Effects of Maternal Stress on Maternal Behavior and Psychological Function

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Abstract

Becoming a mother represents a unique life stage for women, and the physiological and psychological adaptive changes occurring during this period hold significant importance for both maternal and offspring health. Maternal stress constitutes a critical factor that impedes the smooth progression of these maternal adaptive changes. Maternal stress disrupts maternal behavior, cognitive function, and emotion regulation in human mothers and female mammals; this impact is associated with dysregulation of endocrine systems including glucocorticoids, oxytocin, and prolactin; altered neural responses to stimuli in neural circuits such as the maternal circuitry, limbic system, and prefrontal cortex; as well as plasticity changes involving neurogenesis, dendritic and synaptic remodeling.

Full Text

Effects of Maternal Stress on Maternal Behavior and Psychological Function

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Abstract: The transition to motherhood represents a unique life stage during which women experience numerous physiological and psychological changes. These adaptive modifications are crucial for the health of both mother and offspring. Maternal stress represents a significant factor that impedes these adaptive processes. Maternal stress can impair maternal behavior, cognitive function, and emotional regulation in human mothers and female mammals. These disruptive effects are associated with dysregulation of endocrine systems including glucocorticoids, oxytocin, and prolactin; altered neural responses to stimuli in neural circuits such as the maternal circuitry, limbic system, and

prefrontal cortex; and plasticity changes in neurogenesis, dendritic remodeling, and synaptic restructuring.

Keywords: maternal stress; maternal behavior; cognitive function; emotional regulation

Pregnancy, childbirth, and lactation constitute a special period in women' s lives, characterized by extensive physiological and psychological changes. These adaptations are not only essential for ensuring offspring survival but also necessary for mothers to successfully cope with new environmental demands. Simultaneously, the perinatal period represents a high-risk time for psychiatric disorders in women. When normal physiological and psychological adaptations are disrupted, postpartum psychological dysfunction may result. Globally, approximately 5–12% of mothers experience postpartum anxiety, 5–25% develop postpartum depression, and 0.1% suffer from postpartum psychosis (Yang et al., 2015). These conditions severely impact maternal health and mother-infant relationships, exerting long-term negative effects on offspring. Therefore, understanding the factors and mechanisms underlying psychological dysfunction during pregnancy and lactation is urgently needed. Numerous clinical and animal studies have identified maternal stress as a critical contributing factor (Agrati & Lonstein, 2016; Duthie & Reynolds, 2013). However, most previous research on maternal stress has focused on its negative effects on offspring behavior and psychology, with less attention devoted to its impact on mothers' own behavioral and psychological functioning. This paper first briefly outlines maternal stress and its research methodologies, then discusses the effects of maternal stress on maternal behavior, cognitive function, and emotional regulation in human mothers and female mammals, along with advances in understanding the underlying neuroendocrine, circuitry, and plasticity mechanisms. By elucidating these effects and mechanisms, we aim to enhance understanding of dysfunctional mother-infant relationships and postpartum psychiatric disorders, and to establish a foundation for preventing and treating such behavioral and psychological dysfunctions.

1 Maternal Stress

Stress is a series of neuroendocrine responses dominated by sympathetic nervous system activation and increased pituitary-adrenal cortex secretion, triggered by various internal and external stimuli, which subsequently induces functional and metabolic changes (Rice, 1998; Yang, Hou, Yang, & Zhang, 2011). Based on its nature and severity, stress can be categorized as physiological or pathological. Physiological stress represents an adaptive defensive response to mild environmental changes and psychosocial stimuli that mobilizes organismal potential without causing serious harm. Pathological stress, conversely, causes non-specific damage and can lead to stress-related diseases (Zhao, Wang, Zhang, & Wang, 2011). For women, pregnancy, childbirth, and lactation themselves constitute a stressor that significantly reduces the responsiveness of the brain and endocrine systems to stressors (Brunton, Russell, & Douglas, 2008;

Pawluski, Lambert, & Kinsley, 2016). These adaptive changes benefit maternal and offspring health. The maternal stress discussed in this paper specifically refers to pathological stress experienced by women or female mammals during pregnancy through the postpartum lactation period.

