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Perinatal Anxiety and Depression: Associations with Oxytocin and Mother-Infant Interactions

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1. Introduction

There is converging evidence that heightened maternal anxiety and depression during the perinatal period affects early bonding and mother-infant interactions, exerting an important impact on the later development, competencies and mental health of the child. Yet there is a limited understanding of the biological mechanisms underpinning the link between perinatal mood and mother-infant interaction difficulties. This chapter will review the literature on the role of oxytocin in mediating bonding difficulties and infant outcome in women with perinatal anxiety and depression.

2. Maternal bonding and subsequent infant outcomes

The nature and quality of early parent-child relationships has been the subject of nearly a century of research stemming from attachment theory through to current biological approaches. Axiomatic to all approaches is the idea that successful human development is predicated on the establishment of a "bond". That is, the parent and child recognise, attend to and approach each other, are responsive to each other's states and communications, and experience feelings of 'closeness' and love (Brockington et al., 2001). All mammals are driven to seek predictable nurturing relationships with caregivers, and the successful negotiation of such early relationships lay the foundation for reaching maximum potentials in cognitive and social competence, and physical health (Meaney, 2001; Suomi, 1997). Large epidemiological studies show that early exposure to dysfunctional parenting is the single most significant (known) risk factor for childhood and later-onset mental disorders including depression, anxiety, disruptive behaviour, and substance abuse disorders (Green et al., 2010).

Both child and parent factors contribute to and derive benefits from, the successful establishment of such bonds. There is some evidence that the more sensitively the parent responds to their infant's cues the greater the likelihood that the infant will become securely attached (Atkinson et al., 2005; Bakermans-Kranenburg, van Ijzendoorn, & Juffer, 2003; De Wolff & van Ijzendoorn, 1997; Pederson, Gleason, Moran, & Bento, 1998; Seifer, Schiller, Sameroff, Resnick, & Riordan, 1996). Much of this research is based upon a classification system developed by Ainsworth that paid particular attention to the reaction of infants

following a stressful situation (Ainsworth, 1979; Ainsworth, Blehar, Waters, & Wall, 1978). Attachment classifications are accorded in relation to the strength to which an infant uses their primary caregiver as a secure base, and also how avoidant or resistant the infant is when they resume contact with their mother after spending time with a stranger (see Ainsworth, et al., 1978; for full procedure). Secure attachment styles generally develop following positive maternal bonding experiences, including responsive and sensitive parenting. Once established, this style remains relatively consistent across the lifespan and if secure, will generally protect children from developing psychopathology such as anxiety, depression and aggression (Shaw, Owens, Giovannelli, & Winslow, 2001; Urban, Carlson, Egeland, & Sroufe, 1991; Warren, Huston, Egeland, & Sroufe, 1997); as well as enhancing their social competence and emotional regulation later in life (Elicker, Englund, Sroufe, Parke, & Ladd, 1992; Sroufe, 2005). The reverse is also the case with a meta-analysis revealing a significant association between an insecure maternal attachment in early childhood and later externalizing problems in children (Fearon & Belsky, 2011). Thus, improving maternal attachment and bonding within this early period can alter the trajectory of development towards more resilience in childhood and reduce the risk of developing mental health problems.

3. A role for oxytocin in maternal bonding

The biological basis of the "attachment-caregiving system" is slowly being elucidated with oxytocin continually being identified as the key component (Insel, 1997; Taylor et al., 2000). Oxytocin, as a neurohypophyseal hormone, is involved in the control of labour and secretion of milk via interaction with its receptors located in the uterus, mammary glands and peripheral tissues (Russell & Leng, 1998). Central oxytocin on the other hand has a key role in establishing and maintaining social affiliative behaviours (Lim & Young, 2006). The social affiliative properties of oxytocin are conserved across mammalian species (Keverne & Curley, 2004) along with the neural network of the oxytocinergic system (Tost et al., 2010) whereby oxytocin is produced within the Para ventricular nucleus and projects to limbic sites including the amygdala, ventral striatum, hypothalamus, nucleus accumbens and the mid brain (Sofroniew, 1983). Oxytocin facilitates the motivation to approach and engage others; it increases attention to, and the accurate perception of, salient social information; as well as improving social recognition. These are all essential processes in the formation of attachment bonds (Insel & Young, 2001). Conversely, there is emerging evidence that disruptions in oxytocin function are associated with impairments in social functioning and affiliative behaviours, including maternal bonding (Heinrichs & Gaab, 2007; Meyer-Lindenberg, Domes, Kirsch & Heinrichs, 2011). Given its well-researched, central role in initiating and maintaining successful bonding behaviours in non-human mammals (Insel, Krasnegor, & Bridges, 1990; Insel, Shapiro, Pedersen, Caldwell, & Jirikowski, 1992; Pedersen, Caldwell, Jirikowski, & Insel, 1992), oxytocin is proposed to also play an important role in the understanding and management of disrupted parent-child bonding in humans.

