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Transgenerational effects of maternal exposure to trauma: The roles of parenting and cortisol as potential mediators

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An abstract of

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Abstract

Research suggests that maternal exposure to trauma may produce negative outcomes in offspring born post-trauma, including problems with anxiety; however, this body of literature is limited, and findings have been inconsistent. Even among those studies suggesting that such a relationship does exist, a consideration of mechanisms that might explain the process of intergenerational transmission of trauma effects has been largely absent. The current study aimed to explore the relationship between maternal exposure to trauma and child anxiety and to investigate two potential mediators of this relationship: parenting quality and child cortisol levels. By identifying mediators that might be involved in the intergenerational effects of maternal trauma exposure, we hoped to better understand points in the development of child anxiety that might be promising for prevention and intervention purposes. These aims were examined within a sample of 153 mother-child dyads who participated in a longitudinal study assessing maternal trauma history, parenting, and child cortisol reactivity at preschool age (ages 2.5 to 5.5 years) and maternal and secondary caregiver reports of child anxiety at school age (ages 6 to 10 years). We hypothesized that (1) maternal exposure to trauma would be positively associated with anxiety levels in offspring, and that (2) parenting quality and child cortisol reactivity would mediate the relationship between maternal exposure to trauma and child anxiety. Maternal exposure to trauma was found to be significantly related to child anxiety. Parenting, but not cortisol reactivity, was found to mediate this relationship. It was also found that type, timing, and cumulative trauma exposures might be differentially related to parenting, cortisol reactivity, and child anxiety outcomes. Clinical and research implications of these findings are discussed.

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Transgenerational effects of maternal exposure to trauma: The roles of parenting and child cortisol reactivity as potential mediators

Throughout the history of psychology, there has been an interest in trauma and the negative consequences it may have on the individual. This interest has, in some cases, been extended to trauma experienced in childhood as in Ferenczi's (1949) analysis of the effects of childhood sexual abuse on child outcomes and Levy's (1945) discussion of the "psychic trauma" that results when children undergo operative procedures. Only recently, however, has the scientific conversation turned to a consideration of transgenerational trauma effects, or the effects that parental exposure to trauma might have on offspring. This line of research has witnessed mounting interest with time, particularly as tragedies, such as the Holocaust and World War II, have become distant enough to study their effects in the offspring of those who experienced these events. For example, Rosenheck, in 1986, published work in which he examined the experiences of children who had grown up in households with fathers who suffered from Post-Traumatic Stress Disorder (PTSD) as a result of exposure to combat trauma in World War II. His study suggested that these veterans' offspring displayed transgenerational effects related to their father's combat exposure. Similarly, Wiseman and colleagues (2006) explored intergenerational effects of trauma among offspring of Holocaust survivors, specifically exploring parental overprotectiveness and maladaptive communication patterns as potential mediators of this relationship. Similar work has been conducted among the offspring of individuals exposed to the Vietnam War, Hurricane Katrina, and the Iraq-Iran War aimed specifically at examining offspring adjustment, distress, aggression, and anxiety levels as outcomes (e.g., Ahmadzadeh & Malekian, 2004; Beckham, Braxton, Kudler, Feldman, Lytle, & Palmer, 1997; Casseli & Motta, 1995; Davidson & Mellor, 2001; Davidson, Smith, & Kudler,

1989; Goodman & West-Olatunji, 2008; Harkness, 1993; Jordan et al., 1992; Motta, Joseph, Rose, Suozzia, & Leiderman, 1997; Parsons, Kehle, & Owen, 1990; Rosenheck & Fontana, 1998a; Rosenheck & Fontana, 1998b; Ruscio, Weathers, King, & King, 2002; Souzzia & Motta, 2004; Westerink & Giarratano, 1999). Notably, results of these studies have been mixed with some demonstrating transgenerational effects of trauma and others finding no differences between offspring of trauma-exposed parents and controls. More research is needed in order to better understand the potential impact of parental exposure to trauma on child outcomes and to understand this impact in the context of more commonly experienced traumas than genocide, natural disaster, or war.

Theoretical Arguments

Much of the work in this area has been couched in theories of attachment and bonding. Namely, this work rests on the assumption that parental exposure to trauma leads to stable and salient changes in the parent that will ultimately result in poor attachment and bonding between parent and child (Dekel & Goldblatt, 2008). This theoretical framework suggests that the distress that results from exposure to a traumatic event can lead to feelings of helplessness, anxiety, and fear in a parent that pose long-lasting vulnerabilities and risks. These vulnerabilities may result in intergenerational transmission of trauma effects in a number of ways. First, parents who have been exposed to trauma may "pass along" trauma effects directly via the child's direct exposure to their parent's symptoms. This idea is often associated with social learning theories, whereby children learn how to react to their surroundings, other people, and the world based on their observations of their parents' behaviors in childhood (Dekel & Goldblatt, 2008; Jordan et al., 1992). It has also been posited that parents, as a result of their trauma exposure, may engage in behaviors that create a more stressful environment for their children than there would be

otherwise. This might occur through means as severe as perpetration of abuse by the parent (particularly among parents who are victims of childhood abuse themselves) or as subtle as a parent having difficulty functioning emotionally and/or difficulty maintaining stable relationships with others (Banyard, Williams, & Siegel, 2003). Alternatively, a parent may simply have diminished ability to understand, be sensitive to, and respond to their child in an adaptive way. The creation of this unstable environment by the parent disrupts bonding and may eventually lead to negative outcomes in the child (Banyard, Williams, & Siegel, 2003).

A separate, more recently proposed theory concerning the intergenerational transmission of trauma involves biological mechanisms, including disruptions to the hypothalamic-pituitary-adrenal (HPA) Axis. Namely, this theory posits that mothers who have been exposed to trauma may develop permanently altered cortisol levels that predispose their offspring to negative outcomes prenatally. This theory is based on research demonstrating that adults with PTSD have reduced cortisol levels in comparison to controls (Yehuda, 2002), research demonstrating a relationship between maternal PTSD symptoms experienced during pregnancy and an infant's altered cortisol levels (Yehuda et al., 2005), and research demonstrating effects of maternal trauma exposure on both maternal and infant cortisol levels (Brand, Brennan, Newport, Smith, Weiss, Stowe, 2010).

