fMRI and Endocrinological Studies of Depression and Anxiety Following the Birth or Adoption of a Child: Towards a Model of Feminist Science[1]

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(Received: 16th January 2024; Revised: 30th April 2024; Accepted: 10th May 2024)

Abstract

This paper reviews selected literature between 2000 and 2015 on efforts, through fMRIs and endocrinological studies, to ascertain the causes of depression and anxiety following the birth or adoption of a child and to improve treatment. Typically, only the brains of postpartum women have been studied to determine whether depression and anxiety after the birth or adoption of a child can be associated with changes in the brain. Similarly, endocrinology studies have been limited to women who have recently given birth, and sometimes result in sexist stereotypes about both the causes and impacts of postpartum depression and anxiety, which may compound barriers to recovery. Studying only postpartum women's brains and attempting to isolate a cause particular to women's hormones contributes to damaging stereotypes of women, is likely to discourage men from seeking help, and to date does not seem to be productive in leading to effective treatment. Further, the lack of attention to social factors may result in less effective treatment. To improve diagnosis and treatment and to move towards a more equitable model of science, diagnosis of postpartum depression should examine the role of social factors, include others experiencing parental depression besides postpartum women as subjects, and avoid essentialist conclusions.

[1] Author note: This analysis was influenced by my experience with post-partum depression and anxiety in 2008. I would like to thank Dr. Carla Fehr for piquing my interest in this area of study through a 2015 graduate seminar on feminist philosophy of biology. I would also like to thank my cohort from the HASTAC Feminist Scholars Digital Workshop for providing valuable feedback on the second draft in 2017. Finally, I would like to thank the three anonymous peer reviewers and the editors for providing generously substantial critique and contextualization of that critique.
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Depression following the birth or adoption of a child is a serious public health issue. Postpartum depression, a "depressive episode that begins in or extends into the postpartum period" which includes symptoms of "anxiety, guilt, negative maternal attitudes, and poor parenting self-efficacy" (Lanes et al., 2011), has been characterized as a "significant public health problem" (Moses-Kolko et al., 2010). Postpartum depression affects 10 to 15 percent of people who have recently given birth (Brummelte & Galea, 2010; Anderson & Maes, 2013), though some studies estimate up to 19 percent may be affected (O'Hara & McCabe, 2013), with other studies citing 15-20% of those who have recently given birth affected (Leuner et al., 2014, p.1). Yet another study posits that due to the difficulties associated with diagnosis (such as stigma, lack of screening, etc.), the rates of postpartum depression may be even higher than 20-25% (Lonstein, 2007, p.116). A number of studies of paternal postpartum depression and anxiety published from 2005-2015 by Barbara Figueiredo and Ana Condes (2011), Jane Iles et al. (2011), James F. Paulson and Sharnail D. Bazemore (2010), Beatrice P.Y. Lai et al. (2010), Luciana Quevedo et al. (2011) Sharon Bond (2010), and Karen Wynter et al. (2013) – just to name a few - suggest that men also experience mental health changes in the postpartum period and that paternal depression is recognized as a condition. Further, research has demonstrated that depression also occurs in parents following the adoption of a child. However, the majority of studies addressing parental depression focus on postpartum depression. Depression following the birth or adoption of a child has consequences not just for the individual with the condition but also for their children and families (Moses-Kolko et al., 2010). Due to the prevalence and severity of postpartum depression, researchers cite a need to understand the role of neurobiology in postpartum response (Strathearn et al., 2009, Moses-Kolko et al., 2010, O'Hara & McCabe, 2013). A review of the literature of a recent 15-year period (2000-2015) reveals a number of articles dealing either with fMRI studies relating to postpartum depression/ anxiety or neuroendocrinology and postpartum depression/anxiety, one of which is a comprehensive review of prior literature on postpartum women and fMRI studies (Moses-Kolko et al., 2014).

1. Overview

In the second section, I provide an account of the literature reviewed. These studies do not comprise, nor do they intend to comprise, a representative sample of the literature from 2000 through 2015 on depression following the birth or adoption of a child, fMRIs, and endocrinology. Rather, they merely illustrate prevalent attitudes from the time period about parental depression, how it should be diagnosed, and how it should be treated.

In the third section, I detail critiques of these studies - both in terms of how the studies were conducted, the results sections, and how the studies might be perceived and articulated in public discussions. These studies dichotomize men and women. They also insist that the cause of postpartum depression/anxiety is unique to women's hormones, thus supporting stereotypes about women's hormones and their behavior. These studies are limited to postpartum women, and so decrease the likelihood of treatment-seeking behaviors and diagnosis in men (as well as women partners of a woman who has recently given birth to a child) and adoptive parents. I also argue against an etiology-only focus on researching postpartum depression/anxiety. An etiology-only method, while understandable given the potential consequences of postpartum depression/anxiety on women and children, also has multiple shortcomings. I discuss some justifications for a focus on etiology and then discuss the shortcomings. These include the contention that focusing only on a brain-based or hormone-based etiology may lead to further stigma for women with postpartum depression/anxiety. Further, I argue that

additional limitations of the studies include that rats are not a good animal model and that the studies do not take into account potential differences across race and levels of education.

