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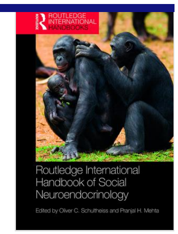
Routledge International Handbook of Social Neuroendocrinology



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Description

The *Routledge International Handbook of Social Neuroendocrinology* is an authoritative reference work providing a balanced overview of current scholarship spanning the full breadth of the rapidly developing field of social neuroendocrinology. Considering the relationships between hormones, the brain, and social behavior, this collection brings together groundbreaking research in the field for the first time.

Featuring 39 chapters written by leading researchers, the handbook offers impressive breadth of coverage. It begins with an overview of the history of social neuroendocrinology, before discussing its methodological foundations and challenges. Other topics covered include state-of-the-art research on dominance and aggression; social affiliation; reproduction and pair bonding (e.g., sexual behavior, sexual orientation, romantic relationships); pregnancy and parenting; stress and emotion; cognition and decision making; social development; and mental and physical health. The handbook adopts a lifespan approach to the study of social neuroendocrinology throughout, covering the role that hormones play during gestation, childhood, adolescence, and adulthood. It also illustrates the evolutionary forces that have shaped hormone-behavior associations across species, including research on humans, non-human primates, birds, and rodents.

The handbook will serve as an authoritative reference work for researchers, students, and others intrigued by this topic, while also inspiring new lines of research on interactions among hormones, brain, and behavior in social contexts.

Reviews

"Social neuroendocrinology is a rapidly growing scientific discipline that has revolutionized our understanding of the biological bases of all social processes. The *Routledge International Handbook of Social Neuroendocrinology* offers the most comprehensive and most authoritative review of this field of research to date. A must-read for all behavioral scientists." - *Dario Maestripieri, The University of Chicago, USA*

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About the Editors

Oliver C. Schultheiss is a professor of psychology at Friedrich-Alexander University, Erlangen, Germany. His research focuses on the implicit motivational needs for power, achievement, affiliation, and sex, and their interactions with the endocrine system.

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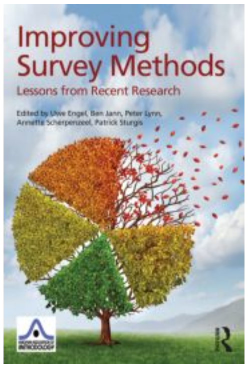

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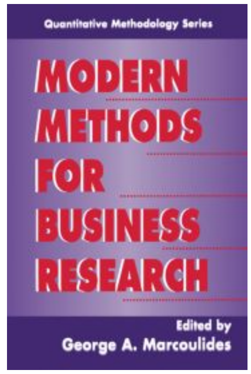

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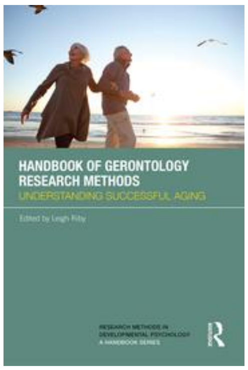

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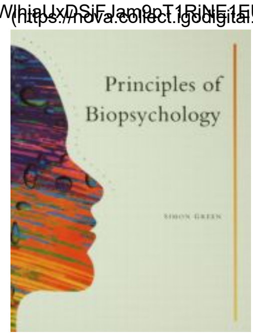

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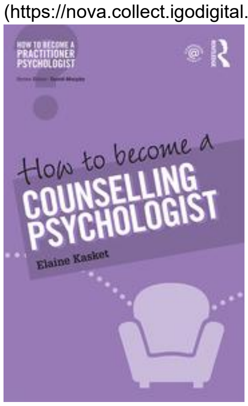

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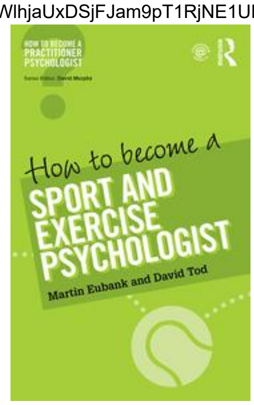