Effects of parental exposure to trauma on offspring

Within the past few decades, studies have begun to show that parental exposure to trauma may influence child behavioral and emotional outcomes (see Hickman, 2012, for a review). However, this literature has been largely limited in three ways. First, the body of literature supporting intergenerational effects of trauma has generally focused on samples of parents who have a PTSD diagnosis, examining the relationship between PTSD symptoms and child

outcomes, rather than studying the effects of general exposure to trauma without a diagnosis of PTSD (e.g., Ahmadzadeh & Malekian, 2004). Secondly, this literature has focused on the effects of combat or war-related traumas rather than traumas spanning other domains, such as physical or sexual abuse (e.g., Van Ee, Kleber, & Mooren, 2012). Finally, this literature has largely studied the effects of *paternal* rather than *maternal* trauma exposure, likely as a result of the emphasis on combat trauma. A recent review conducted by a group at Harvard suggests that, while the link between fathers' PTSD symptoms and child outcomes has been well demonstrated, there is surprisingly little knowledge of the effects of maternal trauma on the child (Dekel & Goldblatt, 2008). Given that mothers often play the most prominent role in a child's upbringing, it would seem important to gain a better understanding of the influence of maternal trauma exposure on the development of child anxiety. That is the primary aim of the current study.

While limited in quantity, some research has focused on the relationship between *maternal* childhood trauma and child anxiety-related outcomes. For example, Smith and colleagues (2009) found that maternal exposure to sexual abuse in childhood was associated with higher levels of emotional and anxiety-related problems in offspring. Maternal exposure to childhood abuse has also been found to be related to physiological measures of anxiety in children as well. Javanovic and colleagues (2011) demonstrated that children's dark-enhanced startle responses were higher if their mothers had been exposed to childhood physical abuse. This literature suggests that maternal exposure to trauma may, in fact, influence anxiety-related outcomes in offspring. However, despite these questions now being asked in the literature, much work remains to be done in better understanding the mechanisms involved in the relationship between maternal exposure to trauma and child anxiety-related outcomes. The secondary aim of

this study is to examine two such potential mechanisms, child cortisol reactivity and parenting.

Maternal Trauma and Mother and Offspring HPA Axis Functioning

One system often activated when an individual is exposed to acute stress or trauma is the hypothalamic-pituitary-autonomic (HPA) axis—an intrinsic component of the body's stress system and one that is responsible for aiding the body in returning to homeostasis. When an individual is exposed to trauma, long-term alterations in the body's stress system may occur. Activation of the body's stress system results in the secretion of glucocorticoids, which have the ability to cross the blood-brain barrier (Gunnar & Quevedo, 2007), creating long-term effects in the individual's neurobiology. Previous studies have suggested that exposure to stressors or traumas contribute to dysregulated HPA axis functioning (Fisher, Kim, Bruce, & Pears, 2012) and that dysregulation of the HPA axis is involved in the pathogenesis of many psychological disorders (e.g., Tyrka et al., 2012). Less research, however, has been conducted demonstrating the effects of trauma on HPA axis regulation intergenerationally. Still, it is well substantiated that prenatal exposure to disrupted HPA functioning can affect infant cortisol levels via fetal programming—the process by which the development of the fetus is altered as a result of the conditions of its immediate environment (e.g., Seckl & Holmes, 2007). Therefore, it seems plausible that a mother's exposure to trauma may potentially later alter her offspring's HPA axis functioning in this way.

Levels of regulation within the HPA axis can be assessed via cortisol, a hormone secreted by the HPA axis, which increases sugars (glucose) in the blood system in an effort to regulate the body and brain's stress response. Dysregulation of cortisol and HPA activity can result in impaired ability to respond appropriately to encountered stressors (e.g., Shea, Walsh, Macmillan, & Steiner, 2005)—an impairment that might also place an individual at risk for subsequent

development of various forms of psychopathology (e.g., anxiety disorders, depression). While there has been extensive research conducted on the relationship between stress and cortisol in general populations (e.g., Meinlschmidt & Heim, 2005), far less has been investigated with regards to potential influences of maternal trauma exposure on maternal and child cortisol levels and subsequent child outcomes. Still, limited research provides support along these lines. For example, Shea and colleagues (2007) found that childhood maltreatment (as measured by the Childhood Trauma Questionnaire) was negatively correlated with baseline cortisol levels (cortisol awakening response, CARS) in mothers during pregnancy and explained 12% of the variance in this relationship. Additionally, our research group has demonstrated that maternal trauma exposure may have intergenerational effects in its association with cortisol hyporeactivity among trauma-exposed mothers following an infant stressor paradigm and cortisol hyperreactivity in their infants (Brand et al., 2010). Therefore, it appears that maternal exposure to trauma may have effects on both mothers' and children's HPA axis functioning. While studies along these lines have begun demonstrating relationships between maternal trauma and physiological outcomes, results have been mixed. Despite some studies showing steeper decline in cortisol reactivity or lower baseline cortisol levels, alternate findings have linked childhood trauma to HPA axis *hyper*activity, rather than hypoactivity, particularly in the mothers themselves (e.g., Heim et al., 2000). Therefore, much more research is needed in this area to delineate the relationship and direction of HPA axis and cortisol reactivity with maternal trauma exposure. Furthermore, additional research is needed to extend the relationship between maternal trauma exposure and cortisol levels to subsequent behavioral outcomes in offspring.

Cortisol and Child Anxiety Problems

HPA axis functioning and cortisol levels have proven to be pertinent in this picture of

transgenerational trauma effects, largely because they have been shown to predict emotional and behavioral outcomes in children. Many studies have demonstrated associations between HPA axis dysregulation and persistent anxiety problems in children and adolescents. (e.g., Greaves-Lord, Ferdinand, Oldehinkel, Sondeijker, Ormel, & Verhulst, 2007). Furthermore, it has also been shown that cortisol levels (particularly cortisol awakening response) may be highly heritable (Bartels, de Geus, Kirschbaum, Sluyter, & Boomsma, 2003). Therefore, maternal exposure to trauma has the potential of influencing child cortisol levels and subsequent anxiety problems through heritability of cortisol patterns between mother and child.

The conclusion that maternal trauma exposure may influence child cortisol levels and subsequent anxiety in the child may be implicitly drawn from various pieces of the literature demonstrating relationships between maternal trauma and maternal or child cortisol patterns and, separately, between child cortisol patterns and subsequent anxiety problems. Smith and colleagues (2009) demonstrated relationships between maternal trauma and child anxiety and, separately, between decreased cord blood cortisol levels and offspring anxiety in their study. However, very few studies have examined relationships among all three of these variables of interest (maternal trauma exposure, child cortisol levels, and child anxiety problems) in relation to one another within a mediation model. Our research group has published one study exploring the relationship between maternal trauma exposure and maternal and child cortisol levels in infancy, demonstrating that maternal trauma exposure may, indeed, affect both mothers' and children's cortisol patterns. However, it is important to now assess these relationships beyond infancy and into childhood given the tendency for psychological problems to emerge (or be more easily observed) later in development (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000).

