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Maternal sensitivity and infant autonomic and endocrine stress responses

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ABSTRACT

Background: Early environmental exposures may help shape the development of the autonomic nervous system (ANS) and hypothalamic–pituitary–adrenal (HPA) axis, influencing vulnerability for health problems across the lifespan. Little is known about the role of maternal sensitivity in influencing the development of the ANS in early life.
Aims: To examine associations among maternal sensitivity and infant behavioral distress and ANS and HPA axis reactivity to the Repeated Still-Face Paradigm (SFP-R), a dyadic stress task.
Study design: Observational repeated measures study.
Subjects: Thirty-five urban, sociodemographically diverse mothers and their 6-month-old infants.
Outcome measures: Changes in infant affective distress, heart rate, respiratory sinus arrhythmia (RSA), and T-wave amplitude (TWA) across episodes of the SFP-R were assessed. A measure of cortisol output (area under the curve) in the hour following cessation of the SFP-R was also obtained.
Results: Greater maternal insensitivity was associated with greater infant sympathetic activation (TWA) during periods of stress and tended to be associated with greater cortisol output following the SFP-R. There was also evidence for greater affective distress and less parasympathetic activation (RSA) during the SFP-R among infants of predominantly insensitive mothers.
Conclusions: Caregiving quality in early life may influence the responsiveness of the sympathetic and parasympathetic branches of the ANS as well as the HPA axis. Consideration of the ANS and HPA axis systems together provides a fuller representation of adaptive versus maladaptive stress responses. The findings highlight the importance of supporting high quality caregiving in the early years of life, which is likely to promote later health.

1. Introduction

A growing body of evidence supports the developmental origins of health and disease hypothesis, which purports that early environmental factors influence mental and physical health into adulthood [1]. Physiological systems involved in the human stress response, particularly the endocrine system and the autonomic nervous system (ANS), have received attention for their potential malleability by early environmental influences and their hypothesized etiological involvement in a broad array of disease states (e.g., cardiovascular disease, asthma, metabolic syndrome) as well as emotional and cognitive well-being (e.g., post-traumatic stress disorder, depression) [1–3].

Maternal caregiving quality has been identified as a robust programming agent of child stress response systems in early life, with sensitive maternal behaviors, including accurate reading of the child's signals and contingent, timely, emotionally supportive responding, linked to more optimal stress responding, and insensitive behaviors, including inaccurate interpretation of the child's needs and withdrawn, intrusive, and hostile behaviors, linked to maladaptive stress responding throughout life [4,5]. The majority of research on caregiving effects on offspring

Abbreviations: ANS, autonomic nervous system; HPA, hypothalamic–pituitary–adrenal; HR, heart rate; RSA, respiratory sinus arrhythmia; RSA_c, respiratory sinus arrhythmia corrected for respiration rate and tidal volume; RSA_u, respiratory sinus arrhythmia uncorrected for respiration rate and tidal volume; SFP, Still-Face Paradigm; SFP-R, Repeated Still-Face Paradigm; TWA, T-wave amplitude.

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stress response systems has focused on the hypothalamic–pituitary–adrenal (HPA) axis [5–7]. Less is known about the impact of maternal sensitivity on ANS activity. Existing studies on infant ANS stress reactivity have focused on the cardiac vagal system (as part of the parasympathetic branch of the ANS), following hypotheses about its role in early life attention, communication, affect, and coping [7,8]; (however, see [9]). In this context, cardiac vagal withdrawal is hypothesized to support the greater metabolic demand associated with an active coping response. However, work in adult cardiovascular psychophysiology suggests that overactivity of the sympathetic system in particular may be linked to long-term adverse health outcomes [10]. Therefore, research is needed to understand the potential influence of caregiving behaviors on both the parasympathetic and sympathetic branches of the ANS.

Notably, evidence shows that responding across and within stress systems is not necessarily correlated [11,12] Studies that have examined the effects of maternal behaviors on child stress reactivity have tended to study stress systems in isolation. Thus, there is a need for studies that examine maternal caregiving effects on a comprehensive assessment of infant stress reactivity, including affect/behavior, the HPA axis, and the sympathetic and parasympathetic branches of the ANS. The resultant findings may enhance our understanding of the mechanisms by which early experiences influence long-term health and inform the development of more efficacious interventions to prevent disease across the lifespan.

One of the most commonly used paradigms for assessing the infant stress response is the Still-Face Paradigm (SFP), during which the mother is asked to interact normally with the infant (baseline play episode), then withhold interaction (stressor still-face episode), and then resume interaction (recovery reunion episode) [13]. The SFP has been shown to reliably produce a stress response, as reflected in increases in observable negative affect/distress and heart rate and decreases in respiratory sinus arrhythmia (RSA; interpreted as an indicator of cardiac parasympathetic activity) during the still-face episode; and increases in cortisol output following the procedure [7,14–17]. More recently, investigators have discovered that the reunion episode may provide evidence of the dyad's ability to reduce the infant's stress response following cessation of the still-face episode, as evident by decreases in negative affect and heart rate and increases in RSA, though not necessarily back to baseline play levels [7,14–18]. Infant affective and behavioral responses to the SFP (e.g., amount of positive and negative affect, gaze aversion) have been linked to parental caregiving history and to future adaptation (e.g., attachment quality, emotional and behavioral problems) [16].

