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Maternal Posttraumatic Stress Symptoms and Infant Emotional Reactivity and Emotion Regulation

Michelle Bosquet Enlow, Robert L. Kitts, and Emily Blood

Children's Hospital Boston and Harvard Medical School

Andrea Bizarro and Michelle Hofmeister

Children's Hospital Boston

Rosalind J. Wright

Brigham and Women's Hospital and Harvard School of Public Health

Abstract

The current study examined associations between maternal posttraumatic stress disorder (PTSD) symptoms and infant emotional reactivity and emotion regulation during the first year of life in a primarily low-income, urban, ethnic/racial minority sample of 52 mother-infant dyads. Mothers completed questionnaires assessing their own trauma exposure history and current PTSD and depressive symptoms and their infants' temperament when the infants were 6 months old. Dyads participated in the repeated Still-Face Paradigm (SFP-R) when the infants were 6 months old, and infant affective states were coded for each SFP-R episode. Mothers completed questionnaires assessing infant trauma exposure history and infant current emotional and behavioral symptoms when the infants were 13 months old. Maternal PTSD symptoms predicted infants' emotion regulation at 6 months as assessed by (a) infant ability to recover from distress during the SFP-R and (b) maternal report of infant rate of recovery from distress/arousal in daily life. Maternal PTSD symptoms also predicted maternal report of infant externalizing, internalizing, and dysregulation symptoms at 13 months. Maternal PTSD was not associated with measures of infant emotional reactivity. Neither maternal depressive symptoms nor infant direct exposure to trauma accounted for the associations between maternal PTSD symptoms and infant outcomes. These findings suggest that maternal PTSD is associated with offspring emotion regulation difficulties as early as infancy. Such difficulties may contribute to increased risk of mental health problems among children of mothers with PTSD.

Keywords

infant; emotion regulation; reactivity; maternal PTSD

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Correspondence concerning this manuscript should be addressed to the first author at Children's Hospital Boston, 21 Autumn Street, 1st Floor, Boston, MA 02115; phone: (617) 919-4680; FAX: 617-730-0759; michelle.bosquet@childrens.harvard.edu..

Author Note

Michelle Bosquet Enlow and Robert L. Kitts, Department of Psychiatry, Children's Hospital Boston and Department of Psychiatry, Harvard Medical School; Emily Blood, Clinical Research Program, Children's Hospital Boston and Department of Pediatrics, Harvard Medical School; Andrea Bizarro and Michelle Hofmeister, Department of Psychiatry, Children's Hospital Boston; Rosalind J. Wright, Channing Laboratory, Department of Medicine, Brigham & Women's Hospital and Department of Environmental Health, Harvard School of Public Health.

Michelle Hofmeister is now at the Medical College of Wisconsin.

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There has been increasing emphasis in the extant literature on the importance of self-regulation—the ability to manage one’s emotions, attention, physiology, and behavior in a way that promotes competent functioning—in normal development as well as the developmental psychopathology of various mental disorders (Barton & Robins, 2000; Bell & McBride, 2010; Eisenberg, Spinrad, & Eggum, 2010; Gardner, Dishion, & Connell, 2008; National Research Council and Institute of Medicine, 2009). In their landmark report *From Neurons to Neighborhoods*, the National Research Council and Institute of Medicine (2000) concluded that one of the core concepts of development that has emerged from years of research is that “the growth of self-regulation is a cornerstone of early childhood development that cuts across all domains of behavior” (p. 3). Given the importance of self-regulation for multiple aspects of functioning, determining factors that interfere with its attainment is a critical area of research. Evidence suggests that exposure to maternal psychopathology during early development may have a substantial impact on child self-regulation (Brand & Brennan, 2009; Brummelte & Galea, 2010; Davis, Snidman, Wadhwa, Schetter, & Sandman, 2004; Field, 2010; Goodman & Gotlib, 1999). Much of this research has been based on studies of depressed, and to a lesser extent, anxious mothers. The impact of maternal posttraumatic stress disorder (PTSD) on child regulation has received comparatively little attention, though data from multiple sources suggest that exposure to maternal PTSD may have a profound effect on children’s self-regulatory abilities (Bosquet Enlow et al., 2009; Brand, Engel, Canfield, & Yehuda, 2006; Chemtob et al., 2010; Kaitz, Levy, Ebstein, Faraone, & Mankuta, 2009).

1.1. Reactivity and Regulation

Appreciating the potential impact of maternal PTSD on the acquirement of child self-regulation requires an understanding of the related but distinct concepts of “reactivity” and “regulation.” Here, reactivity refers to individual differences in the speed and intensity of initial activation of physiological, attentional, emotional, and motoric responses elicited by stimuli. Developmental theorists have suggested that reactivity reflects underlying biological biases toward particular response patterns that arise from a combination of genes and in utero neuroendocrine influences, though these biases may be modified to some extent by environmental experiences (Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996). Differences in reactivity have been noted as early as the 2nd trimester of the prenatal period, with continuity demonstrable across early and later development (DiúPietro, 2000).

Regulation involves the ability to modify the intensity and duration of physiological arousal, attention, and affective states in order to (a) protect oneself from becoming overwhelmed by stimulation or disorganized by one’s own feelings, (b) modulate one’s emotional expressions, and (c) organize complex behaviors involved in social interactions (National Research Council and Institute of Medicine, 2000; Sroufe, Egeland, Carlson, & Collins, 2005). That is, regulation involves the management of reactivity. Though, like reactivity, regulation may reflect biologically-based differences in the central nervous system (Rothbart, Ahadi, & Hershey, 1994; Rothbart & Derryberry, 1981; Rueda & Rothbart, 2009), the ability to self-regulate is largely theorized to arise out of interactions with primary caregivers during the first years of life (Eisenberg et al., 2010).

In early development, responsibility for child regulation is gradually transferred from the primary caregiver to the dyad to the child. Neonates and young infants are primarily focused on maintaining physiological homeostasis and rely on their caregivers as regulators (Loman & Gunnar, 2010). Physiological stress regulatory systems [e.g., hypothalamic-pituitary-adrenal (HPA) system, autonomic nervous system (ANS)] become organized during the first months through caregiver-infant transactions, with sensitive caregiving promoting effective

physiological regulation of stress (Loman & Gunnar, 2010). By 4 to 6 months of age, infants engage in reciprocal interactions with their caregivers, and by the end of the first year, infants become active participants in the regulation of their arousal and emotional states through their attachment relationships (Schore, 1994). During this period, infants begin to show stabilizing patterns of frontal neural activation and predictable behavioral strategies for emotion regulation (Bell & Fox, 1994; Eisenberg et al., 2010). A history of sensitive caregiving in infancy, reflected in a secure attachment relationship, has been associated with more adaptive self-regulatory abilities and more optimal stress regulation in later development (Kochanska, Philibet, & Barry, 2009; Oosterman, De Schipper, Fisher, Dozier, & Schuengel, 2010). Because poor regulation in early development is thought to be carried forward through later development, disruptions to regulatory processes during the first year of life may have particularly detrimental and enduring effects on later child functioning (Eisenberg et al., 2010).

1.2. Maternal PTSD and Child Regulation

Because the quality of caregiving in early development is critical in shaping a child's emerging self-regulation capacities, factors that interfere with a mother's ability to provide high quality care are expected to negatively influence her child's attainment of these capacities. A caregiver's ability to provide responsive, sensitive care is determined by multiple factors, including her own psychological resources. PTSD is defined by symptoms of hyperarousal (e.g., irritability; angry outbursts; concentration difficulties; hypervigilance), avoidance and numbing of responsiveness (e.g., restricted range of affect; anhedonia; feeling detached from others) and reexperiencing of traumatic events (e.g., flashbacks; intense psychological distress and/or physiological reactivity upon exposure to traumatic reminders) (American Psychiatric Association, 2000). Associated symptoms prevalent among individuals with PTSD, such as emotional lability, difficulties in modulating anger, appropriately expressing emotions, and interpreting others' emotions, and disruptions in attention, memory, and consciousness (Hien, Cohen, & Campbell, 2005) may lead to withdrawn, avoidant, intrusive, irritable, hostile, unresponsive, and non-contingent parenting behaviors. Research shows that such parenting behaviors are associated with major disruptions to young children's self-regulation (Leerkes, Blankson, & O'Brien, 2009; Sroufe et al., 2005; Stams, Juffer, & van IJzendoorn, 2002).

