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Postpartum Rebalancing: A Perspective on Women's Mental Health During Postpartum

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Abstract

For the last fifty years, psychology has been teasing apart that which is normal for women from that which constitutes a mental illness. Women's reproductive experience is no exception. With 75-80% of women experiencing mild to moderate Postpartum depression/ anxiety, perhaps the condition is the normal rebalancing of women's bodies, roles, and expectations in the postpartum period. Dramatic biological changes and societal expectations of motherhood burden the new mother with performance demands when she tends to attribute normal difficulties to her inadequacy. Therefore, those new mothers who could benefit from support, conversation, help, or treatment, are blocked by the stigma of a mental health diagnosis. Perhaps if we labeled the condition postpartum rebalancing, it would be more accurate, would lessen negative attribution, and make treatment-seeking easier.

The new label, Postpartum Rebalancing, is supported by studies on postpartum factors: genetics, hormones, and neurochemicals sleep, nutrition, the social, and psychological factors that impact new mothers. Attention is drawn to the empirical differences between Major Depressive Disorder and Postpartum Depression. Synthesizing this literature suggests that a new mother experiences a major upheaval requiring re-balancing every aspect of her life. The findings also suggest that a non-pejorative label for this difficult period of time in a normal woman's life could lead to the recognition of woman's normally demanding reproductive experiences, thereby, providing needed care without the stigma of mental illness. Just as pregnancy requires special attention, so should the postpartum period yet neither is an illness.

Keywords: post-partum depression and anxiety; PPD; perinatal depression; ante-natal depression; women and childbirth

Postpartum Rebalancing: A Perspective on Women's Mental Health During Postpartum

Pregnancy is not a disease and, yet, requires medical attention. Perhaps that is true for Postpartum Depression (PPD) as well. Depression impacts 5.5% of women globally. In the United States, 8 – 16% of reproductive age women have depression [1]. Yet more than 80% of new mothers experience depressive symptoms and 57% experience anxiety symptoms after giving birth [2]. Mild depressive symptoms in postpartum (often called baby blues) remediate within 4 to 6 weeks [3] and impact about 50% of new mothers [4]. Mild PPD peaks at 3 – 4 weeks postpartum, usually reducing to normal by 4 months [5]. However, some mild and most moderate depressive symptoms last longer and about 40% of mothers have moderate depressive symptoms which typically peak at 4 months and reduce to normal by 12 months. However, the variance is so great, that these peaks can emerge at any time during the first 12 months after the birth [3]. Worse yet, these symptoms can begin or advance to severe postpartum depression in 11 – 25% of new mothers, especially influenced by ethnicity, poverty, social support, age, and delivery problems.

The onset of depressive symptoms occurs during the last weeks of pregnancy in 33.4% of the women6and in the remaining 66% anytime from 2 weeks to 12 months. Although PPD is treatable, Ko et al. [1] found that of those who did have depressive symptoms, 65.9% remain undiagnosed and 49.6% of those who were diagnosed went untreated. However, a precise trajectory is unknown. Post-partum anxiety (PPA), seldom drawing the attention of professionals, has been reported at 54% lasting as long as 18 months [6] and little is known about the dynamics, symptoms, etiology, onset, and progression or impact of this postpartum anxiety.

In addition to the suffering of women who experience such symptoms, the impact of post-partum depression on infants has been well-documented and includes lower cognitive functioning, less creative play, less emotion regulation, with higher rates of anxiety and depression and more relationship problems in adolescence and adulthood [7] .Postpartum depressive and anxious symptoms also impact women's marriages [8] and their immune system [9]. In some populations, the leading cause of deaths among new mothers is suicide [10] during postpartum.
Reducing distress in the postpartum period and its impact is so significant that the National agenda for Healthy People 2020 and the International World Health Organization agendas for 2020 have targeted a reduction of PPD. The CDC began the PRAMS epidemiological study in 2004 to assess the ongoing progress in reaching the Healthy People goal. They too found that nearly 60% of women with depressive symptoms, go undetected and almost 50% of those go untreated in this large data-base [11]. With 3.3 million births in the US annually, it means that each year 2,640,000 women are suffering.

Despite the high number of women who suffer annually and the long-range impact of not treating PPD and PPA, research is limited in the psychological literature. Psychology in the United States has focused mainly on the impact of PPD on infants and child development, leaving investigations of the condition itself, mostly to the medical system as well as other countries, especially Australia, China, Holland, & the United Kingdom.