In human research, maternal stress primarily involves psychosocial stressors including: stressful life events (e.g., domestic violence, adverse pregnancy outcomes, marital relationships, economic conditions), catastrophic events (e.g., hurricanes, earthquakes), daily hassles, parenting stress (e.g., excessive infant crying), subjective stress perception, and chronic strain (e.g., work stress, job insecurity) (Yim, Tanner Stapleton, Guardino, Hahn-Holbrook, & Dunkel Schetter, 2015). Human maternal stress is typically measured using stress-related scales that assign scores reflecting stress levels. For example, the Parenting Stress Index-Short Form (PSI-SF) assesses parenting stress, including feelings of distress from parenthood, unmet parental expectations, dysfunctional parent-child interactions, and perceptions of difficult child behavior. A total score above 90 indicates clinically significant parenting stress (Paris, Bolton, & Weinberg, 2009).

In rodent studies, common maternal stress paradigms include (Li & Chou, 2016): (1) gestational restraint, typically involving 1 hour of daily restraint from gestational days 10–20; (2) repeated maternal separation, where dams are separated from pups for 3–4 hours daily from postpartum days 3–14, modeling social relationship loss in humans; (3) chronic social stress, a chronic social defeat paradigm where an unfamiliar male intruder is placed in the cage for 1 hour daily during lactation days 2–16; (4) learned helplessness, involving 80 inescapable electric shocks on postpartum day 3; and (5) chronic corticosterone administration during gestation (days 10–20) or postpartum (days 2–24). Most animal studies of maternal stress employ one or a combination of these methods.

Although the severity of maternal stress's impact depends on the stressor, infant (offspring) stimuli, individual genetic susceptibility, and in humans, personality traits and social support (Agrati & Lonstein, 2016), maternal stress generally disrupts the expression of maternal behavior and affects cognitive function and emotional regulation.

2 Effects of Maternal Stress on Maternal Behavior

Maternal behavior constitutes a primary component of parental care, encompassing all activities through which females provide protection and maintenance for their own or related offspring—that is, all behaviors associated with parturition and offspring rearing. As an ecologically valid and complex behavioral system, human and animal maternal behaviors share many similar features (Fleming & Corter, 1988; Chen et al., 2017). This innate behavior is highly motivated and well-organized, enhancing offspring survival and playing a vital role in species continuity and population stability (Li, Sun, Zhang, & Hu, 2010; Numan, 2007).

Clinical studies on maternal stress and behavior have primarily examined moth-

ers with postpartum mood disorders, who typically exhibit higher stress levels and poorer maternal behavior than normal mothers. For example, mothers experiencing postpartum depression and suicidal ideation often cannot fulfill maternal responsibilities, show poor mother-infant bonding, engage in less touching and communication with infants, display fewer facial expressions, share less joint attention, and demonstrate reduced sensitivity and responsiveness to infant cues (Paris et al., 2009) (see Table 1). Additionally, maternal stress from preterm birth can trigger mood disorders, reducing care quality, increasing interference, decreasing sensitivity, and consequently elevating infant attachment insecurity (Castel et al., 2016). Ionio and Di Blasio investigated the relationship between postpartum PTSD symptoms and mother-infant interaction, finding that mothers with persistent postpartum PTSD made less eye contact with their infants and consistently described infant states negatively (Ionio & Di Blasio, 2014). Challacombe et al. found that mothers with postpartum obsessive-compulsive disorder lacked confidence, reported more marital problems and less social support than controls, were less willing to breastfeed, and showed lower sensitivity during infant interactions (Challacombe et al., 2016). Similar results have been found in postpartum anxiety; Nicol-Harper, Harvey, and Stein examined mother-infant interactions in standardized play scenarios at 10–14 months postpartum, revealing that high-trait-anxiety mothers displayed lower sensitivity than low-anxiety mothers (Nicol-Harper, Harvey, & Stein, 2007).

Livestock animals frequently experience maternal stress during husbandry, which reduces maternal behavior. For instance, frequent disturbance during mid-gestation increases time spent lying prone during early lactation, hindering piglets' access to milk and suggesting reduced nursing motivation in sows (Ringgenberg, Bergeron, Meunier-Salaun, & Devillers, 2012). Ewes roughly handled during late gestation showed increased lamb grooming within 24 hours postpartum, but fear reduced ewe-lamb proximity when humans were present (Hild, Coulon, Schroeer, Andersen, & Zanella, 2011). A study of captive gorillas found that stress from disorderly conditions and poor housing caused mothers to neglect their infants (Bahr, Pryce, Dobeli, & Martin, 1998).