Maternal sensitivity during the SFP can be assessed [16]. The few studies specifically linking maternal sensitivity to infant affective and physiological responses to the SFP have produced mixed findings. Some studies have linked greater maternal sensitivity to lower infant negative affect and lower heart rate during the still-face and reunion episodes or to a more attenuated HPA axis response following the SFP [6,7,15,19,20]. Others have found associations between lower maternal sensitivity and greater increases in infant heart rate from the still-face to the reunion episode [19]. However, others have found no associations between maternal sensitivity and infant affect or cortisol [7,15], and yet others have found results contrary to prediction, such as decreases in RSA from the play episode to the reunion episode only for infants of sensitive mothers [7]. Notably, studies have been inconsistent in how maternal sensitivity is measured in the context of SFP studies. Some studies consider maternal behaviors during the play episode only [20], some focus on the reunion [6], some create composite sensitivity scores across play and reunion episodes [15], and some consider behaviors during the play and reunion episodes separately [19].

Currently there are numerous gaps in the literature assessing associations between maternal sensitivity and infant affective and physiological stress reactivity in response to the SFP. Only two studies have assessed the infant HPA axis response [15,20], with one collecting only one cortisol measure pre-SFP and with one collecting only two cortisol measures, the first pre-SFP and the second 20-minutes post-SFP. [15]. Studies examining ANS reactivity have focused on heart rate (parasympathetically and sympathetically influenced) and RSA (parasympathetically influenced) and have not included measures of sympathetic reactivity. Furthermore, such studies have often failed to control for motor activity, impeding efforts to distinguish possible stress effects on autonomic measures from metabolic costs of increased motor activity, which often accompanies distress [21]. Measures of maternal sensitivity have most frequently been examined in relation to infant responses *within* episodes rather than in relation to changes *across* episodes, though examining changes across episodes is important to understanding infant stress responses [7,16]. Finally, only one study has considered separately the impact of maternal sensitivity under both non-distress (play) and distress (reunion) conditions on infant SFP responses, though maternal behaviors under each of these conditions may differentially influence child outcomes [6,19]. For example, the developmental literature suggests that maternal sensitivity during times of distress predicts mother–infant attachment quality, problem behaviors, social competence, and physiological and behavioral regulation; that maternal sensitivity under non-distress conditions predicts child cognitive development; and that maternal sensitivity under non-distress and distress conditions together predict child affect regulation [19,22–24]. More studies that consider maternal sensitivity under both non-distress and distress conditions to determine if they have differential influences on the various infant stress response systems are needed.

The goal of the current study was to address these gaps by examining associations between maternal sensitivity under non-distress and distress conditions and infant affective and physiological stress reactivity. The recently developed repeated version of the Still-Face Paradigm (SFP-R), which includes two still-face and reunion episodes, was utilized to ensure sufficient opportunity to observe an infant stress and recovery response. Measures of infant responding over the course of the SFP-R included affective distress, heart rate, RSA (parasympathetic activity), and T-wave amplitude (sympathetic activity). In addition, cortisol was measured before and repeatedly after completion of the SFP-R to characterize the HPA axis response. We hypothesized that, on average, infants would show increases in behavioral distress, attenuation of the T-wave, and decreases in RSA during the stressful still-face episode. We further hypothesized that maternal insensitivity would be associated with greater infant distress, T-wave attenuation, and decreases in RSA during the still-face episodes; poorer recovery (i.e., more limited decreases in distress, T-wave attenuation, and increases in RSA relative to the preceding still-face episode) during the reunion episodes; and greater overall cortisol output following completion of the SFP-R. Based on the one prior study to examine the differential contributions of maternal sensitivity during the play and reunion episodes to infant biobehavioral reactivity during the SFP [19], we expected that maternal sensitivity during the reunion episode would have stronger associations with infant biobehavioral reactivity than maternal sensitivity during the play episode.

2. Method

2.1. Participants

Participants were mothers and their 6-month-old infants (M = 27.8 weeks, SD = 1.3 weeks) enrolled in a longitudinal study examining the impact of maternal trauma on infant emotional, behavioral, and physiological stress reactivity in the first year of life. Pregnant women receiving prenatal care at two major Boston hospitals and three affiliated urban community health centers and women attending Women, Infants and Children (WIC) programs associated with the health centers were recruited during the 1st or 2nd trimester between August 2006 and September 2009. Inclusion criteria included a) mother aged ≥18 years at the child's birth and b) single gestation birth. Exclusion criteria included (a) mother not sufficiently fluent in English to complete study measures; (b) infant at increased risk for