At this time, women's distress in the postpartum period is classified as either "Baby Blues" or "Postpartum Depression." These labels carry significant implications. If a woman is experiencing "baby blues" she attributes the distress to her own inadequacies as a mother [12] and powers through. If a woman is among the 11%- 25% who experience serious symptoms, she may or may not recognize it herself or be recognized and may or may not be treated [13] but again attribute the distress to her inadequacies and hide because of the stigma of being mentally ill.

Just as pregnancy is not a disease, yet requiring special care of the mother and infant, we submit that the postpartum period is a condition requiring special care of the mother, just as the infant, and is not a disease. We propose that this vulnerable period in a woman's life be called postpartum re-balancing. The purpose of this paper is to explain why the condition seen in almost all postpartum women can be labeled postpartum re-balancing. Postpartum re-balancing refers to the emotional, physical, and social experience of a new mother in the first year after birth. Postpartum re-balancing ranges from mild to moderate to severe with depressive and anxious symptoms. While some symptoms of postpartum mothers and depressive disorders remain similar, their etiology, pathways, and influencers are different. For example, 63.3% of women experience nausea during the first trimester [14], yet this symptom, which is due to normal estrogen flooding, is not categorized with other stomach problems. Exploring this range as a re-balancing complex of symptoms, somewhat different than regular depression considers the biological components, reduces the stigma of the symptoms and may lead to more women seeking and following through on treatment. Just as pregnancies have few to major complications on a continuum, so does postpartum re-balancing, yet despite the severity, never becomes labeled as a disease, but as complicated re-balancing.
There are four considerations that undergird our arguments: the prevalence, the hormonal and neurochemical changes, the

physical demands on a new mother, and societal expectations of motherhood. We will show how the impact of these factors in the postpartum period leads to feeling depressed, anxious, and overwhelmed with compromised self-efficacy and self-worth, yet are different from depressive disorders.

Epidemiological Differences

Prevalence rates. One would expect that a complex of symptoms labeled a disease would inherently be defined in part because of its deviance from normal functioning. With up to 85% of women suffering from symptoms in the postpartum period, this fact alone calls into question the deviance from normal functioning criteria. Postpartum rebalancing may be normal functioning for women, albeit on a continuum, and requiring attention and care.

Prevalence of Disparities. Considering the 8.7% of women in the general population diagnosed with routine depression and the 11%-25% diagnosed with postpartum depression, it is reasonable to believe that the same percentage of new mothers would demonstrate depression as in population samples, given that these epidemiological studies do not exclude women based on reproductive events. The fact that there is likely a number of women who fit the depression diagnosis prior to giving birth would be the same, the fact that the postpartum depression rate is 3 -10% higher, suggests that there is something happening that cannot be classified solely as depression. Postpartum re-balancing is more than routine depression, despite prior depression being a risk factor [13].

Biological Influencers of Postpartum Re-balancing

Hormonal and Neurochemical Changes

With the population-based incidence of Major Depressive Disorder (MDD) for women at 8.7% and Severe Post-partum Depression (PPD) at 11% - 25%, it is undeniable that women experience both to a much greater degree than men (5%). And the fact that differences emerge during prepuberty, and show distinct pathways at critical reproductive times, suggest that women's reproductive cycle influences the higher rates of depression. Evidence shows that reproductive hormones cross the blood-brain barrier and influence neural activity, some of which are governed by genetics. Yet many of the biological mechanisms (hormonal, genetic, neurochemical, and circuit-level changes) operate differently in the post-partum period than in Major Depressive Disorder (MDD). Indeed, Payne and Maguire [15] in search of biomarkers for PPD conducted a review of neurobiological pathways research leading to several conclusions: the mechanisms are highly interrelated, there is a high probability that numerous mediating mechanisms intervene, and there is biological and environmental epigenesis involved.

Reproductive hormone levels are obviously implicated in anything related to pregnancy and childbirth. However, studies have demonstrated inconsistent results regarding hormone levels and PPD. Estrogen and progesterone flood the system in pregnant women and drop precipitously within a few hours to days of giving birth [16]. When testing the actual levels of hormones, only those women with a history of PPD had different levels than those with no history, but women who had depressive symptoms for the first time had no significant differences in actual levels of these hormones [17]. Both the introduction of these hormones or their withdrawal triggered symptoms in women with a depressive history but not in controls [18]. While actual levels were not indicated, perhaps the swiftness of the drop triggered depressive symptoms. This idea was also explored, yet there are no consistent findings implicating the swiftness of the drop [19]. However, there is evidence that these women may be differentially sensitive to the effects of these hormones when withdrawal from estradiol and progesterone increased their depressive symptoms [19]. There is some evidence that oxytocin levels predicted PPD and severity [20] and some evidence that oxytocin levels only predicted PPD if the woman was previously diagnosed with MDD [21]. While these studies may establish some support for the sensitivity to dramatic hormone drops hypothesis [17], this does not account for the 70% of mild to moderate depression and anxiety seen in most mothers. The results on studies of reproductive hormones do not show a direct relationship and are inconclusive at this point yet may involve other mechanisms indirectly affected by the estradiol and progesterone levels.