In experimental animal research, rats and mice serve as common model organisms, with maternal behaviors including nest building, nursing, licking, and pup retrieval. Daily 1-hour restraint stress during gestational days 10–20 significantly reduced time spent in arched-back nursing and pup retrieval postpartum (Smith, Seckl, Evans, Costall, & Smythe, 2004). Similarly, postpartum maternal stress disrupts maternal behavior expression. Aguggia, Suarez, and Rivarola separated dams from pups for 4.5 hours daily beginning on postpartum day 1, continuing for 3 weeks until weaning, finding that separated dams took nearly 2.5 times longer to retrieve entire litters, showed difficulty focusing on pup-related activities (e.g., sniffing and carrying pups), and exhibited impaired nest building with more exploratory behavior. Their latency to first pup contact was also longer than controls, indicating hesitation in recognizing and caring for pups (Aguggia, Suarez, & Rivarola, 2013). Boero et al. subjected dams to gavage intubation during gestational days 12–20 and 3-hour maternal separation during

postpartum days 3–15, finding that combined gestational and lactational stress significantly reduced arched-back nursing, licking, nest building, and grooming, severely disrupting maternal behavior (Boero et al., 2018).

The disruptive effects of maternal stress on maternal behavior are closely linked to its impact on maternal cognitive function and emotional regulation.

3 Effects of Maternal Stress on Maternal Cognitive Function

Maternal cognitive function is crucial for child-rearing, enabling mothers to perceive and understand infant intentions and needs while adjusting their own behavior accordingly in complex environments with competing stimuli. Cognitive skills such as planning, organization, working memory, cognitive flexibility, sustained and appropriate selective attention regulation, and decision-making are considered essential foundations for parenting and key to maternal sensitivity. Human and rodent mothers outperform non-mothers on many cognitive tasks related to attention, spatial learning and memory, and working memory (Pereira & Ferreira, 2016). However, maternal stress or its induced mood disorders can disrupt these adaptive cognitive changes.

In human mothers, Meena, Soni, Jain, Jilowa, and Omprakash administered cognitive function, depression, anxiety, and stress scales to 200 mothers at 7 days postpartum, finding significantly higher depression, anxiety, and stress levels than controls. Cognitive testing revealed significantly lower scores in attention, processing speed, visual scanning, executive function, psychomotor performance, and perceptual organization, demonstrating clear cognitive deficits (Meena, Soni, Jain, Jilowa, & Omprakash, 2016). Hampson et al. screened pregnant women at 34–36 weeks gestation for depression and assessed working memory, finding that depression severity significantly predicted working memory performance—higher depression correlated with poorer working memory. Conversely, non-depressed pregnant women performed equivalently or even better than non-pregnant controls (Hampson et al., 2015). Kataja et al. divided second-trimester pregnant women into high, medium, and low emotional symptom groups (reflecting varying maternal stress levels) based on self-reported depression and anxiety symptoms, then tested cognitive function. Mothers with high and moderate emotional disturbances made more errors on visuospatial working memory and executive function tests (Kataja et al., 2017).

Research on maternal stress and cognitive function is limited and primarily uses rodent models. Mice subjected to restraint and bright light exposure three times daily for 45 minutes during mid-to-late gestation showed impaired olfactory memory, with maternal stress disrupting consolidation and retrieval processes (Belnoue, Malvaut, Ladeveze, Abrous, & Koehl, 2016). Additionally, mice exposed to intermittent 3000 Hz noise stress or combined restraint and elevated platform stress during gestational days 12–16 exhibited severe spatial learning and memory impairments at postpartum day 30, demonstrated by prolonged

swimming times, slower speeds, and reduced probe trial performance in the Morris water maze (Jafari, Mehla, Afrashteh, Kolb, & Mohajerani, 2017). Aguggia et al. found that prolonged postpartum maternal separation resulted in significantly shorter latencies in the step-down inhibitory avoidance task, indicating cognitive impairment affecting both short- and long-term memory (Aguggia et al., 2013). Sung et al. reported consistent findings, showing that 6-hour daily separation for 14 days produced shorter inhibitory avoidance latencies and reduced hippocampal neurogenesis with increased apoptosis, suggesting maternal stress affects neural circuits involved in memory acquisition, consolidation, and retrieval (Sung et al., 2010).