Trauma and Parenting

In addition to the possible neuroendocrinological mechanism of cortisol, research has begun to uncover ways in which the relationship between trauma exposure and offspring outcomes might be related through the mechanism of disrupted parenting. Specifically, studies have shown that an individual's history of trauma exposure might impact the ways in which they eventually parent their children. For example, Zalewski and colleagues (2013) have recently shown that maternal trauma exposure (specifically emotional abuse and emotional neglect) is associated with lower rates of parental acceptance and higher levels of psychological control among parents. Other studies have demonstrated that an individual's exposure to childhood sexual abuse is associated with the increased use of physical punishment as a parent, perpetration of sexual and physical abuse on their own children, problems surrounding perceptions of children and the parent-child relationship, less support of one's child in their disclosing of their own abuse, and more negative views of one's self as a parent (e.g,. Alexander, Teti, & Anderson, 2000; Appleyard, Berlin, Rosanbalm, & Dodge, 2011; Banyard, 1997; DiLillo & Damashek, 2003; Douglas, 2000; Elliott & Carnes, 2001; Newcomb & Locke, 2001; Oats, Tebbutt, Swanston, Spieker, Bensley, McMahon, & Fung, 1996). Similar findings have been reported on the impact of physical abuse on parenting (e.g., Gara et al., 2000; Merrill, Hervig, & Milner, 1996; Milner, Robertson, & Rogers, 1990). These associations between exposure to trauma and negative parenting have now been so well-demonstrated that the field has turned to potential mediators and moderators of this relationship, finding, for example, that the relationship between trauma exposure and poor parenting may be moderated by one's experience of polyvictimization (spanning at least two different types of trauma; Hickman, et al., 2013; Zalewski, Cyranowski, Cheng, & Swartz, 2013), but not by total *frequency* of traumas in any one category. Despite these studies suggesting that there may be a relationship between trauma exposure and parenting,

few studies explore the effects that either exposure or parenting may then have on child behavioral outcomes. Examining these relationships is critical to understanding the long-term effects that parental trauma exposure may have, not just on the parent, but on his or her offspring as well.

Notably, some of the research in this area has focused on the effects of maternal trauma on parenting *stress,* rather than parenting behaviors. This body of work has shown that maternal trauma appears to result in greater feelings of parenting stress in comparison to controls (Crusto et al., 2010; Gewirtz, Degarmo, & Medhanie, 2011; Hickman et al., 2012). Several explanations have been proposed for this effect, including a parent's sensitivity to negative behaviors exhibited by her child or a bias toward seeing more negative traits and behaviors in the child and more negative dynamics within the parent-child relationship (Sprang, Staton-Tindall, Gustman, Freer, Clark, Dye, & Sprang, 2013). These findings speak to the complex nature of these relationships and the notion that poor parenting resulting from trauma exposure may, in fact, reflect a hypersensitivity to child behaviors that leaves an individual at greater risk for engaging in negative and ineffective parenting behaviors.

Intergenerational Cycle of Abuse

Parental trauma exposure and parenting are often discussed within the framework of the intergenerational cycle of abuse. Parenting is considered a potential pathway by which a mother's exposure to trauma may influence the way she behaves as a parent and, ultimately, affect her child. Broadly speaking, this concept of the intergenerational cycle of abuse refers to the general tendency for parents who have experienced abuse to be more likely to abuse their children themselves (e.g., Egeland, Jacobvitz, & Sroufe, 1988; Faller, 1989; Newcomb & Locke, 2001; Widom, 1989; Zuravin, McMillen, DePanfilis, & Risley-Curtiss, 1996). Several theories

have been proposed to explain why this cycle may take place, namely social learning and attachment theories. Social learning theorists posit that individuals learn that abuse is an acceptable method of parenting and later adopt this form of parenting as their own. Attachment theorists suggest that cognitive representations of early relationships with parents later develop into the same parent-child dynamics once individuals have children of their own. Both theoretical approaches are plausible explanations for the ways in which an individual's own history of abuse may result in negative parenting behaviors later in life.

Gaps in the Literature

While the topic of transgenerational trauma effects is beginning to garner more attention, research has focused on parental exposure to combat or disaster-related traumas, rather than exploring various types of traumas a parent might more commonly experience. One might expect that a parent's exposure to childhood physical or sexual abuse, interpersonal violence, or any number of individually experienced traumatic experiences could have similar effects on offspring as combat or natural disasters might, but this is largely unexplored to date. Secondly, much of the research in this area has focused on fathers' exposure to trauma, rather than mothers', likely due to the field's emphasis on combat trauma. Thirdly, many studies addressing this topic have focused on the effects of parental PTSD symptoms on offspring, rather than parents' general exposure to trauma. While there is a clear need to understand the effects of PTSD symptoms on offspring, it seems equally necessary to understand the impact of parental trauma exposure on children when the parent does not necessarily develop sufficient symptoms for a full diagnosis of PTSD. Studies have estimated that as much as 90 percent of civilian populations have or will be exposed to a traumatic event in their lifetime (e.g., Kessler et al., 1995; Kilpatrick, et al., 2013; Sartor et al., 2012). Furthermore, rates may be even higher among

veteran populations or within impoverished or conflict-ridden communities. Therefore, it is critical to understand any potential impact this exposure may have on later offspring.

In addition to these limitations of the literature, few studies in this area have investigated mechanisms that might be responsible for the relationship between parental exposure to trauma and negative child outcomes. Understanding the mechanisms that might be involved in the intergenerational effects of trauma on offspring is critical for several reasons. Firstly, any progress to be made in identifying appropriate points of prevention of anxiety in childhood and adolescence requires understanding the mechanisms involved in the development of this anxiety. Research has begun to demonstrate a relationship between parental exposure to trauma and negative child outcomes, but very little work has attempted to uncover the ways in which parental trauma exposure might impact child outcomes. By understanding the factors involved in this relationship, we might better design methods of preventing child anxiety among those children at risk. Examining parenting quality as a potential mediator becomes particularly relevant in this regard, because it is a point at which effective intervention may work to prevent the development of child anxiety. There have been many successes in modifying parenting behaviors to effect more positive child outcomes (e.g., Sanders, 1999; Serketich & Dumas, 1996); therefore, this point of intervention is promising. Additionally, studying these mechanisms in childhood rather than in later stages of development is informative for prevention and early intervention programs that can be implemented before the child's behavior problems become chronic or intractable.

Current Study

The current study aimed to address existing limitations in the literature by examining the potential intergenerational effects of maternal exposure (rather than PTSD diagnosis) to

commonly experienced forms of trauma (e.g., physical abuse, sexual abuse) on child anxiety symptoms. In particular, we hypothesized that children of mothers who have been exposed to physical or sexual traumas would have greater rates of anxiety symptoms than children of non-exposed mothers. Secondly, we aimed to examine both cortisol reactivity and parenting as potential mediators of the relationship between maternal trauma exposure and child anxiety. Finally, throughout these analyses, we explored whether the associations between maternal trauma and child anxiety outcomes might differ depending upon the timing (early childhood or lifetime), type (physical abuse or sexual abuse), or number (single or multiple) of traumas experienced. We hoped that by examining all of these issues we might address several critical gaps in the literature, better understand the transmission of trauma effects intergenerationally, and gain insight into the mechanisms by which these risks may be conferred.