Stress Hormones. Due to the fact that women with PPD had three times the rates of daily stressors [22] and women who have experienced multiple adverse life events have three times the rates of PPD [23], stress hormones were also researched as possible biomarkers. Placental corticotropin-releasing hormone was shown to be a strong predictor in some studies [24] but showed an inverse relationship in others [25] and is not a useful biomarker. In MDD, the pathways have been clearly identified and consistent. However, in PPD, there is greater complexity to the relationship of the Cortisol hormones and is an impact on the HP Axis [26].

Alloprogesterone is a neurosteroid that modulates GABA receptors in non-reproductive related depression [18] and fluctuates independently of the reproductive cycle. In a study by Deligiannidis et al [27], women with PPD and normal peripartum women were compared on neuroactive steroids (NAS), cortical GABA, and functional connectivity using fMRI. The results indicated differences in GABA levels and NAS between women with PPD and healthy controls. Interestingly, the parts of the brain affected were those related to some of the psychological symptoms of PPD and PPA. For example, the posterior cingular cortex is related to self-related mental representations, internally directed cognitions [28] and perspective taking [29] linking neurochemical activity to how people self-evaluate. Emotion perception also occurs in this area [30] and women with PPD or PPA tend to self-evaluate negatively. This area of the brain is at the nexus of multiple intrinsic connectivity networks. Plasma GABA concentration increases in the perinatal period and is involved in maternal behaviors. However, the Gaba increase may also enhance the connectivity changes that result in a disturbance to the self-appraisal system in PPD. Seventy percent of the women had co-morbid anxiety and a wider range of GABA concentrations than healthy controls. These studies demonstrate the relationships between neurosteroids related to the reproductive cycle and the cognitions and feelings seen in PPD.

Allopregnanolone is known for having anti-depressant effects [31]. Allopregnanolone increases during pregnancy and drops precipitously after birth [32]. Allopregnanolone decreases correlate with depression and increase when an antidepressant is administered [31]. While there was a negative correlation with PPD in some studies [32], other studies found no decrease in allopregnanolone in women with PPD [27]. Therefore, the role of this stress hormone is unclear but operates differently for PPD than for MDD.

Higher β-endorphin [24], lower platelet serotonin levels [33], increased monoamine oxidase-A density [24] low omega-3 levels [35] and lower Vitamin D [36] have all been associated with greater PPD. However, all these are one-study findings that have yet to be replicated. Stress hormones, while playing a relatively important role in MDD, demonstrate far less promising roles in PPD. Overall, when comparing depressed women with post-partum depression, many of the hormonal and neural activities that coincide with MDD, are not relevant in PPD [19,17]. Presumably, the introduction to a new baby is a stressful event even to adoptive mothers. Mott, Schiller, Richards, O'Hara, & Stuart [37] compared adoptive mothers with birth mothers, resulting in the anticipated 8% of depressed women in the adoptive mothers while the birth mothers' rate was 16%, again suggesting that stress did not increase depression in the postpartum period. These studies suggest that stress hormones are unrelated in the development of PPD, yet, are implicated for other depressive disorders.

Neurochemical Biomarkers

Neuroendocrine Nexis. The onset of PPD may correspond to the dramatic change in hormone levels suggesting that the fluctuations may be more important than actual levels [17]. As shown above, absolute hormone levels do not differ with PPD but sensitivity to the fluctuations may differ [16] but only in those with prior history of PPD. Estrogen signaling (through the HPA) regulates reproductive and stress hormones [38]. Estrogen treatment decreases depressive symptoms during postpartuM [39] and reduces the risk of PPD [40]. Progesterone worsened depression in some studies and decreased a recurrence of PPD in otherS [41]. Oxytocin, regulating emotion, social interaction, stress, and mother-infant relationships [42] and Prolactin, regulating breastfeeding have led to inconclusive results as well. Stress hormones, an obvious risk factor, have been found to accompany altered levels of cortisol, ACTH, and CRH in patients with PPD [16]. These neurochemicals are associated with activity in the HPA axis, yet although they may be triggered, their involvement in the HPA axis in PPD is unproven [25]. Rat studies have shown that it is the inability to suppress the hormonal activation of the HPA axis during the postpartum period that leads to depressive behaviors [43]. The HPA axis responds to environmental stress, possibly supporting a focus on pathways leading to PPD that are affected by the environment.