4 Effects of Maternal Stress on Maternal Emotional Regulation

Clinically, postpartum emotional dysregulation resulting from maternal stress, including anxiety, depression, and other psychiatric disorders, has garnered significant attention. Numerous studies indicate that women experience social and emotional stress during pregnancy that often persists into the postpartum parenting period, representing major risk factors for postpartum emotional disturbances. Hagen (1999) proposed that postpartum depression may occur in women lacking spousal support or experiencing difficult births, leading to infant neglect. Yelland, Sutherland, and Brown surveyed 4,366 mothers at 6 months postpartum, finding that stressful life events and social-medical problems during pregnancy and early postpartum were associated with high maternal anxiety and depression (Yelland, Sutherland, & Brown, 2010). Holditch-Davis et al. examined the relationship between stress and psychological disorders in 177 mothers of preterm infants, finding that preterm birth significantly increased maternal depression, anxiety, and PTSD symptoms (Holditch-Davis et al., 2009). Al Maghaireh et al. surveyed 310 parents of infants in neonatal intensive care units, finding that mothers had significantly higher stress levels than fathers, with stress levels positively correlating with anxiety and depressive symptoms (Al Maghaireh, Abdullah, Chong, Chua, & Al Kawafha, 2017). Conversely, social support from family, friends, partners, or institutions can reduce postpartum anxiety and depressive symptoms (Kong et al., 2013; Norhayati, Hazlina, Asrenee, & Emilin, 2015) and decrease PTSD symptoms in mothers with trauma histories (Ford & Ayers, 2011).

Animal studies show that chronic gestational stress, including restraint, overcrowding, and novel environments, increases avoidance behavior in dams that persists postpartum, disrupting the normal low-anxiety postpartum state and increasing anxiety-like behavior in elevated plus maze and light-dark box tests (Darnaudery, Dutriez, Viltart, Morley-Fletcher, & Maccari, 2004; Hillerer, Reber, Neumann, & Slattery, 2011; Maestripieri, Badiani, & Puglisiallegra, 1991). Repeated prolonged maternal separation similarly reduces open arm entries and time in the elevated plus maze, indicating increased anxiety-like behavior (Aguggia et al., 2013; Maniam & Morris, 2010). Maternal stress also enhances post-

partum depressive-like behavior. Dams exposed to repeated restraint stress during mid-to-late gestation show increased immobility in forced swim tests (Haim, Sherer, & Leuner, 2014; Leuner, Fredericks, Nealer, & Albin-Brooks, 2014; Salari, Fatehi-Gharehlar, Motayagheni, & Homberg, 2016; Smith et al., 2004). Postpartum dams subjected to repeated maternal separation or high-dose corticosterone injection also show significantly increased immobility (Boccia et al., 2007; Brummelte & Galea, 2010; Gobinath et al., 2018; Maniam & Morris, 2010; Sung et al., 2010). Gestational stress or repeated prolonged maternal separation provides effective animal models for postpartum depression research.

5.1 Neuroendocrine Mechanisms

The hypothalamic-pituitary-adrenal (HPA) axis is the primary endocrine system mediating physiological responses to stress, regulating glucocorticoid stress responses (cortisol in humans, corticosterone in rodents). During stress responses, corticotropin-releasing hormone (CRH) secreted by the hypothalamic paraventricular nucleus triggers pituitary adrenocorticotropic hormone (ACTH) release, which stimulates adrenal cortical glucocorticoid secretion into the bloodstream. Blood glucocorticoids exert negative feedback inhibition on hypothalamic CRH or pituitary ACTH release to control secretion and maintain homeostasis. During pregnancy, the maternal HPA axis undergoes dramatic adaptive changes, with blood cortisol increasing nearly threefold by late gestation (Jung et al., 2011). However, elevated cortisol downregulates hypothalamic CRH synthesis, reducing HPA axis responsiveness to physiological or psychological stressors in late pregnancy. Postpartum, maternal plasma cortisol concentrations decline, and HPA axis function gradually returns to pre-pregnancy levels (Duthie & Reynolds, 2013).

