Awareness Day).

[229] *See, e.g.*, Assem. Con. Res. 51, 2003-2004, Reg. Sess. (Cal. 2003) (California Assembly Concurrent Resolution proclaiming May 2003 as Postpartum Mood and Anxiety Disorder Awareness Month).

[230] *See, e.g.*, An Act Prohibiting Denial of Disability Insurance for Treatment for Depression Due to Menopause, Postpartum Depression, and Pregnancy, H. File 634, 79th Gen. Assem., 1st Sess. (Iowa 2001) (Iowa House Bill that would "prohibit[ ] an insurer from completely denying disability insurance coverage on the basis of treatment within the previous five years for depression due to pregnancy, postpartum depression, or menopause. The insurer may, however, in such circumstances, require a waiver of coverage for disability due to depression for a period of time not to exceed five years from the date of coverage.").

[231] H.B. 2964, 80th Leg., Reg. Sess. (Tex. 2007); S.B. 1388, 80th Leg., Reg. Sess. (Tex. 2007). *See generally* Susan S. Night, *A Missed Opportunity to Bring Change for Women Suffering from Postpartum Depression*, HEALTH L. PERSP. (2007), http://www.law.uh.edu/healthlaw/perspectives/2007/post-partumdepressionlegislation.pdf (discussing the value of legislation mandating benefits for treatment of postpartum illness).

[232] H. Con. Res. 47, 74th Leg. (W. Va. 1999).

the West Virginia Senate adopted a concurrent resolution requesting action to increase public awareness of postpartum depression and improve recognition and treatment of postpartum illness, the Senate also found that postpartum depression is a "serious medical condition resulting from the chemical imbalance which is triggered by the abrupt and dramatic drop in a woman's hormonal production following the birth of a baby."[233]

West Virginia is not the only state to suggest that postpartum depression has only one cause; that is, the sharp drop in hormones that occurs within a few days of childbirth. In a 2001 Pennsylvania bill seeking to establish a public awareness, education, screening, and treatment program for postpartum depression, the Pennsylvania General Assembly found that postpartum depression "is the result of a chemical imbalance triggered by a sudden dramatic drop in hormonal production after the birth of a baby . . . ."[234] The same findings are set forth in a pair of 2000 New Jersey bills that would require a New Jersey state agency to establish a public awareness campaign and develop policies and procedures for health care professionals and facilities concerning postpartum depression, and appropriating $50,000 therefor,[235] as well as a 2003 New Jersey Senate Resolution that would declare June 25, 2003 to be Postpartum Depression Awareness Day in New Jersey.[236]

Although the postpartum awareness laws introduced between 1999 and 2003 suggest a unicausal, hormone-based theory of postpartum illness, some federal and state bills and resolutions introduced during and after 2003 recognize the complexity of postpartum illness and suggest a broader range of contributing factors. In a 2003 California Assembly Concurrent Resolution proclaiming May 2003 as Postpartum Mood and Anxiety Disorder Awareness Month, California lawmakers found that,

> The medical community does not fully understand or recognize all factors contributing to postpartum mood and anxiety disorders, but it is believed that these disorders are caused by physiological factors, such as hormone levels, and can be exacerbated by such external risk factors as marital problems, sleep deprivation, and preexisting mental illnesses . . . .[237]

In the federal Mom's Opportunity to Access Health, Education, Research, and Support for Postpartum Depression Act, which Congress designed to ensure that new mothers and their families are educated about postpartum depression, screened for symptoms, and provided with essential services, Congress also recognized the complexity of postpartum illness and suggested a broader range of contributing factors:

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[233] S. Con. Res. 18, 75th Leg. (W. Va. 2000).
[234] S.B. 1172, 185th Gen. Assem., Reg. Sess. (Pa. 2001).
[235] Assem. B. 2775, 209th Leg. (N.J. 2000); S.B. 1111, 209th Leg. (N.J. 2000).
[236] S. Res. 164, 210th Leg. (N.J. 2003).
[237] Assem. Con. Res. 51, *supra* note 229.

> The causes of postpartum depression are complex and unknown at this time; however, contributing factors include: a steep and rapid drop in hormone levels after childbirth; difficulty during labor or pregnancy; a premature birth; a miscarriage; feeling overwhelmed, uncertain, frustrated or anxious about one's new role as a mother; a lack of support from one's spouse, friends, or family; marital strife; stressful events in life such as death of a loved one, financial problems, or physical or mental abuse; a family history of depression or mood disorders; a previous history of major depression or anxiety; or a prior postpartum depression.[238]

Perhaps the most accurate legal understanding of postpartum illness is that of Arizona lawmakers in a 2008 Arizona House Concurrent Resolution that simply stated "The causes of perinatal mood disorders are complex and not fully understood at this time."[239]

Postpartum awareness laws thus provide a third measure of the legal understanding of postpartum illness. Some recent postpartum awareness laws do recognize the complexity of postpartum illness and accurately identify a broad range of possible contributing factors. A second set of postpartum awareness laws remain premised on unicausal, hormone-based theories of postpartum illness that were outdated even at the time of the laws' introduction and enactment. Several possible explanations exist for this second set of laws. First, a lag may exist between the time of an advance in the scientific understanding of an illness and the public and legal understanding of that illness. Even with today's electronic (in advance of print) release of peer-reviewed scientific studies through PubMed and other digital collections of biomedical and life sciences journals, lawmakers may not have the time, desire, or ability to stay abreast of the current scientific literature. In addition, non-scientifically trained lawmakers may have difficulty understanding and accurately describing complex disease etiology in the few sentences that comprise the legislative findings portion of a statute. Finally, simple scientific explanations, including unicausal hormone-based explanations of postpartum illness, may appeal to lawmakers who wish to support their proposed legislation with ostensibly clean, objective, and persuasive authority.

D. A Contemporary Legal Understanding of Postpartum Illness

In this Part, I demonstrated that the legal understanding of the science used to support postpartum law and policy is not always accurate, that these inaccuracies are difficult to remove from the law once established, and that these inaccuracies tend to appear in new law notwithstanding readily acces-

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[238] MOTHERS Act, *supra* note 225, at § 2(7).
[239] S. Con. Res. 1029, 49th Leg., 1st Reg. Sess. (Ariz. 2009).

sible scientific evidence to the contrary. Stated slightly differently, the science used to formulate legislation, contractual provisions, and common law tests in the context of postpartum illness does not always meet the more rigorous standards for scientific evidence required by the common law or federal or state rules of evidence for use in litigation.[240]

Lawmakers who rely on inaccurate science to formulate health law and policy can inadvertently create several potential problems. The first potential problem relates to the abbreviated future of the legislative provision containing the inaccurate scientific reference. More specifically, the discovery of inaccurate scientific references within a law may lead to the repeal of the entire law even if the general policy underlying the law might be right but the science in one portion of the law is wrong. Assume for the sake of argument only that all criminal law scholars agree that female-specific criminal infanticide offense provisions that lower the maximum charge a mother can face for killing her child from murder to a manslaughter-like infanticide are jurisprudentially desirable. As discussed in Part II(A), many infanticide provisions contain two medical bases, that is, infanticide that results from the disturbance of the balance of the mother's mind due to the mother's failure to recover from the effects of giving birth as well as infanticide that results from a mother's impaired judgment due to lactation. In today's parlance, these two medical bases may be referred to as postpartum psychosis and lactational insanity. As discussed in Part I, the scientific literature currently supports the diagnosis of postpartum psychosis but not lactational insanity. When international law reform commissioners learned in the late twentieth century that the concept of lactational insanity was no longer supported by the scientific literature, they began recommending the repeal of their jurisdictions' entire infanticide offenses, not just the second medical basis of lactational insanity.[241] Thus, one risk of relying on inaccurate science to formulate a law is the eventual repeal of the entire law even though all but one of the law's medical bases is accurate. This problem is easily remedied by the repeal of only the inaccurate medical basis.

A second, more significant, concern relates to the development of the wrong health law and policy. The oft-cited controversy surrounding legislation mandating insurance coverage of high-dose chemotherapy with autologous bone marrow transplant ("HDC-ABMT") for treatment of breast cancer nicely illustrates this potential problem.[242] In the HDC portion of the

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[240] *See, e.g.*, Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 589–90 (1993); FED. R. EVID. 702.

[241] *See, e.g.*, LRC, *supra* note 186, at Recommendation 3 (recommending the repeal of the New South Wales infanticide offense provision) and § 3.28 (arguing that the New South Wales infanticide offense provision should be repealed because, "it seems now to be generally doubted that there is any medical basis for the notion of 'lactational insanity.'").

[242] *See, e.g.*, Michelle M. Mello & Troyen A. Brennan, *The Controversy Over High-Dose Chemotherapy With Autologous Bone Marrow Transplant For Breast Cancer*, 20 HEALTH AFFAIRS 101, 106–10 (Sept.–Oct. 2001) (discussing the enthusiastic adoption of

HDC-ABMT procedure, a breast cancer patient is administered a high dose of chemotherapy that is intended to kill the patient's cancer but has the side effect of disabling the patient's immune system.[243] In the ABMT portion of the procedure, the patient is reinfused with bone marrow or stem cells that were extracted prior to the administration of the high dose of chemotherapy in an attempt to restore the patient's immune system.[244] The theory behind HDC-ABMT is that the patient can be given a higher dose of chemotherapy, thus killing more cancer cells, than would otherwise be possible because the patient's immune system will be fortified following the chemotherapy through the transplant process.[245]

Initially, many health insurers refused to cover the approximately $80,000 cost of the HDC-ABMT procedure, relying on early clinical trials finding that HDC-ABMT was not more efficacious than less costly and less toxic standard doses of chemotherapy.[246] Insurers' initial refusals to cover the costs of HDC-ABMT led to intensive political lobbying by patient advocacy and other groups, which led to legislation in several states mandating health insurers to cover or offer coverage for the procedure.[247] The Missouri General Assembly, for example, passed a law in 1995 requiring certain health insurance policies created or renewed after January 1, 1996, to offer coverage for the treatment of breast cancer by HDC-ABMT.[248] The Missouri mandated benefit law remains on the books today notwithstanding the publication since 1998 of several carefully-designed clinical studies concluding that HDC-ABMT does not confer a survival advantage for women with breast cancer relative to standard-dose chemotherapy.[249]

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the unproven HDC-ABMT procedure in the clinical, legal, and insurance contexts); U.S. GEN. ACCOUNTING OFFICE, GAO/HEHS No. 96-83, HEALTH INSURANCE: COVERAGE OF AUTOLOGOUS BONE MARROW TRANSPLANTATION FOR BREAST CANCER 5–6 (1996) [hereinafter GAO REPORT] (discussing the enthusiastic adoption of the unproven HDC-ABMT procedure in the insurance context). *See generally* RICHARD A. RETTIG, PETER D. JACOBSON, CYNTHIA FARQUHAR & WADE M. AUBRY, FALSE HOPE: BONE MARROW TRANSPLANTATION FOR BREAST CANCER (2007) (discussing the enthusiastic adoption of the unproven HDC-ABMT procedure in the clinical, legal, and insurance contexts).

[243] Mello & Brennan, *supra* note 242, at 101.

[244] *Id.*

[245] *Id.*

[246] *Id.* at 102.

[247] *See* GAO REPORT, *supra* note 242, at 11 & n.14 (listing states that, by 1996, had passed or had pending legislation requiring health insurance coverage of HDC-ABMT).

[248] MO. REV. STAT. § 376.1200 (2000).