1.3. Maternal PTSD and Child Reactivity

There is evidence that mothers with PTSD may have infants who show greater reactivity to stimuli, increasing challenges to fostering self-regulation (Brand et al., 2006; Lang, Gartstein, Rodgers, & Lebeck, 2010; Tees et al., 2010). While the mechanisms involved are unclear, this association may be a consequence of disrupted maternal-fetal HPA axis functioning resulting from PTSD. PTSD has been repeatedly associated with dysregulation of the HPA system (Gunnar & Quevedo, 2007; Nemeroff, 2004; Pervanidou & Chrousos, 2010), and animal and human studies suggest that maternal levels of circulating glucocorticoids during pregnancy program the fetal HPA axis (Brand et al., 2010; Davis, Glynn, Waffarn, & Sandman, 2010; Weinstock, 2008). Therefore, offspring of mothers with PTSD may be exposed to nonoptimal levels of glucocorticoids during the prenatal period and consequently develop a hypo- or hyper-reactive stress response system. Yehuda and colleagues (Brand et al., 2006; Yehuda et al., 2005; Yehuda & Bierer, 2008) have documented some evidence for this hypothesis, finding lower postnatal cortisol levels among mothers who developed PTSD symptoms during pregnancy in response to the World Trade Center attacks and among their infants. Furthermore, maternal ratings of infant distress to novelty were positively correlated with maternal PTSD symptoms and negatively correlated with maternal morning cortisol levels (Brand et al., 2006; Yehuda et al., 2005),

suggesting possible infant behavioral consequences of maternal-fetal HPA axis dysregulation resulting from PTSD. Other studies have demonstrated that traumas do not have to occur during pregnancy to influence offspring HPA programming. For example, Brand and colleagues (2010) have documented associations between maternal history of abuse in their own childhood and infant baseline cortisol and cortisol reactivity to stress.

Providing sensitive care to an infant who is highly reactive to stimuli may present a challenge for caregivers due to the infant's need for frequent intervention. These challenges may be magnified for mothers with PTSD who are struggling with their own self-regulation difficulties and who may find that difficult infant behaviors, such as frequent or intense bouts of crying, exacerbate their own symptoms. Conversely, an infant who is minimally reactive may fail to evoke interactions that help to build self-regulatory abilities (Robinson & Acevedo, 2001), particularly from a caregiver who is withdrawn or insensitive to reading her infant's cues due to her own psychological challenges.

1.4. Evidence of Effects of Maternal PTSD on Child Reactivity and Regulation and Mental Health

A limited number of studies have provided evidence supporting associations between maternal PTSD and infant reactivity and regulation. Lang and colleagues (2010) found that, compared to mothers without a childhood maltreatment history, mothers with histories of emotional and/or physical abuse described their 12-month-old infants as more likely to become distressed by limits and less able to recover from distress. Similarly, Tees and colleagues (2010) found positive associations between maternal report of difficult infant temperament at 12 months of age and maternal PTSD and depressive symptoms among mothers exposed to Hurricane Katrina and its aftermath. In a study of the impact of maternal PTSD and depression on preschool children's behavior, Chemtob and colleagues (2010) reported that children of mothers with both PTSD and depression demonstrated poorer emotion regulation and greater behavior problems than children of mothers with depression alone or with neither disorder. A number of other studies have demonstrated increased rates of mental health problems, a possible functional outcome of poor self-regulation, among offspring of parents with trauma histories and/or PTSD, though many of these studies were conducted retrospectively among older children or adult offspring of parents with specific types of trauma histories (e.g., Holocaust survivors, refugees, war veterans) and have relied on one reporter (parent or offspring) for both predictor and outcome measures (Dekel & Goldblatt, 2008; Kaitz et al., 2009; Sack, Clarke, & Seeley, 1995; Zajac & Kobak, 2009).

1.5 Prevalence of PTSD among Mothers of Young Children

Data from epidemiological studies suggest a high prevalence of PTSD among mothers in the general population. Over their lifetime, approximately 90% of Americans experience a potentially traumatic event, and 10-12% of women develop PTSD (Breslau, Kessler, & Chilcoat, 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Resnick, Kilpatrick, Dansky, & Best, 1993). Rates of trauma exposure are especially elevated among low-income, ethnic/racial minority, urban populations (Breslau, Davis, Andreski, & Petersen, 1991; Breslau et al., 1998; Holman, Silver, & Waitzkin, 2000). For example, Hien and Bukszpan (1999) examined lifetime interpersonal violence among a group of urban, low-income, predominantly Hispanic or Black women screened for the absence of psychopathology. Approximately 28% of these women reported a history of childhood abuse, compared to general population estimates of 10%. Urban minority women also experience heightened levels of community violence (Clark et al., 2007; Brown, Hill, & Lambert, 2005). Not surprisingly, given their heightened risk for exposure to trauma, urban low-income minority women experience elevated levels of PTSD compared to the general

population (Breslau et al., 1991; Breslau et al., 1998; Kessler et al., 1995; Stein, Walker, Hazen, & Forde, 1997). An epidemiological survey of women in California revealed that 28% of the women were currently experiencing PTSD symptoms, with higher rates among the poorest women (Kimerling, 2003). Moreover, a significant portion of women caring for young children reported current PTSD symptoms, and 43% of women who were heads of the household with children were experiencing PTSD symptoms. These results are consistent with findings that the perinatal period is a particularly vulnerable time to experience intense psychological symptoms, including PTSD (Seng et al., 2010), especially among low-income women (Beck, 1996; Benoit, Westfall, Treloar, Phillips, & Jansson, 2007; Chaudron et al., 2005; Robertson, Grace, Wallington, & Stewart, 2004).

1.6. The Current Study

Given the high prevalence of PTSD in the general population, and especially among low-income urban mothers, there is a critical need to understand the impact of maternal PTSD on child outcomes. Though multiple lines of evidence within the developmental literature suggest that maternal PTSD may have significant, negative consequences on the development of child self-regulation and, ultimately, mental health, there has been very little research in this area. The goal of the current study was to address this gap by examining associations between maternal PTSD symptoms and child reactivity, regulation, and behavioral and emotional functioning over the first year of life in a sample of urban, primarily low-income minority families. Study measures specifically assessed infant emotional reactivity (i.e., affective responses to stimuli) and emotion regulation (i.e., ability to modify the intensity and duration of emotional reactivity). Unlike the majority of the limited research to date in this area, measures of infant emotional reactivity and emotion regulation included laboratory observations as well as maternal report. Based on the existing literature described above, it was hypothesized that there would be significant associations between maternal PTSD symptoms and infant emotional reactivity and emotion regulation. Specifically, analyses tested the following hypotheses: (a) maternal PTSD is associated with greater infant negative emotional reactivity in response to stress, with quick, intense responses indicative of higher reactivity; (b) maternal PTSD is associated with poorer infant emotion regulation, as reflected in greater time to decrease the intensity and degree of distress following cessation of a stressor; (c) maternal PTSD is associated with symptoms of emotional and behavioral problems by the end of infancy; (d) neither maternal depressive symptoms nor infant direct exposure to trauma accounts for the associations between maternal PTSD and infant emotional reactivity, emotion regulation, and emotional/behavioral symptoms.

