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Postpartum Stress: Current Concepts and the Possible Protective Role of Breastfeeding

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Objective: To review stress during the postpartum and the research supporting that a unique, protective biology exists in breastfeeding mothers that may reduce reactivity to stress.

Data Sources: Publications from nursing and biomedical literature.

Study Selection: Studies reviewed were those that have contributed to concurrent conceptualizations of postpartum stress. Additionally, studies with sufficient participants were analyzed for common findings. Animal literature was reviewed for studies on the stress response in lactating and nonlactating animal models.

Data Extraction: Stress during the postpartum may be conceptualized as physical, intrapersonal, and interpersonal. Animal data and a few recent human studies suggest that the neuroendocrinology of the lactating mother may down-regulate the magnitude of the stress response.

Data Synthesis: A diminished stress response may serve to protect the breastfeeding maternal-infant dyad from environmental stimuli and to direct the physiology of the mother toward milk production, energy conservation, and nurturance.

Conclusion: Nurses understand the benefits of breastfeeding for optimal infant health, but new research suggests that maternal health may also be benefited in a biologic and as yet unexplored way. *JOGNN, 31,* 411–417; 2002.

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The purpose of this review is to examine the stressors that occur in the postpartum period and to explore a possible stress-protecting role of breastfeeding. Research suggests that lactational physiology may produce a diminution of the biologic stress response and even the psychologic perceptions of stress.

Although for most parents there is joy and satisfaction in the birth of a new baby, many situations or events occur that are stressors, with the potential for taxing parents' ability to function. Smith (1989) compared primiparae to multiparae in terms of postpartum adjustments that may lead to stress. For primiparae, the most frequent reported stressors were baby feeding, fatigue, breast soreness, return of the figure, limiting visitors, regulating demands, growth and development, interpreting behavior, physical care of the baby, and labor and delivery. Multiparae reported major stressors of fatigue, regulating demands, emotional tension, sibling jealousy, and the labor and delivery experience.

Stressors

Horowitz and Damato (1999) conducted a triangulated study on postpartum stress. Ninety-five women were studied at Week 6 postpartum. Mothers described experiences that they felt were stressful, which the researchers found fell into four categories (roles, task, resources, and relationships). The following numbers of stressors were reported: 86 reports in the roles category, 60 in tasks (such as child care, housework), 41 in resources, and 13 in

relationships. This study and others cited below support that the specific stressors associated with the postpartum can be divided into somewhat overlapping categories of physical, intrapersonal, and interpersonal phenomena.

Physical Stressors

A variety of physical symptoms occurs in the postpartum, as illustrated by participants in a study by Gjerdingen, Froberg, Chaloner, and McGovern (1993). Those participants reported breast symptoms, fatigue, hemorrhoids, poor appetite, constipation, increased sweating, acne, hand numbness/tingling, dizziness, hot flashes, illness days, respiratory symptoms, sexual concerns, and hair loss. Some physical conditions such as backache (Breen, Ransil, Groves, & Oriol, 1994; MacEvilly & Buggy, 1996), urinary incontinence (Wilson, Herbison, & Herbison, 1996), and sexual dysfunction (Glazener, 1997) have been studied because of their duration, frequency, and/or adverse impact. Physical symptoms may be stressors in themselves or may be due to stress as demonstrated by Lenz, Parks, Jenkins, and Jarrett's (1986) finding that negative life events stress was a predictor of postpartum illness in mothers of 6-month-old infants. Of the physical symptoms reported by mothers, fatigue is consistently identified as a major concern (Brown & Lumley, 1998; Chapman, Macey, Borum, & Bennett, 1985; Gardner, 1991; Gjerdingen et al., 1993; Graef et al., 1988; Gruis, 1977; Ruchala & Halstead, 1994). It has been found to correlate positively with perceived stress in breastfeeding women at 3 days and 3, 6, and 9 weeks postpartum (Wambach, 1998). Pugh and Milligan (1993) proposed that maternal fatigue may alter self-care, mothering behaviors, and infant caregiving. On average, the amount of sleep in a 24-hour period during the 4th week postpartum is 7.53 hours, but only 6.15 hours occur during the night. Furthermore, the sleep pattern is discontinuous (Quillin, 1997). Sleep difficulties continue for some women for many months, and the effects of sleep deprivation can be acute and cumulative (Tulman, Fawcett, Groblewski, & Silverman, 1990). Fatigue is related to both quantity and quality of sleep and can alter, through neuroendocrine pathways, the stress response, immune function, and well-being.

Intrapersonal Stress

A common intrapersonal stress is concern about weight. Stein and Fairburn (1996) found that eating disorder symptoms in a sample of 98 primagravidas increased in the first 3 months postpartum and then plateaued over the next 6 months. Walker's (1998) analysis of 227 new mothers' written descriptions of feelings about weight concluded that 8% had weight-related distress.