[249] *See, e.g.*, Gabriel N. Hortobagyi et al., *Randomized Trial of High-Dose Chemotherapy and Blood Cell Autografts for High-Risk Primary Breast Carcinoma*, 92 J. NAT'L CANCER INST. 225, 231 (2000) (concluding that, "[n]o relapse-free or overall survival advantage was associated with the use of high-dose chemotherapy, and morbidity was increased with its use."); W.P. Peters et al., *A Prospective, Randomized Comparison of Two Doses of Combination Alkyating Agents (AA) as Consolidation After CAF in High-Risk Primary Breast Cancer Involving Ten or More Axillary Lympth Nodes (LN): Preliminary Results of CALGB 9082/SWOG 9114/NCIC MA-13*, 18(1a) PROCEEDINGS AM. SOC'Y CLINICAL ONCOLOGY (1999); Sjoerd Rodenhuis et al., *Randomised Trial of High-Dose Chemotherapy and Haemopoietic Progenitor-Cell Support in Operable Breast Cancer with Extensive Axillary Lymph-Node Involvement*, 352 LANCET 515, 520 (1998); Edward

The enactment and persistence of state legislation mandating health insurance coverage of the HDC-ABMT procedure notwithstanding the initial and continued lack of scientific support for the efficacy and safety of the procedure is referenced in several cautionary law-medicine tales that emphasize (i) the dangers of allowing political pressures to overwhelm science in evaluating new therapies and rationing scarce resources;[250] (ii) the inappropriateness or incompetence of the legal system in resolving health insurance coverage disputes involving complex medical procedures and scientific research studies;[251] and, more generally (iii) the inability of mandated benefit and other health-related legislation to keep pace with evolving clinical standards of care.[252]

These lessons also apply in the context of postpartum law and policy. A legislative desire to provide a lower criminal charge for postpartum women who commit infanticide should not overwhelm or otherwise extend beyond the scientific literature, which no longer supports the concept of lactational insanity. The spring 2009 introduction of Texas H.B. 3318, one portion of which provides a lower criminal charge for women who commit infanticide if their judgment is impaired due to lactation, demonstrates the bill authors' inability to stay abreast of the current scientific literature.[253] If passed, the portion of Texas H.B. 3318 that refers to impaired judgment due to lactation would not only be scientifically incorrect, but would also encourage the introduction of expert testimony regarding future defendants' purported lactational insanity even though the proffered evidence should not survive standards for scientific evidence used in litigation.[254]

A third potential problem associated with the legal misunderstanding of science is the development of conflicts between different laws and policies. If passed, Texas H.B. 3318 would send a message to the Texas public and legal community that lactation can cause impaired judgment that can result in infanticide. This message may contradict the message sent by other Texas laws relating to the maternal, child, and societal benefits associated with lactation. For example, in Texas's Breastfeeding Rights and Policies Act, enacted in 1995, the Texas Legislature stated that breastfeeding "must be encouraged in the interests of maternal and child health and family values,"[255] and that "a woman's choice to breast-feed benefits the family, the

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A. Stadtmauer et al., *Conventional-Dose Chemotherapy Compared with High-Dose Chemotherapy Plus Autologous Hematopoietic Stem-Cell Transplantation for Metastatic Breast Cancer*, 342 NEW ENG. J. MED. 1069, 1074 (2000).

[250] *See, e.g.*, Mello & Brennan, *supra* note 242, at 102, 110–12.

[251] *Id.* at 112–14.

[252] *See, e.g.*, MASSACHUSETTS DIVISION OF HEALTH CARE FINANCE AND POLICY, COMPREHENSIVE REVIEW OF MANDATED BENEFITS IN MASSACHUSETTS: REPORT TO THE LEGISLATURE 1 (2008) ("It can, however, be a challenge for mandated benefits laws to keep apace with evolving clinical standards of care.").

[253] Tex. H.B. 3318, *supra* note 190.

[254] *See id.*

[255] TEX. HEALTH & SAFETY CODE ANN. § 165.001 (Vernon 2001).

employer, and society."[256] Because of the maternal and other benefits associated with breastfeeding, the Texas Legislature officially recognized breastfeeding as the best method of infant nutrition[257] and established the legal right of a mother to breastfeed in any place where she is authorized to be.[258] To the extent the 2009 infanticide bill suggests that lactation causes mental illness and can result in infanticide and the 1995 pro-breastfeeding law states that breastfeeding benefits mothers, babies, and society, the two pieces of legislation may be read as sending conflicting messages regarding the safety and desirability of breastfeeding.

A fourth potential problem associated with the legal misunderstanding of science relates to the perpetuation of outdated sexual stereotypes. When a postpartum awareness law incorrectly attributes postpartum illness solely to hormonal imbalances,[259] the law perpetuates the outdated stereotypes of women as hysterical, unstable, and unable to control their raging hormones.[260] These stereotypes may be used to justify discriminatory treatment against women in a range of employment, education, and other contexts.[261] Less dangerous are postpartum awareness laws that correctly attribute postpartum illness to a range of possible factors, including the biochemical, neuroanatomical, genetic, evolutionary, psychosocial, and sociocultural factors that also play a role in the development of illnesses such as major depressive disorder, bipolar disorder, and schizophrenia that occur in both male and female populations.[262]

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[256] *Id.* § 165.031.

[257] *Id.* § 165.001.

[258] *Id.* § 165.002.

[259] *See, e.g.*, H. Con. Res. 47, *supra* note 232.

[260] *See, e.g.*, ALAN M. DERSHOWITZ, *The PMS Defense Feminist Setback*, *in* THE ABUSE EXCUSE: AND OTHER COP-OUTS, SOB STORIES AND EVASIONS OF RESPONSIBILITY 53, 54–55 (1994) (criticizing legal decisions premised on women's hormonal imbalances on the grounds that they confirm the sexist stereotype of women unable to control their raging hormones); Huang, *supra* note 158, at 364, 363–65 (stating in the context of conversations regarding a premenstrual syndrome (PMS) criminal defense that, "[t]he development of a PMS defense could have retrograde ideological effects by reinforcing a conception of women as inherently irresponsible and unstable . . . which justif[ies] discriminatory treatment of women in employment, education, and political life as well as place[s] women under the insidiously patriarchal control of the medical establishment.") (internal quotation marks omitted).

[261] *See* Huang, *supra* note 158, at 365 (discussing whether recognition of female-specific conditions could lead to discrimination against women). *But see* Michelle Oberman, *"Lady Madonna, Children at Your Feet": Tragedies at the Intersection of Motherhood, Mental Illness, and the Law*, 10 WM. & MARY J. WOMEN & L. 33, 67 (2003).

> To the extent that the legal system considers the circumstances that shape the fabric of the daily life of its litigants who raise postpartum mental illness-related claims, it likely will bring into focus the very factors that led to her legal troubles. In so doing, the law might be able to nudge society in the direction of change, by forcing us to focus on what we can do to alter the circumstances that give rise to the tragedies associated with postpartum mental illness.

Id.

[262] *See, e.g.*, Peter F. Buckley, *Schizophrenia and Bipolar Disorder, Part I: Etiology and Diagnosis*, PSYCHIATRY WEEKLY (Nov. 26, 2007) *available at* http://www.psychiatry

In light of these and other potential problems, lawmakers, judges, and other stakeholders need to recognize the complexity of disease etiology, including the etiology of postpartum illness. To assist in these efforts, I now propose a contemporary legal understanding of postpartum illness that emphasizes the illness's incomplete understanding and likely multifactorial etiology, as well as the elimination or reformulation of common law tests designed to distinguish physical and mental illness.

I begin by proposing the elimination of lactation as a ground for a lesser criminal charge in infanticide legislation. The Infanticide Act of 1938, as amended and effective today in the United Kingdom, continues to allow a mother who kills her child to be charged with a lesser crime if "the balance of her mind was disturbed . . . by reason of the effect of lactation consequent upon the birth of the child."[263] If passed, Texas H.B. 3318 would also allow a Texas mother who kills a child to be charged with a lesser crime if, at the time of the killing, "her judgment was impaired as a result of . . . the effects of lactation following the birth."[264] The current scientific literature does not support a causal relationship between lactation and impaired judgment. I therefore recommend the elimination of the "by reason of the lactation consequent upon the birth of the child" language in the Infanticide Act of 1938, the "effects of lactation following the birth" language in Texas H.B. 3318, and similar language in other jurisdictions' infanticide laws.

I further propose the reformulation of some infanticide laws' remaining medical bases. The Infanticide Act of 1938, as amended and effective today in the United Kingdom, continues to allow a mother who kills her child to be charged with a lesser crime if at the time of the killing "the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child."[265] The Act, perhaps unconsciously, blames the symptoms associated with postpartum depression and psychosis on some women's failure to recover from giving birth. Technically, it is the physiological changes associated with giving birth combined with the influence of other psychosocial, sociocultural, and environmental factors that scientists currently believe contributes to postpartum illness, not a woman's failure to recover from childbirth. Texas H.B. 3318 is less accusatory and allows a mother who kills her child to be charged with a lesser crime if at the time of the killing, her "judgment was impaired as a result of the effects of giving birth."[266] I would therefore amend the Infanticide Act to more

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weekly.com/aspx/article/articledetail.aspx?articleid=640 (stating that both schizophrenia and bipolar disorder have incompletely understood, multifactorial etiologies that likely include genetic, environmental, and other components); Donald M. Hilty, Kathleen T. Brady & Robert E. Hales, *A Review of Bipolar Disorder Among Adults*, 50 PSYCHIATRIC SERVS. 201, 202–03 (1999) (discussing studies investigating the genetic, biochemical, neuroanatomical, and neurofunctional bases of bipolar disorder).

[263] Infanticide Act, 1938, 1 & 2 Geo. 6, c. 36, § 1(1) (Eng.).

[264] Tex. H.B. 3318, *supra* note 190, at § 1.

[265] Infanticide Act of 1938, at § 1(1).

[266] Tex. H.B. 3318, *supra* note 190, at § 1.

closely resemble the Texas bill to permit the application of the lower criminal charge "if the balance of her mind was disturbed as a result of the effects of giving birth" (or, if criminal law and evidence law scholars desire greater clinical specificity, "if the balance of her mind was disturbed due to the hormonal, biochemical, neuroanatomical, genetic, psychosocial, sociocultural, and/or other physiological or environmental factors scientists believe contribute to postpartum psychosis"). In the context of common law tests designed to distinguish physical and mental illness for purposes of applying health and disability insurance coverage provisions, I further propose the elimination of the "treatment provided to the patient test," which results in an almost automatic finding of mental illness if the patient is treated by a psychiatrist even if the psychiatrist uses a drug or device that is designed to produce a physiological response in the brain. In Part III, I suggest that the field of health law should consider a limited, legal merger of physical and mental illness, which would eliminate the need for a replacement common law test.

Finally, in the context of postpartum awareness laws that are designed to improve postpartum illness awareness, education, treatment, research funding, and health and disability insurance coverage, I propose the deletion of outdated references to unicausal theories of postpartum illness. For example, legislators should not include in the legislative findings portion of a postpartum awareness law language stating that, "[p]ostpartum depression is the result of a chemical imbalance triggered by a sudden, dramatic drop in hormonal production after the birth of a baby."[267] Instead, I would include a more accurate report of the current scientific understanding of postpartum illness, such as:

> The causes of postpartum illness are complex and not completely understood at this time. The current scientific literature supports the involvement of a number of different factors in the development of postpartum illness, including hormonal, neurochemical, neuroanatomical, genetic, evolutionary, psychosocial, sociocultural, physiological, and environmental factors. The scientific understanding of postpartum illness is rapidly changing. These legislative findings, as well as the substantive law below, should be reviewed and updated by the Legislature on a regular basis.

III. Legal Implications of Advances in the Scientific Understanding of Postpartum Illness

In the previous Part, I examined the current legal understanding of postpartum illness and found that a range of existing legal authorities, including

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[267] *See, e.g.*, H.R. Con. Res. 47, *supra* note 232 (establishing a unicausal, hormone-based theory of postpartum depression).

criminal infanticide laws, judicial opinions interpreting health insurance policy provisions, and postpartum awareness laws, contain inaccurate understandings of postpartum illness. In this final Part, I turn to the future and explore how advances in the scientific understanding of postpartum illness combined with recent changes to mental health parity law and disability discrimination law may impact the result of future litigation in favor of some women with postpartum illness. More broadly, I use mental health parity law and disability discrimination law as a platform from which to question the appropriateness of health law frameworks that continue to distinguish physical and mental illness, and I conclude by proposing a limited, legal merger of physical and mental illness.

A. Implications for Mental Health Parity Law

Although health insurance plans initially offered physical and mental health benefits under the same terms and conditions,[268] many health insurance plans, including employer-based plans, began reducing their mental health benefits in the 1970s.[269] Insurers and employers justified these benefit reductions on the grounds that mental health treatments were more expensive and less efficacious than treatments for physical illnesses.[270] Patients

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[268] Dana L. Kaplan, *Can Legislation Alone Solve America's Mental Health Dilemma? Current State Legislative Schemes Cannot Achieve Mental Health Parity*, 8 QUINNIPIAC L. REV. 325, 328 (2005). The background information provided in the next two and one-half paragraphs is updated and taken with copyright permission from Tovino, *Remarks, supra* note 87, at 949–50. Copyright permission granted October 29, 2008, 20:19:00 CST, by Jason M. Fuller, Symposium Editor, Akron Law Review.

[269] Kaplan, *supra* note 268, at 328.

[270] *See, e.g.*, RACHEL SETHI, JOANNE JEE, LISA CHIMENTO & D. RICHARD MAUERY, DESIGNING EMPLOYER-SPONSORED MENTAL HEALTH BENEFITS, DHHS Pub. No. SMA-06-4177, at 14 (2006).

> A key concern among employers is that providing better mental health care benefits will result in higher costs and increased utilization of those services. According to a trade journal published by International Society of Certified Employee Benefit Specialists, mental health care costs increased significantly throughout the 1980s and early 1990s; in one year (from 1987 to 1988), these costs grew by 27 percent. Spending for mental health disorders increased three and a half times between 1987 and 2000.