Baseline levels of peripherally measured plasma oxytocin are higher in women demonstrating positive bonding behaviours. Circulating plasma levels of oxytocin demonstrate high intra-individual stability throughout the pregnancy (Feldman, Weller, Zagoory-Sharon, & Levine, 2007; Gordon, Zagoory-Sharon, Leckman, & Feldman, 2010) and

mothers whose levels peak around the birth report the greatest attachment to their unborn babies (Levine, Zagoory-Sharon, Feldman, & Weller, 2007). Around one month following the birth, these mothers demonstrate more maternal behaviours namely eye gaze, positive vocalisations, positive affect and affectionate touch. Moreover, mothers with higher levels of oxytocin also report more positive mental representations of their infants (Feldman, et al., 2007). Across the first six months of the infant's life both the levels of oxytocin and maternal behaviour are conserved, demonstrating a strong positive correlation at this time (Gordon, et al., 2010). Overall, plasma studies carried out in humans thus far mimic results from exhaustive investigations of the role of the neuropeptide in animals such as rats and voles with higher levels of oxytocin facilitatating species specific forms of maternal behaviour in the presence of infants.

4. Oxytocin as a mediator of the intergenerational transmission of attachment styles

Impairment in bonding not only affects the child's immediate psychological and cognitive development, it compromises neural functioning associated with future interpersonal relationships across the lifespan, including with one's own offspring. That is, poor relationship quality with a parent can irreversibly alter neuroanatomical structures in the brain that subsequently reduce the capacity for reinforcement value derived from interacting with one's own infants (Strathearn, Fonagy, Amico, & Montague, 2009; Swain, Lorberbaum, Kose, & Strathearn, 2007). Co-ordinated peripheral oxytocin release in the presence of infants alters as a function of maternal attachment style. Strathearn et al., (2009) found that only mothers with a secure attachment style demonstrated a peak in oxytocin levels during an interaction with their infants, measured when their infants were around seven months old. Mothers with an insecure attachment style showed a decrease in oxytocin levels during these interactive play sessions.

Thus, it seems that a mother's attachment style predicts her level of oxytocin release during interactions with her infant. More substantially, this oxytocin release co-occurs with periods of high levels of touch and affect-synchrony during play sessions, which directly influences her infant's concomitant release of oxytocin. Oxytocin levels in the mothers are mirrored by their infant's salivary oxytocin levels. The reactivity of both parties oxytocin system in response to one-to-one contact is also comparable, with the change scores from pre-post being significantly correlated (Feldman, Gordon, & Zagoory-Sharon, 2010). Furthermore, oxytocin levels are only increased in mothers who engage in high levels of affectionate touch during episodes of close infant contact (Feldman, Gordon, Schneiderman, Weisman, & Zagoory-Sharon, 2010) and increased oxytocin responsiveness is correlated with affect synchrony and infant social engagement (Feldman, Gordon, & Zagoory-Sharon, 2010). As physical touch in other contexts has been found to increase oxytocin levels, eg., during warm partner contact (Grewen, Girdler, Amico, & Light, 2005), 'affectionate touch' may be the necessary and sufficient transmitter of oxytocin levels between mother and infant which influence the responsiveness of the infants' oxytocin system. Oxytocin plasma levels and their pattern of release are transferred to the infant who subsequently adopts a similarly high frequency affectionate touching style with their own offspring.