In section four, I provide an overview of what feminist science does not and does entail. In section five, I argue that studies which considered social risk factors; compared women with postpartum depression/anxiety to men with postpartum depression/anxiety and other, similar conditions such as autism; *and* conducted fMRIs could provide a better model for feminist science. In the final section, I discuss why such a model might provide better treatment strategies for postpartum depression/anxiety and perhaps other conditions that appear to be related as indicated by fMRIs.

2. Account of the research

In these studies, there is an almost unilateral focus on the etiology of postpartum depression, as opposed to focusing on the symptoms and the impacts of those symptoms. With postpartum depression, of course, it is of dire importance that the etiology of the syndrome is given explicit attention, particularly because treatment tends to be ineffective. Just 30-50% of depressed mothers who underwent eight weeks of drug treatment or 12 weeks of "interpersonal therapy" were considered in recovery (Wisner et al., 2006; O'Hara et al., 2000; Moses-Kolko et al., 2010) and it is due to the inefficacy of treatment that Eydie L. Moses-Kolko et al. write that "[g]reater mechanistic understanding of postpartum depression is needed" (2010, p.1373). Greater understanding of the etiology of postpartum depression and anxiety may be necessary for greater access to treatment, as well as more targeted and thus potentially more effective treatment.

A way of "proving" that the depression following the birth or adoption of a child by reference to fMRI may lead more doctors to treat new parents with depression. Additionally, a better understanding of the causes or effects of depression following the birth or adoption of a child as demonstrated by fMRI may lead to better treatment strategies. Cindy-Lee Dennis and Leinic Chung-Lee (2006) note that stigma, as well as practitioners' lack of differentiation between postpartum depression and/or anxiety and "normal maternal adjustment," prevents many women from getting treatment (Dennis & Chung-Lee, 2006, as cited in Moses-Kolko et al., 2010). Miki Bloch et al. (2003, p.234) note that due to "the relative paucity of studies of PPD … many of the important questions in this field remain largely unanswered," including "Are there endocrine abnormalities that characterize women with PPD?" and "What is the biological trigger (if such exists) that is responsible for the onset of depression in the postpartum?" Green et al. (2009, p.259) note that "[d]espite recent interest in PPD, the underlying etiology remains unknown". S. Brummelte and Liisa A.M. Galea cite the justification for their own study (2009) as meeting the need for "further research … to understand the HPA [hypothalamus-pituitary-adrenal] axis' role in treatment and etiology of depression in men and women" (p.767). Hillerer et al. (2011 p.3930) state that "[d]espite the high incidence of" postpartum depression, postpartum anxiety, and postpartum psychosis "and the detrimental outcome for both mother and child, their etiology remains poorly understood" and Joseph S. Lonstein (2007, p.116) states that "[t]he neurobiology underlying why millions of women experience elevated anxiety after giving birth is poorly understood." Therefore, Michael E. Silverman et al. (2007) have valid points when they suggest the need for fMRI studies to better understand the potential ways in which hormones differentially affect postpartum parents:

> [I]t is quite possible that the observed symptoms may correspond to differences in the way the central nervous system responds to various (and possibly interactive) hormonal

> and immunological fluctuations. Nevertheless, although brain response to sex hormones in PPD patients appears central to a neurobiological understanding of PPD's psychopathology, to date, a specific hormonal mechanism has remained elusive. (p. 855).

Some of the studies avoid an absolute and exclusive correlation of hormonal levels and/or areas of brain activation with the cause of postpartum depression. Eydie Moses-Kolko et al. (2010, p.1379) is careful to note that less activity in the dorsomedial prefrontal cortex and decreased connectivity between the amygdala and the dorsomedial prefrontal cortex in women who have been determined to have postpartum depression, as opposed to women who do not, may be due to a cause *or* an effect. Moses-Kolko et al.'s (2014, p.666) review of the literature on neural endophenotypes in postpartum depression delineates the difference between "association" and "causality" in relation to "brain and behavior," stressing that "imaging data provide information on the association." Furthermore, Moses-Kolko et al. (2014, p.665) exposes the "false dichotomy in the early affective neuroscience literature in mothers, whereby one set of studies focuses on standard emotion processing deficits samples of women with major depression and another set of studies focused on maternal responses to infant cues to inform the neural circuitry of maternal caregiving". They critique these initial neuroscience studies of postpartum women because they assume, as does Joseph Lonstein (2007, p.116), that due to noting higher levels of depression among women during the postpartum period, "the causes of postpartum depression and general depression in women are [not] the same".

3. Critique of the research: The risks of an etiology-only, essentialist approach to study design and interpretation

Depression following the birth or adoption of a child is often perceived as affecting only women, and the research on this topic has only studied women's brains. Consequently, it may appear that studies of women with postpartum depression at least begins to avoid the limitations inherent in studies which explicitly search for brain differences between the sexes.

However, an implicit bias exists in the research designs themselves. The majority of these studies are predicated on the idea that only women experience postpartum depression, and that their symptoms are intricately linked to hormonal changes during and after pregnancy - especially the extreme decrease in estradiol and other forms of estrogen after delivery (Galea et al., 2001, p.2; Brummelte & Galea, 2010, p.770; Moses-Kolko et al., 2009). The majority of the studies make claims which associate depression and anxiety following the birth of a child with hormone fluctuations, and very specific hormone fluctuations at that – the decrease in progesterone and estradiol after an increase during pregnancy (Galea et al., 2001, p.2; Brummelte & Galea, 2010, p. 770; Moses-Kolko et al., 2009) - though Joseph S. Lonstein (2007, pp.117-118) states that "there is little evidence for the involvement of ovarian hormones in the etiology of persistent postpartum anxiety" despite the relationship that has been found between postpartum depression and "steroid hormone changes." Such a claim both contributes to stereotypes about women's hormones and their effects, and may contribute to the perception that the partners of postpartum women and adoptive parents do not experience depression and anxiety following the integration of a new child into a family, or if they do, that their symptoms are not severe enough to warrant study.