Neurosteroids also exert effects in the brain related to depression and thought to be mediated by the GABAaRs receptors [31]. A diminished level of allopregnanolone increases the risk of PPD [32] probably triggered by a gene involved in its function [31] and the inability to suppress these hormones [44]. The story is evolving to focus more on the regulatory functioning of pathways along with the differences between PPD and routine depression.

Neurotransmitters. GABA, the primary inhibitory neurotransmitter, levels have been shown to be inversely correlated with depression [27], yet studies on GABA signaling are not definitive for PPD. Glutamate is the primary excitatory neurotransmitter. Levels of glutamate are higher in PPD in the medial prefrontal cortex [45], yet lower in the dorsolateral cortex in PPD women compared to healthy controls [46]. However, this area of study is too new to conclude. Serotonin signaling plays a role in

maternal behaviors but remains unresolved in its role in depressive symptoms in PPD [47]. Dopamine signaling is implicated in maternal behaviors and depression-like behaviors in animal studieS [48,49]. Neurotransmitters, specifically signaling and inhibitory functions regarding maternal behaviors and depressive symptoms are implicated, yet no conclusions are drawn except that the evidence is showing a more complex picture in PPD than with MDD.

Changes in Neuro-pro-inflammatory and Neuro-anti-inflammatory responses occur throughout pregnancy and postpartum and are emerging as possible influential factors [50]. Altered immune functioning has been associated with depressive symptoms. Levels of IL-6 and IL-1βare positively correlated with PPD [51] and T-cell count has been negatively correlated with depressive symptoms in the postpartum period [52]. Other studies did not find these results but did see an association between cytokines and previous adverse life events [53], linking immune function with a well-known risk factor for PPD. Degradation of tryptophan, which regulates serotonin production, was found in women with PPD [54]. Despite conflicting findings, immune system functioning may be relevant yet inconclusive in PPD.

fMRI studies assessed neuron activity during resting state, performing tasks, and responding to pictures of their infant in women with PPD. The amygdala, prefrontal cortex, cingulate cortex, and insula alterations occurred in PPD women [55,55,56]. Deficits in functioning in these areas coincided with differences in processing emotionally-relevant stimuli in women with PPD compared to those without. However, women with PPD did not demonstrate the decreased structural white matter connectivity seen in MDD [58]. Furthermore, alterations in network oscillations have been implicated in MDD but not in PPD [59]. These studies indicate that neural activity in women with PPD may contribute to depression, yet while altered, do not reflect the pattern seen in MDD. Other neural network studies have identified those involved in mediating maternal behavior in the peripartum period.

Genetics

The fact that PPD involves such a broad spectrum of mechanisms, neurological, cognitive, behavioral, hormonal, and environmental, it is likely that genetics play a role. Genetic involvement in PPD is only beginning to be explored. Twin studies have led to some evidence of genetic influence in PPD [60] and certain genes have been identified. The genes targeted for the study were those involved in MDD, and of those, only estrogen signaling and polymorphisms on the hypothalamic-pituitary-adrenal axis (HPA) demonstrated some association with PPD. The estrogen receptor alpha gene (ESDR-1) and mutations in the serotonin transporter (5-HTT) appear in early PPD but only in women who have experienced adverse life events, again suggesting an interaction between genes and the environment in the development of PPD [61]. Monoamine oxidase plays a role in dopamine, norepinephrine, and serotonin. Another polymorphism in genetic coding, Catechol-O-methyltransferase (COMT) is predictive of intermediate stage PPD [62], yet only an inconsistent predictor of MDD [63]. MAOA gene has been associated with PPD [64] yet again only in women with adverse life experiences, again supporting the genetic, environmental link, posing two risk factors for PPD [65].

The specificity of genetic influence is even greater than anticipated. When examining predictive polymorphisms, different regions of the genetic code were associated with either PPD or with depression during pregnancy but not both [66]. These gene expressions are regulated in different areas of the brain [67]. A single nucleotide (SNP) in the gene encoding for oxytocin was predictive of both breastfeeding variation and PPD, yet the oxytocin receptor was predictive only for PPD [68]. As studies shed light on the HPA axis and untangle the genes and environment contributions to PPD, it is becoming clearer that the interaction of environment and biology in PPD is occurring at a genetic level with mutations reflecting personal histories. Studies also suggest that these associations are related to specific time frames during the postpartum period, suggesting that the timing for screening and assessments must be carefully chosen.