Method

This study examined associations among maternal trauma, parenting, and child anxiety. Data used within this sample was collected at two time points: during a visit to our lab when children were preschool-aged (2.5 to 5.5 years) and within an online follow-up study conducted when children were school-aged (6 to 10 years).

Participants

Participants for the follow-up phase of this study were enrolled from an existing sample of 219 mother-child dyads that participated in a preschool study of child development associated with prenatal medication exposures. A total of 153 mothers from this initial sample were enrolled in the follow-up phase of the study, yielding a response rate of 70.8%. Women in the initial sample were recruited from two sources. The majority (81%) were recruited and assessed by the Emory Women's Mental Health Program (WMHP) within the Emory University School

of Medicine's Department of Psychiatry. The WMHP serves as a referral program that provides services and care for women suffering from mental illness with an emphasis on treatment throughout pregnancy, the post-partum period, and the breastfeeding period (Emory University School of Medicine, 2012). The remaining mothers in the preschool sample were recruited from the Emory Child Study Center database. This database includes mothers and children who responded to a mailing requesting participation in psychological studies focused on child development. The majority of the mothers recruited from the WMHP took psychotropic medications in pregnancy; none of the women from the Child Study Center database did so, as their children were explicitly recruited as non-medication exposed community controls. Participants recruited from the WMHP and controls did not differ on any demographics relevant to the current study (mother age, child age, mother/child ethnicity, mother's marital status, education level, number of hours worked per week, number of adults in the household) with the exception of number of children in the home (p = 0.046) with controls having more children in the household than participants recruited from the WMHP.

Children's ages ranged from 2.5 to 5.5 years old (M=3.73; SD=0.92) at the preschool phase of this study and from 6 to 10 years old (M=7.14; SD=1.07) at the time of the online follow-up. Mothers' ages ranged from 21 to 49 years old (M=36.82; SD=5.07) during the preschool phase and from 24 to 53 years old (M=41.06; SD=4.86) at follow-up. Participants spanned a range of ethnic backgrounds (82.6% Caucasian, 10.3% African American, 3.3% Hispanic, 2.3% Asian, 1.4% Biracial), education levels (38.5% graduate/professional degree, 5.2% part graduate/professional education, 33.8% bachelors degree, 6.6% associates degree, 13.1% part college, and 2.8% high school degree or GED), and marital statuses (82.2% married, 8% divorced, 2.3% separated, 7.5% never married). 85% of participating mothers experienced

one or more threshold diagnoses across the lifetime with Major Depressive Disorder being the most common diagnosis (29.1%). 60.1% of mothers were currently undergoing treatment (e.g., individual therapy, psychiatric services, group therapy) at the time of the preschool study.

Procedure

During the preschool phase of this study, participants visited a laboratory at Emory University for a 3-hour protocol during which several measures (described below) were administered to both mother and child and a parent-child interaction (described below) recorded. In the follow-up study, data were collected via a secure online database, called REDCap (Harris et al., 2009), which has been commonly used by research institutions for confidential data collection. Recruitment for the school-aged follow-up was conducted by contacting participants from the original study via email and asking if they would like to take part in a follow-up study with their child. Permission was previously obtained from these participants for follow-up, and contact information was provided for subsequent studies for which they were eligible. The email received by participants included a direct, personal hyperlink to the online measures listed below. Participants were instructed to click on the link, read consent information thoroughly, and complete the online questionnaires if they agreed to all stated study details. Participants were not required to complete all questionnaires in one sitting and were re-contacted if measures were left incomplete for longer than approximately 2 weeks. To supplement our mother-report measures, data was also gathered from an alternate caregiver (e.g., father, grandparent, teacher) using the REDCap procedures outlined above.

Measures

Maternal History of Trauma. Maternal trauma was assessed during the preschool phase of this study using two measures: the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink,

1998) and the Structured Clinical Interview for DSM-4 Axis I Disorders (SCID). The CTQ asks about the occurrence of several traumatic events (e.g., "I believe that I was physically abused." "Someone tried to touch me in a sexual way or tried to make me touch them.") over the course of an individual's childhood and adolescence. These items produced scores across five areas of childhood trauma: Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect. The internal consistencies of scales within this measure were acceptable (Emotional Abuse: Cronbach's $\alpha = 0.770$; Physical Abuse Cronbach's $\alpha = 0.686$; Emotional Neglect Cronbach's $\alpha = 0.898$) with the exception of the Physical Neglect (Cronbach's $\alpha = -0.002$) and Sexual Abuse (Cronbach's $\alpha = 0.005$) scales. These scales likely had such poor internal consistency due to the variability of the items comprising them. For example, the Physical Neglect scale includes the following items: "I didn't have enough to eat." "I knew that there was someone to take care of me and protect me (reverse coded)." "My parents were too drunk or high to take care of the family." "I had to wear dirty clothes." "There was someone to take me to the doctor if I needed it (reverse coded)." Given the variability of items in this scale, it might be expected that they would lack internal consistency. Scales for physical or sexual abuse were used for the current study to maintain consistency with this body of literature (e.g., Brand et al., 2010; DiLillo et al., 2006). On the Sexual Abuse scale, low levels of sexual abuse include scores between 6 and 7, moderate levels include scores between 8 and 12, and high levels include scores of 13 or higher. On the Physical Abuse scale, low levels of physical abuse include scores between 8 and 9, moderate levels include scores between 10 and 12, and high levels include scores of 13 or higher (Bernstein & Fink, 1998). Therefore, consistent with previous literature, individuals having scored either a 6 or above on the Sexual Abuse scale or an

8 or above on the Physical Abuse scale were coded as having experienced abuse in childhood (DiLillo et al., 2006).

In addition to the CTQ, the SCID was administered to mothers in our sample by a licensed clinician and several trained graduate students to assess mothers' mental health histories across the lifespan. Within this clinical interview, mothers were asked whether they had ever experienced an event that was extremely upsetting or life-threatening, "such as a major disaster, very serious accident or fire, being physically assaulted or raped, seeing another person killed, dead, or badly hurt, or hearing about something horrible that has happened to someone you are close to." This score was coded yes/no (1, 0) based on whether or not a mother had ever experienced a traumatic event in her lifetime. This measure allowed us to look at both childhood trauma exposure (as noted on the CTQ) and trauma across the lifespan (as captured by the SCID) separately as predictors. Interrater reliability for maternal reports of lifetime trauma was high (κ =0.98).