Hypotheses suggesting that postpartum depression and anxiety are caused by a sharp drop in hormone levels also has the potential to increase the circulation of stereotypes – however well intentioned – that women's emotions and reasoning are unduly influenced by hormones. Despite the validity of the rationale for focusing on etiology and the potential

benefits this entails, there are considerable risks of an exclusively etiology approach: the assumption that there is no association between postpartum depression and general depression, the potential for further stigma, and the possibility that fMRI and neuroendocrinological diagnoses of postpartum depression may lead to less treatment instead of more. The problems these studies exhibit include dichotomization of the subjects, essentialist claims (for instance, the claim that women suffer more from depression and are more susceptible to stress due to differences in hormones associated with the reproductive cycle, suggesting that there are innate sex-related characteristics as opposed to differences which arise from social experiences), and a focus on etiology which supersedes "the workings of the brain" - an approach which Dussauge and Kaiser (2012, p.136) discourage in the case of fMRIs focusing on identifying differences between how queer and straight men and women process sexual attraction because an explicit focus on etiology does not consider that "there is no such thing as a natural order of gender and sexuality" and because "biological descriptions of the human have been [...] used to legitimize existing forms of oppression." An explicit focus on etiology in the case of postpartum depression/anxiety does not consider differences in the social risk factors for postpartum depression/anxiety, and as such excludes potential opportunities for diagnosis and treatment.

Furthermore, these articles suggesting that women are disparately affected by hormone changes often cite inadequate numbers of studies to demonstrate their claim, or make claims about women which may have more to do with social factors than with hormone levels. Brummelte and Galea (2010, pp.766-767) state that depression affects women two to three times more and that women are more susceptible to stress, apparently not considering that men under-report depression due to social pressure and "gender role conflict and traditional masculinity ideology" (Berger et al., 2006, p.76) and that the extra stress of women might have something to do with their social conditions – for instance, in the case of stress in postpartum, a postpartum woman might have to return to work within six weeks while also providing most of the night-time care for a newborn. Of the aforementioned study, University of Missouri Counseling Psychology Professor Glenn Good says, "I don't think that it's biologically determined that men will seek less help than women…so if that's true, then it must mean that it's socialization and upbringing. Men learn to seek less help" (Winerman, 2005).

Despite Berger et al.'s findings as well as the studies demonstrating postpartum depression in men, all but one of the studies on neuroendocrinology and fMRI included in this review from 2005-2015 focus on finding causes or solutions which have to do specifically with women's hormones levels during and/or after pregnancy. In some cases, the studies make very explicit essentialist claims regarding women's "susceptibility" or "resiliency," without providing much, if any, evidence for their claims. Brummelte and Galea (2010, p.767) claim that men exhibit "differential HPA normalization in response to antidepressant treatment in particular", but cite only one study by Binder et al. (2009). Lonstein (2007, p.116) claims that women are predisposed to "high trait anxiety and low resiliency," but that, since postpartum depression represents a large portion of depression cases and occurs also in women with no history of anxiety, it could not be accurate to say that the causes of postpartum depression and general depression in women are the same. These researchers are determined to isolate a cause particular to women's reproductive activity, if not to their hormones.

While etiology is of course important in the case of disease, it is arguable whether or not fMRI studies of postpartum depression and anxiety have actually contributed to treatment. Studying only women's brains and attempting to isolate a cause particular to women's hormones contributes to damaging stereotypes of women, is likely to discourage men from

seeking help, and to date does not seem to be effective in leading to improved treatment. That fMRI studies on depression following the birth or adoption of a child focus exclusively on postpartum women is therefore actually a weakness rather than a benefit in terms of providing a model for feminist research.

Similarly, women who are diagnosed with postpartum depression via fMRI may be more subject to the stigma that women with postpartum depression are likely to experience. For instance, some of the studies also convey the impression that women with postpartum depression are somehow unilaterally responsible for their condition as well as for the negative effects on their offspring. Douglas (2011, p.1173) argues that "[f]actors impacting on maternal physiology during gestation" (such as those factors associated with postpartum depression and/or anxiety) "cause poor or inappropriate fetal development such that the offspring is susceptible to multiple conditions that threaten its health and well-being later in life; for example, cardiovascular disease, depression, reproductive abnormalities and obesity. Lonstein (2007, p.116) cites higher "[a]nxiety … in mothers at risk for or already abusing their infants". Douglas draws on studies by Dunn et al. (2011), Larsen and Grattan (2010), Welberg et al. (2000), McArthur et al. (2007), and O'Regan et al. (2004). Lonstein cites De Bellis et al. (2001), Nayak et al. (1998), and Whipple et al. (1991). Nevertheless, Douglas and Lonstein are making very strong claims. Douglas's claims in particular could be perceived as ableist or transphobic because depression, reproductive abnormalities, obesity, and sexual dimorphism are cited as undesirable consequences of "poor or inappropriate fetal development," and Douglas also clearly places blame on the mother.