Method

2.1. Participants

Participants were mothers and their infants ($N = 52$ dyads) enrolled in a longitudinal study examining the impact of maternal trauma exposure and related symptomatology on infant emotional, behavioral, and physiological reactivity and regulation in the first year of life. Participants were drawn from the Asthma Coalition on Community, Environment, and Social Stress (ACCESS) project, an epidemiological birth cohort study that examines the effects of early life stress and the physical environment on childhood asthma risk in an urban, primarily low-income sample (Sternthal et al., 2009; Wright et al., 2008). Mothers were originally recruited from two major hospitals in Boston and three urban community health centers during their 1st or 2nd trimester of pregnancy 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 and b) infant at increased risk for neurodevelopmental

disorders (e.g., gestational age < 32 weeks; birth weight < 5.5 lbs; congenital abnormalities; neurological injury) (Minde, 2000). Additionally, ACCESS participants were not approached for participation in the current study if they endorsed drinking 7 or more alcoholic drinks per week or smoking 10 or more cigarettes per day during pregnancy,¹ as usage at or above these thresholds has been associated with infant regulatory difficulties (Testa, Quigley, & Eiden, 2003; Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002). Neither maternal nor infant history of respiratory problems was an inclusion/exclusion criterion. All procedures were approved by the relevant Institutional Review Boards, and written informed consent was obtained from mothers prior to the beginning of study procedures.

2.2. Procedures and Measures

Mothers and infants participated in two laboratory visits, one when the infants were approximately 6 months of age ($M = 27.56$ weeks, $SD = 1.72$ weeks, $n = 50$) and one when the infants were approximately 13 months of age ($M = 56.26$ weeks, $SD = 4.74$ weeks, $n = 46$). Maternal trauma exposure and PTSD and depressive symptoms were assessed during the 6-month assessment.² Infant emotional reactivity and emotion regulation measures administered at 6 months included a maternal-report measure of infant temperament and a dyadic stressor observational protocol. At 13 months, infant lifetime trauma exposure and emotional and behavioral symptoms were assessed via maternal report.

2.2.1. Life Stressor Checklist-Revised (LSC-R; Wolfe & Kimerling, 1997)—Self-reported maternal lifetime exposure to traumatic events was assessed at 6 months² using the LSC-R. The LSC-R assesses exposure and reactions to 30 potentially traumatic events (e.g., experiencing or witnessing a serious accident or natural disaster, death of someone close, maltreatment in childhood), including experiences particularly relevant to women (e.g., sexual assault, interpersonal violence) and has established reliability and validity in diverse populations. A maternal lifetime trauma exposure score (range 0-30) was derived from the number of endorsed events that met Diagnostic and Statistical Manual of Mental Disorders-4th Edition (DSM-IV) PTSD Criterion A (American Psychiatric Association, 2000), i.e., events during which the mother reported thinking that she or someone else could be killed or seriously harmed and that caused her to feel intense helplessness, fear, and/or horror.

2.2.2. Posttraumatic Stress Disorder Checklist-Civilian Version (PCL-C; Weathers, Huska, & Keane, 1991)—Maternal PTSD symptoms over the prior month were assessed at 6 months² using the PCL-C, a 17-item self-report measure that reflects DSM-IV criteria for PTSD. The PCL-C is the most frequently used screening instrument for PTSD in research, particularly with women (Brewin, 2005). Mothers rated each symptom on a five-point Likert scale in terms of how much the symptom had bothered them in the past month, ranging from 1 (not at all) to 5 (extremely). The PCL-C provides a single symptom score based on number and severity of PTSD symptoms (range = 17-85). Additionally, a cutoff method was used to obtain a likely diagnosis of PTSD using DSM-IV criteria, categorizing mothers into a “non-elevated” or an “elevated” PTSD symptoms group.

¹During either the 6- or 13-month assessment described below, mothers were re-interviewed about their use of substances during pregnancy. During this interview, 9.6% of mothers ($n = 5$) endorsed smoking 10 or more cigarettes per day at some point during pregnancy, with two only prior to pregnancy recognition, two during the first trimester, and one throughout the pregnancy. One mother endorsed averaging one alcoholic drink per day during the first trimester. No mothers endorsed the use of any marijuana, opioids, cocaine/crack, or stimulants at any point during the pregnancy. Prenatal substance use was not significantly associated with any of the study predictor or outcome variables and therefore was not included in the analyses.

²Two mother-infant dyads were unable to participate in the 6-month assessment due to scheduling issues. For these two mothers, maternal trauma exposure and psychological symptomatology data obtained at the 13-month assessment were used in analyses.

Mothers who endorsed at least one re-experiencing symptom, three avoidance/numbing symptoms, and two hyperarousal symptoms as moderately to extremely bothersome in the past month were placed in the elevated group. The non-elevated group had a mean total PCL-C score of 26.47, $SD = 7.64$, and the elevated group had a mean score of 55.14, $SD = 9.55$, $t(50) = 11.21$, $p < .001$. The PCL-C has high internal consistency for the total scale and good test-retest reliability and convergent validity with a number of other PTSD scales and with the Clinician-Administered PTSD Scale, a structured clinical interview for PTSD (Weathers, Keane, & Davidson, 2001). Internal consistency in this sample was high, Cronbach's $\alpha = 0.94$.

2.2.3. Edinburgh Postnatal Depression Scale (EPDS; Cox, Holden, & Sagovsky, 1987)—Research has shown that depression is highly comorbid with PTSD (Chemtob et al., 2010; O'Campo et al., 2006) and that maternal depression is associated with infant emotional functioning (Brummelte & Galea, 2010; Davis et al., 2004; Field, 2010; Goodman & Gotlib, 1999). Therefore, demonstrating that maternal PTSD is associated with infant emotional reactivity and emotion regulation after controlling for maternal depressive symptoms would provide a more rigorous test of the proposed hypotheses linking maternal PTSD to infant outcomes. The EPDS, a 10-item self-report questionnaire specifically designed to measure the presence of depressive symptoms in mothers during the postnatal period (Gaynes et al., 2005), was administered at the 6-month study visit² to assess maternal depressive symptoms over the prior week. Each EPDS item is scored for severity from 0 to 3 and then summed to provide a total score (range = 0-30). Additionally, a previously validated (Gibson, McKenzie-McHarg, Shakespeare, Price, & Gray, 2009) cutoff score of 12/13 was used to obtain a likely DSM-IV diagnosis of major depression, categorizing mothers into a “non-elevated” (score ≤ 12) or an “elevated” depressive symptoms group (score ≥ 13). The non-elevated group had a mean total EPDS score of 4.69, $SD = 3.25$, and the elevated group had a mean score of 17.69, $SD = 3.73$, $t(50) = 12.05$, $p < .001$. The EPDS has demonstrated high internal consistency and validity for detecting major depression in the perinatal period (Gibson et al., 2009). Internal consistency in this sample was high, Cronbach's $\alpha = 0.91$.

2.2.4. Traumatic Events Screening Inventory-Parent Report Revised (TESI-PRR; Ghosh Ippen et al., 2002)—Infant lifetime direct exposure to potentially traumatic events was assessed via maternal report at 13 months. The TESI-PRR was specifically developed for use with children ages birth to 6 years. It screens for exposure to a wide range of potentially traumatic events specific to this age group, including accidents, illnesses, injuries, abuse and neglect, witnessing community and domestic violence, separation from the primary caregiver, and attack by an animal. The measure asks the child's age when each endorsed event occurred. The TESI-PRR is a modification of the Traumatic Events Screening Inventory-Parent Report, which has demonstrated good psychometric properties (Ford et al., 2000). The TESI-PRR has been recommended as the best measure for assessing exposure to traumatic events among young children (Stover & Berkowitz, 2005). Two infant trauma exposure scores were obtained, one by summing the number of events experienced by 6 months of age, and one by summing the number of events experienced by 13 months of age.