Child Cortisol Reactivity. Salivary cortisol samples were collected from children in our sample during the preschool visit. Two samples were collected during this lab visit, one of which was collected when the child arrived at the lab (baseline) and one of which was collected 20 minutes after the completion of frustration tasks (reactivity). The frustration tasks utilized during this visit were a green circles task in which a child was repeatedly told that the circle they had drawn was not quite perfect and a clear box task in which a toy was locked in a box and the child given the incorrect key. In order to collect cortisol within the lab, children were asked to chew on a piece of cotton dipped in a small amount of Kool-Aid. Salivary samples obtained with cotton are considered to be reliable and noninvasive techniques of obtaining cortisol, and

given the young age of the subjects at this time (2.5-5.5 years), this method was deemed most appropriate for study feasibility.

After chewing the cotton for one minute, a research assistant retrieved the cotton from the child, put it in the lumen of a 0.5ml syringe, and squeezed out about 1cc of saliva into a small tube. All saliva samples were frozen and stored at -20°C before being transported for assay at the Yerkes National Primate Research Lab at Emory. Upon arrival to Yerkes, saliva samples were stored at -20°C until the day of assay. On the day of assay, samples were thawed, vortexed, and centrifuged to remove any particulate matter.

Salivary cortisol was assayed using an enzyme immunoassay kit (DSL, Webster, TX), catalogue number DSL-10-67100. This assay procedure has an analytical sensitivity of 0.10 mg/dl, using 25 ml of saliva. The intra- and inter-assay coefficients of variation are 4.1% and 7.2%, respectively. Each sample was assayed in duplicate. Duplicate tests with an error of more than 20% were retested. Duplicate test results were averaged, and this value for cortisol will be used in analyses. Cortisol reactivity was measured with residuals calculated by regressing post frustration task cortisol onto lab baseline cortisol.

Parenting Quality. Parenting quality was assessed during the preschool visit using the Dyadic Parent-Child Interaction Coding System (DPICS; Eyberg & Robinson, 1981). Trained research assistants coded two ten-minute segments (one segment of structured play and one segment of unstructured play) of recorded interactions from each mother-child dyad by identifying and recording the frequency of various behaviors in both parent and child. The behaviors coded within this system are observable behaviors exhibited during social interactions between parent and child that vary in the degree of control exerted by the parent. These behaviors span three categories: verbalization, vocalization, and physical behavior.

Parenting variables for the current study were created using Principle Components Analysis (PCA). First, parental verbalizations and vocalizations from both structured and unstructured parent-child play were combined to yield 10 broad categories of behavior (see Table 1). These variables were then entered into the PCA using oblique rotation (Promax) to permit correlation among factors. The resulting pattern matrix can be found in Table 1 with values of 0.40 or higher indicated in bold font. This 0.40 threshold is commonly used in standard PCA methods within samples of this size (Field, 2009). The overall PCA yielded three distinct factors: Factor 1 (Positive Engagement) is comprised of reflective statements, neutral talk, indirect commands, information questions, and descriptive questions. Factor 2 (Negative Engagement) is comprised of negative talk and direct commands. Factor 3 (Positive Reinforcement) is comprised of labeled praise, unlabeled praise, and behavioral descriptions. All parenting behaviors demonstrated good to excellent interrater reliability with the exception of Behavioral Descriptions and Labeled Praise. Interrater reliability could not be calculated for these two categories of behavior due to insufficient frequency. See Table 2 for descriptions of each category of parenting behaviors.

Parenting variables used for analysis were residuals resulting from regressing total mother verbalizations onto the factor scores of positive engagement, negative engagement, and positive reinforcement; this approach controlled for maternal variations in overall levels of speech during the interactions.

Child Anxiety. To assess child anxiety levels, each child's mother and a secondary caregiver (e.g., father, grandparent) were asked to complete the Child Behavior Checklist (CBCL; Achenbach, 1991) during the online follow-up. This 113-item measure assesses children's behavior within the past six months across several domains, including anxiety

problems (e.g. worries, too shy or timid). The anxiety subscale within the CBCL is comprised of 6 items (e.g., "Clings to adults or is too dependent," "fears going to school,") each coded as being either "not true" (0), "somewhat or sometimes true" (1), "or very true or often true" (2). The subscale's minimum possible score is 0 and maximum possible score is 12 with higher scores reflecting higher levels of anxiety. Mother-, and alternate caregiver-reported ratings on the CBCL of children's anxiety behaviors served as criterion measures for comparison to ratings derived from the Pediatric Anxiety form of the Patient-Reported Outcomes Measurement Information System (PROMIS)—a newer measure of anxiety funded by the National Institute of Health (NIH) and designed to screen children for early signs of anxious behaviors. The parent proxy version of this instrument was used for this study, which is deemed valid for children ages 6 to 17. This measure includes 10 items spanning behaviors and feelings that are typical of anxious children (e.g., "In the past 7 days, my child felt nervous."). Each item is coded as occurring "never" (1) in the past 7 days, "almost never" (2), "sometimes" (3), "often" (4), or "almost always" (5). This measure has a minimum score of 10 and a maximum score of 50 with higher scores reflecting higher levels of child anxiety.

By including several measures of child anxiety problems, we hoped to gain a better understanding of the best approaches to measuring anxiety and internalizing behaviors in school-age children. In addition, using multiple reporters allowed us to avoid any biases that might result from relying solely on mothers' reports. Previous research suggests that maternal distortions in reporting on child psychopathology and discrepancies between informants necessitates the inclusion of multi-informant measures whenever possible (e.g., De Los Reyes & Kazdin, 2005).

Life Stress. To assess early life stress, mothers were asked to complete a self-report measure called the Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978) during their lab visit, endorsing any events they had experienced in the past 6 months or since their pregnancy with the enrolled child, as well as how positively or negatively those events impacted them. For the purposes of the current study, it is assumed that any stressor experienced by the mother will affect her child either directly (as in the case of parental conflict or divorce) or indirectly (as with mother's job loss or change in sleep habits). The LES consists of 57 items, each indicating one potentially stressful event that may have been experienced by the mother since the child's birth. Both quantity of stressors and subjective ratings of an event's impact were included in the current analyses.