Id. (internal citations omitted); Allan Beigel & Steven S. Sharfstein, *Mental Health Care Providers: Not the Only Cause or Only Cure for Rising Costs*, 141 AM. J. PSYCHIATRY 668, 668 (1984).

> In 1955 mental health expenditures were estimated to be $1.2 billion, or 6% of all expenditures. By 1977 the total amount of expenditures for mental health care had risen to $19.6 billion, 12% of all expenditures. Even with a correction for population growth and price increases, this amounts to a fourfold increase in mental health expenditures.

Id.; Kaplan, *supra* note 268, at 328 (stating that mental health benefits are two to three times as expensive as physical illness benefits); Cheryl A.C. Brown, *Interpreting Exclusions or Limitations on Coverage for Mental Illness under Health and Disability Policies*, 35 THE BRIEF 52, 53 (Fall 2005).

with mental health conditions, on the other hand, worried that the stigma associated with mental illness, as well as their inability to literally prove the existence of their mental health conditions through routine blood, urine, X-ray, or other diagnostic tests, prompted the less comprehensive coverage.[271] Whatever the cause, the result was referred to as a mental health benefit disparity. Some health insurance plans that covered 365 days of inpatient care for physical illnesses, for example, began covering only 45 days of inpatient care for mental illnesses.[272] Plans that provide unlimited outpatient visits for treatment of physical illnesses might allow only 20 outpatient visits per year for treatment of mental disorders.[273] And plans that covered all or maybe 80% of the cost of treatment for physical illnesses began covering only 50% or less of the cost of treatment for a mental illness.[274]

In the late 1980s, some patients who sought and were denied additional mental health benefits due to these contractual disparities responded by suing their insurers, arguing that their conditions were physical rather than mental in nature and thus covered under the better set of insurance benefits.[275] The *Blake v. UnionMutual Stock Life Insurance Company* case, examined in Part II(B), is one example.[276] In these contract-based lawsuits, the expert witnesses (usually psychiatrists and psychologists) routinely referenced advances in the behavioral and brain sciences to support the arguments of the insured parties.[277] Sometimes the insureds won these lawsuits

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> Until as recently as 1990, the diagnosis and treatment of mental illnesses were relatively unsuccessful. Thus, to avoid the long-term expenses associated with mental illnesses, many policies have addressed the nature of the coverage provided for mental illness. Typically, they exclude or limit coverage, including the period for which benefits are payable.

Id.

[271] *See, e.g.*, The Carter Center, The Carter Center Mental Health Program: Combating the Stigma of Mental Illness, http://www.cartercenter.org/health/mental_health/index.html (last visited Nov. 15, 2009) (explaining that much has changed since the time mental illnesses were shrouded in shame and stigma, but that individuals with mental illness continue to face stigma and discrimination, including as a result of mental health insurance disparities).

[272] *See* Brian D. Shannon, *Paving the Path to Parity in Health Insurance Coverage for Mental Illness: New Law or Merely Good Intentions?* 68 U. COLO. L. REV. 63, 68 (1997).

[273] *See id.*

[274] *See e.g., id.* at 68 n.17; Eric Beal, Note, *It's Better to Have Twelve Monkeys Chasing You Than One Gorilla:* Humana Inc. v. Forsyth*, the McCarran-Ferguson Act, RICO, and Deterrence*, 5 Conn. Ins. L.J. 751, 752 (1998) ("[H]ealthcare insurance contracts often stipulate that the insurer will pay a certain percentage of costs, e.g., 80%, leaving the policyholder with a co-payment of the remaining 20%.").

[275] *See, e.g.*, Blake v. UnionMutual Stock Life Ins. Co., No. 87-0543-CIV, 1989 U.S. Dist. LEXIS 16331 (S.D. Fla. March 10, 1989); Blake v. UnionMutual Stock Life Ins. Co., 906 F.2d 1525, 1525 (11th Cir. 1990); *see also* Tovino, *Neuroscience and Health Law, supra* note 215, at 478–83 (discussing a number of cases in which insured parties who were denied comprehensive insurance coverage responded by suing their insurers, arguing that their conditions were physical rather than mental in nature and thus covered under the more comprehensive set of insurance benefits).

[276] *See, e.g.*, *Blake*, 1989 U.S. Dist. LEXIS at 16331 at *12; *Blake*, 906 F.2d at 1525.

[277] *See* Tovino, *Neuroscience and Health Law, supra* note 215, at 478–83.

and sometimes they did not, but the result usually depended on whether the insured could prove that she had a physical illness through physical evidence.[278]

Frustrated with these piecemeal lawsuits, many mental health patient advocacy organizations began in the early 1990s to lobby Congress and state legislatures for health insurance parity, reasoning that there was no biological justification for the unequal insurance coverage of mental and physical conditions by health insurance plans.[279] By the mid-1990s, proponents of mental health parity had achieved some success at the federal level through the Mental Health Parity Act of 1996 ("MHPA'96"), which required covered group health plans to provide parity with respect to annual and lifetime aggregate spending caps imposed on medical and surgical benefits and mental health benefits within the plan.[280] Because MHPA'96 did not require parity with respect to financial requirements, cost-sharing requirements, and treatment limitations, mental health parity proponents continued to lobby Congress for more complete parity measures over the next twelve years.[281] On October 3, 2008, President George W. Bush responded by signing into law the Emergency Economic Stabilization Act of 2008, one subtitle of which contained the Paul Wellstone and Pete Domenici Mental Health Parity and Addiction Equity Act of 2008 ("MHPA'08").[282] Very generally, MHPA'08 requires covered group health plans that provide both medical and surgical benefits and mental health or substance use disorder benefits to ensure that (1) the financial requirements, such as deductibles and copayments, applicable to mental health and substance use disorder benefits are no more restrictive than the predominant financial requirements applicable to substantially all medical and surgical benefits covered by the plan; (2) no separate cost sharing requirements that are applicable only with respect to mental health or substance use disorder benefits exist; (3) the treatment limitations applicable to such mental health and substance use disorder benefits are no more restrictive than the predominant treatment limitations applicable to substantially all medical and surgical benefits covered by the plan; and (4) no separate treatment limitations that are applicable only with respect to mental health or substance use disorder benefits exist.[283] MHPA'08 applies to covered plans beginning in the first plan coverage year that is one year after the

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[278] *Id.*

[279] Mary Crosby, *Political Lobbying for Child and Adolescent Psychiatry*, 11 CHILD AND ADOLESCENT PSYCHIATRIC CLINICS OF NORTH AMERICA 145, 153–54 (2002) (discussing the push for parity in the 1990s).

[280] Mental Health Parity Act of 1996, Pub. L. No. 104-204, 110 Stat. 2944 (codified as amended at 29 U.S.C. § 1185a (2006)).

[281] *See, e.g.*, Alaska Psychological Association, 2007–2008 Legislative Agenda and Report, at 2, *available at* http://www.ak-pa.org/legislation/2007-2008_legislative_report.pdf (noting that the Alaska Psychological Association's goals for the 2007–2008 Congressional Session include "continu[ing] to lobby for mental health parity . . . .").

[282] Emergency Economic Stabilization Act of 2008, Pub. L. No. 110-343, Subtitle B, §§ 511–12, 122 Stat 3765, 3881 (2008).

[283] *Id.* § 512(a) (amending 29 U.S.C. § 1185a(a) (2006)).

date of enactment. For most covered plans, the effective date of MHPA'08 will be January 1, 2010.[284] As of this writing, at least one health care reform bill, H.R. 3962, would extend the application of certain federal mental health parity provisions beyond the group health plan market to insurance sold on the individual market.[285] The House passed H.R. 3962 on November 7, 2009, although the bill has not yet been considered by the Senate.

Given current mental health parity law, the question becomes how future judicial opinions interpreting federal mental health parity law as well as analogous state parity laws may provide new legal understandings of postpartum illness. The answer lies in the fact that neither MHPA'96 nor MHPA'08 defines the phrase "mental health benefits" other than to refer to "benefits with respect to services for mental health conditions, as defined under the terms of the plan and in accordance with applicable Federal and State law."[286] Because other federal laws do not direct a definition of the phrase "mental health benefits" as used in MHPA'96 and MHPA'08, the application of federal mental health parity law to individuals with particular mental health conditions will depend on the terms of the plan as regulated by state law. Insureds who reside in states that narrowly define these terms may not receive as much, or the same, protection as insureds who reside in states that broadly define these terms.

For example, some state laws define "mental health benefits" in terms of conditions listed in the latest editions of the American Psychiatric Association's ("APA's") Diagnostic and Statistical Manual ("DSM")[287] and/or the World Health Organization's International Classification of Diseases ("ICD"),[288] both of which identify and classify mental disorders. Some states do this even though the current edition of the DSM, the DSM-IV-TR, states in its Introduction that a mental condition's inclusion in the manual should not imply that the condition meets legal criteria for what constitutes a mental disease, disorder, or disability, and that there is an imperfect fit between the law on the one hand and disease classification for clinical diagnostic purposes on the other.[289] Arkansas is an example of such a jurisdiction.

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[284] *Id.* § 512(e)(1) (providing that the law will take effect one year after enactment). As most health insurance plans follow the calendar year, this means the law will take actual effect on January 1, 2010.

[285] Affordable Health Care for Americans Act, H.R. 3962, §214(b), 111th Cong., 1st Sess. (2009).

[286] *See id.* §§ 511–12; Mental Health Parity Act of 1996, Pub. L. No. 104-204, 110 Stat. 2944.

[287] DSM-IV-TR, *supra* note 25.

[288] INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES AND RELATED HEALTH PROBLEMS (World Health Organization 2d ed., 10th Rev., 2007).

[289] DSM-IV-TR, *supra* note 25, at xxxii-xxxiii.

> When the DSM-IV categories, criteria, and textual descriptions are employed for forensic purposes, there are significant risks that diagnostic information will be misused or misunderstood. These dangers arise because of the imperfect fit between the questions of ultimate concern to the law and the information contained in a clinical diagnosis. In most situations, the clinical diagnosis of a DSM-IV

The Arkansas Mental Health Parity Act ("ArkMHPA"),[290] the intent of which is to make insurance coverage for mental illnesses as available as, and at parity with, insurance coverage for other medical illnesses,[291] defines the phrase "mental illness" as an illness or disorder listed in the DSM or the ICD.[292] ArkMHPA then requires health benefit plans to provide benefits for the diagnosis and treatment of mental illnesses under the same terms and conditions as provided for covered benefits offered for the treatment of other medical illnesses or conditions.[293] In Arkansas, then, a woman who requests treatment for a postpartum illness can benefit from federal and Arkansas insurance parity requirements if her condition meets the criteria for a mental disorder listed in the current version of the DSM or the ICD.

The DSM-IV-TR contains a category of mental disorders called "Mood Disorders," which includes the "Depressive Disorders" (including Major Depressive Disorder, Dysthymic Disorder, and Depressive Disorder Not Otherwise Specified ("NOS")), the "Bipolar Disorders" (including Bipolar I Disorder, Bipolar II Disorder, Cyclothymic Disorder, Bipolar Disorder NOS, and Mood Disorder NOS),[294] as well as a category of mental disorders called "Schizophrenia and Other Psychotic Disorders," which includes Schizophrenia, Schizophreniform Disorder, Schizoaffective Disorder, Delusional Disorder, Brief Psychotic Disorder, Shared Psychotic Disorder, and Psychotic Disorder NOS.[295] Although the DSM-IV-TR does not contain a separate category or classification for postpartum illness, the DSM-IV-TR does allow a clinician to add a "Postpartum Onset" specifier to a Major Depressive Disorder, Bipolar II Disorder, or Brief Psychotic Disorder if onset is within four weeks after childbirth.[296] The DSM-IV-TR expressly states, however, that the symptoms of a postpartum-onset episode of a depressive, bipolar, or psychotic disorder do not differ from the symptoms of a non-postpartum-onset episode.[297] An Arkansas woman who requests treat-

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> mental disorder is not sufficient to establish the existence for legal purposes of a "mental disorder," "mental disability," "mental disease," or "mental defect" . . . .

Id.

[290] ARK. CODE ANN. §§ 23-99-501–23-99-511 (2004).

[291] *Id.* § 23-99-502 ("It is the intent of this state that insurance coverage for mental illnesses and the mental health treatment of those with developmental disorders shall be as available and at parity with that for other medical illnesses.").

[292] *Id.* § 23-99-503(6) (" 'Mental illnesses' and 'developmental disorders' mean those illnesses and disorders listed in the International Classification of Diseases Manual and the Diagnostic and Statistical Manual of Mental Disorders . . . .").

[293] ARK. CODE ANN. §§ 23-99-506(a) (LexisNexis Supp. 2009) ("A health benefit plan that provides benefits for the diagnosis and treatment of mental illnesses shall provide the benefits under the same terms and conditions as provided for covered benefits: (1) The duration or frequency of coverage; (2) The dollar amount of coverage; or (3) Financial requirements.").