Although human parenting behaviour is arguably more complex than behaviour seen in non-human animals, this theory is supported by animal data which demonstrates that oxytocin mediates mother-infant bonding through the act of maternal grooming, a behaviour analogous to affectionate touch in humans (Francis, Young, Meaney, & Insel, 2002). Maternal behaviour of increased licking and grooming of pups is associated with increased oxytocin receptor expression in rats. This behaviour is associated with reduced anxiety and better care overall for rat pups, akin to maternal sensitivity in humans (Francis, Champagne, & Meaney, 2000). Furthermore, their offspring resemble them behaviourally and neurobiologically through similarly reduced stress reactivity, denser oxytocin receptor expression in their brains and higher rates of maternal licking and grooming when they rear offspring, compared to pups of low licking/grooming mothers (Champagne, Diorio, Sharma, & Meaney, 2001; Francis, Diorio, Liu, & Meaney, 1999).

Due to the ability to manipulate rearing experiences through cross-fostering studies, the research group were able to show conclusively that environmental factors, in this case parenting style, can alter oxytocin expression in the brain and the subsequent parenting style adopted by offspring. The above pattern was reversed when female pups that were bred to high licking and grooming mothers were raised by low licking and grooming mothers. These infants then displayed the lower oxytocin receptor binding expected of the low licking grooming pups and went on to use the new low licking grooming style with their offspring (Champagne, 2008). This sequence of studies nicely depicts in one species the ever more complex gene by environment interactional explanations required to sequence the development of psychopathology (Champagne, 2011). From these studies we understand that the underlying genetics and maternal environment each play a critical role in predicting which maternal style infants ultimately adopt when they become parents themselves.

Parenting styles in humans have likewise been linked to underlying genetic susceptibility with mothers carrying the less efficient variant of the oxytocin receptor gene demonstrating poorer sensitivity towards their toddlers; acting as a less supportive presence to their children during a problem-solving task (Bakermans-Kranenburg & van Ijzendoorn, 2008). With genetic makeup determining oxytocin expression and parenting style directly influencing oxytocin responsiveness we can argue for a similarly complex gene by environment interplay in humans which ultimately transfers both parenting bonding style and oxytocin receptor expression through levels of affectionate touching. During face to face engagement with one's own infant, mothers differentially respond to social contact dependent upon their reported attachment style with their own parents. At the level of behavioural engagement with the infant, it has been noted that mothers whose oxytocin levels increase during the play engage in affectionately touching and stroking their infants. This would suggest that there is a feedback loop with oxytocin increasing as a function of touch for both the mother and the baby. The proposed mechanism by which this behaviour may be maintained is positive reinforcement derived through activation of the ventral striatum; the interface between mesolimbic dopaminergic and oxytocin systems (Skuse & Gallagher, 2009). A four month follow up of mothers whose oxytocin spiked during play sessions revealed a stronger ventral striatum response to both happy and sad expressions on their infant's face, compared to mothers with insecure attachment styles whose oxytocin levels dipped during the play session. The authors suggested that activation of this reward

centre in the brain renders all stimuli associated with one's own child more rewarding, invoking maternal responsiveness to the infant's needs for securely attached mothers. The regulation of the oxytocin system *in the presence of relevant social stimuli* is therefore important: It was only *in the presence* of their infants that differences were observed in oxytocin levels, as both groups of mothers displayed similar baseline oxytocin levels. Furthermore, it must be *relevant social stimuli* as no differences were seen across all mothers, in ventral striatum activation, when they viewed infants who were not their own (Strathearn, Li, Fonagy, & Montague, 2008).

5. Perinatal anxiety, maternal bonding and oxytocin

Approximately 5-12% of women suffer from clinically diagnosed anxiety around the birth of their child, although sub clinical levels of distress which impact women's experiences of parenting may affect as many as 20-25% of mothers (Lonstein, 2007). Heightened maternal anxiety directly affects early mother-infant interactions (Barnett & Parker, 1985; Kaitz, Maytal, Devor, Bergman, & Mankuta, 2010). Anxiety during the pregnancy itself has the potential to significantly alter parenting such that it remains the single biggest predictor of children's later behavioural and emotional problems and heavily influences the mental competencies of the child (O'Connor, Heron, & Glover, 2002; O'Connor, Heron, Golding, Beveridge, & Glover, 2002). Some mothers find it hard to bond and connect to their new baby, and such failure in the context of anxiety may have long-term effects on the infant (Barnett & Parker, 1986). In this regard, an investigation of parental rearing styles conducted in six European countries found that low parental care and maternal overprotection were linked to anxiety disorders in the offspring, across the countries studied (Heider et al., 2008). It may be through the mechanism of overprotection that transmission of anxiety from mother to child occurs, with higher rates of anxiety diagnoses in children whose mothers suffer from an anxiety disorder (Schreier, Wittchen, Hofler, & Lieb, 2008).