Statistical Approach

Multiple linear regression analyses were used to test all hypotheses. Within these analyses, covariates were included in Block 1 of each model (see Table 3 for a list of all included covariates), maternal trauma variables were entered as predictors in Block 2, and child anxiety was entered as the outcome variable (as measured by mother-reported PROMIS scores, mother-reported CBCL-Anxiety scores, and alternate caregiver-reported CBCL-Anxiety scores). Maternal trauma was measured in several ways (presence of either physical or sexual abuse in childhood, history of trauma across the lifetime, cumulative childhood trauma, childhood physical abuse only, and childhood sexual abuse only) in order to assess differential effects across type, timing, and quantity of traumas. Parenting variables (Positive Engagement, Negative Engagement, and Positive Reinforcement) and cortisol reactivity were each separately tested as potential mediators using regression analyses and indirect effects analyses as well. To demonstrate mediation, linear modeling was used to, first, test the effects between maternal

trauma exposure (independent variable) and either parenting or cortisol (mediators), and between parenting or cortisol (mediators) and mother- and caregiver-reported child anxiety (dependent variable). Subsequently, methods described by Preacher and Hayes (2014) were used to determine the indirect effects of maternal trauma exposure on child anxiety through cortisol reactivity and parenting. Testing indirect effects among variables of interest has come to supersede the more traditional Baron and Kenny (1986) approach to testing mediation given recent work asserting that a direct effect between predictor and outcome variables is not necessary to demonstrate mediation (e.g., Collins, Graham, & Flaherty, 1998; Hayes, 2013). Consequently, bootstrapping methods have come to be seen as more acceptable for testing mediation models. Within this study, MEDIATE for SPSS was used to explore indirect effects among maternal trauma exposure, cortisol reactivity, parenting, and child anxiety given its suitability for testing both continuous and categorical variables (Hayes & Preacher, 2014).

Results

Preliminary Analyses

Sample characteristics of each of the five included trauma variables can be found in Table 4, and descriptive data for all variables of interest can be found in Table 5. Preliminary analyses were conducted to ensure data satisfied the assumptions of multiple linear modeling, to ensure that there were no errors or outliers, and to ensure that variables of interest were not so highly correlated that the analyses might be compromised. Variables found to be significantly skewed were log transformed before being included in analyses. Cortisol reactivity was found to be significantly skewed, and was consequently log transformed. Attrition analyses were also conducted to assess any differences in those participating in the follow-up phase of the study compared to those who did not. These analyses revealed that mothers enrolled in the follow-up

study were more educated than those who were not enrolled in this phase (p=0.015). There were no differences between these two groups on level of trauma exposure (ps > 0.095).

In all analyses, potential confounds (e.g., child's age, gender, or ethnicity) were entered as controls when significantly related to outcome variables at $p < .05$ (see Table 3 for a list of all included covariates). Bivariate correlations between variables of interest can be found in Table 6.

Measures of Child Anxiety

Three measures of childhood anxiety were included as outcomes in these analyses, two of which were completed by mothers of children in the study and one of which was completed by an alternate caregiver (e.g., father, grandparent, teacher). Bivariate correlations were conducted to ensure that these three measures were related and, particularly, to assess the validity of a fairly new measure of anxiety, the PROMIS. Results indicated that these measures were all correlated at $p < 0.001$, suggesting that the PROMIS is indeed a valid a measure of anxiety that appears consistent with the more established and well validated CBCL (see Table 6).

Hypothesis 1

It was first hypothesized that maternal trauma exposure would significantly and positively predict levels of anxiety in school-aged offspring. General exposure to trauma was measured using two variables, presence or absence of physical or sexual abuse in childhood ("Childhood Trauma") and presence or absence of trauma across the lifetime ("Lifetime Trauma"). These variables were each included as independent variables in the prediction of three outcome variables of the three included measures of child anxiety described above (mother-reported PROMIS total scores, mother-reported CBCL-Anxiety ratings, and alternate caregiver-reported CBCL-Anxiety ratings). After exploring relationships between general

exposure to trauma and child anxiety at follow-up, differences across the type of trauma experienced (physical abuse or sexual abuse in childhood) were investigated. Subsequently, timing effects were explored by entering both lifetime trauma and childhood trauma exposure in the same regression model predicting child anxiety, to see if they had differential effects above and beyond one another. Lastly, cumulative trauma (coded as absence of trauma, exposure to only type of trauma (physical *or* sexual trauma), or exposure to both types of trauma (physical *and* sexual trauma)) was examined as a predictor of child outcomes. These analyses were conducted for each of the three included measures of child anxiety (see Table 7).

Trauma Exposure Predicting Child Anxiety. Maternal exposure to trauma (as measured by general trauma exposure across the lifetime) was found to significantly and positively predict child anxiety across CBCL-Anxiety ratings by both mother- and alternate caregiver-report at $p<0.05$. Other measures of maternal exposure to trauma (Childhood Trauma, Cumulative Trauma, Childhood Sexual Abuse Only, and Childhood Physical Abuse Only) were not found to predict child anxiety levels at school age (*p*s>.147) as assessed by any of the three included outcome measures. Given that only Lifetime Trauma predicted child outcomes, whereas all four childhood measures of trauma exposure did not, these results may suggest that more recent traumatic events in mothers' lives have more of an effect on children's behavioral outcomes than more temporally distant, childhood traumas.

Differential Effects by Type of Trauma. Neither Childhood Sexual Abuse nor Childhood Physical Abuse predicted current child anxiety levels (*p*s>.147). Given that neither of these types of trauma predicted child outcomes, it does not appear that there are differential direct effects of maternal trauma on child anxiety based on the type of traumatic experience a mother experienced in childhood.

Differential Effects by Timing of Trauma. Regression analyses were conducted examining whether Lifetime Trauma predicted child anxiety outcomes above and beyond childhood exposure to trauma. Results suggest that, when controlling for Childhood Trauma, Lifetime Trauma does significantly predict outcomes in the expected direction. This is true of both mother- and alternate caregiver reports on the CBCL (β=.215; p=.022 and β=0.277; p=.032, respectively). Again, this suggests that more recent traumatic events may have a larger impact on child outcomes than earlier traumatic events occurring in childhood.

Cumulative Effects of Trauma Exposure. Cumulative levels of maternal trauma exposure were computed by combining childhood physical and sexual abuse variables. In this way, total trauma exposure could reflect absence of any childhood abuse, presence of one type of trauma, or presence of both types of trauma. This cumulative trauma variable did not predict any of the three included outcome measures. Therefore, there does not appear to be a dose-dependent relationship between independent and dependent variables whereby maternal exposure to multiple types of trauma results in significantly higher levels of child anxiety.

Hypothesis 2

Cortisol Reactivity as a Potential Mediator. We hypothesized that cortisol reactivity and parenting would mediate the relationship between maternal trauma exposure and child anxiety. Given that cortisol reactivity was significantly skewed, it was log transformed before being included in analyses. Additionally, this variable was residualized to reflect the portion of cortisol reactivity accounting for each child's stress response rather than their baseline cortisol level. Data was gathered on the time of cortisol collection, as well as the child's food intake on the day of collection, medications taken, level of exercise/activity, and other factors found to influence cortisol levels. The only variables found to be significantly related to cortisol

reactivity in this sample were whether a child had eaten prior to the lab visit and whether a child currently had a loose tooth. Given that these variables were related to cortisol reactivity, they were controlled for in all mediation analyses.