When organisms encounter stressors, the typical physiological response involves rapid glucocorticoid elevation followed by decline. Abnormal HPA axis stress responses, characterized by excessive glucocorticoid elevation and slow recovery, are considered vulnerability factors for future psychiatric illness, particularly depression (Checkley, 1996). Longitudinal cortisol assessments indicate relatively stable glucocorticoid levels during pregnancy (Voegtline et al., 2013). However, self-reported stress, including high anxiety, depression, anger, and daily hassles, can dysregulate the HPA axis, causing significantly elevated cortisol and CRH in mid-to-late pregnancy (Field & Diego, 2008; Valsarnakis et al., 2017). Schreier et al. collected hair from mothers exposed to prenatal stress and trauma, finding significantly higher cortisol content (Schreier et al., 2016). Nierop, Bratsikas, Zimmermann, and Ehlert found that increased cortisol responses to standardized psychosocial stress tests during prenatal care could predict postpartum depression, providing evidence that altered HPA axis reactivity during pregnancy may affect later mental health (Nierop, Bratsikas, Zimmermann, & Ehlert, 2006). Additionally, failure of the postpartum HPA axis to normalize is associated with mood disorders. For example, women with postpartum depression exhibit elevated cortisol and ACTH but substantially

reduced CRH (Duthie & Reynolds, 2013).

Animal studies show that dams exposed to gestational restraint, noise stress, or postpartum maternal separation exhibit increased plasma corticosterone, impaired learning and memory, increased anxiety- and depressive-like behavior, and negative maternal behavior (Jafari et al., 2017; Maniam & Morris, 2010; Smith et al., 2004). Chronic exogenous corticosterone administration during pregnancy or lactation reduces pup care, increases time away from the nest, decreases nursing duration, and increases depressive-like behavior (Brummelte & Galea, 2010). Similarly, high-dose cortisol administration in lactating marmosets impairs maternal motivation and disrupts normal maternal behavior expression (Saltzman & Abbott, 2009).

Normal maternal HPA axis hyporesponsiveness to stress is also regulated by oxytocin and prolactin. Oxytocin synthesized in hypothalamic paraventricular and supraoptic nucleus magnocellular neurons projects to the posterior pituitary for systemic release, while parvocellular neurons project directly to the amygdala, hippocampus, striatum, and brainstem. Oxytocin promotes uterine contractions during delivery and milk ejection during lactation, regulates postpartum maternal behavior, attenuates stress-induced glucocorticoid secretion, and buffers HPA axis activity. Zelkowitz et al. found that in high-risk mothers experiencing psychosocial stress, plasma oxytocin levels negatively correlated with depressive symptoms and positively correlated with maternal sensitivity —higher oxytocin was associated with fewer depressive symptoms and greater sensitivity during mother-infant interactions (Zelkowitz et al., 2014). Prolactin, secreted by the anterior pituitary, primarily promotes milk production. Central prolactin elevation inhibits stress-induced ACTH secretion, thereby reducing HPA axis activity (Kalyani, Callahan, Janik, & Shi, 2017). Research shows that pup stimulation positively correlates with prolactin release during nursing, reducing dams' HPA axis stress responses. Maternal separation prevents physical contact, reduces prolactin release, and induces anxiety-like behavior (Lonstein, 2005). Thus, oxytocin and prolactin likely play important roles in postpartum stress emotion regulation, and abnormalities in these hormone levels may constitute risk factors for postpartum mood disorders and impaired maternal behavior expression.

In summary, the endocrine systems of human mothers and female mammals undergo dramatic adaptive changes during pregnancy through lactation, primarily characterized by reduced HPA axis responsiveness to physiological and psychological stressors and activation of oxytocin and prolactin systems that buffer HPA axis stress responses and promote postpartum maternal behavior expression. However, maternal stress may alter brain oxytocin, prolactin, and HPA axis regulation, leading to abnormal maternal glucocorticoid levels, increased postpartum mood disorders, and disrupted mother-infant bonding.