[294] DSM-IV-TR, *supra* note 25, at 20–21.

[295] *Id.* at 19.

[296] *Id.* at 422–23.

[297] *Id.* at 422.

ment for a postpartum illness thus can benefit from federal and Arkansas parity requirements as long as her condition meets the criteria for a mental disorder, which usually will be one of the Mood Disorders or a Brief Psychotic Disorder, even if its onset occurred after the four-week postpartum period allowed by the DSM-IV-TR. As a result, I do not anticipate that future judicial opinions interpreting state mental health parity law in jurisdictions such as Arkansas will provide further legal discussion or understanding of postpartum illness beyond characterizing it as a depressive, bipolar, or psychotic illness because a finding of postpartum onset is not necessary to the application of mental health parity requirements.

In other jurisdictions, however, the result might be different. Some states do not reference the DSM or the ICD in their definitions of mental health benefits.[298] Instead, some states mandate parity between the benefits offered for physical and mental illnesses and define the mental illnesses that benefit from the parity requirement in terms of whether they are "biologically-based." New Jersey, for example, requires certain health benefit plans to provide benefits for biologically-based mental illnesses under the same terms and conditions as provided for other sicknesses.[299] New Jersey defines a "biologically-based mental illness" as a "mental or nervous condition that is caused by a biological disorder of the brain and results in a clinically significant or psychological syndrome or pattern that substantially limits the functioning of the person with the illness . . . ."[300] A New Jersey woman who is insured by a regulated health benefit plan and requests treatment for a postpartum illness thus can benefit from New Jersey's mental health parity provisions if she can prove that her postpartum condition is caused by a biological disorder of the brain and substantially limits her functioning.[301] I anticipate that in future litigation in which a health insurance company refuses to provide equal insurance coverage for a woman's postpartum illness treatments due to the illness not being biologically-based (along the same

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> The symptoms of the postpartum-onset Major Depressive, Manic, or Mixed Episode do not differ from the symptoms in nonpostpartum mood episodes. Symptoms that are common in postpartum-onset episodes, though not specific to postpartum onset, include fluctuations in mood, mood lability, and preoccupation with infant well-being, the intensity of which may range from overconcern to frank delusions.

Id.

[298] *See, e.g.*, CAL. INS. CODE § 10144.5(d)(1)–(9) (West 2005) (defining "severe mental illness" in terms of certain listed disorders).

[299] N.J. STAT. ANN. § 17B:27A-19.7 (2006) ("Every small employer health benefits plan that provides hospital or medical expense benefits . . . shall provide benefits for biologically-based mental illness under the same terms and conditions as provided for any other sickness under the health benefits plan.").

[300] *Id.* Section 17B:27A-19.7 also provides an illustrative, but not exhaustive, list of conditions that the New Jersey Legislature believes are biologically-based, including schizophrenia, schizoaffective disorder, major depressive disorder, bipolar disorder, paranoia, and other psychotic disorders, obsessive-compulsive disorder, panic disorder, and pervasive developmental disorder or autism. *Id.*

[301] *Id.*

lines as the previously examined *Blake v. UnionMutual Stock Life Insurance Company*, in which the defendant insurer argued that Pam Blake's postpartum depression was not a physical illness),[302] the plaintiff would rely on one or more of the studies presented in Part I of this Article or comparable testimony in an attempt to prove that her postpartum illness has identifiable hormonal, biochemical, neuroanatomical, genetic, or other physical markers and, therefore, is biologically-based. To the extent a court agrees (or disagrees) with the plaintiff's characterization of her postpartum illness as biologically-based, a new legal understanding of postpartum illness exists.

The Arkansas and New Jersey parity provisions discussed above provide two approaches to mental health parity. A third approach, similar to the second approach, is taken by states that expressly tie their definition of protected mental health conditions to those conditions that "current medical science affirms" is caused by an organic or physiological disorder. Nebraska, for example, defines a "serious mental illness" as "any mental health condition that current medical science affirms is caused by a biological disorder of the brain and that substantially limits the life activities of the person with the serious mental illness."[303] If a state-regulated health insurance plan provides coverage for "serious mental illnesses," Nebraska law requires the insurance plan to provide coverage for health care rendered by certain listed individuals as well as health care provided in certain listed facilities.[304] I anticipate that in future Nebraska litigation in which a defendant insurance company refuses to cover health care rendered by a listed individual or within a listed facility for a plaintiff's postpartum illness, the plaintiff may point to one or more of the studies discussed in Part I of this Article or comparable expert testimony in an attempt to prove that current medical science has affirmed that her postpartum illness is caused by a brain-based biological disorder. To the extent a court agrees (or disagrees) that current medical science has affirmed that the plaintiff's postpartum illness is caused by brain-based biological disorder, a new legal understanding of postpartum illness exists.

Assuming for the moment that a particular plaintiff's postpartum illness does substantially limit her functioning, the question becomes whether a court interpreting the New Jersey and Nebraska mental health parity provi-

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[302] Blake v. UnionMutual Stock Life Ins. Co., No. 87-0543-CIV, 1989 U.S. Dist. LEXIS 16331 (S.D. Fla. March 10, 1989); Blake v. UnionMutual Stock Life Ins. Co., 906 F.2d 1525, 1525 (11th Cir. 1990).

[303] NEB. REV. STAT. § 44-792(5)(b) (2004). Section 44-792(5)(b) also provides an illustrative, but not exhaustive, list of conditions that the Nebraska Legislature believes that current medical science affirms are caused by biological disorders of the brain including schizophrenia, schizoaffective disorder, delusional disorder, bipolar affective disorder, major depression, and obsessive compulsive disorder.

[304] *Id.* § 44-793(2). Notably, this section of the statute also contains a caveat providing that "[t]he issuer of a health insurance plan may require a health care provider under this subsection to enter into a contract as a condition of providing benefits." *Id.* The law is silent as to any limits on what the provisions of such a contract may contain.

sions should find that postpartum illness is a "biological disorder of the brain" or that "current medical science affirms [postpartum illness] is caused by a biological disorder of the brain," respectively.[305] Stedman's Medical Dictionary, a standard medical dictionary, defines "biological" as "derived or obtained from living organisms" and "biology" as "the science concerned with the phenomena of life and living organisms."[306] Attempts to define the word "disorder" in the context of mental illness have resulted in vigorous debates and significant controversy due in part to disagreement regarding the corresponding concepts of order and normality, the implications of a definition of "disorder" for health conditions that may be socioculturally bound, as well as the use of a fixed set of criteria for diagnosing mental disorders.[307] Notwithstanding, most standard medical dictionaries define "disorder" as "a disturbance of bodily function, structure, or both."[308]

I believe that applying these definitions to the current scientific understanding of postpartum illness could reasonably support the classification of postpartum illness as a biological disorder of the brain (i.e., a disturbance of brain function, structure, or both). In the context of neurotransmitters,[309] it is helpful to reconsider the 2008 study published by scientists at The University of Pittsburgh and Emory University using positron-emission tomography ("PET") to investigate brain serotonin-1A ("5HT1A") receptor binding and finding that postsynaptic 5HT1A receptor binding in the subjects with postpartum depression was reduced 20% to 28% relative to controls.[310] The scientists concluded that their findings demonstrated a "neurobiological deficit" in women with postpartum depression.[311] In terms of the medical dictionary definition of "disorder" defined above, these findings certainly could be classified as a disturbance in brain function.

Also reconsider the 1998 study published by the group of German scientists who used computed tomography to quantify the CSF spaces in 14 women, 12 of whom had cycloid psychoses with postpartum onset, and finding that certain CSF spaces were significantly larger in patients with postpartum psychosis when compared to age-matched female patients with non-

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[305] *See supra* text accompanying notes 300–303.

[306] STEDMAN'S MEDICAL DICTIONARY 220 (28th ed. 2006).

[307] *See, e.g.*, ALLAN V. HORWITZ & JEROME C. WAKEFIELD, THE LOSS OF SADNESS: HOW PSYCHIATRY TRANSFORMED NORMAL SORROW INTO DEPRESSIVE DISORDER 110–11 (2007) (discussing the controversy surrounding the meaning of the term "disorder" in the context of mental health and mental illness); DSM-IV-TR, *supra* note 25, at xxx ("[A]lthough this manual provides a classification of mental disorders, it must be admitted that no definition adequately specifies precise boundaries for the concept of 'mental disorder.' The concept of mental disorder, like many other concepts in medicine and science, lacks a consistent operational definition that covers all situations.").

[308] STEDMAN'S MEDICAL DICTIONARY at 567, *supra* note 306.

[309] I defined "neurotransmitters" in Part I as endogenous chemicals in the brain that are responsible for relaying, amplifying, and modulating signals between neurons and other cells. *See supra* text accompanying note 76.

[310] *See* Moses-Kolko, *supra* note 80, at 685.

[311] *Id.*

postpartum cycloid psychoses or bipolar affective disorders outside the puerperium.[312] The scientists concluded that their findings "could reflect an unspecific neurostructural vulnerability marker in some patients with postpartum psychosis,"[313] which could reasonably be classified as a disturbance of bodily structure.

In summary, many of the scientific studies presented in Part I of this Article could reasonably be classified as suggesting or finding disturbances in brain structure or function of women with postpartum illness and, thus, suggesting or finding a biological disorder of the brain for purposes of applying mental health parity law in states such as New Jersey and Nebraska.[314] As such, advances in the scientific understanding of postpartum illness may impact the result of future insurance litigation in favor of some women with postpartum illness.

B. Implications for Disability Discrimination Law

In addition to judicial interpretations of mental health parity laws, future legal understandings of postpartum illness may also be found in judicial interpretation of disability discrimination laws, including the Americans with Disabilities Act of 1990 ("ADA")[315] as amended by the ADA Amendments Act of 2008 ("ADAAA").[316] A brief summary of the ADA as amended by the ADAAA is necessary before proceeding.

On July 26, 1990, President George H.W. Bush signed the ADA into law to provide a clear and comprehensive national mandate for the elimination of discrimination against individuals with disabilities.[317] The original ADA provided a three-prong definition of disability (i) a physical or mental impairment that substantially limits one or more major life activities of such individual; (ii) a record of such an impairment; or (iii) being regarded as having such an impairment.[318] Implementing regulations defined "physical or mental impairment" as:

> (1) Any physiological disorder, or condition, cosmetic disfigurement, or anatomical loss affecting one or more of the following body systems: neurological, musculoskeletal, special sense organs, respiratory (including speech organs), cardiovascular, reproductive, digestive, genito-urinary, hemic and lymphatic, skin, and endocrine; or (2) Any mental or psychological disorder, such as

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[312] *See* Lanczik, *supra* note 88, at 45.
[313] *Id.*
[314] *See supra* text accompanying notes 299–303.
[315] Americans with Disabilities Act of 1990, Pub. L. No. 101-336, 104 Stat. 327 (codified as amended at 42 U.S.C. §§ 12101–12206 (2006)).
[316] ADA Amendments Act of 2008, Pub. L. No. 110-325, 122 Stat. 3533 (2008).
[317] Americans with Disabilities Act, Pub. L. No. 101-336, 104 Stat. 327 (1990).
[318] *Id.* § 3(2)(A)–(C) (pre-ADAAA definition of disability).

> mental retardation, organic brain syndrome, emotional or mental illness, and specific learning disabilities.[319]

Traditionally, ADA plaintiffs who claim disability status based on depressive, anxiety, or psychotic disorders have relied on the second clause in the definition of impairment (the "mental or psychological disorder" clause), and not the first clause (the "physiological disorder" clause).[320] The scientific studies presented in Part I of this Article, especially those studies suggesting or finding neuroanatomical vulnerabilities[321] and neurofunctional deficits[322] in women with postpartum depression and psychosis, may provide some support for a future ADA plaintiff with postpartum illness to argue that she has a physiological condition that affects her neurological and endocrine systems and, therefore, that she has a "physiological disorder" within the meaning of the first clause of the definition. However, I do not anticipate that future litigants or courts will spend significant time arguing or resolving the issue whether postpartum illness is a physical versus mental impairment under the ADA because no distinction in legal result exists. Stated another way, an ADA plaintiff who has either a "physiological disorder" or a "mental or psychological disorder" may attempt to qualify as a protected individual with a disability under the ADA.

The original ADA made clear, however, that even if an ADA plaintiff has a physical or mental impairment, the impairment also must substantially limit one or more major life activities of the individual in order for the individual to be a protected individual with a disability for purposes of the ADA.[323] Although regulations implementing the original ADA defined the "major life activities" that must be substantially limited as "functions such as caring for oneself, performing manual tasks, walking, seeing, hearing, speaking, breathing, learning, and working,"[324] the interpretation of the phrase "substantially limit" was left to the courts.