Moreover, if postpartum depression can be explained by a difference in the brain (sex related or not) that may be perceived as fixed or innate (the essentialist perspective addressed earlier), then researchers and doctors alike may respond by paying less serious attention to treatment. The belief that any difference is both innate and fixed may lead to claims that it is impossible to treat the condition and that there is no point in trying (DesAutels, 2010, p.100). Conversely, it is possible that fMRIs will lead to certain cases of postpartum depression/anxiety being deemed serious enough to treat and others not. Moses-Kolko et al. (2014, p.665) states that "there are growing efforts to understand the neurobiology of maternal psychopathology and caregiving deficits, with the objective of establishing biomarkers for illness severity and treatment response."

Additionally, to the concerns that these studies may result in increased stigma of parents with depression and/or anxiety and potentially decrease options for treatment, a major limitation of these neuroendocrinological and fMRI studies of postpartum depression is the use of rats as an animal model. As Cordelia Fine and Giordana Grossi (2012, p.80) note, "[h]umans are cognitively and neurologically dissimilar to rats". Moreover, as Galea et al. (2001, p.6) note, a rodent pregnancy is unlike a human pregnancy in that estradiol levels only "rise during the final few days" and progesterone levels drop throughout gestation in rodents, whereas in humans estradiol levels are high throughout the pregnancy and then increase substantially in the third trimester. The neuroendocrinological and fMRI studies of rats suggest that rats are not good models, in that they are not comparable to humans. A study of chronic gestational stress and the effect on rat dams indicated increased depressive-like symptoms, for instance, but no increase in anxiety-like symptoms (Leuner et al., 2014, p.6). However, "postpartum anxiety disorders are more common than postpartum depression," according to a 2005 study by Amy Wenzel et al. (p.295), with Lonstein's (2007, p. 115) study of neonatal contact as an anxiolyte for postpartum anxiety noting that postpartum depression itself "is frequently associated with elevated anxiety". Since rats do not show an increase in anxiety-like symptoms when exposed

to gestational stress but postpartum anxiety has been shown to be common in humans, it would seem that rats are not a good animal model due to the differences in symptomatology.

However, some studies utilizing rats as the animal model for postpartum depression yield important insights. For instance, a study by Galea et al. (2010, p.7) demonstrates that when exposed to estradiol, rats display fewer depressive-like symptoms and show greater mobility. Oliver Bosch et al. (2006, p.547) find that rat dams bred for anxiety spend more time nursing their pups, and also spend more time on general care activity, including grooming and carrying. Some findings may be relevant to the care giving behaviors of women with postpartum depression – for instance, postpartum depressed women were more likely to breastfeed, or breastfeeding women were more likely to be depressed, according to the results of Moses-Kolko et al.'s study (2010, p.1375).

Despite what we might learn from high anxiety rats about an ethics of care and breastfeeding, rats are not the ideal animal model. Indeed, it may be that animal models should not be used at all, since animals cannot possibly provide a model for the social factors which may play a role in postpartum depression. Therefore, these studies are remiss in using rats as an animal model, both because of the biological differences and because of the lack of ability to reproduce the social risk factors which should be studied in women and men with postpartum depression/anxiety.

The lack of attention to social factors, including the differential ways in which certain groups of women might experience stress postpartum, is also a weakness in some of the fMRI studies on women with postpartum depression. For instance, the study by Moses-Kolko et al. states that 11 out of 16, or 68.8 percent, of healthy mothers are Caucasian and that ten out of 14, or 71.4 percent, of depressed mothers are Caucasian. The race of the non-Caucasian participants is not indicated. The mean years of education for healthy and depressed mothers in Moses-Kolko et al.'s study are 15.2 and 15.1, respectively. The study does not take into account, then, postpartum experiences across race and different levels of education. Similarly, the other studies do not either test a diverse sample of women experiencing postpartum depression, or consider how age, race, ethnicity, and class might impact risk factors, the onset of, and symptomatology of parental depression and/or anxiety.

4. Models for feminist science

What follows is an outline of some of the more recent history of the social consequences of looking for biological differences between the sexes and seeking to identify proof of characteristics such as morality through fMRIs, and the importance of considering lived experience when analyzing the results of fMRIs.

Feminist science indicates that there are dangers in making essentialist claims, including the potential for further stigma. Peggy DesAutels makes such dangers explicit in her discussion of attempts to find morality through brain studies. DesAutels (2010) argues that defining a "moral center" in the brain in terms of what proportion of certain neurochemicals are associated with morality, may have negative consequences for those who are determined *not* to have the proportion of neurochemicals that indicates morality:

> [W]here should this "normal space" be centered? If there are relevant sex-based differences, which sex's brain is considered the prototype for being in control? Of most significance here, I think, is using hormone levels as a parameter for the "normal"

> space…. [A]re the brains of pre-menstrual women destined always to be borderline if not out-of-control? (p.103)

DesAutels posits that if Patricia Churchland's concept of "mapping" a moral center in the brain is realized, it will have to be done so with care:

> Women are much more likely than men to be stereotyped as "hormonal" and irrational. Hormones supposedly rage in women when they are pre-menstrual, menstrual, post-menstrual, pregnant, post-partum, pre-menopausal, and post-menopausal. Women are viewed as being more cognitively swayed by hormone levels, and as more likely to be outside the norm when there are variations…. I do not wish to discourage further pursuit of brain-based understandings of being in control. Instead, the agenda I wish to promote is a more cautious research program, one that is sensitive to individual variation and to sex-based differences, environmental and biological, that shape the brains being observed…. I would also hope those neuroethicists attempting to follow through on Patricia Churchland's proposal take variability within humans, within sex groups, and within individuals very seriously. Even if they do, my concern remains, that attempts to center and circumscribe even quite large neural in-control-spaces privilege those whose spaces fall in the center and quite literally morally marginalize those at the margins. (p.104)

DesAutels concludes by stating that researchers should be cognizant of and acknowledge their "sex-based biases", avoid letting their biases unduly influence "research questions" and interpretation of results, be mindful of how they articulate and where they publish their results given that "findings of even very slight average differences in men's versus women's brains are likely to be used to promote patriarchal and sexist ends" (p.100). She also states that researchers should intervene in public discussions to the extent possible to "correct misuses and misinterpretations of their findings" (p.101).

Similarly, other neuroethicists recommend caution in attempting to prove that there are differences between the brains of men and women because sometimes this research is not conducted in good faith, or the findings are applied in such a way that can compound discrimination towards marginalized and oppressed groups. Robin Bluhm (2015) writes that in the 1970s and the 1980s, scientists showed renewed interest in demonstrating brain differences between the sexes because of the major increases in social change and women's rights. As Ruth Bleier noted when "social movements threaten the social order, it is a recurrent phenomenon that corresponding scientific theories emerge that implicitly defend the *status quo*" (Bleier, 1984, p.1408, as cited in in Bluhm, 2015). That is, these studies were conducted not in the spirit of neutrally testing a hypothesis, but with an ulterior motive. Similarly, Anne Fausto-Sterling (2000, p.115-116) writes that "scientists do not simply read nature to find truths to apply in the social world. Instead, they use truths taken from our social relationships to structure, read, and interpret the natural." Because scientists have employed scientific studies in order to argue for brain differences between men and women which perpetuate the conflation of sex and gender and the concept that women are inferior to men, feminist scientists and feminist philosophers of science have begun to argue that finding brain differences between the sexes should not be a scientific goal.

Cordelia Fine and Giordana Grossi state that it is "problematic to attribute to differences between the 'female brain' and the 'male brain' sex differences in E [empathizing] and S [systematizing] that can be so readily reduced and even eliminated by simple social manipulations that diminish the salience of stereotypical expectations" (2012, p.79).

Though differences in the brain between the sexes should be acknowledged by researchers should they be found to exist, the reverse is also true - that, when a study finds no significant differences between the brains of men and women, that result should be published as well. Because many scientists are looking for brain differences between the sexes, Jordan-Young and. Rumiati recommend "turning away from sex/gender differences" (2011, p.105).

Further, "neuroscience, especially affective neuroscience, really must ask … the organisms being studied what it is *like* for them" because "experience … doesn't only take place in the brain, and is textured by context" (Einstein, 2012, p.157). Einstein recommends "situating" the researcher, study, and participants (pp.164-166) in answering the question, "what would it mean to *do* feminist neuroscience" (p.149) in her study of Somali Canadian women who have undergone ritual female circumcision. Einstein recommends asking and answering the research questions from multiple perspectives, influenced by "how the participant feels about the question", "how [...] the environment" in which the research is taking place and who is asking the question "affect the participant's account", and how "physiological responses relate to what the participant is saying" (p.244). Drawing on Harding's concept of "radical reflexivity" (as cited in Einstein, 2012, p. 246), Einstein also suggests making explicit the "class, race, culture and gender assumptions" and "behaviors" of the researcher. Further, Einstein recommends conducting the research with the understanding that "situated biologies are particular to their context, including: culture, geography, experience, sex, gender, etc." (p.245). Finally, Einstein posits that doing feminist neuroscience well should reduce ignorance about sexed bodies and experiences historically excluded from research, oppose essentialist notions of (sexed) bodies, and conduct studies which are of value to the participants (pp.222-223).

By making explicit the biases of the researcher and the interests and needs of participants, feminist neuroscience has the potential to make neuroscience more ethical and equitable, not just through greater inclusivity of the subjects studied and the methods used, but also through how the results of the research are shared with and directly benefit their subjects.

5. The benefits of considering social factors *with* fMRI and endocrinological studies of parental depression: Aligning fMRI and endocrinological studies following the birth or adoption of a child with feminist science

As noted in section one, there are valid reasons for pursuing an etiology approach to studying and treating depression following the birth or adoption of a child. However, fMRI studies and/or neuroendocrinology studies of postpartum depression should ask several important questions about etiology *besides* how particular areas of brain activation or certain measures of hormones – either basal or hormone turnover measures - might suggest causes of postpartum depression. One question is, is it possible that certain areas of brain activation or certain hormone levels in postpartum depressed women suggest effect rather than cause? That is, are hormone levels/activation of certain areas in the brain representative of a cause or are they an indication of something happening in the brain in response to a cause? This question is important because should it be the case that what is happening in the brain is in response to a cause rather than a cause itself, it is even clearer that social factors need to be more seriously considered. Another question is, if we believe that areas of brain activation in relation to or associated with certain hormonal levels *are* indicative of a cause for postpartum depression, how can we avoid an essentialist claim that in all cases postpartum depression/anxiety is due to particular hormones and particular hormone levels? Such a claim can be damaging because it ignores social factors that have been shown to play a role in the etiology of postpartum

depression/anxiety, such as "stressful life events during pregnancy" or early in the postpartum period, "poor social support, marital conflict … young maternal age, [and] … low partner support" (Pearlstein, 2009, p.358). Finally, how could neuroscientists collaborate with social scientists in order to develop a more comprehensive understanding of the causes of postpartum depression, and then use this understanding to develop better prevention and treatment strategies?

