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The maternal brain and its plasticity in humans

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ABSTRACT

This article is part of a Special Issue "Parental Care".

Early mother–infant relationships play important roles in infants' optimal development. New mothers undergo neurobiological changes that support developing mother–infant relationships regardless of great individual differences in those relationships. In this article, we review the neural plasticity in human mothers' brains based on functional magnetic resonance imaging (fMRI) studies. First, we review the neural circuits that are involved in establishing and maintaining mother–infant relationships. Second, we discuss early postpartum factors (e.g., birth and feeding methods, hormones, and parental sensitivity) that are associated with individual differences in maternal brain neuroplasticity. Third, we discuss abnormal changes in the maternal brain related to psychopathology (i.e., postpartum depression, posttraumatic stress disorder, substance abuse) and potential brain remodeling associated with interventions. Last, we highlight potentially important future research directions to better understand normative changes in the maternal brain and risks for abnormal changes that may disrupt early mother–infant relationships.

Introduction

Maternal caregiving plays a critical role for infant survival and optimal development. After birth, the brain and body of mothers undergo dynamic changes to support the establishment and maintenance of maternal caregiving behaviors. In this review, we focus on neuroimaging studies that revealed neural plasticity among human parents using functional magnetic resonance imaging (fMRI). While animal literature elegantly demonstrate neural plasticity due to pregnancy, parturition, and caregiving (Cohen and Mizrahi, 2015; Fleming et al., 2002; Leuner et al., 2010), our review will be mostly limited to neural plasticity across the postpartum period beyond birth based on available literature in human parents. This review aims to contribute to the current literature by providing an overview of individual differences in brain plasticity and multi-dimensional factors that are associated with such individual differences. We will first review several important sets of maternal brain networks that support sensitive responses to infants. Auditory and visual signals from infants, such as infant cry sounds and infant images, activate brain regions from emotion response and regulation circuits to executive function and attention cortical circuits that function in parental thoughts, empathy and sensitive behavior. Second, we will review environmental, behavioral and hormonal factors that may relate to variations in maternal brain responses to infants. We will also discuss psychopathology in mothers — that may be considered as maladaptive plasticity, including postpartum depression (PPD), posttraumatic stress disorder (PTSD) and substance abuse. Identifying problems with maternal brain plasticity may ultimately offer hope for effective treatments for concerned parents with the development of brain-based targeting of interventions.

Neurocognitive mechanisms underlying parental sensitivity

Sensitive caregiver responses to infant's cues involve an array of complex thoughts and behaviors contingent on infant cues, including recognition and acknowledgment of the child's signals, attribution of salience to the child's cues, maintenance of visual contact, expression of positive affect, appropriate empathy mirroring and vocal quality, resourcefulness in handling the child's distress or expanding the interaction, consistency of style, and display of an affective range that matches the infant's readiness to interact. Such behaviors are likely the result of complex and highly plastic neural networks involved in generating and organizing emotional responses (Kober et al., 2008), as well as dissociable and volitional attention and executive function, reward and motivation, and sensorimotor circuits (Buckner et al., 2008; Seeley et al., 2007; Sripada et al., 2014). At this point, we propose a

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model for maternal brain function based on task-based imaging studies (Swain et al., 2014b; Swain and Lorberbaum, 2008) (Fig. 1) and highlight brain processes that are mechanistically related to certain cortico-limbic circuits and are relevant for healthy parental sensitivity. The studies we reviewed largely use block design of infant stimuli and include analyses focusing on specific regions rather than connectivity. Thus, we would like to acknowledge that our model is necessarily over-simplistic and will require much more work to describe the physiology of key circuit elements, plus the connectivity between them which may itself also be plastic or flexible according to the demands of different stages of parenting and circumstances of psychopathology.

Reward/motivation

Both animal and human research (Numan and Woodside, 2010; Strathearn et al., 2009a) suggests that responses to infants form a model motivational system using dopamine (DA) and oxytocin (OT)-rich pathways. DA contributes importantly to reward-motivated behaviors, reinforcement learning, and drug addiction. Dopaminergic neurons, which originate in the brainstem's ventral tegmental area and substantia nigra, project to the ventral and dorsal portions of the striatum, as well as to the medial prefrontal cortex (PFC), via mesocorticolimbic and nigrostriatal pathways. Natural reward-related stimuli, including food, sex, and faces of one's sexual partner or child, activate the brain's "reward" system (Aharon et al., 2001; Delgado et al., 2000; Melis and Argiolas, 1995; Stoeckel et al., 2008; Strathearn et al., 2008). In mothers, the initial experiences of pleasure and activity in these brain circuits when exposed to their own infants' cues may increase the salience of their infants' stimuli and promote greater attention and bond-formation to ensure continuous engagement in sensitive caregiving (Strathearn et al., 2008, 2009a). Such reward pathways may be relevant very early in the postpartum period, as a mother's positive feelings toward her unborn fetus, as well as her perception of her fetus, have been associated with greater maternal sensitivity to the infant's signals and more affectionate vocalizations and touch (Keller et al., 2003; Keren et al., 2003).