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The Social Neuroendocrinology of Pregnancy and Breastfeeding in Mothers (and Others)

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Introduction

Parenting may be regarded as the keystone upon which all forms of social affiliation rest. Over deep evolutionary time, the fitness advantages of the large brains and flexible cognitive and behavioral repertoires that are the hallmarks of our altricial species compensated for the fitness costs of parental investment over an extended period of child development (Kaplan, Lancaster, & Robson, 2003), favoring the selection of traits which ultimately produced psychobiological adaptations for parental nurturance and defense. The neuroendocrine changes that support human parenting appear to have been co-opted and elaborated, both over evolutionary time and during ontogeny, to support non-parental forms of social attachment and affiliation (e.g., Carter, 2014; Hazan & Shaver, 1987). Thus, while phenomena addressed in this volume such as romantic bonding, friendship, or group identification doubtless constitute complex and distinct topics of study, each plausibly derives from neuroendocrine mechanisms originally evolved to enable parental caregiving. Given the centrality of hormonal changes related to pregnancy and lactation to derived modes of social affiliation, programmatic research on the social neuroendocrinology of these topics is in scandalously short supply. We hope that the present chapter helps to inspire the next generation of researchers to fill this gap.

As the social neuroendocrinology of labor and delivery has been recently and comprehensively reviewed elsewhere (Saxbe, 2017), we concentrate on pregnancy and lactation. We begin with an overview of the dramatic neuroendocrine shifts characteristic of pregnancy, including emerging evidence of their effects on brain physiology, behavior, and social judgment, as well as bidirectional effects of pregnancy-related shifts on fathers and other caregivers. Following childbirth, we review models of the effects of lactation on outcomes for both mother and child, with an emphasis on affiliation, stress-attenuation, and defensive maternal aggression.

We conclude by highlighting a number of translationally vital and theoretically intriguing open questions demanding further research.

Pregnancy

To begin to fathom the dramatic nature of the neuroendocrine changes accompanying pregnancy, consider the remarkable fact that expectant mothers grow an entirely new organ: the placenta. The placenta responds to hormonal signals provided by both mother and fetus, creating reciprocal feedback loops between the neuroendocrine systems of the maternal-fetal dyad and producing a variety of hormones (e.g., CRH, cortisol, estradiol, estrone) to be selectively released into the maternal blood stream or intrauterine environment (Sandman, Davis, Buss, & Glynn, 2011). Key hormones dramatically heighten during pregnancy, with the most marked changes occur during the third trimester when estradiol levels increase by approximately 50-fold, progesterone levels increase by approximately 10-fold (Tulchinsky, Hobel, Yeager, & Marshall, 1972), and prolactin levels increase by approximately 7-fold (Bloch, Daly, & Rubinow, 2003). One of the most dramatic changes in pregnancy arises from the placenta's ability to produce corticotropin-releasing hormone (CRH). CRH produced by the placenta (pCRH) enters the maternal blood stream at levels ranging from 60 to 700 times higher than CRH levels observed before pregnancy (Campbell et al., 1987). In contrast to CRH release in healthy non-pregnant individuals, which ultimately triggers a negative feedback dynamic restoring CRH to baseline levels (Smith & Vale, 2006), pCRH initiates a positive feed-forward process wherein placental tissue exposed to cortisol up-regulates the production of pCRH, causing pCRH levels to heighten exponentially over the course of pregnancy along with relatively modest increases in other HPA products such as ACTH and cortisol (McLean, Thompson, Zhang, Brinsmead, & Smith, 1994). Levels of CRH in cerebrospinal fluid are higher in pregnant compared to non-pregnant women, suggesting that peripheral increases in pCRH elevate CRH in the pregnant brain (Zaconeta et al., 2015).

The dramatic shifts in gonadal and neuropeptide hormones associated with the placenta, and the concomitant effects on HPA activity, halt with the expulsion of the placenta at birth. Levels of CRH typically return to pre-pregnancy levels within days, and other gonadal and steroid hormones typically return to baseline levels within a few weeks (Bloch et al., 2003). Although the dramatic hormonal shifts associated with pregnancy are temporary, evidence derived from both rodent and human studies indicates that having experienced these changes lays the foundation for attentional, behavioral, and motivational shifts away from self-directed effort and toward the provision of parental resources and protection.