5.2 Neural Circuitry Mechanisms

In rodents, specific brain regions in postpartum dams undergo structural and functional changes to enhance maternal motivation and responsiveness to offspring needs. These regions constitute the "maternal circuitry" (Numan, 2007), including the maternal motivation system (comprising the bed nucleus of the stria terminalis (BNST) and medial preoptic area (MPOA)) and non-specific motivation systems (including nucleus accumbens (NAc), ventral tegmental area (VTA), olfactory bulb (OB), medial amygdala (MeA), anterior hypothalamic nucleus (AHN), periaqueductal gray (PAG), and hypothalamic paraventricular (PVN) and supraoptic (SON) nuclei). The maternal circuitry interconnects with the limbic system and medial prefrontal cortex (mPFC) (Slattery & Hillerer, 2016). These brain regions collectively participate in initiating and maintaining maternal motivation, cognitive control, and emotional regulation, ultimately facilitating maternal behavior expression.

Nephew, Caffrey, Felix-Ortiz, Ferris, and Febo used functional magnetic resonance imaging to examine neural responses in lactating dams to male intruders, finding significant activation in the nucleus accumbens, periaqueductal gray, anterior cingulate cortex, anterior thalamus, basolateral amygdala, temporal cortex, prelimbic area, orbital area, and insula (Nephew, Caffrey, Felix-Ortiz, Ferris, & Febo, 2009). Haim, Albin-Brooks, Sherer, Mills, and Leuner subjected dams to restraint stress from gestational days 7–20, finding that dams exhibited depressive-like behavior in forced swim tests at postpartum day 21, with structural changes in nucleus accumbens shell and medial prefrontal cortex that could be reversed by selective serotonin reuptake inhibitors (Haim, Albin-Brooks, Sherer, Mills, & Leuner, 2015). Aguggia et al. found that repeated postpartum maternal separation increased central amygdala cell activity, correlating with increased anxiety-like behavior in the elevated plus maze (Aguggia et al., 2013). Additionally, postpartum maternal separation reduced hippocampal neurogenesis and increased apoptosis, associated with memory impairment (Sung et al., 2010).

The maternal circuitry is conserved across mammals. In human studies, Laurent, Stevens, and Ablow found that after mother-infant separation, reduced HPA axis stress responses to hearing their own infant' s cry were associated with greater activation in the periaqueductal gray, striatum, insula, orbitofrontal cortex, medial prefrontal cortex, and anterior cingulate—regions involved in maternal behavior and emotion regulation. Dysregulated physiological stress responses reduced neural responses to infant cries (Laurent, Stevens, & Ablow, 2011). Schechter et al. showed mothers with PTSD (12–48 months postpartum) videos of their children in helpless separation versus free play while conducting fMRI. When viewing separation videos, PTSD mothers showed overactivation of fear circuits that correlated with higher subjective stress ratings. Compared to healthy mothers, PTSD mothers showed enhanced activity in emotion regulation and empathy regions (bilateral anterior olfactory cortex, left caudate, left insula) and reduced frontal cortical activity (superior frontal gyrus, bilat-

eral parietal cortex) (Schechter et al., 2012). Moses-Kolko et al. found that mothers with postpartum depression showed significantly reduced left dorsomedial prefrontal cortex and amygdala activity when viewing fearful and angry faces. Reduced amygdala responses to negative stimuli correlated with depression severity and maternal attachment, suggesting postpartum depression may disconnect cortical-amygdala circuits (Moses-Kolko et al., 2010). These studies indicate that reduced neural responses in limbic and cortical structures to negative emotional stimuli are associated with maternal emotional dysregulation and insensitivity.

These human and animal findings demonstrate that maternal stress alters neural responses to stimuli in circuits comprising the maternal circuitry, limbic system, and prefrontal cortex. The maternal circuitry participates in activating maternal behavior through a typical model where the medial preoptic area stimulates VTA dopamine release to the nucleus accumbens, inhibiting GABAergic neurons' suppression of the ventral pallidum, thereby activating maternal responses via ventral pallidal output (Chen et al., 2017). The prefrontal cortex and hippocampus, involved in cognitive control, executive function, and memory, play crucial roles in perceiving offspring stimuli, understanding offspring needs, and making appropriate behavioral adjustments—forming an essential foundation for maternal behavior expression. Circuits comprising the amygdala, anterior cingulate, striatum, and insula primarily regulate emotion, empathy, and reward; maternal emotional dysregulation, empathy deficits, and reward loss severely impact maternal behavior expression. The maternal, cognitive, and emotional circuits overlap and interconnect, forming complex interactions (Pawluski, Lonstein, & Fleming, 2017). Thus, maternal stress-induced alterations in these circuits' neural responses collectively affect maternal cognitive function, emotional regulation, and behavior.