The amygdala also interacts with the reward circuit to motivate maternal behaviors. OT receptors are also abundantly present in the amygdala (Viviani et al., 2011). In rodents, during the postpartum period, the increased basolateral amygdala activation provides sensory inputs to the reward circuit including the nucleus accumbens (NAcc) and ventral pallidum (Numan, 2014; Numan and Woodside, 2010). In response to infant stimuli, infant cry and smiles activate the amygdala (Barrett et al., 2012; Seifritz et al., 2003; Swain et al., 2008), which has often been interpreted as a sign of emotional salience (Seifritz et al., 2003; Strathearn and Kim, 2013) or positive emotion associated with attachment (Leibenluft et al., 2004). On the other hand, in virgin rats, activation in the medial nucleus of the amygdala was associated with reduced maternal behaviors (Morgan et al., 1999; Oxley and Fleming, 2000). Thus, while increased activation of the amygdala to infant stimuli is interpreted as a more negative response to infants among typical adults (Riem et al., 2011), in mothers, it can be associated with more positive responses to one's own infant (Barrett et al., 2012).

Another perspective on maternal motivations and rewards involve preoccupations that may be part of healthy maternal responses to their infants that draw them close in order to meet the infant's physical and psychological needs (Bowlby, 1969; Winnicott, 1956). This suggests the importance of "checking and worrying" brain circuits overlapping with those hyperactive in obsessional anxiety (Leckman et al., 2004). Indeed, parental anxiety peaks immediately after childbirth and then begins to diminish during the first three to four months postpartum (Feldman et al., 1999; Kim et al., 2013; Leckman and Mayes, 1999). This matches apparent increased responses to baby cry in postpartum anxiety circuits including the basal ganglia and orbitofrontal cortex that diminish over the first 4 months postpartum (Swain et al., 2014b, under review). This plasticity in anxiety circuits may be part of a healthy range of threat detection and harm avoidance (Feygin et al., 2006), yet also perhaps an opportunity for problems to occur with such adaptations — in which abnormally reduced or excessive worry may be part of postpartum psychopathology.

Emotion regulation

During interactions with an infant, particularly a distressed infant, it is critical for mothers to perceive the distressed cues of infants appropriately, and manage their own distress in response to their infants' negative emotions. A mother's sensitivity to distress has been a better predictor of the child's outcome than her sensitivity to non-distress cues (Joosen et al., 2012; Leerkes, 2011; Leerkes et al., 2009; McElwain and Booth-Laforce, 2006).

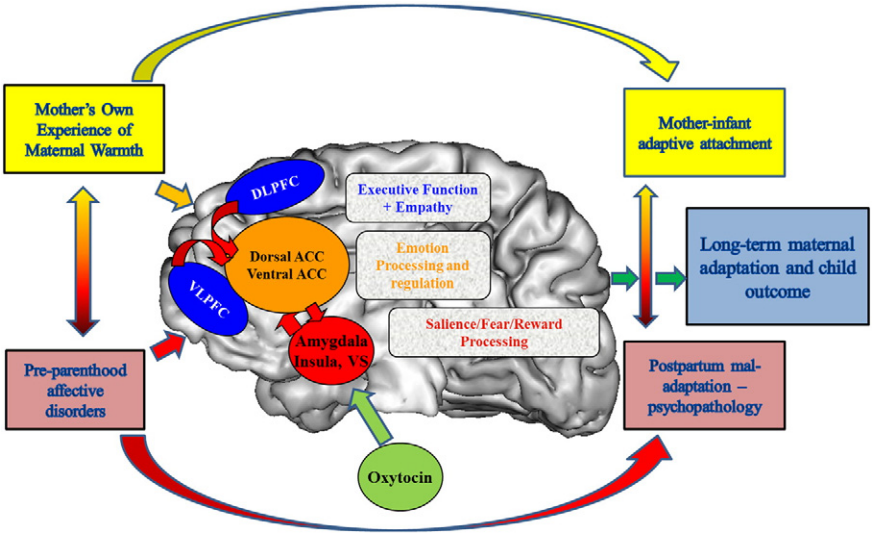

Fig. 1. Plasticity in the maternal brain–early life factors, such as experience of parental warmth and previous mental health affect plastic brain circuits that ultimately regulate maternal affective regulation capacity and caregiving outcomes. Plastic or adaptable circuits, some of which are overlapping, include those for Emotion Response and Processing [Amygdala, Dorsal Anterior Cingulate Cortex (ACC), Ventral ACC] and Salience/Fear/Motivation Processing [Amygdala, Insula, Ventral Striatum (VS)] working with cortical executive function [Ventrolateral Prefrontal Cortex (VLPFC), Dorsolateral Prefrontal Cortex (DLPFC)] and empathy [Medial Prefrontal Cortex (MPFC), Precuneus, Superior Temporal Sulcus] circuits.
Adapted from Moses-Kolko et al. (2014).