Pregnancy and the Maternal Brain

Natural selection appears to have shaped the pattern of endocrinological changes typical of pregnancy not only to enable gestation, but to alter the social brain in parentally adaptive ways (Glynn, 2010; Kinsley & Lambert, 2008). In rodents, nulliparous females produce lasting maternal behavior following blood transfusions from pregnant rats (Terkel & Rosenblatt, 1972), and the time-sequence of changes in both the increasing levels and relative ratios of estrogen and progesterone track the onset of maternal behaviors in rats (Bridges, 1984) and macaques (Maestripieri & Zehr, 1998). Illustrative of the causal impact of pregnancy hormones on maternal motivation in nonhuman animals, treating marmosets with estradiol and progesterone in patterns parallel to those characterizing pregnancy causes non-pregnant females to press bars more often to see an infant or to silence infant distress cries (Pryce et al., 1993).

Estradiol and progesterone exposure during pregnancy may induce maternal behavior indirectly by heightening sensitivity to oxytocin and prolactin. Pedersen and Prange (1979) administered oxytocin to virgin rats, finding that only those females whose estradiol levels were relatively high at the time of transfusion exhibited maternal behavior. Follow-up studies confirmed that administering estradiol prior to oxytocin administration caused full maternal behavior to emerge in virtually all animals, possibly because estradiol exposure in virgin rats significantly increases oxytocin receptor binding in the medial preoptic area (arguably the region most closely linked with maternal caregiving) and the lateral septum (a key motivational region) (Champagne, Diorio, Sharma, & Meaney 2001). Interestingly, rats whose mothers exhibited greater licking and grooming behaviors evinced increased oxytocin receptor density in the medial preoptic area and lateral septum, whereas rodents lacking comparable experience of early maternal grooming do not show changes in oxytocin receptor density in these regions despite estradiol exposure, suggesting a biological pathway for intergenerational transmission of individual differences in maternal behavior (Champagne et al., 2001). In a similar pattern, infusions of prolactin into the medial preoptic area induce maternal behavior in virgin rats previously given estradiol and progesterone—but not in untreated females (Bridges, Numan, Ronsheim, Mann, & Lupini, 1990). In sum, classic comparative work sketches how estradiol and progesterone exposure during pregnancy may similarly lay the foundations for oxytocin and prolactin exposure at the end of pregnancy to induce maternal behavior in humans.

The limited extant work in humans broadly accords with a preparatory function of estradiol and progesterone during pregnancy. In a largest longitudinal study, Glynn and colleagues (2016) assessed estradiol and progesterone at five points during pregnancy, then investigated whether hormonal trajectories predicted mothers' sensitivity towards their infants during play sessions one year post-birth. Mothers with lower mid-gestational estradiol-to-progesterone ratios at 20-34 weeks of pregnancy were more sensitive to their infants' cues, whereas higher absolute levels of estradiol and lower absolute levels of progesterone predicted reduced maternal sensitivity. These results correspond with prior findings that mothers with lower estradiol-to-progesterone ratios in early pregnancy self-report greater infant attachment at four days postpartum (Fleming et al., 1997). Glynn and colleagues also found that gonadal hormone exposures were more important predictors of maternal sensitivity in first-time mothers, a parity effect that may owe to the fact that new mothers evinced higher levels of progesterone than multiparous women, possibly—at least in part—to enact long-term changes in the maternal brain by increasing oxytocin receptor density in relevant regions. Higher levels of plasma oxytocin over the course of human pregnancy predict maternal vocalizations (e.g., cooing), self-reported positive emotion, infant-directed eye gaze, and infant ideation (Feldman et al., 2007), and elevated oxytocin levels similarly predict more frequent affectionate maternal touch during play (Feldman et al., 2010). In a complementary finding, the magnitude of the increase in oxytocin from early to late pregnancy positively predicts the quality of maternal-infant attachment (Levine et al, 2007).