Studies of postpartum depression and anxiety, particularly in relationship to fMRI and neuroendocrinology, should avoid essentialist claims. Not only do essentialist claims have the potential to cause further harm to women with postpartum depression and decrease the likelihood that men with postpartum depression will seek help, but the results of various studies trying to find neuroendocrinological differences in women demonstrate a potential contradiction which complicates the relationship between hormone levels and the onset of postpartum depression. Some studies suggest that reproductive related depression actually begins *during* pregnancy (Gaynes et al., 2005, p.3), so how can postpartum depression/anxiety be definitively linked to the sharp drop in hormone levels after delivery? Further limitations include the use of rats as a model for human cognition and behavior, and the general lack of diversity and awareness of subjects' situated identities, including the social factors which might play a role in postpartum depression.

The disadvantage, both of the studies intended to model postpartum depression in rats and of the fMRI studies, is that they do not adequately consider social factors in postpartum depression. Is there anything to be salvaged from these studies; that is, do they indicate something useful about postpartum depression, particularly for a feminist model of science? Also, what would a study which incorporated both a consideration of social factors and fMRI look like? I posit that a study which incorporated both would look somewhat like the studies by Moses-Kolko et al., except that instead of looking for differences between postpartum depressed women and women without postpartum depression, such a study might look for similarities in the brain between postpartum depressed women and people with general depression, similarities between women with postpartum depression and people on the autism spectrum, or similarities between women and men presenting symptoms of postpartum depression. Such a study would both provide the diagnosis that women with postpartum depression need, and move away from an essentialist, potentially sexist argument about the absolute causal role of hormones.

How might the tenets of feminist science be applied to fMRI studies of parental depression to reduce the likelihood that these studies will lead to reification of sexist concepts, promote more humanistic inquiry, and increase the likelihood that these studies will result in more inclusive and effective treatment of parents with depression? As Ellen Leibenluft notes (2010, p.1295), "research on the circuitry mediating postpartum depression and/or maternal-child interactions in healthy and depressed women will have the greatest impact if it suggests new treatment strategies." Therefore, studies of postpartum depression and the brain should include both men and women as research subjects. fMRI studies should also in all cases be conducted in conjunction with an understanding of the social factors involved in depression and/or anxiety that follows the birth or adoption of a child.

An understanding of the social factors may lead to alternative and/or preventative treatment strategies, while the use of fMRI may lead to better strategies for diagnosis, more uptake of postpartum depression/anxiety research, and new treatment strategies for women who are already suffering from postpartum depression/anxiety. Using fMRI along *with* contextualizing risk factors as socially situated can demonstrate that people besides postpartum

women experience depression following the integration of a new child into the family. The causes may be different, but fMRI can show that the effects are the same. fMRI studies also demonstrate that postpartum depression bears some similarity to autism. The same areas of the brain – particularly the amygdala - show less activation in both postpartum depressed women (Moses-Kolko et al., 2014, p. 667-669) and autistic subjects (Baron-Cohen et al., 2000, p.360). A study which makes the potential connection between postpartum depression and autism more explicit is Brian E. Eisinger et al.'s 2013 study, which demonstrates that there is a "genetic link between the maternal brain and pathways involved in autism" (p.6). Another study which makes explicit a potential connection is Inga D. Neumann's 2007 study, which details the potential of studying oxytocin's "modulat[ion] of social behaviors" and treatment of "numerous psychiatric illnesses, for example social phobia, autism, and postpartum depression" (231). Because fMRI can show similarities in the brain among subjects experiencing different, but related, symptoms, fMRI studies can show that depression and anxiety following the birth or adoption of a child may have very little to do with women's hormones, and may still provide a model for feminist science.

Similarly, conducting fMRIs of parents experiencing depression and/or anxiety following the birth or adoption of a child in conjunction with attending to the social factors which may play a role in etiology and symptomatology may lead to increased awareness, increased and improved prevention efforts, and improved treatment strategies. For instance, if fMRIs of parents experiencing depression and/or anxiety following the birth or adoption of a child demonstrated that depressed parents who are financially insecure have differential brain activation as opposed to depressed parents who are not financially insecure, these results could be used to bolster intervention and prevention strategies for parents who are financially insecure.

6. Implications for treatment

What can be learned from these various studies in terms of potential treatments? In addition to providing the diagnosis that will make it more likely for treatment to be initiated, some of the studies – despite their limitations – offer intriguing possibilities for treatment. Galea et al. (2001, p.5) demonstrate that estradiol administered after delivery is effective "at alleviating 'depressive-like' symptoms in a 'post-partum model' of depression in rodents" and that the result "complements those that find supplemental estrogen given to women with PPD or with a history of PPD significantly alleviates their depressive symptoms." Understanding the role of fMRI and endocrinology studies in postpartum depression/anxiety alongside fMRI studies which include parents who have not given birth, however, might lead to development of treatments that work not just for women with postpartum depression/anxiety, but perhaps for all parents experiencing depression and/or anxiety following the birth or adoption of a child, and for others who experience difficulty processing emotion in relationships due to a lack of connectivity between the amygdala and the dorsomedial prefrontal cortex.

