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The association between breastfeeding, the stress response, inflammation, and postpartum depression during the postpartum period: Prospective cohort study

Sukhee Ahn [a], Elizabeth J. Corwin [b,*]

[a] College of Nursing, Chungnam National University, Daejeon, Republic of Korea
[b] Nell Hodgson Woodruff School of Nursing, Emory University, 1520 Clifton Road NE, Room 244, Atlanta, GA 30322, United States

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ABSTRACT

Background: Research suggests that exclusive breastfeeding may have a stress-protective role in postpartum depression; however, less is known about the underlying mechanisms by which this protection may occur or whether the protective relationship holds for women who mix breast and bottle feeding.
Objectives: To examine patterns of the stress response, inflammation, and depressive symptoms among women predominantly breastfeeding or bottle feeding their infants at 6 months postpartum.
Design: A part of a larger longitudinal study across 6 months postpartum investigating the psychoneuroimmunology (PNI) of postpartum depression.
Setting: Prenatal clinics and community.
Participants: One hundred nineteen postpartum women who met inclusion/exclusion criteria and followed up from the prenatal period to postpartum 6 months.
Methods: Data were collected during seven home visits occurring during the 3rd trimester (weeks 32–36) and on postpartum days 7 and 14, months 1, 2, 3, and 6. Women completed stress and depression surveys and provided blood for pro- (IL-1β, IL-6, IL-8, TNF-a, IFN-γ) and anti-inflammatory (IL-10) cytokines, and collected saliva for diurnal cortisol.
Results: Self-report of predominant breastfeeding during 6 months postpartum ranged from 91.9% at day 7 to 70.6% at month 6 postpartum. There were no associations between the pattern of feeding and depressive symptoms. Biological differences, however, existed between the groups, with levels of salivary cortisol at 8 AM and 8:30 AM at month 6 higher and levels of IL-6 at month 6 lower in women who primarily breastfed compared to those who primarily bottle fed their infants after controlling for confounding variables.
Conclusions: Breastfeeding was not related to postpartum depression however differences in stress and inflammatory markers are apparent at month 6 postpartum.

* Corresponding author. Tel.: +1 404 712 9805; fax: +1 404 712 6945.
E-mail addresses: sukheeahn@cnu.ac.kr (S. Ahn), ejcorwi@emory.edu (E.J. Corwin).

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What is already known about the topic?

- Research suggests that exclusive breastfeeding may have a stress-protective role in postpartum depression.
- There is no research available to date that uses a PNI perspective to explore the effect of predominantly breastfeeding on postpartum depression, stress, and inflammatory response in women at 6 months postpartum.

What this paper adds

- Predominantly breastfeeding does not impact depressive symptoms or perceived stress compared to predominantly bottle feeding.
- Our data do identify psychoneuroimmune effects of predominantly breastfeeding versus bottle feeding, including decreased level of the pro-inflammatory marker IL-6 and increased levels of salivary cortisol at 8 AM and 8:30 AM that may have implications for health.

1. Introduction

Symptoms of depression are prevalent during the perinatal period (Beck, 2008; Lee et al., 2007) and may have a significant impact on maternal and neonatal outcomes (Barker et al., 2011; Field et al., 2010a). Elevated depressive symptoms are estimated to occur in up to 37.1% of women during pregnancy (Lee et al., 2007), while in the postpartum period, as reviewed by Beck (2008), estimates of prevalence vary widely from 11% in US population (Rychnovsky and Beck, 2006) to 42.6% in Taiwanese mothers (Chen et al., 2007) depending on data collection time points, the population, and the instruments used. Regardless of prevalence, postpartum depression (PPD) has adverse consequences for both mother and child that may last a lifetime. Untreated PPD poses a serious threat to the emotional well-being of the mother and her confidence and capacity to care for her infant (Goodman, 2007; Lovejoy et al., 2000) including her success in maternal role attainment (Cooke et al., 2007). In addition, children of depressed mothers are at risk for delays in growth and development and reduced cognitive, neuropsychological, social and emotional skills across childhood and into adolescence (Feldman and Eidelman, 2009; Murray et al., 2011; Verbeek et al., 2012). Given the profound disruptive influences of depression for both mother and child, the detection and early treatment of vulnerable women at risk for PPD is essential.