Anxiety experienced by children with regard to separation from central attachment figures is of particular relevance in theoretical models of the transmission of attachment styles across generations. Individuals with separation anxiety have extreme anxiety about separations, actual or imagined, from significant others, usually, but not always, attachment figures (Manicavasagar, Silove, Curtis, & Wagner, 2000; Silove, Manicavasagar, & Drobny, 2002). Childhood separation anxiety is a DSM classified disorder (American Psychiatric Association, 2000). Anxiety specifically focused upon bonding with attachment figures in childhood activates a mental model of separation anxiety resulting in insecure attachment patterns of behaviour practiced throughout their lifetime (Manicavasagar, Silove, Marnane, & Wagner, 2009). Hence, researchers have argued for the continuity of separation anxiety in to adulthood, namely 'Adult Separation Anxiety Disorder' (Manicavasagar, Silove & Wagner, 2000). It has been suggested that there is a potential interaction between such anxiety and attachment style, with mothers having insecure attachment representations reporting heightened levels of maternal separation anxiety (Lutz & Hock, 1995). Mothers who report secure forms of attachment and who demonstrate more sensitive behaviour towards their infants are less likely to have children who develop separation anxiety (Dallaire and Weinraub, 2005). Thus, outcomes for children are mediated by maternal sensitivity. Support for this assertion comes from a finding that mother's mental representations of the attachment relationship were predictive of both their own level of

maternal responsiveness and their child's attachment security demonstrated in the strange situation procedure at 18 months (Fonagy, Steele, & Steele, 1991). Maternal anxiety therefore exerts influence over a variety of crucial cognitive and behavioural repertoires in the mother, affecting subsequent mother-infant interactions and infant outcomes.

A complex gene-environment interaction has been proposed in the pathogenesis and intergenerational transmission of anxiety disorders (Eapen et al., 2005; Manicavasagar et al., 2001; Silove et al., 1995), a relationship which may be mediated through specific neuroendocrine modulators, including oxytocin and the hypothalamic-pituitary-adrenal (HPA) axis (Lonstein et al., 2007). Separation anxiety disorder in humans has been linked to a polymorphism of the oxytocin receptor gene (Costa et al., 2009) and one study has shown lower circulating levels of plasma oxytocin in patients with co-morbid depression and anxiety (Scantamburlo, et al., 2007). However, the majority of research has been carried out in non-human animals and supports the role of oxytocin in attenuating stress responses (eg. Lim and Young, 2006). A negative correlation between the levels of oxytocin and cortisol is not consistently observed, leading to speculation of a more complex interaction between oxytocin and the HPA axis. Instead of supporting a linear relationship between oxytocin and cortisol levels, research thus far appears to support a 'dysregulation' of both systems which results in inter-individual variability in how these systems respond during periods of stress and social affiliation. In reaction to stressful situations, peripheral oxytocin is typically released to attenuate stress (Nishioka et al., 1998; Wotjak et al., 1998). Although the mechanism of action is not entirely clear, it is suggested that high central oxytocin activity is anxiolytic and that such anxiolytic effects may partly be mediated through oxytocin's ability to modulate the release of excitatory amino acids (Ebner et al., 2005), and GABA (Brussaard, Wossink, Lodder, & Kits, 2000). Intracerebral oxytocin release in response to anxiogenic stimuli depends on personal past experiences and the inherent responsiveness of the mother's HPA axis (Bosch et al., 2005). In this regard, oxytocin knock-out mice exhibit more anxious behaviours and they also demonstrate reductions in maternal care toward their offspring (Mantella, Vollmer, Li, & Amico, 2003; Pedersen, Vadlamudi, Boccia, & Amico, 2006). Thus, both a mothers' oxytocin profile and her HPA axis regulation are important factors in determining her level of stress and social approach behaviours which may be directly transmitted to the foetus during periods of high antenatal anxiety (Huizink, Mulder, & Buitelaar, 2004).