The development of maternal identity (Rubin, 1984) and maternal role attainment, defined by Mercer (1986, p. 198) as a process in which "the mother achieves competence in the role and integrates the mothering behaviors into her established role set, so that she is comfortable with her identity as a mother," are clearly intrapersonal challenges in the postpartum period. Maternal stress impedes sensitivity to the infant. Ragozin, Basham, Crnic, Greenberg, and Robinson (1982) found a relationship between high levels of maternal stress at 1 month postpartum and decreased maternal sensitivity to cues at 4 months postpartum. In a study of role adaptation (Dormire, Strauss, & Clarke, 1989), adolescent primiparae, when highly stressed, demonstrated less sensitivity to the messages conveyed by their infants' behaviors and less effective maternal behaviors.

> The postpartum is a time of joy and satisfaction and also a time of vulnerability or even of crisis.

A dramatic intrapersonal consequence of stress is postpartum depression. With an incidence consistently estimated at more than 10%, postpartum depression has a deep and persistent negative impact on mothers, infants, partners, and other children. Hall, Kotch, Browne, and Rayens (1996) found the effects of common, everyday stressors on depressive symptoms in 738 women 1 to 2 months postpartum were both direct and mediated by self-esteem. Everyday stressors were more strongly associated with depressive symptoms than were major life events. Beck's (1996) meta-analysis of 44 studies of depression and various predictor variables found that child care stress and life stress were major predictors of depression and only prenatal depression had a greater effect size.

Interpersonal Stress

Pregnancy and childbirth are periods of stressful transition with respect to women's social roles. Gruis (1977) found that at 1 month postpartum almost all mothers were concerned about their abilities to meet the needs of family members, take effective and safe care of their infants, and find time for themselves. This finding was replicated by Harrison and Hicks (1983) in mothers 3 to 8 weeks postpartum. In a study of work and postpartum health, McGovern et al. (1997) found that 90% of women reported role function limitations and that time off from work of more than 20 weeks had a positive effect on maternal mental health and role function.

Longer term effects on functional status (the performance of behaviors associated with roles) is indicated by the finding that even at 6 months postpartum, 20% of women had not resumed their full level of household

activities and 30% had not fully resumed their social and community activities (Tulman et al., 1990). The multiplicity and overlapping of roles often provokes conflict and stress (Spurlock, 1995). Thomas (1997) suggested that stress associated with women's role commitments may contribute to immunosuppression and disease vulnerability. This would support Gjerdingen et al.'s (1993) suggestion that increased gynecologic and breast symptoms among working women at 9 to 12 months postpartum may represent a delayed response to the stress of combining multiple roles of employment, new parenthood, and marriage.

A decrease in satisfaction with or strain in the marital relationship following birth is common (Lewis, 1988), and Affonso and Mayberry (1990) found mate/spouse interactions to be stressors of high intensity. Mercer (1986) found that maternal perception of overall family functioning (among and between individuals, dyads, and family-social units such as work and school) was adversely affected by life events stress, which was measured in women at both high and low risk at 8 months postpartum. Unrealistic cultural assumptions about motherhood result in work overload, difficulty with transition to motherhood, stress, guilt, and sometimes depression (Lee, 1997).

Biologic Factors: Lactation and Stress Responses

The postpartum is associated with significant stressors, and excessive stress poses a threat to maternal, child, and family well-being. It is important to determine if postpartum biology plays a role in the mother's stress response and in stress's potentially deleterious effects on her health. Stress must be understood in terms of both the biologic processes that mediate response and the psychologic processes that affect perception, appraisal, and coping.

This article focuses on the biologic processes mediating stress reactivity and alterations or adaptations in those processes occurring during lactation. Basics of the physiologic response are illustrated in Figure 1. In general, a stressor, which may be physiologic or psychosocial or both, simultaneously activates the sympathetic adrenomedullary system leading to release of the catecholamines epinephrine and norepinephrine and the hypothalamic-pituitary-adrenal system (HPA axis) leading to release of corticotropin, cortisol, and endogenous opioid peptides. Specifically, the hypothalamus secretes corticotropin releasing hormone (CRH), which stimulates the anterior pituitary hormone to secrete adrenocorticotropic hormone (ACTH). This ACTH stimulates the adrenal cortex to synthesize and secrete glucocorticoids. All of these phenomena are influenced by the neuroendocrine and immune physiology of the postpartum woman.

It is interesting to note how few studies of postpartum stress and adaptation or even postpartum physiology distinguish lactating from nonlactating women. All postreproduction animal studies are necessarily of lactating animals, whereas in humans breastfeeding is a choice. Clearly, the neuroendocrinology of a lactating mother is profoundly different from that of a mother who is not lactating. From a biobehavioral perspective, it is reasonable to propose that the postpartum stress response might differ in breastfeeding and bottle-feeding mothers. Available literature supports this premise.