Nine years after the original ADA's enactment, the United States Supreme Court in *Sutton v. United Air Lines* held that the phrase "substantially limits" requires that an individual be presently—not potentially or hypothetically—substantially limited in a major life activity.[325] According to *Sutton*,

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[319] 29 C.F.R. § 1630.2(h)(1)–(2) (2009).

[320] *See, e.g.*, Schneiker v. Fortis Ins. Co., 200 F.3d. 1055, 1061 (7th Cir. 2000) (analyzing the ADA plaintiff's major depression under the "mental or psychological disorder" language); Snead v. Metro. Prop. & Cas. Ins. Co., 237 F.3d 1080, 1088 & n.8 (9th Cir. 2001) (analyzing the plaintiff's depression under the "mental or psychological disorder" language and noting that, "[a]t least four other circuits agree that depression can constitute a mental impairment under the ADA").

[321] *See, e.g.*, Lanczik, *supra* note 88, at 45.

[322] *See, e.g.*, Moses-Kolko, *supra* note 80, at 685; Maurer-Spurej, *supra* note 78, at 23–25, 27.

[323] Americans with Disabilities Act, Pub. L. No. 101-336, § 3(2)(A), 104 Stat. 327 (1990).

[324] *See* 29 C.F.R. §1630.2(i) (2009).

[325] Sutton v. United Air Lines, Inc., 527 U.S. 471, 482 (1999).

an individual whose physical or mental impairment is corrected by medication or other mitigating measures does not have an impairment that presently substantially limits a major life activity and, therefore, is not a protected individual with a disability.[326] Twelve years after the original ADA's enactment, the Supreme Court in *Toyota Motor Manufacturing, Kentucky, Inc. v. Williams* further narrowed the broad scope of protection intended to be afforded by the ADA and held that an individual's impairment must be permanent or long-term and must prevent or severely restrict the individual from doing activities that are of central importance to most people's daily lives.[327]

Following the publication of *Sutton* and *Toyota* but before the enactment of the new ADAAA, the Supreme Court and lower courts across the country found that a number of individuals with a wide range of physical and mental impairments, including depressive and anxiety disorders, were not protected individuals with disabilities[328] in part because their medications, psychotherapy, and other treatments constituted mitigating measures.[329] In cases specifically involving individuals with postpartum depression, the courts tended to deny disability status on the ground that the plaintiff's postpartum depression imposed only a short-term or temporary restriction on a woman's major life activities.[330]

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[326] *Id.* at 482.

> Looking at the Act as a whole, it is apparent that if a person is taking measures to correct for, or mitigate, a physical or mental impairment, the effects of those measures—both positive and negative—must be taken into account when judging whether that person is "substantially limited" in a major life activity and thus "disabled" under the Act.

Id.

[327] Toyota Motor Mfr. Kentucky, Inc. v. Williams, 534 U.S. 184, 185 (2002).

[328] ADA Amendments Act of 2008, Pub. L. No. 110-325, § 2(a), 122 Stat. 3533 (2008) (explaining that following the publication of the Sutton and Toyota cases, "lower courts have incorrectly found in individual cases that people with a range of substantially limiting impairments are not people with disabilities . . . .").

[329] *See e.g.*, Albertson's, Inc. v. Kirkingburg, 527 U.S. 555, 565–66 (1999) (declaring that mitigating measures encompass not only artificial aids, such as medications and devices, but also measures undertaken, whether consciously or not, with the body's own systems, including subconscious mechanisms for compensating and coping with visual impairments); Murphy v. United Parcel Service, Inc., 527 U.S. 516, 521 (1999) (noting that the determination of whether petitioner's impairment substantially limited one or more major life activities was properly made with reference to the mitigating factor of blood pressure medication); Orr v. Wal-Mart Stores, Inc., 297 F.3d 720, 724 (8th Cir. 2002) (dismissing the claims of a pharmacist with diabetes who controlled his condition with insulin injections and a controlled diet); Chenoweth v. Hillsborough Co., 250 F.3d 1328, 1330 (11th Cir. 2001), *cert denied*, 534 U.S. 1131 (2002) (dismissing the claims of a nurse with focal onset epilepsy controlled by medication); Boerst v. Gen. Mills Operations, No. 01-1483, 2002 WL 59637 at *408 (6th Cir. Jan. 15, 2002) ("[The plaintiff's] own testimony shows that he suffered no substantial limitation on his ability to work when Zoloft's mitigating effects are taken into account."); Nordwall v. Sears Roebuck & Co., No. 01-1691, 2002 WL 31027956 (7th Cir. Sept. 6, 2002) (dismissing the claims of an administrative assistant with diabetes who controlled her condition to some degree by daily blood sugar tests and daily injections of insulin).

[330] *See infra* text accompanying notes 331–340.

In the Sixth Circuit case of *Novak v. MetroHealth Medical Center*, for example, an employee sought leave under the federal Family and Medical Leave Act ("FMLA") to care for her 18-year-old daughter who allegedly had postpartum depression.[331] The FMLA authorizes leave for employees to care for a child 18 years of age or older if the child is suffering from a serious health condition and is incapable of self-care because of a "physical or mental disability."[332] According to regulations adopted by the Department of Labor interpreting the FMLA, the phrase "physical or mental disability" as used in the FMLA means a "physical or mental impairment that substantially limits one or more of the major life activities of an individual," as defined in the ADA.[333] Stated another way, an employee may take FMLA leave to care for an adult child only if that child has a disability under the ADA.[334] The Sixth Circuit thus had to determine whether the daughter's claimed postpartum depression constituted a disability under the ADA in order to resolve the underlying FMLA claim.

The Sixth Circuit provided three overlapping reasons in support of its holding that the daughter's postpartum depression did not constitute a disability under the ADA.[335] First, the mother did not provide sufficient evidence (other than her daughter's nonspecific, nonexpert testimony that she could not "follow the doctor's orders without some help" and that she was afraid she might "freak out and not know how to deal with a newborn") that her daughter's postpartum depression was severe.[336] Second, evidence from a physician's brief certification form and the daughter's own testimony showed that the daughter's postpartum depression only lasted a week or two, and under *Toyota*, short-term restrictions on major life activities do not constitute disabilities.[337] Third, the mother failed to provide any evidence showing that the daughter's postpartum depression "inflicted any permanent or long-term impact on her health" or caused the daughter to "endure[ ] any long-term adverse effects."[338]

In a similar case, *Shalbert v. Marcincin*, the Eastern District of Pennsylvania also was asked to determine whether the plaintiff's postpartum depression constituted a disability under the ADA.[339] Like the *Novak* court, the *Shalbert* court found that the plaintiff (who began feeling better two months after she was prescribed the drug Paxil®) failed to produce evidence show-

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[331] 503 F.3d 572, 575–76 (6th Cir. 2007).

[332] *Id.* at 581 (citing 29 U.S.C. § 2612(a)(1)(C) (2006) and 29 U.S.C. § 2611(12)(B) (2006)).

[333] *Id.* (citing 29 C.F.R. § 825.113(c)(2)).

[334] *Id.*

[335] *Id.* at 582.

[336] *Id.*

[337] *Id.*

[338] *Id.*

[339] Shalbert v. Marcincin, No. 04-5116, 2005 U.S. Dist. LEXIS 16564, at *17 (E.D. Pa. Aug. 9, 2005) (finding that the plaintiff failed to produce evidence showing that her postpartum depression was long-lasting or permanent).

ing that her postpartum depression was long-lasting or permanent. The *Shalbert* court concluded that, "temporary, non-chronic impairment of short duration is not a disability covered by the ADA . . . ."[340]

To remedy the narrow application of the ADA in these and other cases, President George W. Bush signed the ADAAA into law on September 25, 2008, with the stated goals of (1) restoring the original intent and scope of the ADA by reinstating a broad scope of protection; (2) overturning the Supreme Court's ruling in *Sutton* and related cases that disabilities should be determined with reference to the ameliorative effects of mitigating measures; (3) rejecting the Supreme Court's holding in *Toyota* that the ADA requires that an impairment severely restrict major life activities; and (4) expressing Congress' intent that the focus of ADA compliance and litigation should be on whether covered entities have complied with their ADA obligations.[341]

To achieve these ends, the new ADAAA retains the ADA's basic three-prong definition of disability (that is, with respect to an individual (i) a physical or mental impairment that substantially limits one or more major life activities; (ii) a record of such impairment; or (iii) being regarded as having such an impairment),[342] but clarifies the interpretation of several phrases and several clauses within this definition. First, the ADAAA expressly rejects the *Sutton* and *Toyota* courts' interpretation of the phrase "substantially limits"[343] and clarifies that the phrase is intended to be interpreted broadly and that an impairment that substantially limits one major life activity need not limit other major life activities in order to be considered a disability.[344] Second, the ADAAA clarifies that an impairment that is episodic or in remission remains a disability so long as it substantially limits a major life activity when active.[345] Third, the ADAAA clarifies that the determination of whether an impairment substantially limits a major life activity should be made without regard to the ameliorative effects of mitigating measures such as medication or learned behavioral or adaptive neurological modifications.[346]

Fourth, and perhaps most importantly, the ADAAA provides a new statutory definition of "major life activity," which is broader than the regulatory definition established by the EEOC in its regulations implementing the original ADA.[347] Under the EEOC regulations implementing the original ADA,

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[340] *Id.* at *17.

[341] ADA Amendments Act of 2008, Pub. L. No. 110-325, § 2(b), 122 Stat. 3533 (2008).

[342] *Id.* § 4(a) (amending 42 U.S.C. § 12102 (2006)).

[343] *Id.* § 2(b)(3)–(4).

[344] *Id.* § 4 (amending 42 U.S.C. § 12102 (2006)).

[345] *Id.*

[346] *Id.*

[347] *Id.* Although the original ADA did not define the phrase "major life activity," regulations promulgated by the EEOC did. *See also* Ray v. Glidden Co., 85 F.3d 227, 229 (5th Cir. 1996) ("The ADA does not define 'substantially limits' and 'major life activities.' But, regulations promulgated by the EEOC under the ADA define both.").

"major life activities" were defined as "functions such as caring for oneself, performing manual tasks, walking, seeing, hearing, speaking, breathing, learning, and working."[348] The ADAAA not only contains an expanded list of "major life activities," including "caring for oneself, performing manual tasks, seeing, hearing, eating, sleeping, walking, standing, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, communicating, and working," but also identifies certain "major bodily functions" that are to be included within the definition of "major life activities," including "the operation of a major bodily function, including but not limited to, functions of the immune system, normal cell growth, digestive, bowel, bladder, neurological, brain, respiratory, circulatory, endocrine, and reproductive functions."[349] On September 23, 2009, the Equal Employment Opportunity Commission issued proposed regulations implementing the ADAAA in the context of employment.[350] If adopted in final form, these regulations would clarify that certain impairments, including major depression, bipolar disorder, and schizophrenia, will consistently meet the definition of disability due to their substantial limitation of the major life activities of brain function, thinking, concentrating, interacting with others, sleeping, or caring for oneself.[351]

Although ADA plaintiffs generally were not successful in arguing that postpartum depression constituted a protected disability under the original ADA,[352] I anticipate that future plaintiffs with postpartum illness who are operating under the authority of the ADAAA may attempt to present hormonal, neurochemical, neuroanatomical, or genetic research or evidence to argue that they have substantially impaired neurological and/or endocrine functions and, therefore, that their postpartum illnesses substantially limit a major bodily function. And, if the EEOC's proposed regulations are adopted in final form, the regulations would almost automatically qualify individuals with major postpartum depression or postpartum psychosis as individuals with disabilities. What should not matter, according to the ADAAA and the proposed regulations, is evidence showing that a drug has reduced the woman's symptoms of postpartum depression or psychosis, or evidence showing that the mother was not permanently depressed or psychotic.

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[348] *See* 29 C.F.R. § 1630.2(i) (2009).

[349] ADA Amendments Act of 2008, Pub. L. No. 110-325, § 4, 122 Stat. 3533 (amending 42 U.S.C. § 12102 (2006)).

[350] Regulations To Implement the Equal Employment Provisions of the Americans With Disabilities Act, as Amended, 74 Fed. Reg. 48431 (Sept. 22, 2009), *available at* http://edocket.access.gpo.gov/2009/pdf/E9-22840.pdf.

[351] *Id.* at 48441 (proposing new 29 C.F.R. 1630.2(j)(5)(H) (providing examples of impairments that will consistently meet the definition of disability)).

[352] *See, e.g.*, Novak v. Metrohealth, 503 F.3d 572, 582 (6th Cir. 2007); Shalbert v. Marcincin, No. 04-5116, 2005 U.S. Dist. LEXIS 16564, at *17 (E.D. Pa., Aug. 9, 2005).