We have seen that maternal attachment style predicts the responsiveness of the oxytocin system in the presence of infants, providing support for oxytocin facilitating positive social engagement and increasing the reward from the interaction. However, oxytocin release may be triggered by the stress of the situation and be acting as an anxiolytic (Uvnäs-Moberg, 1998). At this stage enough is not known about the direction of action between oxytocin and the HPA axis (Bicknell, 2003). Women with the highest rates of trait anxiety who may be the most likely to benefit from the anxiolytic effects of continual infant contact, are the least likely to initiate and persist in infant contact, including breastfeeding (Clifford, Campbell, Speechley, & Gorodzinsky, 2006; Forster et al., 2006; Papinczak & Turner, 2000). The lack of breastfeeding reduces the release of oxytocin by their infants suckling (Matthiesen, Ransjö-Arvidson, Nissen, & Uvnäs-Moberg, 2001) and as a lack of breast feeding is associated with decreased levels of oxytocin (Grewen, Davenport, & Light, 2010; Light et al., 2000; Mezzacappa & Endicott, 2007) these mothers would not be receiving the beneficial

anxiolytic effects from breastfeeding. However, there is limited and inconclusive evidence in humans that physical contact with infants, even without breastfeeding, is an important contributor to positive mood and reduced anxiety in mothers (Lonstein, 2007).

6. Perinatal depression, maternal bonding and oxytocin

Between 10-20% of mothers suffer from perinatal depression (O'Hara & Swain, 1996) and many more experience subclinical levels of depression around the birth of their child. When identified risk factors for depression are controlled for, women who have given birth are 1.6 times more likely to develop depression as compared to women who have not had children (Eberhard-Gran, Eskild, Tambs, Samuelsen, & Opjordsmoen, 2002). Regarding the aetiology of depression in the postpartum period, similar risk factors to other episodes of depression have been identified, including: a past history of depression; previous treatment for emotional problems; a poor relationship with the partner; and experiencing a large number of negative life events in the previous 12 months (Eberhard-Gran, et al., 2002; Leigh & Milgrom, 2008; Milgrom et al., 2008; Whiffen & Gotlib, 1993). However, primiparity and antenatal anxiety are risk factors unique to postnatal depression, independent of previous episodes of depression (Eberhard-Gran, et al., 2002; Leigh & Milgrom, 2008). Twenty-nine per cent of women who experience antenatal depression also report significant depressive symptoms in the postpartum period (Milgrom, et al., 2008). Furthermore, mothers who report excessive anxiety or worry during the antenatal period are more likely to report postnatal depression (Austin, Tully, & Parker, 2007). Cooper and colleagues (2007) failed to find significant phenomenological differences in the clinical symptom presentation for three groups of women who were all suffering from recurrent episodes of clinical depression: those who were currently suffering from postnatal depression; those who had suffered from an episode of postnatal depression; or mothers who were suffering recurrent episodes of depression but had not had an episode following the birth of their child/ren (Cooper et al., 2007). Some minor differences were noted in that postnatally depressed women were less likely to report poor appetite, morning wakening and slowed activity. However, the authors noted that this could be explained by a general increase in appetite during breastfeeding, generally disrupted sleep after the child's birth and increased activities relating to childrearing. Mixed results for symptom course and duration are reported with some studies finding a longer duration for depressive episodes following the birth of a child whilst others reporting that depressive episodes across their sample had a similar pattern of remission within a six-month period (Cooper, et al., 2007; Kumar & Robson, 1984; Whiffen & Gotlib, 1993). Whiffen and Gotlib (1993) also observed that episodes of postnatal depression are generally milder than other episodes of depression.

Therefore, for the most part, postnatal depression closely resembles other episodes of depression. The most noteworthy difference is that the psychiatric disturbance occurs at a critical period in the infant's development, which singles out maternal postnatal depression as a worthwhile enterprise for research attention. Depression experienced by a parent at any time impacts on a child's development, however it is even more potent at this early stage in development when an infant is acquiring the basic skills required for all subsequent language, cognitive, behavioural, social and emotional development. Postnatal depression has been found to have adverse consequences across multiple domains of the mother's life. Higher levels of postnatal depression have been associated with reduced maternal sleep

(Meltzer & Mindell, 2007) and increased parental stress (Milgrom & McCloud, 1996). Depressed mothers do not experience the joys and challenges of parenting in the same way as non-depressed mothers; reporting lower levels of competence, poorer emotional attachment to their infant, poorer health and more restricted and more socially isolated feelings than non-depressed mothers. Furthermore, they were more likely to report poorer marital quality, higher tension, confusion, anger, fatigue and lower levels of vigour than non-depressed mothers (Milgrom & McCloud, 1996). Although self-reported depression decreased over the study period of 3-12 months, negative ratings of their relationship with their child and their spouse were maintained.