5.3 Neural Plasticity Mechanisms

The brains of female mammals undergo plasticity changes during pregnancy through lactation to ensure successful transition to motherhood. These changes include neurogenesis in the hippocampal dentate gyrus granule cell layer and lateral ventricle subventricular zone, plus dendritic and synaptic remodeling in the hypothalamus, hippocampus, olfactory bulb, medial prefrontal cortex, and nucleus accumbens (Hillerer, Jacobs, Fischer, & Aigner, 2014; Slattery & Hillerer, 2016).

Maternal stress affects these naturally occurring plasticity changes, with research primarily focused on rodents. Pawluski et al. examined effects of early gestational stress on hippocampal granule cell layer proliferation, immature neurons, and cell survival in late gestation, finding that stressed dams had significantly increased hippocampal cell proliferation and immature neuron numbers, but no effect on new cell survival over 20 days (Pawluski et al., 2015). Late gestational stress produced consistent results (Pawluski, van den Hove, Rayen, Prickaerts, & Steinbusch, 2011). Furthermore, Hillerer, Neumann, Couillard-Despres,

Aigner, and Slattery subjected postpartum dams to 12 days of restraint stress, finding increased hippocampal volume and proliferating cell numbers with elevated corticosterone, but no difference in surviving cell numbers at postpartum day 21 compared to unstressed controls. Further examination revealed higher cell death proportions in stressed versus unstressed dams (Hillerer, Neumann, Couillard-Despres, Aigner, & Slattery, 2014).

Beyond neurogenesis, maternal stress affects neuronal dendritic morphology and dendritic spine characteristics. Gestational stress causes hippocampal apical dendritic atrophy in late gestation, reducing branch points and dendritic length in CA3 (Pawluski, Valenca, et al., 2012). In late lactation, mPFC layer 2/3 pyramidal neurons show 13% and 18% reductions in apical and basal dendritic spines, respectively (Leuner et al., 2014). At postpartum days 7 and 21, dams exhibit reduced branch point numbers and shortened lengths of nucleus accumbens shell spiny neuron dendrites, with reduced spine density at day 7 (Haim et al., 2014). Postpartum maternal stress also disrupts hippocampal dendritic development and orientation to the dentate gyrus (Hillerer, Neumann, et al., 2014). High-dose postpartum corticosterone administration reduces complexity of hippocampal CA3 pyramidal cell basal dendritic arbors while increasing mushroom spine density in CA3 apical and basal regions (Workman, Brummelte, & Galea, 2013).

Although the precise functions of these plasticity changes in the maternal brain remain largely unclear, their scope suggests critical roles in maternal physiological and psychological health and offspring survival. Maternal stress-induced alterations in brain plasticity likely underlie changes in maternal behavior, cognition, and emotion. Previous research found that enhanced hippocampal-dependent learning in normal dams (Pawluski, Walker, & Galea, 2006) correlates with dendritic remodeling in hippocampal CA1 and CA3 regions (Pawluski & Galea, 2006). Better performance on attentional set-shifting tasks in postpartum dams corresponds with increased mPFC pyramidal neuron dendritic spines (Leuner & Gould, 2010). Moreover, stressed dams with fewer mPFC total intersections and thin spines show increased immobility in forced swim tests and reduced arched-back nursing (Leuner et al., 2014). Gestational stress-induced reductions in mPFC-NAc pathway dendritic morphology and spines may contribute to sustained postpartum depressive-like behavior (Haim et al., 2014). Similar to chronic stress effects, high-dose corticosterone administration during lactation reduces maternal behavior and correlates with decreased hippocampal CA3 dendritic complexity and increased mushroom spine density (Workman et al., 2013).