C. A Limited, Legal Merger of Physical and Mental Illness

Advances in the scientific understanding of postpartum illness combined with recent developments in mental health parity law and disability discrimination law thus may impact future insurance and disability discrimination litigation in favor of some women with postpartum illness. Mental health parity and disability discrimination law also can serve as platform from which I can question the appropriateness of health law frameworks that continue to distinguish physical and mental illness.

Some legal distinctions between physical and mental illness have no legal consequences. Consider the ADA's definition of "physical or mental impairment," which contains one clause describing the types of "physiological disorders" that may qualify as impairments and a second, separate clause that describes the types of "mental or psychological disorders" that also may qualify as impairments.[353] I suggested in Part III(B) that the current scientific literature could support the classification of postpartum illness as either a "mental or psychological disorder" or a "physiological disorder" of the neurological or endocrine systems.[354] Regardless of how a court decides to categorize postpartum illness, however, a woman with postpartum illness still may attempt to qualify as a protected individual with a disability. Stated another way, even if a court finds that a woman's postpartum illness is a "mental or psychological disorder," the woman still may be a proper ADA plaintiff if she can show that her postpartum illness substantially limits a major life activity.

Although some commentators have suggested that the existence of mental health parity law is evidence that lingering legal distinctions between physical and mental illness have no legal consequences, a careful review of mental health parity law reveals several important legal consequences. At the federal level, neither MHPA'96 nor MHPA'08 regulates the health benefit packages of small employers (those with 50 or fewer employees) or insurance offered in the individual market.[355] Individuals who have employer-sponsored group health plan coverage but work at small employers as well as individuals who purchase health insurance in the individual market may continue to be subject to inferior mental health benefits. Second, federal mental health parity law only requires group health plans that provide both medical and surgical benefits and mental health or substance use disorder benefits to adhere to parity requirements.[356] Because federal law does not

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[353] 29 C.F.R. § 1630.2(h)(1)–(2) (2009).

[354] *See supra* text accompanying notes 321–322.

[355] *See* 29 U.S.C. § 1185a(c)(1) (2006) (small employer exemption); *id.* § 1185a(a)(1)–(2) (regulating only group health plans); Emergency Economic Stabilization Act of 2008, Pub. L. No. 110-343, Subtitle B, § 512(a)(3), 122 Stat 3765, 3881 (2008) (slightly revising but retaining the small employer exemption); *id.* § 512(a)(1)–(2) (regulating only group health plans).

[356] *See* 29 U.S.C. § 1185a(a)(1)–(2) (2006) (regulating only a "group health plan . . . that provides both medical and surgical benefits and mental health benefits"); Emergency

require covered group health plans to actually offer mental health or substance use disorder benefits, individuals with mental illness who belong to a group health plan that does not offer mental health or substance use disorder benefits will not have access to such benefits unless they purchase them separately on the individual market, which federal parity law does not regulate. Although some states have responded to these loopholes by enacting state parity laws that apply to a broader range of insurance plans and/or that require the offering of mental health benefits,[357] other states do no more than federal law; that is, other states only require large group health plans to implement parity if they offer mental health benefits.[358] Even in those states that have parity laws that apply to a broader range of insurance plans or that require the offering of mental health benefits, the parity provisions may only apply to certain classes of mental illness, such as "biologically-based mental illness"[359] or "serious mental illness."[360] In summary, legal distinctions between physical and mental illness and among the different types of mental illness continue to have significant legal consequences in the context of health insurance.

The two-part question becomes whether (1) continuing legal distinctions between and among physical and mental illness are valid given our current scientific literature; and (2) whether varying legal consequences based on such distinctions, such as equal or comprehensive insurance coverage versus no insurance coverage, are normatively appropriate. I believe that as the scientific understanding of illness (as well as the efficacy of a range of treatments) continues to progress, legal distinctions between and

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Economic Stabilization Act of 2008, at § 512(a)(1) (regulating only a "group health plan . . . that provides both medical and surgical benefits and mental health or substance use disorder benefits").

[357] *See, e.g.*, National Conference of State Legislators, State Laws Mandating or Regulating Mental Health Benefits, http://www.ncsl.org/IssuesResearch/Health/StateLaws MandatingorRegulatingMentalHealthB/tabid/14352/Default.aspx (last visited Nov. 15, 2009) (online resource categorizing state mental health parity laws by whether they require (1) the offering of health benefits for all mental illnesses; (2) the offering of health benefits for some mental illnesses, such as biologically-based mental illnesses; or (3) parity between physical health benefits and mental health benefits if the regulated plan offers mental health benefits).

[358] *See, e.g.*, ARIZ. REV. STAT. ANN. § 20-2322(A) (2002) (requiring "any health benefits plan . . . that provides services or health benefits that include mental health services or mental health benefits . . ." to comply with the State's parity requirements).

[359] *See, e.g.*, IOWA CODE § 514C.22.3 (2007) (defining "biologically-based mental illness" as "the following psychiatric illnesses: Schizophrenia, Bipolar disorders, Major depressive disorders, Schizo-affective disorders, Obsessive-compulsive disorders, Pervasive developmental disorders, [and] Autistic disorders . . ."); and *id.* § 514C.22.1 (requiring certain health plans to provide coverage benefits for treatment of biologically based mental illness if certain criteria are satisfied).

[360] *See, e.g.*, DEL. CODE ANN. tit. 18 § 3343(a)(3) (1999 & LexisNexis Supp. 2008) (defining "serious mental illness" as "any of the following biologically based mental illnesses: schizophrenia, bipolar disorder, obsessive-compulsive disorder, major depressive disorder, panic disorder, anorexia nervosa, bulimia nervosa, schizo affective disorder and delusional disorder . . ."); and *id.* § 3343(b) (requiring regulated health insurance carriers to provide coverage for such serious mental illnesses).

among physical and mental illness are becoming less valid and, as a result, varying legal consequences less appropriate.

Stated another way, regardless of whether the comparative focus is illness etiology, effect of illness, treatment modality, or treatment provider, few distinctions remain between illnesses traditionally classified as "mental" and "physical." Let us test these four methods of comparing a range of health conditions, some of which have been traditionally classified as "physical" (including epilepsy, Parkinson's disease, and brain cancer), and some of which have been traditionally classified as "mental" (including major depression, schizophrenia, obsessive-compulsive disorder, and eating disorders). The first method of comparison is illness etiology, which is the primary focus of state mental health parity law and federal disability law and one area of focus of courts that have interpreted health insurance policy provisions.[361] The causes of many health conditions, like postpartum illness, are incompletely understood at this time, although scientists currently believe that most illnesses, including both those traditionally classified as "physical" and "mental," have multifactorial etiologies that may include genetic, biological, and environmental factors. For example, the etiology of epilepsy is complex and not completely understood in all cases. Depending on the person, epilepsy may be caused by a head injury, cerebrovascular disease, a prenatal injury, heredity, an imbalance in neurotransmitters, or a combination of these and other unidentified physiological or environmental causes.[362] Likewise, the etiology of major depression also is complex and incompletely understood at this time. Scientists currently believe that major depression has genetic, biological, and psychosocial factors.[363] Notwithstanding considerable scientific investigation, the etiology of Parkinson's disease remains unclear, and scientists continue to debate the role and contribution of genetic, biological, and environmental factors.[364] The etiology of schizophrenia also remains under investigation, although both biology and environmental stress are considered probable factors.[365] Genetics and the environment also are likely factors in the etiology of brain cancer, which

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[361] *See supra* Part II(B) (judicial interpretation of health insurance policy provisions) and Parts III(A) and (B) (mental health parity law and federal disability law).

[362] *See, e.g.*, World Health Organization, Epilepsy: Key Facts, *available at* http://www.who.int/mediacentre/factsheets/fs999/en/index.html (last visited Nov. 15, 2009).

[363] *See, e.g.*, Department of Health & Human Services, Mental Health: A Report of the Surgeon General 251 (Dec. 13, 1999) *available at* http://www.surgeongeneral.gov/library/mentalhealth/home.html [hereinafter Surgeon General Report].

[364] *See* Kathy Steece-Collier, Eleonora Maries & Jeffrey H. Kordower, Commentary, *Etiology of Parkinson's Disease: Genetics and Environment Revisited*, 99 PROCEEDINGS NAT'L ACADEMY SCIENCES 13972, 13972 (2002) ("Despite many years of focused research, the causes of [Parkinson's disease] remain to be elucidated . . . . The relative contributions of genetic versus environmental factors regarding the cause of [Parkinson's disease] have been hotly debated.").

[365] *See, e.g.*, Herbert Y. Meltzer & Ariel Y. Deutch, *Neurochemistry of Schizophrenia*, *in* BASIC NEUROCHEMISTRY, MOLECULAR, CELLULAR, AND MEDICAL ASPECTS 1053, 1065 (6th ed., George J. Siegel et al. eds., 1999).

likewise remains incompletely understood.[366] Obsessive-compulsive disorder also has likely, although not certain, biological and psychosocial factors.[367] In summary, many health conditions have complex, multifactorial etiologies that would preclude illness etiology from serving as a singular, distinguishing feature. More broadly, the "illness etiology" test fails to take into account current approaches to and models of science including the "unity of science," which deemphasizes traditional distinctions between and among the social sciences (including sociology and psychology) and the natural sciences (including biology, chemistry, and physics), and emphasizes the ability of scientists and others to understand phenomena in physics in terms of chemistry, chemistry in terms of biology, psychology in terms of biology, and so on.[368]

A second possible method of comparison requires an analysis of the effects, symptoms, or consequences of the illness. The application of this test, however, does not always lead to the most consistent or rational results. For example, an individual with an eating disorder who displays symptoms of dehydration and malnourishment may be classified as an individual with a physical illness whereas an individual who displays the high and low moods

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> [T]he etiology of schizophrenia may involve pathological processes which begin in utero or perinatally and continue to unfold until the brain approaches its adult anatomical state as a result of extensive neuronal loss and synaptic pruning during early and late adolescence. These neurodevelopmental abnormalities are proposed to lead to the activation of pathological neural circuits during adolescence or young adulthood, perhaps due to severe stress, leading to the emergence of positive or negative symptoms or both.

Id.

[366] *See, e.g.*, ClinicalTrials.gov, A Case Referent Study of Brain Tumors in Adults, Oct. 6, 2008, http://clinicaltrials.gov/ct2/show/NCT00339300 ("The etiology of brain cancer is largely unknown, although several chemicals and occupations have been associated with the disease."); Paul Kleihues, Adriano Aguzzi & Hiroko Ohgaki, *Genetic and Environmental Factors in the Etiology of Human Brain Tumors*, 82–83 TOXICOLOGY LETTERS 601, 601–05 (1995) (discussing the genetic and environmental factors in the etiology of human brain tumors).

[367] *See, e.g.*, Mark F. Eddy & Gordon S. Walbroehl, *Recognition and Treatment of Obsessive-Compulsive Disorder*, 57 AM. FAMILY PHYSICIAN 1623, 1624 (1998).

> The etiology of obsessive-compulsive disorder is uncertain, but it appears to include a combination of neurologic and psychologic factors. The dominant neurochemical theory of obsessive-compulsive disorder suggests that the neurotransmitter serotonin plays a central role in the development of the condition. Drugs that increase the availability of serotonin in the body are effective in ameliorating the symptoms of obsessive-compulsive disorder, while nonserotonergic medications have been found to have little or no effect.

Id.

[368] *See generally* EDWARD O. WILSON, CONSILIENCE: THE UNITY OF KNOWLEDGE (1998) (deemphasizing gaps between and among the natural and social sciences, as well as the arts, and encouraging scholars to bridge those gaps in future research); Martha Farah, Ph.D., Director, University of Pennsylvania Center for Neuroscience & Society, Opening Lecture and PowerPoint, Neuroscience Boot Camp, Philadelphia, Pennsylvania (Summer 2009) (providing an overview of the relationship between neuroscience and society as well as various approaches to understanding relationships between different scientific disciplines, including the "unity of science" concept).

associated with bipolar disorder may be classified as an individual with a mental illness.[369] By further example, an individual with severe depression who engages in self-cutting or who presents to an emergency department following a failed suicide attempt may be classified as an individual with a physical illness whereas an individual with severe depression who presents to an emergency department with suicidal ideations may be classified as an individual with a mental illness.[370]

A third possible method of comparison relates to method of treatment. According to some common law tests, patients who receive individual or group counseling or other forms of psychotherapy, or who are prescribed psychoactive drugs or electroconvulsive therapy, are considered individuals with mental illnesses,[371] whereas patients who have received other types of treatments are considered individuals with physical illnesses. The application of the "method of treatment" test also can lead to somewhat illogical results, especially in the context of drugs, therapies that rely on electricity, and surgery, all of which are designed to and do produce physiological changes in the brain. For example, an individual who has Parkinson's disease and is treated with dopamine-enhancing drugs[372] may be classified as an individual with a physical illness whereas an individual who has schizophrenia and is treated with psychoactive drugs[373] may be classified as an individual with a mental illness. By further example, a woman who has epilepsy and is treated with an implanted vagus nerve stimulator that delivers an electrical pulse to her brain[374] may be classified as an individual with a physical illness whereas an individual who has medication-resistant depression and is treated with electroconvulsive therapy that delivers electricity to his brain[375]

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[369] *See* Tovino, *supra* note 215, at 478–83 (referencing courts that have applied the "symptom" test to a range of illnesses).