Mothers who suffer from postnatal depression also interact differently with their infants. This has been well documented in numerous studies comparing the behaviour of depressed and non-depressed women. They demonstrate less sensitive, less affirming and more negating behaviours towards their infants (Murray, Fiori-Cowley, Hooper, & Cooper, 1996) and are more likely to show increased negative affect, less imitation and game playing, and less contingent responses (Field et al., 1985). Affectionate contact is also reduced and there are less vocalisations and responses to the infant's vocalisations (Fleming, Ruble, Flett, & Shaul, 1988). On the whole, they demonstrate less sensitive responding to their infant's cues (Cox, Puckering, Pound, & Mills, 1987).   Evidence exists for mother's negative attitudinal and behavioural repertoires noticeably altering infant's behaviour over the short and long term. Infants of postnatally depressed mothers show less engagement (Murray, Fiori-Cowley, et al., 1996), they are drowsier and fussier, and they appear less relaxed and show less content expressions (Field, et al., 1985) when observed during early interactions with their mothers. Their performance on object concept tasks are impaired at 12 months (Murray, 1992) and this finding has been supported by other follow up studies which observed these infants to be suffering from cognitive developmental delays compared to their age matched peers (Lyons-Ruth, Zoll, Connell, & Grunebaum, 1986; Murray, Fiori-Cowley, et al., 1996). When infants were assessed at 19 months, those with a mother who had suffered from postnatal depression during their first year were found to be angrier, they showed less affectionate sharing and were less sociable with strangers (Stein et al., 1991). These children were also more at risk for developing emotional and behavioural problems during early childhood (Billings & Moos, 1983; Lynne, 1992; Murray, 1992; Murray, Hipwell, Hooper, Stein, & Cooper, 1996).

The negative observed outcomes at 18-19 months were also demonstrated in infants whose mother's depression remitted within three to six months (Murray, 1992; Stein, et al., 1991), indicating that it is the changed interaction style established between a mother and her infant during this time, which persists beyond the depressive episode, that is responsible for influencing infant outcomes. A five year follow up of children of depressed mothers supported this theory with maternal insensitivity predicting poorer outcomes for children, independently of the mothers' level of depression (Murray, Hipwell, et al., 1996). Mothers suffering from depression are also at an increased risk of developing bonding difficulties. A third of women with postnatal depression will report bonding problems with their infants, and even mild levels of depression are associated with increased bonding difficulties (Brockington, et al., 2001; Moehler, Brunner, Wiebel, Reck, & Resch, 2006). A subsample of these women will report severe rejection of, and anger toward, their infant (Loh & Vostanis, 2004).

Children whose mothers experience bonding difficulties and respond less sensitively to them aren't as securely attached to their mothers around two years of age (Bakermans-Kranenburg, et al., 2003). In addition, exposure to maternal depression early in life is an independent significant predictor of poorer attachment (Martins & Gaffan, 2000). All of the researchers who have investigated this relationship have concluded that depression in the mother has significant adverse outcomes on the infant's attachment to her and therefore results in less secure forms of attachment as measured by the strange situation procedure, or a variant thereof (Lyons-Ruth, et al., 1986; Murray, Fiori-Cowley, et al., 1996; Radke-Yarrow, Cummings, Kuczynski, & Chapman, 1985; Teti, Gelfand, Messinger, & Isabella, 1995)..