2.2.5. Repeated Still-Face Paradigm (SFP-R; Haley & Stansbury, 2003; Tronick, Als, Adamson, Wise, & Brazelton, 1978)—The infant's emotional reactivity to a stressor and the dyad's ability to regulate infant distress following cessation of the stressor were assessed at 6 months using the repeated Still-Face Paradigm (SFP-R). The Still-Face Paradigm (SFP) is a videotaped observational procedure used to study infants' behavioral, emotional, and physiological responses to brief, moderate levels of induced stress (Adamson

& Frick, 2003). The SFP involves three 2-minute episodes during which the infant is seated in an infant seat across from the mother. The mother was instructed to play with her infant for 2 minutes ("Play" = baseline), followed by a still-face episode, during which the mother was instructed to maintain a neutral facial expression and avoid touching or vocalizing at the infant ("Still-Face" = stressor). The still-face episode is hypothesized to be a stressor for the infant because the mother is no longer providing behavioral cues needed for the infant to maintain an organized social and affective state (Weinberg & Tronick, 1996). The mother was then instructed to resume playing with the infant during a "reunion" episode ("Reunion" = recovery), which provided an opportunity for the infant to recover, with the mother's assistance, from any stressful effects of the still-face episode. The repeated version of the SFP (SFP-R) includes a second still-face episode and reunion episode administered immediately after the cessation of the first reunion episode. The SFP-R was designed specifically to assess infant stress reactivity and regulation (Haley & Stansbury, 2003). The still-face episodes were curtailed if the infant displayed one minute of continuous fussing or 30 seconds of continuous crying to prevent the infant from becoming excessively distressed. The second still-face and reunion episodes were only administered if the infant had returned to a non-distressed state by the end of the first reunion.

The infants' affective states were coded second-by-second from videos by trained coders blind to the other study data. Codes were mutually exclusive and consisted of the following: hard crying, crying, fussing/negative affect without vocalizations (called "fussing" from this point forward), neutral, positive, very positive, mixture of positive and negative, unclassifiable, unobservable/asleep, or autonomic indicator (see Appendix). One coder coded all of the sessions, and a second coder coded 44% of the sessions for inter-rater reliability. The weighted kappa was 0.77 between the two raters. For the current analyses, percentage of time spent in hard crying, crying, fussing, and positive affect (positive + very positive) were scored for each infant for each SFP-R episode. Latency to negative affect (fussing, crying, or hard crying), that is, the amount of time in seconds to display any negative affect following initiation of the still-face, was also calculated for each of the still-face episodes.

2.2.6. Infant Behavior Questionnaire-Revised (IBQ-R; Gartstein & Rothbart, 2003)—Maternal perception of infant temperament was assessed at 6 months using the IBQ-R. The IBQ-R was developed based on the conceptualization of temperament as biologically-based differences in reactivity and regulation in response to stimuli (Gartstein & Rothbart, 2003). The 191-item measure is organized into 14 subscales. For the current analyses, two of the subscales were used as measures of emotional reactivity and emotion regulation, respectively: (a) distress to limitations, which assesses fussing, crying or showing distress while in a confining place or position, involved in caretaking activities, or unable to perform a desired action, and (b) falling reactivity, which assesses rate of recovery from peak distress, excitement, or general arousal and ease of falling asleep (Gartstein & Rothbart, 2003). The IBQ-R has demonstrated good internal consistency (Parade & Leerkes, 2008) and has been found to provide complementary information to laboratory observations regarding infant reactivity and regulation (Forman et al., 2003). Cronbach's α values for the current sample were 0.76 and 0.84 for the distress to limitations and falling reactivity subscales, respectively.

2.2.7. Infant-Toddler Social and Emotional Assessment (ITSEA; Carter & Briggs-Gowan, 2006)—Infant social and emotional symptoms were assessed at 13 months using the ITSEA, a parent-report measure of social-emotional and behavioral problems and competencies in 12- to 36-month-old children. The 166-item measure is organized into four broad domains of behaviors: Externalizing, Internalizing, Dysregulation, and Competence. The Externalizing domain is composed of Activity/Impulsivity,

Aggression/Defiance, and Peer Aggression scales. Because 26% of the infants in the current sample were missing data for the Peer Aggression scale due to lack of exposure to peers, this scale was excluded in the calculation of the Externalizing domain scores. The Internalizing domain includes Depression/Withdrawal, General Anxiety, Separation Distress, and Inhibition to Novelty scales. The Dysregulation domain includes Sleep, Negative Emotionality, Eating, and Sensory Sensitivity scales. The Competence domain includes Compliance, Attention, Imitation/Play, Mastery Motivation, Empathy, and Prosocial Peer Relations scales. The ITSEA has demonstrated good internal consistency, particularly with regard to the Externalizing, Internalizing, and Dysregulation domains (Carter, Briggs-Gowan, Jones, & Little, 2003), and adequate test-retest reliability. The validity of the ITSEA is supported through associations with other problem behavior checklists, observed child behavior, and evaluator ratings of problem behaviors (Carter & Briggs-Gowan, 2006). Internal consistency in the current sample was high: Cronbach's $\alpha = 0.84, 0.80, 0.91$, and 0.83 for the Externalizing, Internalizing, Dysregulation, and Competence domains, respectively.

2.2.8. Potential confounders—Several demographic variables were considered as potential confounders, including maternal age, educational attainment, marital status, parity, financial strain, race, and ethnicity, and infant sex, birthweight, gestational age, race, and ethnicity. Educational attainment was dichotomized based on whether the mother completed any education beyond high school. Parity was dichotomized into primiparous and multiparous. Maternal age, birthweight and gestational age were considered as continuous variables, and race, ethnicity and marital status as categorical variables. Financial strain was based on a 3-item index of economic difficulties scored on a 5-point Likert scale that was summed (range 1-15); higher scores indicate greater financial strain (Sternthal et al., 2009).

2.3. Data Analytic Plan

Data analyses proceeded in several steps to address the study hypotheses. First, descriptive data are presented to show the distribution of maternal trauma exposures and PTSD and depressive symptoms. Potential demographic differences between the non-elevated and elevated maternal PTSD symptoms groups were explored and considered as possible confounders in subsequent analyses. Next, infant responses to the SFP-R were tested for differences in infant emotional reactivity and emotion regulation by maternal PTSD group, controlling for maternal depressive symptoms. Then, associations between maternal PTSD symptoms at 6 months and infant IBQ-R scales at 6 months and infant ITSEA scales at 13 months were examined, controlling for maternal depressive symptoms where indicated. Finally, analyses tested whether infant direct exposure to trauma may account for any noted associations between maternal PTSD and infant emotional reactivity and emotion regulation. The specific statistical techniques employed to address each of these steps are described below immediately preceding the relevant results.

Results

3.1. Descriptive Results

Distributions of maternal trauma exposure and maternal PTSD symptoms and likely diagnostic status were examined. Associations among maternal trauma exposure, PTSD, and depressive symptoms were assessed. Distributions of the demographic variables were examined for the entire sample and by maternal PTSD group. Demographic variables that differed significantly by maternal PTSD group were then tested in relation to the outcome variables. Variables that were significantly associated with both maternal PTSD and infant outcome were considered as potential confounders in multivariate analyses.

3.1.1. Maternal trauma exposure, PTSD, and depressive symptoms—Mothers reported experiencing a range of Criterion A traumatic events, with 19% reporting exposure to no traumatic events, 23% to one event, 12% to two events, 21% to three events, and 25% to four or more events, $M = 2.62$ (2.48), range = 0-10. The most frequently reported events included having a serious accident or accident-related injury (27%), being abused or physically attacked after age 16 (19%), learning about a traumatic event happening to a loved one (19%), seeing a serious accident (17%), witnessing a robbery, mugging, or attack (17%), being robbed, mugged, or attacked (15%), being emotionally abused or neglected (14%), being abused or physically attacked before age 16 (14%), and witnessing violence between family members before age 16 (14%).

Mothers reported significant variability in current symptoms of PTSD [$M = 34.19$ (15.18), range = 17-77], with 27% of participants meeting criteria for a likely diagnosis of PTSD. Similar variability in depressive symptom scores were found [$M = 7.94$ (6.59), range = 0-24], with 25% surpassing the cutoff score for a likely diagnosis of major depression. As shown in Table 1, trauma exposure was moderately correlated with PTSD symptoms, and PTSD and depressive symptoms were strongly correlated. There was a high degree of overlap between probable diagnoses of PTSD and depression, with 71% of the mothers with a probable diagnosis of PTSD also having a probable diagnosis of major depression, and 77% of mothers with a probable diagnosis of major depression having a probable diagnosis of PTSD, $p < .001$, two-tailed Fisher's exact test.