At the climax of pregnancy, the act of giving birth may also play an important role in shaping the maternal brain (see Saxbe, 2017, for a review). For example, the spike in oxytocin required for uterine contractions during parturition, occurring within the context of a brain primed with gonadal hormones, may help to explain observations of heightened maternal sensitivity in mothers who give birth naturally. Swain and colleagues (2011) asked six mothers who delivered vaginally and six mothers who had planned cesarean sections to listen to their own babies' cries during neuroimaging, finding that vaginal childbirth was associated with greater sensitivity to their infant's cries in areas of the brain related to sensory processing, empathy, arousal, motivation, and habit-regulation—in short, key brain correlates of maternal attention and care. However, as this study was low-powered and suffered the confound that individual differences in maternal motivation prior to childbirth may covary with the decision to give birth naturally, further research is required to ascertain whether and to what extent the mode of delivery determines changes to the maternal brain.

Beyond domain-specific effects on maternal caregiving, there is also limited evidence suggesting that pregnancy-related endocrine changes may shape more general aspects of social cognition. For example, Jones et al. (2005) found higher preferences for facial cues associated with health in pregnant compared to non-pregnant women akin to a similar preference observed in women taking progesterone-laced birth control or during high-progesterone phases of the menstrual cycle. Women also appear to favor in-group over out-group members during early pregnancy, as expectant mothers have been found to display more intense ethnocentrism during the first trimester relative to the second and third trimesters of pregnancy (Navarrete, Fessler & Eng, 2007). These shifts in preferences away from cues of ill-health or out-group membership have been hypothesized to reflect functional design to avoid disease, particularly during the immunosuppressed period of early pregnancy when pathogen-exposure would pose a greater risk, or to affiliate with in-group members for access to protection and resources. While intriguing, these interpretations should be treated with caution pending further replication, including direct measures of pregnancy hormones. Whatever the ultimate merits of these particular functional interpretations, there do appear to be real effects and hence a need for further research into plausible pregnancy-related, hormonally mediated shifts in outcomes such as partner intimacy, kin-preferences, or wariness toward strange males (Hahn-Holbrook et al., 2011).

From Mothers to Others

Highlighting the apparent functional aspects of the neuroendocrinology of pregnancy and lactation risks creating the impression that parents who did not gestate or lactate consequently experience less love for, or commitment to, their children. To the contrary, fathers, adoptive parents, step-parents, and other caregivers can evidently develop close bonds with children via pathways independent of those neuroendocrinologically mediated by pregnancy, natural childbirth, or breastfeeding. However, and intriguingly, emerging evidence suggests that the neuroendocrinological shifts associated with motherhood can biologically and behaviorally spread to other caregivers—and in turn be influenced by their social attributes—in a kind of mutual psychobiosocial contagion.

Comparative and human studies suggest that the degree of correlation between fathers' and mothers' hormones may index paternal investment and long-term relationship commitment (Edelstein et al., 2015; Saxbe et al., 2016). For example, father's hormones correlate with maternal hormones such as prolactin, estradiol and cortisol during pregnancy (Storey et al., 2000), indicating that relationship closeness with the expectant mother—including the concomitant exposure to her maternal hormones—may help to biologically prepare males for fatherhood. In further evidence that men are hormonally influenced by shifts within their pregnant partners, Edelstein and colleagues (2015) observed significant associations between prenatal maternal and paternal cortisol and progesterone. This human work accords with comparative work in mice and hamsters finding that female biobehavioral cues help to initiate and sustain paternal care behaviors (Gubernick & Nelson, 1989).