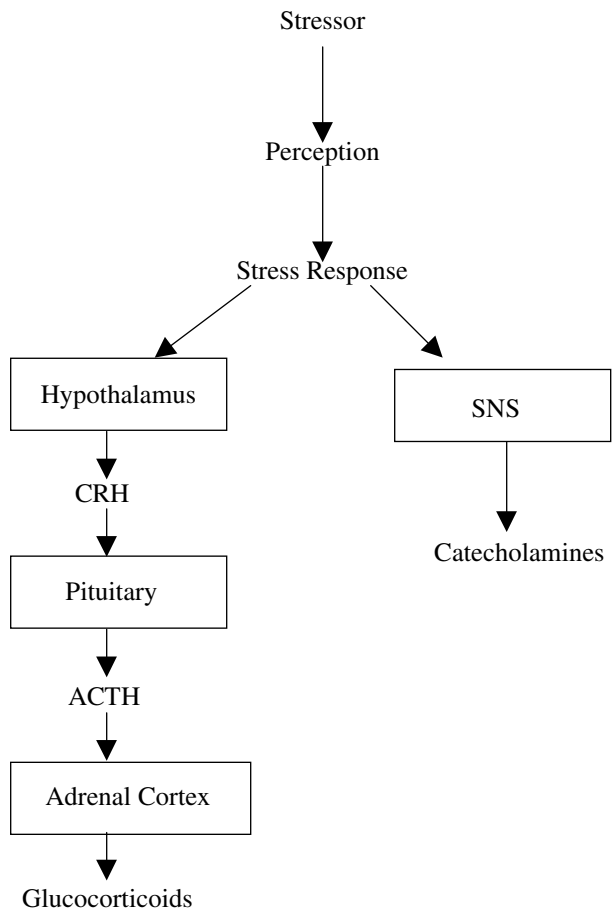

FIGURE 1
The physiologic response to stress.

Note. SNS = Sympathetic nervous system. CRH = corticotropin releasing hormone. ACTH = adrenocorticotropic hormone.

Lactating animals have a well-described diminished physiologic reactivity to stressors. Human studies, although few in number, support the existence of the same response in breastfeeding mothers. Although mothers may be subjected to many stressors in the postpartum, nature has apparently designed a way to decrease the magnitude of their physiologic response to stress if they breastfeed. The unique endocrine and immune state of the lactating mother is directed toward protection of the maternal-infant dyad, reducing maternal reactivity to the environment, promoting calmness and nurturing behavior, maintaining quantity and quality of milk, enhancing

immune function, and opposing glucocorticoid-mediated catabolism (Altemus, Deuster, Galliven, Carter, & Gold, 1995). It is characterized by down-regulated stress responses in both adrenergic and HPA reactivity, which return to normal very quickly when lactation ceases (Windle, Brady, et al., 1997) as do blood levels of cortisol, ACTH, and prolactin (Fischer, Patchev, Hellbach, Hassan, & Almeida, 1995).

Stressors during the postpartum may be divided into somewhat overlapping categories of physical, intrapersonal, and interpersonal phenomena.

The decrease in stress reactivity appears to originate in late pregnancy, when there is a surge in cortisol, due in part to placental CRH. This CRH from the placenta stimulates ACTH and glucocorticoid secretion. At the end of pregnancy, cortisol levels are very high and may approach levels seen in clinical depression or anorexia nervosa or in athletes who exercise strenuously (Magiakou, Mastorakos, Webster, & Chrousos, 1997).

Cortisol facilitates many physiologic and behavioral adaptations of late pregnancy, including catabolic mobilization of maternal energy stores to meet fetal needs and preparation of the breasts for later lactation (Atkinson & Waddell, 1995). In humans, CRH, which has risen throughout pregnancy, peaks at labor and falls dramatically at 1 day postpartum (Smith, Chan, Bowman, Harewood, & Phippard, 1993).

During pregnancy, there is a central suppression of CRH, as increasing placental CRH appears to suppress CRH release from the hypothalamus. This suppression and the gradual return to normal as the HPA axis "resets" itself may be implicated in the genesis of postpartum dysphoria. Lower levels of afternoon cortisol in the early postpartum have been associated with postpartum depression (Harris et al., 1996), as has the longer and more severe postpartum blunting of ACTH response to exogenous CRH administration (Magiakou et al., 1997).

The blunting of the HPA axis may be due to neuroendocrine mechanisms. Levels of estrogen and progesterone that were high during pregnancy drop dramatically at birth and remain low if a female lactates. Prolactin and oxytocin levels rise after birth and remain high if the mother lactates. Estrogen is an important factor in the secretion of ACTH, as it directly stimulates CRH production (Magiakou et al., 1997). The low levels of estrogen during lactation may therefore suppress CRH release by the hypothalamus. Plasma catecholamines also appear to be reduced in response to stressors in lactating rats. Higuchi, Negoro, and Arita (1989) found that immobilization stress produced a smaller elevation in both plasma epinephrine and norepinephrine in lactating compared with nonlactating rats.