In addition to increasing the likelihood that men as well as women will seek diagnosis and treatment for postpartum depression/anxiety, and potentially developing treatments which are beneficial for general depression and for autistic people, conducting fMRI along with contextualizing social risk factors for postpartum depression/anxiety has other uses. Conducting fMRI along *with* contextualizing risk factors for postpartum depression/anxiety as socially situated also has the potential for demonstrating that, despite claims to the contrary, postpartum depression among women in developing countries can be considered analogous to postpartum depression among women in more developed countries.

Conclusion

In this paper, I have argued that though fMRI studies of women with postpartum depression do not yet provide a model for feminist science, they could. They do not currently provide a model for feminist science because they dichotomize men and women, make essentialist claims which have the potential to harm both women and men, and focus only on the etiology of postpartum depression/anxiety in terms of hormones and other sex-based differences. Such a narrow focus on etiology may provide some useful treatment options, but it also excludes men and non-birth parents from diagnosis and treatment options, and increases the circulation of stereotypes about women's hormones and their effects on women's behavior. Moreover, rodent models of postpartum depression/anxiety and a lack of diversity (in terms of race and educational levels) are weaknesses of these studies. They are weaknesses because they do not consider the range of social risk factors which may contribute to postpartum depression/anxiety.

To provide a model for feminist science and to provide better and more diagnosis and treatment options, fMRI studies of postpartum depression/anxiety should include both men and women as research subjects and look for similarities between them. They should also in all cases consider the social risk factors that may lead to postpartum depression/anxiety. Considering social risk factors, including men and other parents who have not given birth as research subjects, and conducting fMRIs alongside endocrinological studies, may lead to a model which may be used in the future for analyzing the similarities between postpartum depression/anxiety and other conditions - thus potentially developing more effective treatments which could be used for a range of conditions.

REFERENCES

Anderson, G. & Maes, M. (2013). Postpartum depression: psychoneuroimmunological underpinnings and treatment. *Neuropsychiatric disease and treatment, 9*. doi: http://doi.org/10.2147/NDT.S25320

Baron-Cohen, S., Ring, H. A., Bullmore, E. T., Wheelwright, S., Ashwin, C., & Williams, S. C. (2000). The amygdala theory of autism. *Neuroscience and biobehavioral reviews, 24*(3), 355–364. https://doi.org/10.1016/s0149-7634(00)00011-7

Berger, J. M., Levant, R. F., McMillan, K. K., Kelleher, W., & Sellers, A. (2008). "Impact of gender role conflict, traditional masculinity ideology, alexithymia, and age on men's attitudes toward psychological help seeking": Erratum. *Psychology of Men & Masculinity, 9*(3), 192. https://doi.org/10.1037/1524-9220.9.3.192

Bloch, M., Daly, R. C., & Rubinow, D. R. (2003). Endocrine factors in the etiology of postpartum depression. *Comprehensive psychiatry, 44*(3), 234–246. https://doi.org/10.1016/S0010-440X(03)00034-8

Bluhm, R. (2015). Feminist Philosophy of Science and Neuroethics. In Clausen, J. & Levy, N. (Eds.), *Handbook of Neuroethics.* doi: 10.1007/978-94-007-4707-4_158.

Bosch, O. J., Krömer, S. A., & Neumann, I. D. (2006). Prenatal stress: opposite effects on anxiety and hypothalamic expression of vasopressin and corticotropin-releasing hormone in rats selectively bred for high and low anxiety. *The European journal of neuroscience, 23*(2), 541–551. https://doi.org/10.1111/j.1460-9568.2005.04576.x

Brummelte, S. & Galea, L. A. M. (2010). Depression during pregnancy and postpartum: Contribution of stress and ovarian hormones. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 34*(5). doi: http://doi.org/10.1016/j.pnpbp.2009.09.006

DesAutels, P. (2010). Sex differences and neuroethics. *Philosophical Psychology, 23*(1).

Douglas, A. J. (2011). Mother-offspring dialogue in early pregnancy: Impact of adverse environment on pregnancy maintenance and neurobiology. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 35*(5). doi: http://doi.org/10.1016/j.pnpbp.2010.07.024

Dussauge, I. & Kaiser, A. (2012). Re-Queering the Brain. In Bluhm, R., Jacobson, A. J. & Maibom, H. L. (Eds.), *Neurofeminism: Issues at the Intersection of Feminist Theory and Cognitive Science.* Palgrave McMillan.

Einstein, G. (2012). Situated Neuroscience: Exploring Biologies of Diversity. In Bluhm, R., Jacobson, A.J. & Maibom, H.L. (Eds.), *Neurofeminism: Issues at the Intersection of Feminist Theory and Cognitive Science.* Palgrave McMillan.

Eisinger, B. E., Saul, M. C., Driessen, T. M., & Gammie, S. C. (2013). Development of a versatile enrichment analysis tool reveals associations between the maternal brain and mental health disorders, including autism. *BMC neuroscience, 14*, 147. https://doi.org/10.1186/1471-2202-14-147

Fausto-Sterling, A. (2000). *Sexing the Body: Gender Politics and the Construction of Sexuality.* Basic Books.