Just as maternal hormones and behaviors appear to biologically influence paternal caregivers, so can the support provided by others influence mothers' hormones in pregnancy and related health outcomes. A study of 210 mothers found that perceived social support during pregnancy protected women against postpartum depression by dampening maternal pCRH trajectories during pregnancy (Hahn-Holbrook et al., 2013). Specifically, women who perceived themselves as enjoying greater support from their families during pregnancy evinced slower rises in pCRH during late pregnancy, mediating fewer postpartum depressive symptoms compared to women who perceived themselves as possessing relatively poor family support. In a somewhat surprising twist, however, the perceived support of fathers did not significantly influence pCRH trajectories in this sample, although perceived paternal support did attenuate subsequent depressive symptoms. Future work may nonetheless detect effects of perceived paternal (or non-paternal partner) support on pCRH and other maternal hormones, as the null results may owe to the notable heterogeneity in types of male partner support in this sample.

Lactation

Notwithstanding their aforementioned roles in parental motivation, oxytocin and prolactin are the primary hormones involved in lactation. Oxytocin facilitates smooth muscle contraction and thereby facilitates the release of breastmilk; prolactin, as the term suggests, is the primary hormone responsible for the production of milk in breast tissue. Oxytocin is released into the blood stream to aid in milk ejection prior to breastfeeding (White-Traut et al., 2009), and this dynamic appears driven by infant cues, as mothers separated from their infants prior to feeding do not evince a prefeeding oxytocin release (McNeilly, Robinson, Houston, & Howie, 1983). As tactile nipple stimulation occurs during feeding, oxytocin and prolactin are released in pulsating patterns directed by nerve fibers linked to the hypothalamus (Gimpl & Fahrenholz, 2001). The supply/demand dynamic intrinsic to infant suckling means that timing is key in research on the social neuroendocrinology of breastfeeding. For example, oxytocin is upregulated in response to each breastfeeding session and returns to baseline shortly thereafter. Consequently, mothers who breastfeed once daily will display similar baseline oxytocin levels to non-lactating mothers unless they are either about to nurse or have recently nursed. By contrast, mothers who breastfeed in short sessions every fifteen minutes or so during waking hours, as has been reported in several small-scale horticulturalist societies, and as is not unusual for mothers who feed on demand, may display highly elevated levels of oxytocin throughout the day. Thus, investigators should never regard breastfeeding status alone as a reliable proxy for oxytocin or other hormone levels. Dose-dependent hormonal increases have also been noted with regard to prolactin, which peaks approximately forty minutes post-feeding. Nursing mothers typically exhibit higher overall prolactin levels than women who are not breastfeeding, although prolactin levels, like oxytocin levels, vary in proportion to breastfeeding frequency and spacing (Battin, Marrs, Fleiss, & Mishell, 1985). Researchers should take care to consider potential moderators such as participants' breastfeeding or breast-pumping frequency, number of breastfeeding or pumping sessions over the preceding 24 hours, time since and duration of last breastfeeding or pumping session, anticipated time of the next feeding session, and percentage of the child's diet composed of breastmilk. In addition, studies should take the age of the child into account, as milk production transitions from primarily endocrine control to autocrine control at around six months postpartum.

Breastfeeding and the Maternal-Child Bond

One of the chief motives for breastfeeding reported by new mothers is the desire to further bond with their infants (Arora et al., 2000). Indeed, breastfeeding is frequently assumed to foster maternal behavior in the scientific literature (e.g., Jansen et al., 2008). Folk intuition notwithstanding, surprisingly few studies have actually tested this hypothesis.

The release of oxytocin and prolactin linked with lactation appears integral for evoking maternal caregiving in many nonhuman species (Kendrick, 2000). In rats, injection of oxytocin (Pedersen et al., 1992) or prolactin (Bridges et al., 1985) into the brain after pregnancy experimentally triggers maternal behaviors, whereas the injection of oxytocin- or prolactin-blocking agents shortly after giving birth prevents the onset of maternal behaviors (Bridges et al., 2001; van Leengoed et al., 1987). Heightened prolactin, characteristic of lactation, also promotes maternal responses in marmosets (Dixson & George, 1982), tamarins (Ziegler et al., 2000), and mice (Voci & Carlson, 1973). Notably, however, lactation-associated hormonal shifts appear less determinative of maternal caregiving in primates, for whom social and developmental experience are more relevant to later maternal behavior (Pedersen, 2004). For instance, among Rhesus monkeys, the injection of oxytocin antagonists into the brain impairs select maternal behaviors while leaving others in place (Boccia et al., 2007). Likewise, maternal behaviors often emerge in nonhuman primates in the absence of lactation (e.g., nulliparous females often carry or groom others' infants) (Hrdy, 1999).