3.1.2. Potential confounders—Table 2 presents the demographic characteristics for the total sample and by maternal PTSD group. As shown in Table 2, the PTSD groups did not differ significantly in maternal age, education, marital status, parity, race, or ethnicity or infant sex, birthweight, gestational age, or ethnicity. The PTSD groups differed on financial strain, with the elevated group reporting greater financial strain than the non-elevated group. The PTSD groups also differed significantly in distribution of infant race, with the non-elevated group having greater numbers of White and Black infants and the elevated group having greater numbers of Asian and Multiracial infants.

To determine if financial strain or infant race should be included in analyses as covariates, given their significant associations with maternal PTSD status, associations between these variables and the outcome variables were examined. Neither financial strain nor infant race was significantly associated with either of the IBQ-R temperament scales at 6 months or with any of the ITSEA scales at 13 months, all $ps \geq .05$. Therefore, none of the covariates were considered further in analyses.

3.2. Maternal PTSD and Infant Emotional Reactivity and Emotion Regulation to Stress (SFP-R) at 6 Months

Data from 43 infant-mother dyads were included in the SFP-R analyses. Of the remaining nine dyads, two did not participate in the 6-month assessment, one mother did not consent to videotaping, one dyad lacked sound on the video, and four infants were too distressed and one infant was too sleepy to complete at least one of the still-face episodes. Among the 43 dyads, 31 (72%) were in the non-elevated maternal PTSD symptoms group and 12 (28%) were in the elevated maternal PTSD symptoms group. Thirty-one dyads [23 non-elevated (74%), 8 elevated (26%)] completed all five episodes of the SFP-R; the SFP-R was curtailed during an earlier episode for 12 dyads due to infant distress. There were no differences between dyads who did and did not participate in the SFP-R or between dyads who did and did not complete all five episodes of the SFP-R on maternal PTSD or depressive symptoms or infant IBQ-R scores, all $ps > .35$. All available SFP-R data were used in analyses.

To test whether maternal PTSD was associated with infant emotional reactivity and emotion regulation in response to the SFP-R, mixed models that accounted for repeated measures within participants across the SFP-R episodes were implemented. The models included main effect terms for PTSD and episode indicators as well as interaction terms between PTSD and the episode indicators. Analyses compared infants of mothers in the non-elevated and elevated PTSD symptoms groups. Contrasts of coefficients and associated F -tests were performed to examine specific hypotheses, as detailed below. Where appropriate, estimated mean difference scores between groups (i.e., the difference between non-elevated and elevated maternal PTSD groups in magnitude of change between episodes) and associated standard error scores were calculated as an indicator of magnitude of effect. To control for the effects of maternal depressive symptoms, analyses included maternal depressive symptoms as a covariate.³ Additionally, Mann Whitney U tests were run to explore differences between maternal PTSD groups in infant latency to negative affect during the still-face episodes.

3.2.1. Infant affective changes across SFP-R by maternal PTSD group—Infants of mothers in the non-elevated PTSD symptoms group and infants of mothers in the elevated PTSD symptoms group showed expected changes in affect across the SFP-R episodes. Specifically, both groups showed significantly more positivity during the play and reunion episodes than during the still-face episodes, $F(4, 144) = 32.81, p < .0001$ and $F(4, 144) = 15.10, p < .0001$ for the non-elevated and elevated groups, respectively (Figure 1). Infants in the two groups did not differ in their overall amount of positivity during the SFP-R, $F(5, 144) = 0.88, p = .50$. Infants in both groups also showed significantly more fussing [$F(4, 144) = 10.81, p < .0001$ and $F(4, 144) = 4.33, p = .002$ for the non-elevated and elevated groups, respectively] and crying [$F(4, 144) = 11.14, p < .0001$ and $F(4, 144) = 6.15, p = .0001$ for the non-elevated and elevated groups, respectively] during the still-face episodes than during the play and reunion episodes. The two groups did not differ in overall amounts of fussing, $F(5, 144) = 1.40, p = .23$ (Figure 2), or crying $F(5, 144) = 0.40, p = .85$ (Figure 3), over the course of the SFP-R. Infants in the two groups showed different amounts of hard crying across the SFP-R episodes, $F(5, 144) = 5.34, p = .0002$ (Figure 4). Infants in the non-elevated group showed very low levels of hard crying across all episodes, with no differences between play and reunion versus still-face episodes, $F(4, 144) = 0.84, p = .50$. Infants in the elevated group showed greater amounts of hard crying during the play and reunion episodes versus the still-face episodes $F(4, 144) = 8.13, p < .0001$. An examination of Figure 4 shows that infants in the elevated group showed low levels of hard crying during the play and first still-face and reunion episodes and higher levels during the second still-face episode and even higher levels during the second reunion episode.

3.2.2. Infant emotional reactivity by maternal PTSD group—To assess differences in infant emotional reactivity by maternal PTSD group, contrast tests were run to examine whether infants in the non-elevated and elevated maternal PTSD symptoms groups differed on the following: (a) magnitude of increase in negativity (fussing, crying, or hard crying) during the still-face episodes relative to the play episode, and (b) magnitude of increase in negativity during the second still-face episode relative to the first still-face episode. Additionally, Mann-Whitney U tests were run to test group differences in latency to negative affect (fussing, crying, or hard crying) during the still-face episodes. Greater reactivity was assumed from greater negativity during the still-face episodes relative to the play episode and during the second still-face episode relative to the first still-face episode

³Analyses were also run controlling for maternal depression scored categorically, using 12/13 as the cutoff for non-elevated/elevated classification. Results using the categorical approach revealed nearly identical results to those using a continuous approach, with no differences between approaches as to which analyses reached statistical significance. Results from analyses using the continuous measure of depressive symptoms are presented.

and from shorter latency to negative affect during the still-face episodes. Infants in the elevated maternal PTSD symptoms group tended to show greater reactivity than infants in the non-elevated maternal PTSD symptoms group as assessed by greater levels of hard crying during the second still-face episode relative to the first still-face episode, $F(1, 144) = 2.80, p = .096$, mean difference between PTSD groups = 8.96 (5.36). The remaining contrast tests were not significant, with ps ranging from .14 to .88. The groups also did not differ on Mann Whitney U tests of latency to negative affect during the first, $Z = 1.26, p = .21$, or second, $Z = 0.33, p = .74$, still-face episode.

3.2.3. Infant emotion regulation by maternal PTSD group—To assess differences in infant emotion regulation by maternal PTSD group, contrast tests were run to examine whether infants in the elevated and non-elevated maternal PTSD symptoms groups differed on the following: (a) magnitude of decline in negativity from each still-face episode to the subsequent reunion episode; (b) amount of negativity during reunion episodes relative to the play episode; (c) amount of negativity in the second reunion relative to the first reunion. Greater difficulty in emotion regulation was assumed from smaller declines in negativity during reunion episodes, greater negativity during reunions relative to play, and greater negativity during the second reunion relative to the first reunion.

Group differences emerged in the expression of the mildest (i.e., fussing) and most extreme (i.e., hard crying) forms of distress. As demonstrated in Figure 2, infants in the non-elevated maternal PTSD symptoms group showed increases in fussing during the second reunion relative to the first reunion, whereas infants in the elevated maternal PTSD symptoms group showed decreases in fussing between these two episodes, $F(1, 144) = 3.93, p = .049$, mean difference between PTSD groups = -19.98 (10.08). There were no group differences in the decline in the amount of fussing from the first still-face to the first reunion episode or from the second still-face to the second reunion episode or in the amount of fussing during the play episode versus either reunion episode, with ps ranging from .16 to .92.