While psychosocial risks for depression have been recognized for some time (Beck, 2003, 2008), accumulating evidence also suggests biological risk factors. For example, research suggests that the volatility in reproductive and hypothalamic–pituitary–adrenal (HPA) axis hormones (Glynn et al., 2013; Yim et al., 2009) that occur toward the end of pregnancy and in the early postpartum period may contribute to the development of PPD as well, at least in some women (Bloch et al., 2000). More recently, researchers interested in psychoneuroimmunology (PNI) have linked physical and psychological stressors that both stimulate the HPA axis response (Lommatzsch et al., 2006; Mastorakos and Ilias, 2003) and increase the production of pro-inflammatory cytokines (Raison et al., 2006) with an increased risk of depression in non-pregnant, non-postpartum populations (Schiepers et al., 2005; Wilson and Warise, 2008). Given that both stress and inflammation accompany labor and delivery and the important role of inflammation in postpartum healing (Dunn et al., in press), researchers have also hypothesized that PPD may likewise have a PNI etiology (Corwin et al., 2008; Corwin and Pajer, 2008; Glynn et al., 2013; Groer and Morgan, 2007). The literature remains mixed, however, as to whether higher or lower levels of cortisol or pro-inflammatory markers (Osborne and Monk, 2013) do indeed associate with depressive symptoms in new mothers. For example, while studies suggest that postpartum depressed women demonstrate a down regulated HPA axis, with lower levels of salivary cortisol compared to non-depressed mothers (55% breastfeeding) during postpartum 4–6 weeks (Groer and Morgan, 2007), other reports identify higher levels of serum cortisol in the group of depressed mothers (Lommatzsch et al., 2006). Depressed mothers also have been reported to have lower serum levels of the pro-inflammatory marker interferon-gamma (IFN-γ) and the ratio of pro-/anti-inflammatory levels (IFN-γ/IL-10), suggesting possible depressed cellular immunity (Groer and Morgan, 2007). Added to this, a very recent publication from our group provides evidence that neither inflammation nor cortisol alone explains PPD risk, but instead dysregulation in the bidirectional feedback circuit between the HPA axis and the production of pro-inflammatory cytokines after delivery underlies PPD symptom development (Corwin et al., 2015).

At the same time, given the impact of breastfeeding on both stress and immunity (Kendall-Tackett, 2007), current literature also suggests that breastfeeding may have an important role to play in the mental health of new mothers, also within the PNI framework. Lactation has been identified as a variable affecting the pattern of cortisol output, and suckling has been reported to provide a neural stimulus that dampens the HPA axis circadian rhythm and reduces the stress response (Groer and Davis, 2006; Hahn-Holbrook et al., 2013; Slattery and Neumann, 2008; Tu et al., 2006), although other studies are conflicting (Dennis and McQueen, 2007; Taylor et al., 2009). These differences are perhaps related to different definitions of what encompasses breastfeeding: i.e., exclusive breastfeeding, mixed feeding, or breastfeeding only at night.

The recent 2014 "Breastfeeding Report Card" from the Centers for Disease Control concludes that most women combine breast and bottle feeding (Center for Disease Control, 2014). Specifically, in the United States, the rate of breastfeeding at 6 months was reported as 60.6%, while the rate of exclusive breastfeeding through 6 months was reported as only 25.5%. Little is known as to whether the protective effects of breastfeeding on mood hold for women who both breast and bottle feed, and no information is available on PNI function in this group. Moreover, few prospective studies exist that evaluate the association between breastfeeding and level of depression over time or consider how it changes, and again, none in