6 Summary and Outlook

The reviewed literature demonstrates common effects of maternal stress on maternal behavior and psychological function in human mothers and female mammals: reduced maternal motivation, disrupted cognitive function, disturbed emotional regulation, impaired maternal behavior expression, and psychologi-

cal dysfunction. These effects likely represent a complex product of interactions among neuroendocrine, circuitry, and plasticity mechanisms. Maternal stress may cause abnormal HPA axis stress responses and dysregulation of endocrine systems including glucocorticoids, oxytocin, and prolactin, leading to altered neurogenesis and synaptic plasticity in key brain regions (e.g., hippocampus, medial prefrontal cortex, nucleus accumbens) that affect neural circuit responses to stimuli related to cognitive function, emotional regulation, and maternal motivation initiation. However, these mechanisms interact complexly: altered neural circuit activation may further disrupt endocrine systems and plasticity, ultimately impairing maternal cognition, emotional regulation, and behavior. Additionally, maternal stress may affect synaptic signaling by altering amino acid neurotransmitters (glutamate, GABA) and monoamine neurotransmitters (dopamine, serotonin) and their receptors, thereby influencing synaptic and neural circuit plasticity in brain regions involved in postpartum cognitive control, emotional regulation, and maternal motivation (Payne & Maguire, 2019; Slattery & Hillerer, 2016). For example, postpartum maternal separation reduces 5-HT1A receptors in the maternal brain' s hippocampus, prefrontal cortex, medial preoptic area, and central amygdala (Pawluski, Li, & Lonstein, 2019). Although monoamines, particularly serotonin, are implicated in depression pathophysiology and their reuptake inhibitors are clinically used, research on relationships between amino acid/monoamine neurotransmitters and maternal mood disorders during the perinatal period remains limited and requires further validation.

Furthermore, maternal stress can shorten gestation, trigger inflammatory responses, reduce milk quantity and quality, decrease litter size, and lower offspring birth weight (Brummelte & Galea, 2010; Cook, Ayers, & Horsch, 2018; Dole et al., 2003; Ingman, Glynn, & Hutchinson, 2014). Since mother-infant relationships involve dynamic interactions with innate motivational responses from both parties, stress responses from one alter the other' s physiological and psychological states. Research on maternal stress effects on offspring shows that stressed offspring exhibit increased stress sensitivity, anxiety, depression, anhedonia, cognitive impairment, and substance abuse in adulthood (Lehmann & Feldon, 2000; Mohammadian, Najafi, & Miladi-Gorji, 2019; Pryce & Feldon, 2003). Thus, understanding maternal stress effects on maternal behavior and psychological function is crucial for both maternal and offspring health.

However, research on maternal stress effects on mothers themselves has several limitations requiring further investigation. (1) **Basic research considerations**: Animal studies indicate that maternal stress effects depend heavily on the stress model used, including duration, intensity, timing, and measurement timepoints. For example, repeated gestational restraint stress has non-significant effects on anxiety- and depressive-like behavior in dams (Baker et al., 2008; Pawluski et al., 2015; Pawluski et al., 2011) and increases hippocampal proliferating cells in late gestation, but this effect appears transient with minimal impact on hippocampal neurogenesis after weaning (Pawluski, Charlier, et al., 2012). These findings suggest differential effects of maternal stress during gestation versus lactation. Additionally, different stress methods produce incon-

sistent results for postpartum anxiety- and depressive-like behavior (see Table 1), making cross-study comparisons difficult and leaving experimental conditions and findings controversial. Future basic research should employ identical stress models across multiple parameters to reveal coordinated effects on maternal behavior, psychology, physiology, neural mechanisms, and genetics. (2) **Translational considerations**: Clinical research faces methodological limitations due to ethical concerns and potential health risks to mothers and infants. While animal models allow controlled variables and quantitative behavioral description, they cannot simulate human social complexity, and their cognitive, emotional, and maternal behaviors are far less complex than humans' . Therefore, basic research must develop more effective methods to simulate human social stress. (3) **Clinical prevention and treatment considerations**: First, given maternal stress' s negative impact on emotional regulation, efficient maternal stress measurement methods warrant further research to identify populations at risk for postpartum psychiatric disorders. Second, interventions to reduce maternal stress during pregnancy and lactation require development, such as increasing infant cue stimuli to improve mother-infant interaction patterns and promote maternal motivation, or strengthening social support for women during this critical period through public health institutions. Additionally, considering adverse effects of antipsychotic medications on pregnant and lactating women and infants, non-pharmacological interventions hold promise. For example, exercise and high-fat diets have proven effective in improving depressive-like behavior in animal models of maternal stress (Gobinath et al., 2018; Maniam & Morris, 2010). These research directions will deepen understanding of postpartum psychiatric disorder pathophysiology and provide feasible clinical prevention and treatment strategies.

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