Galea, L. A., Wide, J. K., & Barr, A. M. (2001). Estradiol alleviates depressive-like symptoms in a novel animal model of post-partum depression. *Behavioural brain research, 122*(1), 1–9. https://doi.org/10.1016/s0166-4328(01)00170-x

Gaynes, B. N., Gavin, N., Meltzer-Brody, S., Lohr, K. N., Swinson, T., Gartlehner, G., Brody, S., & Miller, W. C. (2005). Perinatal depression: prevalence, screening accuracy, and screening outcomes. Evidence report/technology assessment (Summary), (119), 1–8. https://doi.org/10.1037/e439372005-001

Green, A. D., Barr, A. M., & Galea, L. A. (2009). Role of estradiol withdrawal in 'anhedonic' sucrose consumption: a model of postpartum depression. *Physiology & behavior, 97*(2), 259–265. https://doi.org/10.1016/j.physbeh.2009.02.020

Grossi, G., & Fine, C. (2012). The Role of Fetal Testosterone in the Development of the 'Essential Difference' Between the Sexes: Some Essential Issues. In Bluhm, R., Jacobson, A. J. & Maibom, H. L. (Eds.), *Neurofeminism: Issues at the Intersection of Feminist Theory and Cognitive Science.* Palgrave McMillan.

Hillerer, K. M., Neumann, I. D., & Slattery, D. A. (2012). From Stress to Postpartum Mood and Anxiety Disorders: How Chronic Peripartum Stress Can Impair Maternal Adaptations. *Neuroendocrinology, 95*(1). doi: http://doi.org/10.1159/000330445

Lanes, A., Kuk, J. L., & Tamim, H. (2011). Prevalence and characteristics of postpartum depression symptomatology among Canadian women: a cross-sectional study. *BMC public health, 11*, 302. https://doi.org/10.1186/1471-2458-11-302

Leibenluft, E. (2010). Editorial: The Ties That Bind: Maternal-Infant Interactions and the Neural Circuitry of Postpartum Depression. *Am J Psychiatry, 167*(11).

Leuner, B., Fredericks, P. J., Nealer, C., & Albin-Brooks, C. (2014). Chronic gestational stress leads to depressive-like behavior and compromises medial prefrontal cortex structure and function during the postpartum period. *PLoS One, 9*(3). doi:http://dx.doi.org/10.1371/journal.pone.0089912

Jordan-Young, R. M. & Rumiati, R. I. (2011). Hardwired for Sexism? Approaches to Sex/Gender in Neuroscience. In Bluhm, R., Jacobson, A. J. & Maibom, H. L. (Eds.), *Neurofeminism: Issues at the Intersection of Feminist Theory and Cognitive Science.* Palgrave McMillan.

Lonstein, J. S. (2007). Regulation of anxiety during the postpartum period. *Frontiers in Neuroendocrinology, 28*(2-3). doi: http://doi.org/10.1016/j.yfrne.2007.05.002

Moses-Kolko, E. L., Horner, M. S., Phillips, M. L., Hipwell, A. E., & Swain, J. E. (2014). In search of neural endophenotypes of postpartum psychopathology and disrupted maternal caregiving. *Journal of neuroendocrinology, 26*(10), 665–684. https://doi.org/10.1111/jne.12183

Moses-Kolko, E. L., Perlman, S. B., Wisner, K. L., James, J., Saul, A. T., & Phillips, M. L. (2010). Abnormally reduced dorsomedial prefrontal cortical activity and effective connectivity with amygdala in response to negative emotional faces in postpartum depression. *The American journal of psychiatry, 167*(11), 1373–1380. https://doi.org/10.1176/appi.ajp.2010.09081235

Neumann, I. D. (2007). Oxytocin: The Neuropeptide of Love Reveals Some of its Secrets. *Cell Metabolism, 5*(4).

O'Hara, M. W., & McCabe, J. E. (2013). Postpartum Depression: Current Status and Future Directions. *Annual Review of Clinical Psychology, 9*. doi: http://doi.org/10.1146/annurev-clinpsy-050212-185612

Pearlstein, T., Howard, M., Salisbury, A., & Zlotnick, C. (2009). Postpartum depression. *American journal of obstetrics and gynecology, 200*(4), 357–364. https://doi.org/10.1016/j.ajog.2008.11.033

Silverman, M. E., Loudon, H., Safier, M., Protopopescu, X., Leiter, G., Liu, X., & Goldstein, M. (2007). Neural Dysfunction in Postpartum Depression: An fMRI Pilot Study. *CNS Spectrums, 12*, pp 853-862 doi: 10.1017/S1092852900015595

Strathearn, L., Fonagy, P., Amico, J., & Montague, P. R. (2009). Adult attachment predicts maternal brain and oxytocin response to infant cues. *Neuropsychopharmacology, 34*(13), 2655–2666. https://doi.org/10.1038/npp.2009.103

Wenzel, A., Haugen, E. N., Jackson, L. C., & Brendle, J. R. (2005). Anxiety symptoms and disorders at eight weeks postpartum. *Journal of anxiety disorders, 19*(3), 295–311. https://doi.org/10.1016/j.janxdis.2004.04.001

Winerman, L. (2005). Helping men to help themselves: Research aims to understand why men are less likely than women to seek mental health help, and what psychologists can do to change that. *Monitor on Psychology, 36*(6). Web.