As shown in Figure 4, infants in the non-elevated maternal PTSD symptoms group showed low levels of hard crying throughout the SFP-R, whereas infants in the elevated maternal PTSD symptoms group showed an increase in hard crying during the second half of the SFP-R. These different patterns were evident in significant group differences in the amount of hard crying during the second reunion versus the play, $F(1, 144) = 15.19, p = .0001$, mean difference between PTSD groups = 22.16 (5.69); the first reunion, $F(1, 144) = 18.13, p < .0001$, mean difference between PTSD groups = 24.21 (5.69); and the second still-face episodes, $F(1, 144) = 5.81, p = .02$, mean difference between PTSD groups = 14.16 (5.88). There were no group differences in the magnitude of decline in hard crying from the first still face episode to the first reunion episode or during the play episode versus the first reunion episode, with ps ranging from .69 to .83. No group differences emerged from any of the analyses involving changes in crying across episodes, with ps ranging from .31 to .77 (Figure 3).

3.3. Maternal PTSD Symptoms and Infant Temperament (IBQ-R) at 6 Months

Maternal PTSD symptoms at 6 months were tested for associations with infant IBQ-R scales at 6 months by calculating and testing Pearson correlation coefficients. Significant correlations were followed with multiple regression analyses including maternal depressive symptoms as a covariate if maternal depressive symptoms were significantly correlated with the outcome.

As shown in Table 1, maternal PTSD symptoms were not significantly associated with infant distress to limits in bivariate correlational analyses. Both maternal PTSD and depressive symptoms were significantly correlated with infant falling reactivity. When both

maternal PTSD and depressive symptoms were included in a multiple regression analysis predicting falling reactivity, neither variable was significant: PTSD symptoms, $\beta = -.22$, $t(46) = -1.10$, $p = .28$; depressive symptoms $\beta = -.17$, $t(46) = -0.84$, $p = .41$; overall $R^2 = .13$, $F(2, 44) = 3.15$, $p = .05$.

3.4. Maternal PTSD Symptoms and Infant Social and Emotional Symptoms (ITSEA) at 13 Months

Maternal PTSD symptoms at 6 months were tested for associations with infant ITSEA scales at 13 months by calculating and testing Pearson correlation coefficients. Significant correlations were followed with multiple regression analyses including maternal depressive symptoms as a covariate if maternal depressive symptoms were significantly correlated with the outcome.

As shown in Table 1, maternal PTSD symptoms at 6 months were significantly associated with infant externalizing, internalizing, and dysregulation symptoms but not competence behaviors at 13 months. Maternal depressive symptoms at 6 months were significantly correlated with internalizing and dysregulation symptoms. When both maternal PTSD and depressive symptoms were included in a multiple regression analysis predicting infant internalizing symptoms, neither variable was significant: PTSD symptoms, $\beta = .34$, $t(43) = 1.53$, $p = .13$; depressive symptoms $\beta = .04$, $t(43) = 0.20$, $p = .84$; overall $R^2 = .14$, $F(2, 41) = 3.32$, $p = .046$. When both maternal PTSD and depressive symptoms were included in a multiple regression analysis predicting infant dysregulation symptoms, PTSD symptoms remained significant but depressive symptoms were not significant: PTSD symptoms, $\beta = .50$, $t(43) = 2.34$, $p = .02$; depressive symptoms, $\beta = -.07$, $t(43) = -0.34$, $p = .74$; overall $R^2 = .20$, $F(2, 41) = 5.13$, $p = .01$.

3.5. Contribution of Direct Infant Exposure to Trauma to Infant Emotional Reactivity and Emotion Regulation

Using maternal report on the TESI-PRR, rates of infant direct exposure to potentially traumatic events by 6 and by 13 months of age were calculated. To determine if any associations between maternal PTSD and infant emotional reactivity or emotion regulation may be due to infant direct exposure to trauma, associations between infant trauma exposure and maternal PTSD symptoms and between infant trauma exposure and IBQ-R and ITSEA scales were examined by calculating and testing Pearson correlation coefficients. Multiple regression analyses and mixed model analyses were then run to determine whether maternal PTSD contributed to the prediction of any of the infant outcomes after adjusting for infant direct exposure to trauma.

Mothers reported that their infants were exposed to a range of potentially traumatic events over the first year of life on the TESI-PRR. By 6 months of age, 65% had not been exposed to any events, 30% had been exposed to one event, 2% to two events, and 2% to three events. By 13 months of age, 35% had not been exposed to any events, 33% had been exposed to one event, 24% to two events, 7% to three events, and 2% to four events. The most frequently reported events included undergoing a serious medical procedure/treatment by a paramedic or in an emergency room/overnight hospitalization/life-threatening illness (39%), separation from a primary caregiver for several days or under stressful circumstances (17%), seeing or hearing acts of war or terrorism on the television or radio (9%), being in a serious accident (9%), and death of someone close to the infant (4%).

Mothers in the non-elevated and elevated PTSD symptoms groups reported similar levels of infant trauma exposure at 6 and at 13 months of age: $M = 0.38$ (0.55) and 0.50 (0.90) events by 6 months of age, non-elevated and elevated groups, respectively, Mann-Whitney U test Z

= 0.02, $p = .99$; $M = 1.12$ (0.98) and 1.00 (1.21) events by 13 months of age, non-elevated and elevated groups, respectively, Mann-Whitney U test $Z = 0.62$, $p = .54$.

As shown in Table 1, trauma exposure by 6 months was not significantly associated with either of the IBQ-R scores using Pearson correlation coefficients (both $ps > .30$), and trauma exposure by 13 months was not significantly associated with any of the ITSEA scales using Pearson correlation coefficients (all $ps > .06$).

Multiple regression analyses predicting infant IBQ-R and ITSEA scores were run including maternal PTSD symptoms and infant trauma exposure as predictor variables. Also, the mixed models described in Section 3.2 that were implemented to analyze the associations between maternal PTSD symptoms and infant SFP-R responses were re-run to include infant trauma exposure as a covariate. In all analyses, the inclusion of infant trauma exposure had minimal impact on the magnitude of association between maternal PTSD symptoms and infant outcome and did not alter the study conclusions.

Discussion

The ability to regulate one's emotional responses to internal and external stimuli underlies mental health and competent functioning throughout life (National Research Council and Institute of Medicine, 2000, 2009). Emotion self-regulation largely develops over the first few years of life within the context of the caregiver-child relationship. Consequently, factors that may negatively impact maternal functioning, such as psychopathology, may impede the development of optimal child self-regulation abilities. Numerous studies have documented the negative impact that maternal depression may have on a child's emotional reactivity and emotion regulation (Beck, 1996; Brand & Brennan, 2009; Davis et al., 2004; Field, 2010). However, far fewer studies have examined the impact of maternal trauma exposure and PTSD on child emotional reactivity and emotion regulation. The primary goal of this study was to address this gap by testing associations between maternal PTSD symptoms and various measures of child emotional reactivity and emotion regulation in the first year of life, accounting for maternal depressive symptoms and infant direct trauma exposure.

Overall, analyses demonstrated that maternal PTSD symptoms were not associated with measures of infant emotional reactivity but were associated with measures of infant emotion regulation. Maternal PTSD symptoms were not significantly correlated with maternal report of the infant's tendency to become distressed in response to limits (reactivity) but were significantly correlated with maternal report of the infant's ability to recover once distressed (regulation) on a measure of infant temperament, the IBQ-R. Results from the repeated Still-Face Paradigm (SFP-R) were consistent with maternal report. That is, maternal PTSD showed minimal associations with infant emotional reactivity but was associated with infant recovery from distress. Specifically, the only difference between infants of mothers with and without elevated symptoms of PTSD in emotional reactivity to the SFP-R was a trend for the infants of mothers with elevated symptoms of PTSD to show a greater increase in hard crying during the second still-face episode relative to the first still-face episode. Thus, in general, infants of mothers with and without elevated symptoms of PTSD showed similar levels of distress in response to the stressor of the still-face episodes.