Genetics may influence PPD through stress hormone production as well. No one can deny that having a new baby is stressful. Both twin and family studies support a moderate genetic basis [69]. A recent study [70] found that father support interacted with the OXTR genotype in the mother and was a moderating factor in the development of PPD. OXTR has been linked with PPD symptoms in other studies [71]. There are very few studies that explore this relationship between psychological stress, and genetics while specific risk factors are largely unknown [72].

However, again the HPA, hypothalamic-pituitary-adrenal axis is implicated as the regulator of both exogenous and endogenous stress. Corticotropin-releasing hormone (CRH) releases adrenocorticotropic hormone (ACTH) which then triggers the release of cortisol. While cortisol levels rise and fall in a predictable daily cycle they otherwise remain stable until

a stressor is introduced. However, in pregnancy, this rise and fall peaks two times higher than in non-pregnant women [73], then reduces to baseline within a few weeks of delivery. However, altered HPA axis correlates have been associated with PPD [74] both in mid-pregnancy and in postpartum. There are several genes influencing this process, specifically one study tested the association between PPD and the Haplotype-tagging single nucleotide polymorphism in key HPA axis geneS [75]. Findings show that SNPs are associated with stress hormones during pregnancy. Findings also demonstrated a difference between peripartum and postpartum depression, identifying distinct phenotypes for PPD. Although this research is in its infancy, we may conclude that a mechanism exists between genetics and reproductive depression, differently at different times during the cycles, thus distinguishing it from MDD.

Sleep as Influencer

While women typically obtain adequate sleep during most of their pregnancy, sleep deteriorates in the third trimester when their bodies are taxed with the size, weight, and movement of the growing fetus. Of course, infant demands interfere greatly with sleep in the postpartum period. Misaligned circadian rhythms are also a feature of mood disorders [76]. The sleep cycle is strongly influenced by melatonin. Melatonin is released in the prefrontal cortex and triggers arousal and temperature. The pineal gland operates as a time cue [77] that triggers the secretion of melatonin in the blood in reaction to the light coming into the eyes. Melatonin binds in the brain to MT1 receptors and acts via the G coupled protein pathway that enhances the binding of GABA to GABA receptors. Both the amount of melatonin and melatonin timing decreased in antepartum depression and increased in postpartum depression. Adjusting the timing of melatonin onset and sleep onset in antenatal women reduced depression in 40-60% of the pregnant women but in postpartum women, depression increased. Exactly the opposite response happened when the timing of postpartum melatonin to aid sleep onset was manipulated to reduce depression. These findings suggest a) the important link between depression and sleep and b) that postpartum depression responds differently to sleep and sleep aids than in routine depression [78].

Sleep patterns are significantly interrupted in postpartum and lack of sleep has consequences that are typical in this period, such as the "Mommy brain" deficits in concentration. Melatonin is produced in the ovary and placenta [79]. Melatonin levels are increased during delivery to induce the uterine contractability then decreases within days of the birth [80]. Women who did not give birth were compared to women with newborns. Both were awakened three times/ night for 30 minutes each. There were no differences in their mood, REM sleep or melatonin after 1 night, however, after two weeks, melatonin had decreased even further in mothers of infants and not in other women [81], but the relationship between melatonin and depression was bi-directional, not causal as studies have shown in routine depression [82]. Lighting decreases melatonin as well. Women often use TV and cell phones during nightly breast feeding [83], not realizing that the blue light suppresses melatonin [84]. These studies suggest that melatonin plays a different role in the sleep cycles of new mothers than in routine depression.

Fatigue and depression are highly correlated, however, Wilson, Wynter, Fisher, & Bei [85] found that in a sample of women seeking support for difficult infant behaviors, depression and fatigue were separate constructs, despite both being predicted by sleep efficiency. This may indicate a need to assess fatigue both as a diagnostic predictor of depression and as independent of depression.