[370] *Id.*

[371] *See, e.g.*, Blake v. UnionMutual Stock Life Ins. Co., No. 87-0543-CIV, 1989 U.S. Dist. LEXIS 16331, at *12 (S.D. Fla. Mar. 10, 1989) (noting that the plaintiff's postpartum depression was properly considered a mental illness because "[s]he was treated primarily by psychiatrists receiving well recognized psychiatric treatment, including individual psychotherapy, psychoactive drug therapy, electroconvulsive therapy and participation in group sessions.").

[372] *See, e.g.*, Murata, *supra* note 216, at S17–20 (discussing the use of Levodopa, a dopamine-enhancing drug, for the treatment of Parkinson's disease).

[373] *See, e.g.*, Rune A. Kroken, Erik Johnsen, Torleif Ruud, Tore Wentzel-Larsen & Hugo A. Jorgensen, *Treatment of Schizophrenia with Antipsychotics in Norwegian Emergency Wards, a Cross-Sectional National Study*, 9 BMC PSYCHIATRY 24, 24 (2009) (study evaluating the treatment of patients with schizophrenia with antipsychotics in the Scandinavian public health system).

[374] *See, e.g.*, S.M. Kabir, C. Rajaraman, C. Rittey, H.S. Zaki, A.A. Kemeny & J. McMullan, *Vagus Nerve Stimulation in Children with Intractable Epilepsy: Indications, Complications and Outcome*, 25 CHILD'S NERVOUS SYSTEM 1097, 1097–1100 (2009) (retrospectively analyzing the indication, complications and outcome of vagus nerve stimulation in intractable childhood epilepsy and concluding that vagus nerve stimulation is a relatively safe and potentially effective treatment for children with medically intractable epilepsy).

[375] *See, e.g.*, Paula Barros Antunes, Moacyr Alexandro Rosa, Paulo Silva Belmonte-de Abreu, Maria Inês Rodrigues Lobato & Marcelo P. Fleck, *Electroconvulsive Therapy*

may be classified as an individual with a mental illness. Finally, a woman who has brain cancer that is surgically removed[376] may be classified as an individual with a physical illness whereas a man who has obsessive-compulsive disorder and is surgically implanted with a deep brain stimulator that stimulates his ventral caudeate nucleus[377] may be classified as an individual with a mental illness. In summary, the "method of treatment" test fails to recognize that drugs, electricity-based therapies, and surgeries produce similar physiological changes in the brain when applied to individuals with illnesses traditionally classified as both "physical" and "mental." Even in the context of psychotherapy, which traditionally has been considered a treatment for mental illness, scientists are uncovering neurobiological findings that support prior, first-person, subjective reports of efficacy.[378]

A fourth possible method of comparison relates to treatment provider. According to some judicial opinions, individuals who are treated by psychiatrists or psychologists should be classified as individuals with mental illnesses, whereas individuals who are treated by other non-psychiatrist physicians should be classified as individuals with physical illnesses.[379] This test ignores the fact that many individuals with illnesses traditionally classified as "mental" receive treatment solely from a non-psychiatrist primary care physician or general practitioner and that the majority of psychoactive drugs are prescribed by non-psychiatrist physicians, including primary care physicians and general practitioners who do not have extensive training in psychiatry.[380] Application of the "treatment provider" test to two different

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in Major Depression: Current Aspects, 31 (Supp.1) REVISTA BRASILEIRA DE PSIQUIATRIA S26, S26–33 (May 2009) (study published in the *Brazilian Review of Psychiatry* designed to demonstrate the role of electroconvulsive therapy ("ECT") in the treatment of depression, finding that ECT remains a highly efficacious treatment in treatment-resistant depression and with improvements in technique has become safer and more useful for the both the acute phase and for the prevention of new depressive episodes).

[376] *See, e.g.*, A. Romano, G. D'Andrea, G. Minniti, L. Mastronardi, L. Ferrante, L. M. Fantozzi & A. Bozzao, *Pre-Surgical Planning and MR-Tractography Utility in Brain Tumour Resection*, 19 EUROPEAN RADIOLOGY 2798, 2798 (2009) (study finding that MR-tractography can provide neurosurgeons with new anatomical views that may impact surgical resection planning for brain neoplasms).

[377] *See, e.g.*, Bruno Aouizerate, Corinne Martin-Guehl, Emmanuel Cuny, Dominique Guehl, Helene Amieva, Abdelhamid Benazzouz, Colette Fabrigoule, Bernard Bioulac, Jean Tignol & Pierre Burbaud, *Deep Brain Stimulation for OCD and Major Depression*, 162 AM. J. PSYCHIATRY 2192, 2192 (2005) (stating that deep brain stimulation of the ventral caudate nucleus continues to be a promising strategy for the treatment of refractory cases of both OCD and major depression).

[378] *See, e.g.*, LOUIS J. COZOLINO, THE NEUROSCIENCE OF PSYCHOTHERAPY: BUILDING AND REBUILDING THE HUMAN BRAIN (2002) (arguing that many forms of psychotherapy are supported by neuroscientific findings).

[379] *See, e.g.*, Blake v. UnionMutual Stock Life Ins. Co., No. 87-0543-CIV, 1989 U.S. Dist. LEXIS 16331, at *12 (S.D. Fla. March 10, 1989) (noting that the plaintiff's postpartum depression was properly considered a mental illness because "she was treated primarily by psychiatrists receiving well recognized psychiatric treatment . . .").

[380] *See, e.g.*, RALPH REISNER, CHRISTOPHER SLOBOGIN & ARTI RAI, LAW AND THE MENTAL HEALTH SYSTEM: CIVIL AND CRIMINAL ASPECTS 110 (4th ed. West 2004) ("[T]he 'bulk of psychotropic medications are in fact' not being prescribed by psychiatrists, but

individuals with the same condition thus could lead to different or illogical results if one individual receives treatment from a psychiatrist and the second individual receives treatment from a non-psychiatrist physician for the same condition. In addition, the "treatment provider" test assumes that an individual with a primary diagnosis will only see one type of health care provider when, in reality, many ill individuals see multiple physicians with different areas of expertise. An estimated 25% of cancer patients meet clinical criteria for major depression, for example, and are encouraged to and do obtain treatment by psychiatrists and other mental health care providers affiliated with their institutional cancer care providers.[381] By further example, scientists continue to investigate rather strong associations between depression and osteoporosis, which suggests that individuals with depression also may need treatment from an endocrinologist, orthopedist, family practitioner, an expert in physical medicine and rehabilitation, or other physician who specializes in osteoporosis care.[382] In summary, application of the "treatment provider" test may not lead to the most consistent, rational results. The "treatment provider" test also fails to recognize the push within some medical and scientific circles to merge the disciplines of neurology and psychiatry.[383]

As the empirical study of the cost and efficacy of mental health care advances, legal consequences to individuals with illnesses traditionally classified as "mental" also become less appropriate. Old data cited by insurance companies and employers finding that treatments for mental versus physical

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instead 'are being prescribed by primary care physicians who have no extensive training in the treatment of mental disorders.'").

[381] *See, e.g.*, Karen Stuyck, *Psychotherapy Extends Cancer Care Beyond the Physical*, 53 ONCOLOG 1–2 (2008) *available at* http://www2.mdanderson.org/depts/oncolog/pdfs-issues/08/oncolog6-08.pdf (reporting that "[a]nywhere from 20% to 70% of cancer survivors experience some level of depression and/or anxiety, depending on other stressors in their life; and the U.S. National Cancer Institute estimates that 25% of cancer patients experience major depression" and referencing the psychiatric services available through The University of Texas M. D. Anderson Cancer Center).

[382] *See, e.g.*, Williams, *supra* note 142, at 16–25 (reviewing the relationship between depression and bone metabolism); National Osteoporosis Foundation, How to Find a Doctor, http://www.nof.org/patientinfo/finding_doctor.htm (last visited Nov. 15, 2009).

> There is no single type of healthcare provider or medical specialty that focuses on osteoporosis. Also, there is no certification program for health professionals who treat the disease. Over time, some healthcare providers in different medical specialties have gained the knowledge and expertise to diagnose and treat people with osteoporosis. These specialties include endocrinology, family practice, geriatrics, gynecology, internal medicine, orthopedics, physical medicine and rehabilitation, and rheumatology.

Id.

[383] *See, e.g.*, Mark Moran, *Psychiatry, Neurology Urged to Become One Discipline*, 43 PSYCHIATRIC NEWS, Nov. 21, 2008, at 1, 4 (quoting Dr. Stuart Yudofsky as stating that, "I believe psychiatry and neurology should be combined into one profession . . . ."). *But see* Thomas Szasz, *Letter to the Editor: Merger of Psychiatry, Neurology*, 44 PSYCHIATRIC NEWS, March 6, 2009, at 25, 25 (explaining that psychiatry and neurology formerly comprised a single discipline referred to as "mad-doctoring" or "neuropsychiatry," and arguing that the reestablishment of psychiatry as a medical specialty is redundant).

illnesses compared unfavorably in terms of cost[384] are being replaced with new data showing that mental health treatments are not more expensive and that the implementation of managed mental health care and mental health parity can result in lower total health care costs.[385] Old data suggesting that treatments for mental versus physical illness compared unfavorably in terms of efficacy[386] are being replaced with new data, including a recent report by the Surgeon General finding that mental illness is very treatable.[387] Old concerns regarding the ability of some individuals to malinger, or fake, mental illness,[388] are being replaced with new data showing that psychological tests can successfully detect malingering[389] and that urine, blood, and radiological diagnostic tests soon may provide a method of diagnosing the existence of, or a predisposition or vulnerability to, mental illness.[390]

Given advances in the scientific understanding of illness and treatment efficacy as well as the empirical study of the cost and efficacy of mental health care, I thus propose a limited, legal merger of physical and mental illness. I will first address the limitations on such merger. I do not currently propose the elimination of distinctions between types of individual or institutional health care providers or illnesses to the extent that these distinctions attempt to identify patient populations with particular needs, encourage the development of diagnostic and treatment services for such patients, or attempt to route patients with particular health conditions to the most appropriate care provider. For example, many states have enacted mental health

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[384] *See* SETHI, *supra* note 270, at 14; Beigel, *supra* note 270, at 668; Brown, *supra* note 270, at 53; Kaplan, *supra* note 268, at 328.

[385] *See, e.g.*, William Dodge, *Health Coverage Should Include Mental Illness*, S. F. CHRON., Apr. 22, 1999, at A27 (referencing several studies finding that the implementation of managed mental health care and mental health parity, as well as the emphasis on primary mental health care, can lower total health care costs).

[386] *See, e.g.*, Brown, *supra* note 270, at 53 ("Until as recently as 1990, the diagnosis and treatment of mental illnesses were relatively unsuccessful. Thus, to avoid the long-term expenses associated with mental illnesses, many [health insurance] policies have addressed the nature of the coverage provided for mental illness. Typically, they exclude or limit coverage, including the period for which benefits are payable.").

[387] *See, e.g.*, SURGEON GENERAL REPORT, *supra* note 363, at 64 ("Mental disorders are treatable, contrary to what many think.").

[388] *See, e.g.*, Pamela Kulbarsh, *Why Would Anyone Want to Fake Mental Illness?*, LAWYER.COM, June 16, 2009, http://www.lawofficer.com/news-and-articles/columns/Kulbarsh/malingering.html# (defining "malingering" as the "intentional production of false or exaggerated physical or psychological complaints with the goal of receiving a reward," noting that malingering has been estimated to occur in 7.5% to 33% of all disability claims, and stating that malingering depletes Social Security, disability, workers compensation, and insurance funds).

[389] *See, e.g.*, David J. Schretlen, *The Use of Psychological Tests to Identify Malingered Symptoms of Mental Disorder,* 8 CLINICAL PSYCHOL. REV. 451, 451 (1988) (noting the importance of detecting malingering in both the medical and legal contexts and finding that the majority of studies show that psychological tests can accurately detect malingering).

[390] *See supra* text accompanying notes 66–70 (discussing the potential of a hormone test to predict the onset of postpartum illness) and 90–95 (discussing the potential of neuroimaging to identify vulnerability to postpartum illness).

code provisions that permit counties, hospital districts, local agencies, and other entities to establish community mental health centers that provide diagnosis and treatment services for individuals with mental illness and substance use disorders and that encourage the referral of patients with certain diagnoses to these centers.[391] These code provisions are premised on findings showing that patients with illnesses traditionally classified as "mental" are underserved and have fewer treatment and placement options than patients with illnesses traditionally classified as "physical."[392] To the extent all patient populations become equally served and patients with particular needs are always routed to the most appropriate care providers, this proposal should be revisited.