In addition, oxytocin and prolactin, which are high during lactation, may inhibit the HPA axis. Cook (1997) demonstrated this effect when he found that cortisol release decreased in both lactating and nonlactating sheep after infusion with oxytocin and prolactin and exposure to the stress of a barking dog. The stress increased prolactin in the nonlactators but not in lactating rats. Lightman and Young (1989) showed a similar effect on oxytocin release.

The antistress nature of lactation may be governed, in part, by the hormone oxytocin, which is maintained at high levels throughout lactation. Oxytocin is also released upon HPA axis activation, particularly in response to emotional stress. In stressed male rats, the oxytocin level rises remarkably within the brain, even though peripheral levels may not change (Engelmann, Ebner, Landgraf, Holsboer, & Wotjakl, 1999). Oxytocin infusion in both male and female rats produces sedation, lower blood pressure, decreased corticosteroid levels, and evidence of increased vagal activity. A variety of stimuli produce an elevation in oxytocin release in nonlactating animals, which may account for certain behavioral phenomena such as positive mood and prosocial behavior (Uvnas-Moberg, 1997). Infusion of oxytocin in 182 healthy men produced attenuated arousal and anger (Pietrowsky, Krug, Fehm, & Born, 1992). In human mothers, this hormone has been found to be amnestic and to reduce anxiety and produce sedation. The possibility of oxytocin-induced pain reduction is suggested by animal studies (Carter & Altemus, 1997) and supported by our knowledge of a mother with a chronic pain syndrome who reported that the only time she was free of debilitating pain was when she was breastfeeding.

A difference in behavioral response to stress has been clearly demonstrated in lactating and nonlactating animals exposed to a mild stress (10 minutes of white noise). Virgin rats showed HPA activation and increased activity levels, displacement grooming behaviors, and rearing, whereas lactating rats showed an entirely different repertoire of behaviors that were directed toward their pups. They did not show activation of the HPA response in response to the stress (Windle, Woods, et al., 1997). During lactation, there may also be increased central nervous system gamma-aminobutyric acid, a major inhibitory neurotransmitter, which may inhibit the affective state and behavior of the lactating animal (Qureshi, Hansen, & Soderstein, 1987).

The small amount of research conducted on the reactivity to stressors in lactating humans supports the premise that the breastfeeding mother has a diminished

response to stressors. Weisenfeld, Malatesta, Whitman, Grannose, and Vile (1985) found that compared to bottle-feeding mothers, breastfeeding mothers showed reduced galvanic skin response and heart rate changes while listening to a tape of a crying infant. Lactating women were found to have lower plasma levels of ACTH, cortisol, glucose, and norepinephrine than did nonlactating women following 20 minutes of graded treadmill exercise as a stressor (Altemus et al., 1995).

Mezzacappa, Kelsey, and Katlin (1999) studied blood pressure and continuous impedance cardiography in breastfeeding and bottle-feeding mothers and found a greater cardiovascular response to psychologic stressors in the mothers who bottle-fed their infants. They also reported lower incidences of psychiatric and cardiovascular disorders in the breastfeeding group. Light et al. (2000) found that breastfeeding mothers had lower blood pressure compared with bottle-feeding mothers, both during a laboratory stress experiment and later at home. As predicted from animal studies, they found that oxytocin release during stress was higher in lactating mothers and this hormone suppressed stress responsivity.

Summary

This review has provided evidence for consideration of the postpartum as potentially stressful for a variety of physical, intrapersonal, and interpersonal factors. Nevertheless, postpartum women may be protected from the deleterious effects of stress through biologic mechanisms that have been extensively studied in animals but not in humans.

> Lactating animals have a diminished physiologic reactivity to stressors, and a few human studies confirm this for breastfeeding mothers.

This protection appears to be governed by the neuroendocrinology associated with lactation, which is the natural, biologic normative behavior of all postpartum animal mothers. This protection makes evolutionary sense in that the maternal-infant dyad is protected and supported by these responses. Little attention has been paid to this obvious biologic difference in studies of human mothers. Lactational state has rarely been a variable in studies of the human postpartum, although only in humans is lactation a choice. We encourage maternal-child researchers to analyze their data with this perspective in mind.

Practice implications of these concepts include the possibility that the bottle-feeding mother might be physiologically more fragile and more susceptible to postpartum illness. Further research is required to provide sufficient evidence for this effect. Of course, nurses need to support all postpartum mother-infant dyads and to promote nurturant behaviors, including feeding, whether it is by breast or bottle.

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