Nutrition as an influencer

Pregnant and lactating women have higher demands for nutrition and are regularly prescribed vitamin supplements. Studies are scarce on how nutrition impacts mental health, specifically in postpartum women, who may be reducing their supplements at this time. Vitamin D has been implicated in depression. Vitamin D consists of a complex of secosteroid hormones produced through sunlight exposure, fungi, or animal tissue, but has limited sources through diets. It is metabolized in the liver and forms (25- hydroxy vitamin D in the blood (25OHD). Research has shown an association during pregnancy and post-partum between 25 OHD and depressive symptoms [86,87]. Multiple studies across countries and ethnicities have shown that deficiencies in 25 OHD in the first trimester predicts depression in 2nd and 3rd trimesters, even in locations with high sun exposure [88]. In a review of the antenatal period, depression was also associated with a deficiency in Vitamin D [89]. An RCT [90] demonstrated improvement of depression as the OHD 25 improved by the end of pregnancy and 2 months postpartum. Lamb, et al. [91] found that OHD 25 deficiency at third trimester predicted postpartum depression at 10 weeks. Vitamin D impacts both the immune system and the Hypothalamic-pituitary-adrenal axis that exert a regulatory role in depression [92]. Although Vitamin D has been implicated in other forms of depression as well, it is likely that Vitamin D deficiency is exacerbated if a woman is housebound during the early months of her infant's life [93].

Taken together these studies indicated that while some of the same stress and sleep hormones and the neurochemicals are similar in both PPD and MDD, their pathways are quite different. Responses to neurochemical and hormonal shifts operate in demonstrably different ways in MDD and in PPD. Medical interventions also affect women differently in the two diagnoses. There is an overlay of dramatic hormonal shifts and sleep deprivation occurring in PPD that is distinct from depression. Stress hormones reflect both the woman's state in the postpartum period as well as her past environmental experiences. Genetic studies, although in its infancy, are pointing towards differences between depression and postpartum depression or postpartum re-balancing. There is a growing body of evidence for significant biological distinctions between depression and the postpartum condition, which supports the notion that PPD is different from other depressive disorders. We suggest that Postpartum Rebalancing would be more accurate and would encompass the mild and moderate symptomatology seen in the majority of new mothers.

Environmental influences on motherhood as an exogenous factor

The above studies demonstrate that there is an interaction of the biological and environmental, even at the genetic level. Not only does the biology affect women's well-being in postpartum, but external demands and influences pose enormous pressure and stress on mothers at a time when they need gentle care. Societal pressures include the expectations of perfection in mothers, the barrage of media presentations of things that can go wrong with a baby, the lack of support or lack of quality support, and isolation. Amidst all this, there is a powerful stigma involved in mothers who are struggling. This section will explore how the myths of motherhood, the isolation, the stigma, and other psycho socio cultural factors distinguish PPD from other depressive disorders.

Myths about motherhood abound in every culture, but most centers on a belief that mothers should be 'perfect' in multiple ways and mothers expect themselves to live up to this perfection. This perfection is unattainable, of course, yet many new mothers use this standard to self-evaluate. One qualitative study found that as mothers with PPD recovered, they shifted their standard to balance their needs and the child's needs. This study concluded that healing occurs through de-idealizing the myths [94]. Another study demonstrated the loss of identity as she centered her life on childcare in an effort to attain the status of perfection believed necessary to care for a baby. Her self-evaluation shifted from a more nuanced and holistic appraisal of whom she was focusing solely on her ability as a mother [95]. Mothers with infants who suffered colic and cried more were more likely to have PPD [96]. Women's actual subjective experiences presented a very different image of motherhood than their perception of the norm97. Women are not born into natural mothering and the ability to care for their infant [98]. These myths persist even in the face of more progressive ideas [99] and this conflict between the actual experience and perceived norm has been shown to manifest in postpartum depression [100]. In the face of these myths and to the degree a woman subscribes to these myths as truths, she attributes inadequacies to herself, which is a major component of postpartum depression [101]. A new mother, however, is not the mere recipient of culturally imposed myths but attempts to resolve the myth versus reality conflict. Social support is an enormously important factor in reducing PPD [70]. With social support, she is more likely to become agentic in her self-evaluation as a mother, shore up her identity, and reduce depression and anxiety.

Isolation influences new mothers. There is ample evidence that social support in the form of intimate connection and practical assistance counteracts post-partum depressive symptoms and anxiety [102]. Women typically work before giving birth and many of their friendships are with coworkers. Maternity leave disrupts their support systems. Work migration separates extended families who may live at a distance at a time when new mothers must adjust to the isolation of being housebound with a new infant.

The second form of isolation is the one experienced even if people are nearby and that is when a woman feels the need to hide what she is feeling. Emotional isolation and withdrawal accompany stigma, guilt, and shame leading to more depressive symptoms to hide. Women thrive in connection with others and when the connection is diminished, the loneliness can be acute [103]. Although spouses/ partners are encouraged to help, they have little direction on how to help. Lamaze, LaLeche, and infant-focused parenting classes do not address specific things that Dads can do to help. When distress and fatigue enter the picture, couple communication suffers leaving the new father in a quandary about how to support the new mother, forming a wedge between the couple [104]. Marriages that are arranged show no difference from those based on other ways of choosing one's spouse [105] in the amount of support provided and in PPD.