Significant group differences were evident in the dyads' abilities to regulate infant distress following termination of the still-face episodes. Infants of mothers with elevated symptoms of PTSD showed greater amounts of hard crying during the second reunion than during the baseline play episode, the first reunion, and the second still-face episodes. Infants of mothers without elevated symptoms of PTSD showed higher amounts of fussing in the second reunion relative to the first reunion but low levels of hard crying throughout the procedure.

Fussing and hard crying represent different levels of distress. During bouts of hard crying, infants were in a highly dysregulated state, often with their eyes closed while engaging in behaviors such as distressed breathing, screaming, and back arching. When in this state, infants had difficulty accepting soothing behaviors from their mothers and, consequently, calming to a non-distressed state. Infants of mothers with elevated symptoms of PTSD showed the greatest amount of this state of extreme dysregulation during the second reunion episode, which should have been a recovery stage. Infants of mothers without elevated symptoms of PTSD showed increases in fussing in later episodes of the procedure; unlike hard crying, such displays may reflect adaptive efforts to communicate distress without becoming dysregulated. During bouts of fussing, infants were often able to continue to engage with their mothers through eye contact, which allowed them to accept comforting gestures offered by the mother, reflected in decreases in negative affect among infants of mothers without elevated symptoms of PTSD during the reunion episodes.

The fact that maternal PTSD symptoms were more predictive of difficulties with infant emotion regulation than emotional reactivity may reflect the impact of maternal PTSD on quality of caregiving. As noted in Section 1.1, reactivity is frequently conceptualized as reflecting underlying biological response tendencies whereas regulation is hypothesized to arise through repeated interactions with the primary caregiver(s). Recent work has demonstrated that the quality of maternal caregiving when the infant is in a distressed state, such as following the still-face episodes, may have particular impact on the development of the child's psychophysiological functioning and, consequently, ability to self-regulate (Conradt & Ablow, 2010). More research is needed to determine if and how PTSD interferes with mothers' abilities to provide the type of sensitive care that promotes the development of child regulation abilities.

By 13 months of age, maternal PTSD symptoms were significantly associated with infant symptoms of externalizing, internalizing, and dysregulation symptoms as assessed by maternal report using the ITSEA. These results suggest that, by the end of the first year of life, infants of mothers experiencing elevated PTSD symptoms may be exhibiting behaviors indicative of emerging mental health difficulties, a potential functional implication of poor emotion regulation. As noted in Section 1.4, a number of studies have demonstrated increased rates of mental health problems among older children and adult offspring of parents with trauma histories/PTSD (Dekel & Goldblatt, 2008; Kaitz et al., 2009; Sack et al., 1995; Zajac & Kobak, 2009). More research is needed to determine what the mechanisms of these intergenerational effects are and how early in development such transmission occurs. The current findings suggest that there may be processes occurring very early in development that adversely impact the attainment of child self-regulation abilities, thereby increasing risk for the emergence of mental health difficulties.

The analyses did not provide support for the hypothesis that one of the mechanisms by which maternal PTSD increases risk for poor infant outcome is increased child exposure to trauma. Analyses revealed no significant associations between maternal PTSD and infant trauma exposure or between infant trauma exposure and infant emotional reactivity, emotion regulation, or emotional and behavioral symptoms. Moreover, including infant trauma exposure in the analyses had minimal impact on the associations between maternal PTSD and any of the infant outcomes.

The findings suggest that maternal PTSD symptoms may have associations with infant emotion regulation that are independent of maternal depressive symptoms. An elevated score on maternal PTSD symptoms was a significant predictor of infant emotion regulation during the SFP-R even after controlling for maternal depressive symptoms. Maternal PTSD symptoms but not depressive symptoms were associated with infant externalizing

symptoms, and maternal PTSD symptoms were predictive of infant dysregulation symptoms even after accounting for maternal depressive symptoms. That the association between maternal PTSD symptoms and infant internalizing symptoms was reduced to non-significance once maternal depressive symptoms were considered may be attributable to the strong correlation between maternal PTSD and depressive symptoms and/or lack of power given the relatively small sample size. However, notably, the standardized beta was greater for maternal PTSD symptoms than for maternal depressive symptoms in predicting infant internalizing symptoms.

More research is needed to determine how the impact of maternal PTSD may differ from, modify, and/or magnify the impact of maternal depression on child emotional functioning. To date, studies of maternal depression have largely ignored maternal PTSD symptoms, though the two conditions are highly comorbid (Chemtob et al., 2010; O'Campo et al., 2006). Therefore, associations between maternal depression and infant emotional functioning reported in the literature may be attributable, at least in part, to the effects of unexamined comorbid maternal PTSD, particularly in high-risk samples. Prior work suggests that maternal depression in the absence of comorbid psychiatric diagnoses or life adversity (e.g., poverty, dangerous environments) may confer limited increased risk to the mother-infant relationship (Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001). In Chemtob and colleagues' (2010) study of maternal PTSD and depression and child regulation, they were unable to examine the impact of maternal PTSD outside of depression due to an insufficient number of mothers with "pure" PTSD (3 of 116), highlighting the high prevalence of depression among mothers with PTSD. As noted above in Section 1.4., the authors did find that children of mothers with PTSD and depression showed poorer emotion regulation and greater behavior problems than children of mothers with depression alone or with neither disorder, leading the authors to conclude that maternal PTSD may result in greater child difficulties than maternal depression alone due to specific impairments that each disorder confers on maternal functioning or due to nonspecific additive burden from having both disorders.

The rates of trauma exposure and PTSD symptoms found within the current sample are consistent with prior findings among urban, low-income, ethnic/racial minority participants (Breslau et al., 1991, 1998; Brown et al., 2005; Clark et al., 2007; Hien & Bukszpan, 1999; Holman et al., 2000; Kessler et al., 1995; Kimerling, 2003; Stein et al., 1997). Therefore, the current findings are relevant for segments of the population most affected by trauma exposure and PTSD.

4.1 Limitations

The most significant limitation of the current study is the relatively small sample size. Despite the small sample size, significant associations were found between maternal PTSD and various measures of infant emotion regulation. A larger sample size may have revealed significant associations between maternal PTSD and infant emotional reactivity. As noted in Section 1.3, other studies have demonstrated associations between maternal PTSD and infant stress reactivity, possibly through prenatal programming of infant physiological systems by maternal stress hormones. Associations between maternal PTSD and infant emotional reactivity may have been stronger if maternal PTSD symptoms had been assessed during pregnancy rather than infancy, particularly if infant emotional reactivity is influenced by prenatal programming mechanisms.

The current analyses may have obscured findings if maternal PTSD is associated with both high and low levels of infant emotional reactivity, both of which, as noted in Section 1.3, may lead to difficulties in establishing self-regulatory abilities. Statistical methods that can account for such non-linear associations, possible with larger samples, may uncover

associations between maternal PTSD and different types of infant emotional reactivity. Other methods of measuring infant reactivity (e.g., peak cortisol response following SFP-R) may also reveal associations with maternal PTSD (Brand et al., 2010). Of note, a previous examination of ANS responsivity across the SFP in the current sample found that infants of mothers with high and low levels of perinatal traumatic stress showed similar patterns of ANS activation from the Play to the Still-Face, but the infants of mothers with high levels of perinatal traumatic stress showed poorer ANS recovery during the Reunion (Bosquet Enlow et al., 2009), again demonstrating associations between maternal posttraumatic stress and infant regulation but not reactivity.

The study hypotheses should be tested in larger samples to determine if the pattern of results replicate when there is greater power to detect effects. A larger sample size may also provide the power to detect any meaningful differences between infants who do and do not complete the SFP-R. The mixed models method employed to analyze the SFP-R data produces unbiased results if missing data are missing at random, and there was no evidence that the infants who did and did not complete the SFP-R differed on maternal PTSD or depressive symptoms or infant reactivity or regulation as assessed by maternal report. However, the possibility remains that the completers and non-completers differed on other unmeasured variables.