I also do not currently propose the elimination of distinctions between types of individual or institutional health care providers or illnesses in health care regulation to the extent the purpose of the regulation is to protect patients with illnesses traditionally classified as "mental" due to findings of heightened abuse, neglect, and exploitation in this patient population, as well as the inability of such patients to have and exercise basic human and patients' rights. For example, many state legislatures have passed laws that give patients with illnesses traditionally classified as "mental" the same rights as non-patients, such as the right to vote, the right to sue and be sued, and the right to religious freedom.[393] Congress and many state legislatures also have passed laws that give patients with illnesses traditionally classified as "mental" the same rights as other patients, including the right to medical privacy and the right to be free from unnecessary restraint and seclusion.[394] To the extent patients with illnesses traditionally classified as "mental" become no more vulnerable than other patients and have and are able to exercise their basic human and patients' rights, this proposal should be revisited.

I also will not identify as a "first target" for revision legal distinctions between physical and mental illness, such as the ADA's distinction between "physiological disorders" and "mental or psychological disorders" in its definition of "physical or mental impairment," when such distinctions do not have legal consequences; however, these distinctions should be removed as time and resources permit to enable the language of the law to more closely resemble the current scientific understanding of illness.

I do, however, propose the immediate elimination of distinctions between types of individual or institutional health care providers or illnesses in health law to the extent the purpose of the distinction is to provide less legal

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[391] *See, e.g.*, TEX. HEALTH & SAFETY CODE § 534.001(a)–(b)(1) (Vernon 2003 & West Supp. 2009).

[392] *See, e.g.*, TEX. HEALTH & SAFETY CODE § 534.0015(b) (West Supp. 2009).

[393] *See, e.g.*, TEX. HEALTH & SAFETY CODE § 576.001(a) (Vernon 2003 & West Supp. 2009); *id.* at § 576.001(b) ("A person with mental illness in this state [of Texas] has the rights, benefits, responsibilities, and privileges guaranteed by the constitution and laws of the United States and this state.").

[394] *See, e.g.*, 42 U.S.C. § 10841 (2006) (establishing the "Restatement of Bill of Rights for Mental Health Patients").

protection or fewer benefits to individuals with illnesses traditionally classified as "mental." One method of implementing this proposal would be to repeal current federal mental health parity law and enact in its place a new provision that would prohibit all health insurers, public and private, from distinguishing physical and mental illness in their individual and group health insurance policies and plans. This proposal would eliminate the need for the concept of mental health parity and would remedy the contractual problem presented in the *Blake v. UnionMutual Stock Life Insurance Company* litigation discussed at length in Part II(B), in which the health insurance policy issued to plaintiff Pam Blake distinguished physical illness (defined as an "illness or disease . . . [including pregnancy] unless excluded elsewhere") from mental illness ("any mental, nervous or emotional diseases or disorders") and limited Blake's coverage of mental illness to only 30 days of inpatient care and $1,000 worth of outpatient treatments. This proposal also would eliminate the need for common law tests designed to distinguish physical and mental illness, including the "treatment provided to the patient test," the "symptoms of illness test," and the "origin of disease test."

To achieve the same result without repealing current mental health parity law, a second method would be to amend existing parity laws to require all insurers, public and private, that offer individual and group medical and surgical insurance benefits to also offer mental health and substance use disorder benefits and to make those benefits equal to offered medical and surgical benefits. To implement this proposal, Congress could (1) delete the introductory clause ("In the case of a group health plan (or health insurance coverage offered in connection with such a plan) that provides both medical and surgical benefits and mental health and substance use disorder benefits—") from 42 U.S.C. § 1185a(a)(1) (2006) (regulating aggregate lifetime limits), 42 U.S.C. § 1185a(a)(2) (2006) (regulating annual limits), 42 U.S.C. § 1185a(a)(3) (2006) (regulating financial requirements and treatment limitations), and 42 U.S.C. § 1185a(a)(5) (2006) (addressing care provided in the out-of-network context) and replace it with the following language: "In the case of a public or private individual or group health plan (or health insurance coverage offered in connection with such a plan) . . . "; (2) delete the construction language currently set forth at 42 U.S.C. § 1185a(b) (2006) that provides, "Nothing in this section shall be construed—(1) as requiring a group health plan (or health insurance coverage offered in connection with such a plan) to provide any mental health or substance use disorder benefits"; (3) delete the small employer exception at 42 U.S.C. § 1185a(c)(1) (2006); (4) create a new paragraph at 42 U.S.C. § 1185a(a)(6) (2006) that provides, "In the case of a public or private individual or group health plan (or health insurance coverage offered in connection with such a plan) that provides medical and surgical benefits, the plan shall also provide mental health and substance use disorder benefits."; and (5) make conforming changes to 42 U.S.C. § 1185a(b)(2) (2006) as well as other Medicare and

Medicaid provisions, as necessary. I emphasize that my proposals would apply to both private and public health plans in light of current federal law, which provides fewer Medicare and Medicaid benefits for inpatient psychiatric hospital services and care received at institutions for mental disease, respectively,[395] compared to inpatient non-psychiatric hospital services.

D. Conclusion

In this Article, I used postpartum illness as an example of a health condition that has an evolving scientific understanding to demonstrate that the science lawmakers use to support and interpret health law and policy is not always accurate. Further, I suggest that lawmakers who rely on outdated scientific findings risk developing inappropriate health laws and policies, encouraging the introduction of expert testimony that will not meet evidentiary standards, establishing conflicts between different health laws and policies, and supporting the public misunderstanding of health conditions. I hope that readers will project the concerns I have expressed in the context of postpartum law and policy to other areas of health law including, but certainly not limited to, compulsory immunization law and policy, which is currently based on scientific findings regarding the safety and efficacy of early childhood vaccines but is being challenged by some parents who believe that their children are developing autism and other disorders as a result of vaccines;[396] disease reporting law and policy, which is based on epidemiological findings relating to communicable diseases such as the swine flu and

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[395] *See* 42 C.F.R. § 409.62 (2009) (federal Medicare regulation limiting inpatient psychiatric hospital services to 190 lifetime days); 42 U.S.C. § 1396d(i) (2006) (federal Medicaid provision defining the term "institution for mental diseases" as "a hospital, nursing facility, or other institution of more than 16 beds, that is primarily engaged in providing diagnosis, treatment, or care of persons with mental diseases, including medical attention, nursing care, and related services"); 42 C.F.R. § 435.1009(a)(2) (2009) (federal Medicaid regulation providing that federal financial participation is not available for services provided to "individuals under age 65 who are patients in an institution for mental diseases unless they are under age 22 and are receiving inpatient psychiatric services"). *See generally* Sara Rosenbaum & Joel Teitelbaum, George Washington University School of Public Health and Health Services Issue Briefs*, Issue Brief #2: An Analysis of the Medicaid IMD Exclusion* 2 (Dec. 19, 2002) *available at* http://www.gwumc.edu/sphhs/departments/healthpolicy/CHPR/downloads/behavioral_health/reports/IMD%20Report%201202.pdf) (noting that the Medicaid limitation on coverage of care provided at institutions for mental diseases raises several policy questions and poses several important questions: "As researchers gain an increasing understanding of the biological basis of many forms of mental illness, does the exclusion continue to have meaning other than as a financial penalty? If conditions classified as 'mental diseases' in fact increasingly are treated through medical therapies and treatments that require limited to no traditional psychiatric intervention, is there still a justification for the exclusion?").

[396] *See, e.g.*, U.S. Court of Federal Claims, Omnibus Autism Proceeding, *available at* http://www.uscfc.uscourts.gov/omnibus-autism-proceeding (last visited Nov. 15, 2009) (providing information about lawsuits filed by parents against the federal government seeking compensation for the injuries allegedly sustained by their vaccinated children).

sexually-transmitted diseases;[397] complementary and alternative medicine and the law and policy, conservative forms of which are loyal to traditional allopathic medicine and are being challenged by individuals who seek a more holistic approach to care, including individuals who seek access to non-physician acupuncturists, chiropractors, homeopaths, naturopaths, massage therapists, and midwives;[398] mental health law and policy, which is based on scientific findings regarding the safety and efficacy (or lack thereof) of controversial therapies such as physical restraint, chemical restraint, deep brain stimulation, electroconvulsive therapy, and psychosurgery;[399] and abortion law and policy, which is based in part on scientific findings relating to fetal viability, maternal health risks associated with abortion, and maternal health risks associated with carrying a child to term.[400] In all of these contexts, lawmakers need to be able to understand the medical and scientific bases of the health conditions, theories of health care, and medical or surgical interventions under consideration, use those understandings to inform and shape (but not dictate) the development of appropriate law and policy, and, perhaps most importantly, revisit existing law when its medical and scientific foundations have changed.

More broadly, I have attempted to demonstrate that differences between scientific and legal understandings in health law may relate less to the complexity of the substantive topic under review than to differences between the *process* of fact finding in science compared to law. As illustrated in Part I, the scientific investigation of postpartum illness has been characterized by an open-ended search for knowledge with the recognition that scientific findings published one day are subject to revision the next. In almost all of the published scientific studies and review articles referenced in Part I, the scientist authors emphasized the limitations of their findings, the fact that the etiology of postpartum illness remains poorly understood, and the fact that additional research is needed, and identified possible directions for future

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[397] *See, e.g.*, TEX. HEALTH & SAFETY CODE § 81.041 (Vernon 2001 & West Supp. 2009) (requiring the reporting of certain communicable diseases in the name of health promotion and disease prevention).

[398] *See, e.g.*, Suzanne Hope Suarez, *Midwifery Is Not the Practice of Medicine*, 5 YALE J.L. & FEMINISM 315, 315 (1993) (arguing that states should legalize traditional midwifery).

[399] *Compare* TEX. HEALTH & SAFETY CODE § 578.003(b) (Vernon 2003) (requiring patients considering electroconvulsive therapy to be informed by their physicians that a division of medical opinion as to the efficacy of electroconvulsive therapy exists) *with Branning v. Branning*, 285 Ill.App.3d 405, 411 (Ill. App. 1996) (finding a "significant" "liberty interest in refusing unwanted ECT, psychosurgery and services of an 'unusual, hazardous, or experimental' nature.").

[400] *See, e.g.*, TEX. HEALTH & SAFETY CODE § 170.001(3) (Vernon 2001 & West Supp. 2009) (statutory definition of viability); *id.* § 171.012(a)(1)(B), (D) (statutory informed consent provisions addressing maternal health risks).

research.[401] The understanding and expectation of these authors is that future studies may yield different findings and disprove the authors' hypotheses.[402]

As demonstrated in Parts II and III, legislators and judges also have sought to understand postpartum illness as necessary to make laws that affect and adjudicate disputes involving new mothers, although legal understandings of postpartum illness tend to be characterized by factual findings that are fixed in time. In very few of the statutes and judicial opinions referenced in this Article did the legislators and judges recognize that postpartum illness is a complex health condition that remains incompletely understood. In none of the statutes and judicial opinions referenced in this Article did the legislators and judges state that their findings should be revisited as the scientific understanding of postpartum illness evolves. This result is to be expected in a common law system that is based on the concept of precedent, in which judges through opinions establish principles and rules that other judicial bodies rely on when deciding subsequent cases involving similar issues. The result, however, is the eventual discrepancy between scientific and legal findings in the context of postpartum illness. For these reasons I do not conclude, as do so many other health law scholars, that lawmakers simply need to improve their understanding of the particular scientific relationship, technology, or health condition (in this case, postpartum illness) under review. Instead, I also recommend improving the awareness of health law and policymakers of the existence of differences between scientific and legal methods and the need to revisit legislative and judicial findings as their medical and scientific foundations evolve.

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[401] *See, e.g.*, Charles B. Nemeroff, *Understanding the Pathophysiology of Postpartum Depression: Implications for the Development of Novel Treatments*, 59 NEURON 185, 185 (2009) ("Depression during pregnancy and in the postpartum period is . . . poorly understood in terms of pathophysiology."); Lorberbaum, *supra* note 96, at 99 ("Future work in this area may help: (1) unravel the functional neuroanatomy of the parent-infant bond; and (2) examine whether markers of this bond, such as maternal brain response to infant crying, can predict maternal style (i.e., child neglect), offspring temperament, or offspring depression or anxiety.").

[402] *See, e.g.*, ROBIN DUNBAR, THE TROUBLE WITH SCIENCE 19 (1995) (referencing Australian philosopher Karl Popper's observation that scientists proceed by attempting to disprove, not prove, previously generated hypotheses).