Before testing indirect effects among maternal exposure, cortisol reactivity, and child anxiety, preliminary regression analyses were conducted to explore the direct effects between these variables of interest. First, relationships between predictor variables (representing various forms of maternal trauma exposure) and cortisol reactivity were tested. Of the five included predictor variables (Childhood Trauma, Lifetime Trauma, Cumulative Trauma, Childhood Sexual Abuse Only, and Childhood Physical Abuse Only), only Childhood Sexual Abuse (β=-0.210; p=0.019) predicted cortisol reactivity. This relationship was negative, suggesting that children of mothers having experienced sexual abuse in childhood showed lower cortisol reactivity in response to lab stressor tasks. No other predictor variables were found to be associated with cortisol reactivity (ps>0.170). Second, relationships between cortisol reactivity and all three outcome variables (mother-reported PROMIS scores, mother-reported CBCL-Anxiety scores, and alternate caregiver-reported CBCL-Anxiety scores) were tested. Cortisol reactivity was not found to be significantly associated with any of these three outcome variables (ps>0.313).

Following these analyses of the direct effects among maternal trauma exposure, cortisol reactivity, and child anxiety variables, indirect effects were tested using MEDIATE. Results within MEDIATE are considered to support an indirect effect from predictor variable to outcome variable through the proposed mediator if confidence intervals do not include a null value of zero. Fifteen potential indirect pathways were tested including cortisol reactivity as a mediator (e.g., Childhood Trauma→Cortisol Reactivity→Mother-reported PROMIS scores, Lifetime

Trauma→Cortisol Reactivity→Mother-reported PROMIS scores, Childhood Sexual Abuse Only→Cortisol Reactivity→Alternate Caregiver-reported CBCL Anxiety scores). Of these tested pathways, none was found to be significant when including cortisol reactivity as a mediator (see Table 8).

Parenting as a Potential Mediator. Before testing indirect effects among maternal exposure, parenting, and child anxiety, preliminary regression analyses were conducted to explore the direct effects between these variables of interest. Parenting behaviors were represented by the three factors derived from a principal components analysis of maternal behaviors observed during two parent-child interactions. These factors include Positive Engagement, Negative Engagement, and Positive Reinforcement (see Tables 1 and 2 for descriptions of the behaviors that constitute these factors). Each of these three variables was then converted to its residual form. This enabled us to assess differences between mothers that were attributable to the type of parenting behaviors exhibited, rather than differences attributable to the overall amount of speaking a mother engaged in during the parent-child interaction. These residualized variables were used in all mediation analyses.

Findings from our analysis of direct effects among these variables were mixed. Negative Engagement was found to be significantly and positively predicted by Lifetime Trauma (β=.236; p=.007), such that mothers with lifetime trauma exposure displayed greater levels of negative engagement with their children during parent-child interactions. Negative Engagement was not significantly predicted by Childhood Trauma (β=.215; p=.022), but trended toward significance (β=.157; p=.068), suggesting that more recent lifetime traumas may have greater impact on mothers' parenting behaviors. Additionally, Negative Engagement was significantly and positively predicted by Cumulative Trauma (β=.270; p=.001), Childhood Sexual Abuse Only

(β=.199; p=.019), and Childhood Physical Abuse Only (β=.240; p=.005). Positive Engagement was found to be significantly and negatively predicted by Lifetime Trauma (β=-.227; p=.018) and Childhood Sexual Abuse Only (β=-.204; p=.040). These findings suggest that mothers with lifetime histories of trauma or childhood sexual trauma are less likely to display positive engagement in interactions with their children. Cumulative Trauma also significantly predicted Positive Engagement (β=-.209; p=.034). Positive Engagement was not found to be significantly predicted by Childhood Trauma (β=-.125; p=.206) or Childhood Physical Abuse (β=-.126; p=.189). Positive Reinforcement was found to be significantly predicted by Childhood Physical Abuse (β=-.256; p=.003) and by Cumulative Trauma (β=-.192; p=.029). No other trauma variables were found to be associated with Positive Reinforcement (ps>.187). With regards to outcome variables, Negative Engagement was found to significantly and positively predict child anxiety as measured by both mother-reported PROMIS scores (β=.222; p=.012) and mother-reported CBCL-Anxiety scores (β=.197; p=.025), but not alternate caregiver-reported CBCL-Anxiety scores (β=.087; p=.462). Positive Engagement trended toward significance in predicting mother-reported PROMIS scores (β=-.169; p=.058) and significantly predicted mother-reported CBCL-Anxiety scores (β=-.177; p=.045), but was not predictive of alternate caregiver-reported CBCL-Anxiety scores (β=-.112; p=.339). Lastly, Positive Reinforcement was not found to be significantly associated with any outcome variables (ps>.467).

Following these analyses of the direct effects among maternal trauma exposure, parenting, and child anxiety variables, indirect effects were tested using MEDIATE. Results within MEDIATE are considered to support an indirect effect from predictor variable to outcome variable through the proposed mediator if confidence intervals do not include a null value of zero. 45 potential indirect pathways were tested including each of the three parenting

variables as a potential mediator (e.g., Childhood Trauma→Negative Engagement→Mother-reported PROMIS scores, Lifetime Trauma→Positive Engagement→Mother-reported CBCL-Anxiety scores, Childhood Sexual Abuse Only→Positive Reinforcement→Alternate Caregiver-reported CBCL Anxiety scores). Of these analyses, several indirect effects were found to be supported (see Table 9-11). Childhood Trauma, Lifetime Trauma, Cumulative Trauma, Childhood Sexual Abuse, and Childhood Physical Abuse were each found to significantly predict mother-reported PROMIS scores via Negative Engagement. Additionally, Lifetime Trauma, Cumulative Trauma, and Childhood Sexual Abuse were found to significantly predict mother-reported PROMIS scores via Positive Engagement. Cumulative Trauma and Childhood Sexual Abuse were also found to significantly predict mother-reported CBCL Anxiety scores via Positive Engagement. There were no significant indirect effects found supporting the role of Positive Reinforcement as a mediator, nor were there any significant effects observed predicting alternate caregiver reported CBCL-Anxiety scores as an outcome.

Hypothesis 3 (Post-hoc/Exploratory)

Post-hoc analyses were conducted to explore whether parenting might act as a moderator in addition to acting as a mediator in the relationship between maternal trauma exposure and child anxiety. In order to conduct these analyses, interaction terms were computed within SPSS by centering each independent variable of interest around its mean and computing the product of the two variables predicted to interact (i.e., maternal trauma exposure x parenting). Within these models, main effects of maternal trauma exposure and parenting were entered, as well as the interaction term between maternal trauma exposure and parenting, in predicting child anxiety levels. None of the interactions that were tested were statistically significant ($ps > 0.316$).