Furthermore, certain cultures restrict women's activities and confine her to the house for a month. In those mothers who accept this tradition, there is less PPD [106] but in other

mothers, this tradition correlates with a higher risk of PPD [107]. Other cultures require a new mother to stay with her husband's family for the first 100 days of the child's life. She has the choice to remain there or leave her child with them. Support for the new mother may or may not be adequate.

Psychosocial influencers

Psychiatric Diagnoses. Diagnoses of MDD or a family history of MDD are predictive of PPD and may manifest during pregnancy, especially in the last month prior to delivery [4]. Women previously diagnosed with bipolar require special attention and should be followed closely during the pregnancy and first year of a child's life, especially since she must make some critical decisions about her medication during pregnancy [108]. While these prior diagnoses were the highest predictors of PPD across studies, they still only accounted for 8% of the variance [109]. As expected, having a new baby is a life stressor and women with these vulnerabilities are susceptible to stronger reactions to stressful events, yet studies on depression associated with other reproductive events, such as Pre-menstrual Dysphoric Disorder, mood instability secondary to oral contraceptives, or puberty are inconsistent in their findings, thus there is no conclusive evidence of a direct relationship [110].

Psychological Features. The research on psychological factors is notably sparse. Negative body image [109] and adverse life events have been repeatedly shown to accompany PPD [111] specifically if a woman suffered from physical, emotional, or sexual abuse [112]. Perceived stress [113], poor quality of life [110], the intention of going back to work [114], unplanned or unwanted pregnancy, and negative attitude towards having a child [115] are risk factors. However, the psychological impact and dynamics of these associations have not been tested and do not explain these associations. However, women who are more resilient, perceive less stress, and have lower cortisol levels regardless of some of this factors [116].

Socio-environmental features. In developed countries, SES was not associated with PPD but in developing countries, low income, low education, and low status were associated with increased PPD [117]. Younger age was significant in developing PPD in developed countries (<25 had 5 times higher risk, [118] but not in developing countries [119]. The section on genetics in this paper demonstrated that certain environmental situations led to gene mutations but little is known about these interactions.

Social support has consistently been a protective factor against PPD [120] in all countries and its importance cannot be underestimated. The stability of the union was a prominent protective factor [121]. If the baby's father was the main source of support, there was an interaction with oxytocin genotype that reduced the risk of PPD [70]. Polygamous marriage was found to be a risk factor [106]. Migration status was associated with a higher risk of PPD [122]. Domestic violence was found to be a factor [40] and, unfortunately, pregnant and lactating women are more likely to be a victim of domestic violence and partner abuse ranging from 12.9% in Ireland, 17% in the UK, 18% in India, and 27.7% in Uganda [123]. While many factors cross borders, there are also cultural factors that uniquely affect PPD. In countries that have strong social preferences for a boy, those mothers delivering girls had a greater risk of developing PPD (this relationship was culture-bound with China being strongest, and Turkey being mixed). More broadly, women in communities that lack knowledge about child care [124] and breast feeding [125] have a higher incidence of PPD.

Stigma. Routinely screening mothers for PPD serves to identify those in serious need. However, in a study of 6,437 mothers, only 6.3% were identified using the Edinborough, a gold standard assessment in this area, compared to 11% identified in epidemiological studies world-wide, indicating serious under-reporting in screening.

Mothers who suffer do not recognize their distress as PPD and often see the problem as their own inadequacies as a mother [126]. Many do not seek treatment [127]. In another study, the worse mothers felt, the less self-care they engaged in, believing that family must come first [129]. Mothers feared that people would think they are "crazy" [126]. The stigma prevented other mothers from discussing depressive symptoms due to guilt and shame [128]. Mothers also feared a negative impact on their child and even that protective services might take their child [129]. Cost of services and few providers with expertise contributed to making treatment seeking difficult [127]. In addition, making appointments on time in the first three months is extremely hard to manage. Women also do not speak with others about what is happening to them. Perhaps identifying this condition with depression, a mental illness, is too stigmatizing for women to obtain help. Many women who experience PPD and PPA, especially mild and moderate levels, have always been considered as sane, well-balanced, effective members of society [130]. Taking a non-pathologizing approach may reduce stigma [130], increase women's awareness, encourage discussion, and lead more women to treatment. Research is needed on overcom-

ing the obstacles to treatment of PPD, especially public campaigns to reduce stigma, disassociating PPD from the sensationalized and isolated stories about mothers killing their babies, and relabeling the condition as Postpartum Rebalancing.