Only a handful of studies have investigated whether oxytocin or prolactin predict maternal behavior in humans. Plasma oxytocin levels assessed postpartum (and during pregnancy) correlate with maternal positive vocalisations (e.g., cooing), self-reported positive emotion, infant-directed eye gaze, and infant ideation (Feldman et al., 2007). Bewilderingly, research on the effects of prolactin on parenting motivation in humans has focused almost exclusively on fathers (Rilling & Young, 2014). In the lone study of human mothers that we are aware of, prolactin levels increased more after holding a doll in pregnant and postpartum women than in women without children (Delahunty, McKay, Noseworthy & Storey, 2007). To date, no studies have provided evidence that breastfeeding-induced increases in oxytocin or prolactin influence caregiving.

Although far from direct neuroendocrine evidence, breastfeeding and bottle-feeding mothers behave detectably differently during infant interaction. Else-Quest and colleagues (2003) reported that breastfeeders displayed more positive mother-infant interactions at 12 months than formula-feeding mothers, and Nishioka and colleagues (2011) found that mothers whose infants derived most of their nutrition from breastmilk during the first five months reported heightened maternal bonding relative to mothers who primarily relied on formula or did not breastfeed at all. In a complementary set of findings, Britton et al. (2006) found that those mothers who were not breastfeeding by the third month reported less sensitivity to their infants' needs than breastfeeders, and independent observers note greater infant sensitivity in mothers who were still breastfeeding at three months during a 30-minute mother-infant interaction at six months after birth (Jonas et al., 2015). Intriguingly, the latter association between breastfeeding and heightened maternal sensitivity was only evident among mothers who experienced a high degree of psychological stress—mothers who reported little stress evinced relatively high maternal sensitivity regardless of breastfeeding behaviors (Jonas et al., 2015). With respect to maternal sensitivity to cues of infant distress, a large longitudinal study of 675 mother-infant dyads found that the amount of time spent breastfeeding predicted greater sensitivity at 14 months (Tharner et al., 2012), and a functional neuroimaging paradigm revealed greater activation of brain regions implicated in maternal-infant bonding and empathy in response to hearing their infant cry among exclusively breastfeeding mothers compared to exclusively formula-feeding mothers at one month after childbirth (Kim et al., 2011).

Although the foregoing results broadly accord with the claim that breastfeeding facilitates maternal bonding and caregiving, no firm conclusions may yet be drawn given the potential self-selection factors independent of the effects of breastfeeding. Indeed, mothers who express a willingness to breastfeed while pregnant also report greater maternal sensitivity at three months, and this prior willingness to breastfeed correlates with the reported strength of the mother-infant bond (Britton et al., 2006). Conversely, the quality of mother-infant bonding observed two days following parturition predicts exclusive breastfeeding at six months (Cernadas et al., 2003), suggestive that it is bonding that contributes to breastfeeding rather than the reverse. Further, it is unclear at the time of writing whether the heighted maternal sensitivity associated with breastfeeding may be somewhat accounted for by the experience of frequent, close contact between mothers and infants as opposed to the neuroendocrine effects of lactation. It is also important to stress that, regardless of whether breastfeeding may help to facilitate maternal sensitivity, the formula-feeding mothers in the studies discussed above displayed maternal sensitivity within a normal range. Thus, additional research is required to clarify both the extent and the means by which breastfeeding may influence bonding or related outcomes such as maternal sensitivity.