Another limitation of the current study is the use of maternal report for maternal symptoms of PTSD, infant temperament, and infant emotional and behavioral symptoms, potentially inflating correlations between maternal symptoms and infant outcome variables due to maternal reporting bias. However, objective scoring of the SFP-R showed a pattern of findings consistent with maternal report, namely that maternal PTSD symptoms were associated with differences in infant emotion regulation but not infant emotional reactivity. Of note, this is one of the only studies of maternal PTSD and child functioning to include an objective measure of child emotion regulation.

That the subjects were recruited from the community rather than mental health clinics increases the generalizability of the findings. The findings, however, may not generalize to other populations that have different resources or sources of support. The study hypotheses should be tested in more diverse (e.g., non-English speaking, different levels of socioeconomic status) samples to determine if the findings are applicable to other populations.

4.2. Conclusions

This is one of a few studies to examine associations between maternal PTSD and infant emotional functioning, demonstrating that maternal PTSD may have a negative impact on the development of offspring emotion regulation from the first year of life. Given the prevalence of trauma exposure and PTSD in the general population and especially in low-income urban populations as well as the importance of optimal emotion regulation skills for later development and mental health, this is an important area for future study. Further research is needed to determine the mechanisms by which maternal PTSD may impact child emotion regulation so that appropriate interventions may be designed to minimize intergenerational effects.

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Appendix

Repeated Still-Face Paradigm Infant Affect Codes

Code	Descriptor
Hard Crying	<ul style="list-style-type: none"> ◦High rhythmicity in crying ◦Screaming while crying ◦Lack of sound while crying due to large breath inhalations
Crying	<ul style="list-style-type: none"> ◦Intense negative facial expression while crying: deep vertical wrinkles between brows, brows drawn sharply down and together, thick lines in forehead, eyes tense and closed for extended periods of time, open, angular mouth ◦Rhythmic negative vocalizations with sad or angry facial expression: eyes squinted or briefly closed, pronounced bulge between brows, mouth tense, corners drawn straight back
Fussing/Negative Affect without Vocalizations	<ul style="list-style-type: none"> ◦Makes fussing noises/whimpers without tears or rhythmicity of crying ◦Displays negative facial expression (anger, grimace, sad): inner corners of brow raised, mild wrinkling between brows, mouth may be open or closed
Neutral	<ul style="list-style-type: none"> ◦Facial expression neither positive nor negative ◦Expression of interest ◦Forehead smooth, eyes open, mouth relaxed
Positive	<ul style="list-style-type: none"> ◦Smiles ◦Coos, babbles with positive expression (corners of mouth slightly drawn back, round, open mouth, brows slightly raised)
Very Positive	<ul style="list-style-type: none"> ◦Large smiles (open mouth, corners of mouth raised high, cheeks raised, eyes crinkled) ◦Laughs ◦Squeals with positive expression (eyes squinted, forehead smooth, corners of mouth drawn back)
Mixture Positive/Negative	<ul style="list-style-type: none"> ◦Shows a mixture of positive and negative affect (e.g., smiling while fussing)
Unclassifiable	<ul style="list-style-type: none"> ◦Shows affect that cannot be categorized into one of the above affect codes
Unobservable/asleep	<ul style="list-style-type: none"> ◦Face is obscured such that critical parts of face cannot be seen and affect cannot be inferred from vocalizations ◦Asleep
Autonomic Indicator	<ul style="list-style-type: none"> ◦Infant yawns or sneezes, and affect is not codable

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Research Highlights

- Maternal PTSD symptoms are associated with infant emotion regulation difficulties.
- Maternal depression does not account for maternal PTSD—infant regulation link.
- Infant trauma exposure does not account for maternal PTSD—infant regulation link.
- Poor regulation may increase mental health risk among children of mothers with PTSD.

Figure 1.

Mean rate of infant positive affect over the course of the repeated Still-Face Paradigm (SFP-R) by maternal PTSD group.

Note. Plots based on least squares means from the estimated mixed model.

Figure 2.

Mean rate of infant fussing over the course of the repeated Still-Face Paradigm (SFP-R) by maternal PTSD group.

Note. Plots based on least squares means from the estimated mixed model.

Figure 3.

Mean rate of infant crying over the course of the repeated Still-Face Paradigm (SFP-R) by maternal PTSD group.

Note. Plots based on least squares means from the estimated mixed model.

Figure 4.

Mean rate of infant hard crying over the course of the repeated Still-Face Paradigm (SFP-R) by maternal PTSD group.

Note. Plots based on least squares means from the estimated mixed model.

Table 1

Correlations Among Study Variables

Variable	1	2	3	4	5	6	7	8	9
1. Maternal trauma exposure (LSC-R)	--								
2. Maternal PTSD symptoms (PCL-C)	.46**	--							
3. Maternal depressive symptoms (EPDS)	.39**	.76***	--						
4. Infant trauma exposure (TESI-PRR) [†]	.13	-.02	.01	--					
5. Infant distress to limitations (6 months, IBQ-R)	-.10	.21	.24	.16	--				
6. Infant falling reactivity (6 months, IBQ-R)	-.32*	-.33*	-.32*	-.12	-.45**	--			
7. Infant externalizing symptoms (13 months, ITSEA)	-.12	.32*	.22	.28	.43**	-.16	--		
8. Infant internalizing symptoms (13 months, ITSEA)	-.11	.37*	.30*	.16	.45**	-.21	.61***	--	
9. Infant dysregulation symptoms (13 months, ITSEA)	-.01	.45**	.31*	.03	.49**	-.14	.68***	.70***	--
10. Infant competence behaviors (13 months, ITSEA)	.09	.16	.09	.16	-.03	.21	.31*	.11	.04

[†] All correlation coefficients involving infant trauma exposure were calculated using infant trauma exposure by 13 months of age except for the correlation analyses with the IBQ-R variables, which used infant trauma exposure by 6 months of age.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 2
Participant Demographic Characteristics

Variable	Total Sample (<i>N</i> = 52 dyads)	Non-Elevated PTSD Group (<i>n</i> = 38 dyads)	Elevated PTSD Group (<i>n</i> = 14 dyads)	<i>p</i> Value ^a
Maternal Age [<i>M</i> (<i>SD</i>)]	26.69 (6.10) years	26.34 (5.55) years	27.66 (7.56) years	.56
Maternal Education (%)				
High school, GED, or less	40%	32%	64%	.10
Some college or greater	44%	50%	29%	
Not reported	15%	18%	7%	
Maternal Marital Status (%)				
Single/Separated/Divorced/Widowed	40%	37%	50%	.60
Living with partner	15%	13%	21%	
Married	31%	34%	21%	
Not reported	14%	16%	7%	
Maternal Parity, % Primiparous	46%	53%	29%	.21
Financial Strain	5.99 (3.16)	5.13 (2.86)	8.21 (2.89)	.003
Maternal Race (%)				
White	52%	53%	50%	.14
Black	42%	45%	36%	
Asian	4%	0%	14%	
Multiracial	2%	3%	0%	
Maternal Ethnicity (%)				
Hispanic	25%	26%	21%	1.00
Non-Hispanic	75%	74%	79%	
Infant Sex, % Male	54%	53%	57%	1.00
Infant Birthweight [<i>M</i> (<i>SD</i>)]	3391 (466) grams	3389 (454) grams	3396 (514) grams	.96
Infant Gestational Age [<i>M</i> (<i>SD</i>)]	39.24 (1.87) weeks	39.16 (2.04) weeks	39.46 (1.34) weeks	.53
Infant Race (%)				
White	39%	45%	21%	.03
Black	42%	45%	36%	
Asian	4%	0%	14%	
Multiracial	15%	11%	29%	
Infant Ethnicity (%)				
Hispanic	33%	34%	29%	1.00
Non-Hispanic	67%	66%	71%	

^a *p* values comparing maternal non-elevated and elevated PTSD symptoms groups. Variables presented as means and standard deviations were compared using *t*-tests. Variables presented as percentages were compared using two-tailed Fisher's exact tests.