These three major psychosocial influencers, the myth of motherhood, the isolation, and the stigma, are all societally-induced stressors playing havoc on a woman's psyche during this vulnerable time. Yet, she continues to blame herself without realizing how much these influencers manipulate her self-efficacy at a time when she is prone to more emotional lability. These contributors to the postpartum condition require re-balancing how she perceives herself in the face of a new, very responsible, role in her life. None of these are inherently within the woman's internal state and that may be why nearly 85% of women have distress during the post-partum period. The impact of these environmental factors does not constitute a mental illness.

Limitations in the Literature

In both meta-analyses and reviews on PPD, studies in 40 countries have reported 0% - 82% for clinical postpartum depression [131]. While the prevalence is certainly high, research has failed to be consistent in reporting, especially when examined across countries. With such variation in prevalence rates, the instruments used to measure these rates could be questioned. Most studies used scales designed to measure either MDD or PPD, yet the scales used seldom corresponded to DSM criteria to diagnose or identify PPD. Various cut-off scores were used depending on the researcher, the country, and the culture. Most of these scales were validated using the SCID, yet correlations between the SCID and these instruments are low. The two instruments considered the gold standard for screening PPD, the EPDS, and PDSS, contain some aspects of PPD not in the DSM. Another problem emerges from the fact that the majority of biological studies were conducted on already diagnosed women, usually using the SCID. At this point, we have promising but not enough evidence yet for certain biomarkers. While the search for objective identifiers of PPD goes on, on a qualitative level, one can speak to almost any woman who has given birth and find the subjective experience with PPD and PPA, yet never defined it as such, never spoke about it, and never sought help [95].

Implications for screening &treatment

Screening. To meet the goals of reducing postpartum depression in mothers by 2020 the CDC recommended improved screening, referral, and treatment [11]. PPD is identifiable, preventable, and treatable. Several states have recently passed legislation for Ob/Gyn physicians to screen all new mothers. Insurers are also mandated in some states to pay for PPD services [132]. However, there are problems with screening in that any device used has many false negatives even for severe PPD [133]. Many physicians use an unvalidated two question process [132] and only 2.5% of their patients are referred on average.

Boyd, Mogul, Newman, and Coyne [134] identified several major barriers against referring the screened positive mothers to treatment. On the clients' side, the stigma of mental health disorders and logistical impediments for mothers restricted clients from seeking help. More accommodation of services like a home visit or free treatments would have a positive impact. Systemically, few mobile or crisis unit services are available, clinics have long waitlists, and lack of resources and staff in agencies causes more challenges in successfully referring the client to get services. Office-based screening usually results in identifying about 6% of women as having diagnosable PPD. However, this rate falls significantly short of the actual population rates, resulting in about 11% of false negatives and does little to help women in the mild to moderate range. Of those identified as high moderate to severe, only 55% of the women sought treatment when referred. The rate of follow-through depended on the level of integration between the referral source and the medical practice. Only 32% of pediatrician referrals reached treatment, while76% of ob/GYN referrals were treated but only to an onsite therapist [135]. While convenience contributed, the "warm handoff" connection is likely to have reduced the stigma. Efforts to improve screening beyond these self-report instruments are currently underway using machine learning (ML) to identify utilization markers in women's use of social media that identify and follow the trajectory of postpartum rebalancing.

To date, the treatments for postpartum depression have used those shown to be effective for MDD, both pharmacotherapy and CBT. Many pregnant and lactating women do not want to use medications for fear it will affect their infants [136]. The only drug developed specifically for PPD is Brexalonone, which requires an overnight hospital stay to treat.

Based on the idea that women go through a vulnerable period of re-balancing their bodies, their social sphere, and their identities in the postpartum period, treatments should target this view of their condition. More importantly, however, rather than taking the default position of using CBT to treat as any other depression, new approaches are necessary that take into consideration the re-balancing of a woman's system that is a major influence of PPD. Also, PPD is an interactive problem in that the

mother's condition is associated with the baby's condition [4] and as such, treatment should center on this interaction as well. Practitioners could learn about Postpartum Depression and Anxiety and understand the full scope of biological, psychological, and social influencers that are affecting new mothers. Technology-based solutions are becoming available that accommodate a new mother's difficulty in scheduling and overcoming the stigma.

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