Approaching maternal-infant bonding from the infant's perspective, breastfeeding appears likely to facilitate attachment for a number of reasons, including direct skin-to-skin contact between mother and child, encouragement of early maternal-child social interactions, and the calming effect of the sucking reflex. Here again, however, surprisingly few studies have investigated the putative relationship between breastfeeding and infant attachment, and those that have reveal a mixed empirical picture (Janson et al., 2008). For example, in a study of 152 mother-infant pairs examining the association between breastfeeding and the quality of infant attachment by the first year of life, infants who had been breastfed displayed comparable secure maternal attachment to infants who had been formula-fed (Britton et al., 2006). This result may reflect the fact that infants are evolutionarily motivated to develop secure attachment relationships with non-lactating caregivers (e.g., fathers). However, breastfed infants have also been found more likely than formula-fed infants to orient their bodies towards gauze pads carrying the scent of their mothers than to gauze pads carrying the scent of unfamiliar breastfeeding women at the second week of life, consistent with the premise that breastfeeding facilitates early recognition of and attraction to maternal scent (Cernoch & Porter, 1985).

Moving from mode of consumption to the biopsychosocial effects of breastmilk itself, emerging research suggests that exposure to bioactive hormones in breast milk may shape infant temperament. In a recent study, for example, infants exposed to higher levels of milk cortisol scored higher in negative affect in comparison to infants whose milk possessed relatively low cortisol (Grey, Davis, Sandman, & Glynn, 2013), a relationship which was not accounted for by covarying environmental factors (e.g., maternal education, age, or income) nor by negative maternal affect (e.g., depression and perceived stress). This finding converges with research showing that circulating infant cortisol levels are more closely correlated with circulating maternal cortisol in breastfed infants compared to formula-feeders (Glynn et al., 2007). Interestingly, a somewhat distinct effect of milk cortisol on infant temperament has been reported among Rhesus macaques (Hinde et al., 2015), in that higher levels of milk cortisol were found to predict greater social confidence. While further work is needed, this overall pattern of findings suggests that lactation may be regarded as a veritable “fourth trimester” during which breastmilk provides a direct conduit between the endocrine systems of mother and infant.

Breastfeeding Attenuates Maternal Social Stress

Maternal caregiving can be intensely challenging, particularly during the initial months following childbirth. Maternal stressors run the gamut from sexual dysfunction and sleep deprivation (Gjerdingen et al., 1993) to preoccupations with being a ‘good’ mother, and new parents also typically experience a sustained state of heightened vigilance toward potential hazards to children largely instantiated within neurobiological stress systems (Hahn-Holbrook et al., 2011). Understandably, given the array of demands placed on new mothers, approximately

20% report depressive symptoms within the first year following childbirth (Gavin et al., 2005). Deleterious levels of maternal stress adversely affect child health and behavior outcomes, suggesting that selection pressures favored mechanisms to attenuate maternal stress during early childhood, and which might be expected to efficiently co-opt time-matched adaptations for lactation. Indeed, breastfeeders display lower cardiovascular stress reactions (e.g., higher cardiac parasympathetic control, lower basal systolic blood pressure) than formula-feeders or nulliparous women to the Trier Social Stress Task (Altemus et al., 2001), and similar cardiovascular benefits have been observed in breastfeeding mothers during the period of anxious anticipation prior to the Trier ordeal (Light et al., 2000). The stress-reducing effects of lactation appear to be particularly notable immediately following feeding sessions, as mothers who breastfeed just before the Trier task display blunted hormonal cortisol responses in comparison to control breastfeeding mothers who hold their infants without nursing (Heinrichs et al., 2001). A number of studies indicate that breastfeeders experience greater emotional equanimity and positive mood, as well as diminished anxiety, relative to formula-feeding mothers (Altshuler et al., 2000; Carter & Altemus, 1997). Crucially, the aforementioned differences between breastfeeders and formula-feeders obtain when controlling for potential confounds such as differences in income, age, health, or employment (Mezzacappa & Katlin, 2002).