The link between oxytocin and depressive symptoms has not been illuminated with research returning either null findings or a positive correlation between the two (Scantamburlo et al., 2007; van Londen et al., 1997). Researchers have speculated upon a link with oxytocin as it has direct effects upon the HPA axis, the dysregulation of which is heavily implicated in major depressive disorder (Pariante & Lightman, 2008). Cyranowski et al., (2008) found that depressed women did not display the same pattern of oxytocin release during emotional and stress inducing tasks, arguing for the dysregulation of oxytocin release in these women. Recent evidence supporting a negative correlation between oxytocin and depressive symptoms has emerged from a study directly investigating women's oxytocin levels during late pregnancy. Skrundz and colleagues (Skrundz, Bolten, Nast, Hellhammer, & Meinlschmidt, 2011) found that in approximately 8/10 cases they were accurately able to predict a women's risk of developing postnatal depressive symptoms at two weeks postpartum from an analysis of her oxytocin levels. Women with lower levels of oxytocin were susceptible to low mood in the early postpartum. Some of these women are prone to developing bonding difficulties with their infant and this data ties in with Feldman's results of decreased oxytocin levels being associated with poorer bonding behaviours around this time (Feldman, 2007; Feldman, et al., 2007).

This relationship is further complicated by breastfeeding status in these women as we have seen that oxytocin levels increase during feeding and depressed mothers cease breastfeeding earlier than mothers who have not suffered from postnatal depression. Ninety-three percent of these women report ceasing breastfeeding at the time of, or after the onset of postnatal depression (Henderson, Evans, Straton, Priest, & Hagan, 2003). Additionally, mothers who are not breastfeeding report higher levels of postnatal depression than those who do breastfeed (Eberhard-Gran, et al., 2002). Therefore, mothers with depression may not be receiving the beneficial effects of increased oxytocin (Grewen, et al., 2010; Matthiesen, et al., 2001), and it may be that this is due to a neurobiological deficit in naturally circulating levels of oxytocin which acts as a risk factor for both postnatal depression and breastfeeding difficulties. This remains speculative as longitudinal research is yet to be carried out within this population.

7. Conclusion

It is plausible that mothers with anxiety and depression mediated through low levels of oxytocin would be particularly vulnerable to negative mother-infant interactions as they find the interactions with their child less rewarding or more anxiety provoking. These patterns may set the stage for the development of psychopathology in the developing child leading to adverse mental health outcomes in adulthood. Affected adults in turn will have

attachment difficulties in rearing their own infants creating a transmission of parenting styles across generations. The investigation of oxytocin in human maternal affiliation and separation behaviours is in its early stages. However there is emerging evidence to implicate oxytocin in patterns of maternal bonding, with higher oxytocin levels in mothers with secure attachment rendering stimuli associated with their own child more rewarding, thereby facilitating maternal sensitivity and responsiveness and improving the quality of bonding and attachment with the infant. Nevertheless, the role of maternal mood in this process is less clear and further studies examining relevant neurobiological parameters, especially the association of maternal anxiety, depression and attachment behaviours with oxytocin, and other indices of hypothalamic-pituitary-adrenal axis functioning, are indicated. Such research would open up the possibility of early psychological interventions and pharmacological therapies targeting oxytocin and the HPA axis that would improve maternal mood and prevent the development of potentially maladaptive mother-infant interactions resulting in better maternal and infant outcomes.

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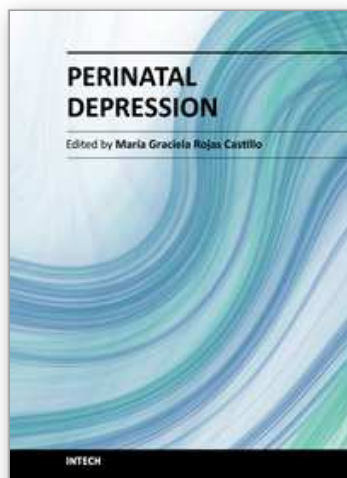
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Perinatal Depression

Edited by Dr. María Graciela Rojas Castillo

This book presents ten chapters that give us important information about epidemiological, biological, clinical and psychological aspects of common mental disorders during pregnancy and in the postnatal period. Some of the issues covered in this book are: detecting postnatal depression using different instruments at the right time, which is very important to avoid the negative effects on the children of depressed mothers; understanding the impact of anxiety and depression during pregnancy and in the postnatal period; biological issues of perinatal anxiety and depression; epidemiological information about perinatal mental health problems among minorities, like immigrant population and underserved rural women. Some information is also provided on postnatal depression in men, which is frequently overlooked.

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