Oxytocin and prolactin appear integral to the stress-attenuation effects of breastfeeding. Oxytocin mediates lactation-related reductions in stress in rodent experiments (Neumann et al., 2000), with comparable results observed with regard to prolactin (Freeman et al., 2000). Similarly, correlational studies in human samples show that both higher plasma oxytocin and prolactin predict reductions in early postpartum anxiety (Nissen et al., 1998). Complementarily, mothers who release relatively greater levels of oxytocin in response to suckling display lower cortisol

(Chiodera et al., 1991). In sum, the overall pattern of findings derived from both comparative and human studies suggests that lactation attenuates social stress, and that these effects are likely mediated by increases in oxytocin and prolactin, although direct evidence for the roles played by oxytocin and prolactin in buffering human maternal stress is quite thin at present.

Breastfeeding and Maternal Aggression

As a singularly altricial species, humans invest enormous resources in relatively few, highly vulnerable offspring who require protection over a period of years. Given the high fitness stakes, human mothers are likely to have evolved mechanisms to facilitate protection of their young against potentially dangerous conspecifics and predators, particularly during the early period of greatest infant helplessness. Fellow humans posed a serious risk to infants in the ancestral past, particularly unrelated males whose reproductive incentives conflicted with those of mothers and their children (Hrdy, 1999; Hahn-Holbrook et al., 2010). In many other mammals, lactation coincides with a period of heightened defensive aggression toward hostile conspecifics or predators characteristic of the months following birth (Lonstein & Gammie, 2002). In rats, the release of oxytocin and prolactin via lactation disinhibits aggression toward potentially threatening conspecifics (Hansen & Ferreira, 1986), and a parallel dynamic has recently been documented in human mothers. In a behavioral paradigm, breastfeeders evinced lower systolic blood pressure (a proxy of stress) and were more aggressive (i.e., administering louder, longer aversive sound bursts) during a conflictual encounter with a confederate than were either formula-feeding mothers or women who had never given birth, suggesting that you may not want to steal a breastfeeder's parking space (Hahn-Holbrook et al., 2011).

Conclusions

The discoveries regarding the social neuroendocrinology of pregnancy and lactation accumulated to date have both challenged common assumptions and illuminated key biopsychosocial determinants of maternal and infant well-being. Remarkable though the extant literature may be, the relative dearth of human studies and over-reliance on indirect proxies rather than actual biological measures are frankly appalling given the theoretical and translational importance of these issues. Consider, for example, that only one study to date has investigated how prolactin influences maternal behavior (Delahunty et al., 2007), whereas many studies have examined the effects of prolactin on fathers. Without speculating unduly into the biases underlying such an asymmetry—which is absurd on its face given the central role of pro*lactin* in *lactation* and the abundance of comparative work indicating an important role in maternal anxiety-reduction and maternal motivation—it seems fair to suggest that the community of scholars may have deprioritized understanding the psychobiology of human motherhood.

On a more optimistic note, any number of enticing research opportunities are now available to the enterprising social neuroendocrinologist. To cite just one direction, the emerging indications of a rich bidirectional interplay between the endocrine systems of expectant mothers and others raises provocative possibilities with regard to effects on mothers' alloparenting kin (e.g., siblings) and friends. Similarly, with respect to breastfeeding, researchers might examine the extent to which personality adaptations to mothers' local environments (e.g., harsh, resource-poor, and/or dangerous social settings) may be transmitted to their infants via mother's milk, with potentially lifelong personality effects (e.g., risk-tolerance as an adaptive response to uncertain environments). The gold standard for future research needed to test such hypotheses will be longitudinal studies incorporating both biological and psychosocial measures. However, experimental approaches are also invaluable given the inherent limitations of correlational research. In particular, experimentally manipulating natural parental behaviors (e.g., randomly assigning mothers to breastfeed vs. hold their baby) affords social neuroendocrinologists ready alternatives to reliance on methods such as the administration of synthetic oxytocin or prolactin, which may not invoke physiologically normal or ecologically valid responses. Finally, very little research has yet been conducted on the impact of either pregnancy or breastfeeding on mothers' social interactions with romantic partners, fellow caregivers, unrelated children, or strangers, although the limited work that has been done hints at the existence of a range of functional shifts. From our vantage point here in the early 21st century, research on how mind, brain, body, and social context conspire to produce parental behavior has taken important first steps, but there's considerable growing up left to do.

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