Exploratory analyses were conducted to examine whether the relationship between

cortisol and child outcomes might be moderated by the stress present in children's environments. Therefore, stressful life events (within the past six months and since the child's birth) were tested as moderators in this relationship using multiple linear regression analyses. In order to conduct these analyses, interaction terms were computed within SPSS by centering each independent variable of interest around its mean and computing the product of the two variables predicted to interact (e.g.,, cortisol reactivity x life stress). Within these models, main effects of cortisol reactivity and life stress were entered, as well as the interaction term between the two, in predicting child anxiety levels. Only one of the tested interactions was significant (cortisol reactivity x stress since the child's birth; β=-0.179; p=0.042), but when the sample was split on levels of lifetime stress to explore the nature of this interaction, both subsamples yielded negative, non-significant associations between cortisol reactivity and child anxiety. Therefore, it does not seem that life stress moderates this relationship in any meaningful or interpretable way.

Discussion

To better inform research in the area of transgenerational effects of maternal exposure to trauma, relationships among maternal trauma exposure, cortisol patterns, parenting, and child anxiety were examined within this study. A relationship between maternal exposure to trauma and child anxiety was demonstrated lending support to our first hypothesis. This direct relationship appeared to be most salient for lifetime, rather than childhood, traumatic events. Our second aim was to examine the roles of parenting and cortisol reactivity as potential mediators in the relationship between maternal trauma exposure and child anxiety levels. While findings support the role of parenting as a mediator of this relationship, the same was not found to be true for cortisol reactivity. Lastly, parenting was not found to moderate the relationship between maternal trauma exposure and child anxiety, nor was life stress found to moderate the

relationship between cortisol reactivity and child anxiety.

The findings from the current study address several gaps in an existing body of literature that focused primarily until now on the effects of PTSD diagnosis among fathers having experienced war-related traumas. Additionally, given that our study design measures variables of interest across multiple observers (mothers, alternate caregivers, and observational data collected by researchers in a controlled lab setting), the findings presented in this study are considered to be a valuable contribution to this body of literature.

Relationship between Maternal Trauma Exposure and Child Anxiety

Our first hypothesis was supported in that maternal trauma exposure positively predicted child anxiety levels in the case of mother and alternate caregiver-reported ratings. Supplemental analyses were conducted to assess whether maternal trauma exposure predicted child anxiety above and beyond children's levels of life stress (as measured by the LES), and findings remained the same with inclusion of life stress (both recent stress in the past 6 months and stress across the child's lifetime) in our models. This suggests that maternal trauma exposure can impact children above and beyond the stress that children themselves experience in their immediate environments. Therefore, in understanding the development of child anxiety, it is important to consider not only environmental stress and risks faced by children themselves, but also risks that may be transmitted intergenerationally.

Support of a relationship between maternal trauma exposure and child outcomes also sheds light on a question that is tremendously important given the prevalence of traumatic experiences in our lives: how does trauma affect not only the individuals experiencing it, but also those within their care? A recent study assessed trauma exposure within a national sample of nearly three thousand American adults according to DSM 5 criteria and found that 89.7 percent

of them had experienced at least one traumatic event in their lifetime with multiple traumas being the norm rather than the exception (Kilpatrick, Resnick, Milanak, Miller, Keyes, & Friedman, 2013). Given this incredibly high prevalence, it is essential that we understand all the effects associated with traumatic experiences not just on the individuals experiencing them, but also on the children who are in their care. It is particularly critical that we understand the effects of these events on mothers given the traditional role of mother figures as most active in childrearing, as well as the impact mothers can have on offspring prenatally. These findings also extend a body of literature that has largely focused on combat trauma and paternal figures until now. Within this study, we see that these effects are not only noticeable when parents experience traumas within the context of wars, genocide, or natural disasters, but also childhood physical and sexual abuse and lifetime traumas as well.

Results of this study also suggest that type, timing, and the number of traumas experienced may yield differential effects on child outcomes, though results in these areas were very much mixed. Specifically, outcomes within this study appeared to yield the strongest direct effects when lifetime trauma exposure, rather than childhood trauma exposure, was included as a predictor. This result negates findings in the trauma literature that suggest that there are no timing effects involved in the impact of trauma on the individual or that cumulative trauma is more predictive of negative outcomes than recent traumas (e.g., Dubowitz et al., 2001). Within our study, both cumulative trauma and lifetime trauma were analyzed, and the latter appeared to be more impactful in predicting outcomes. However, it is important to note that our measure of lifetime trauma did not explicitly assess for age or time that traumatic events were experienced, so this finding should be interpreted with caution. While there were differential effects observed based on when a trauma was experienced, there did not appear to be any

consistent effect of the type of trauma experienced on outcomes. Results involving relationships between physical and sexual abuse that included cortisol and parenting variables were mixed such that, at times, childhood sexual abuse predicted proposed mediators, while childhood physical abuse did not, and at times, the opposite was found to be true. Therefore, no conclusions about differential effects of the types of trauma experienced by mothers can be drawn based on results of this study.

Parenting and Cortisol Reactivity as Potential Mediators

. Our hypothesized mediation models were partially supported. Significant indirect effects were found supporting the role of parenting as a mediator in the relationship between maternal trauma exposure and child anxiety; however, results did not lend support for cortisol reactivity acting as a mediator. Among the tested parenting variables, positive engagement and negative engagement were found to mediate the relationship between maternal trauma exposure and child anxiety, whereas positive reinforcement was not found to mediate this relationship. These results suggest that the mechanisms by which maternal trauma exposure leads to child anxiety involve mothers' use of more negative engagement and less positive engagement in interactions with their children compared to mothers without histories of trauma. Notably, these indirect effects were found to be true for each trauma variable (childhood, lifetime, cumulative, sexual abuse, and physical abuse), suggesting that these mechanisms may be generalizable across various types of trauma occurring at various times in the lives of the mothers who experienced them.

Specifically when involving *childhood* trauma exposure, trauma-exposed mothers tendency to engage in lower levels of positive engagement and greater levels of negative engagement may reflect these mothers' adoption of ineffective parenting styles that they

themselves were subjected to as children. Thus, even if these mothers have not gone on to abuse their children in accordance with the intergenerational cycle of *abuse* theory, these findings may still suggest an intergenerational cycle of *poor parenting* that leads to similarly negative child outcomes. These findings are tremendously helpful for elucidating the mechanisms by which we may see intergenerational effects of maternal trauma exposure transmitted to offspring. Moreover, they suggest that even when actual abusive behaviors are not adopted by mothers who have been exposed to trauma themselves, ineffective parenting behaviors adopted by these mothers can still lead to unfavorable outcomes in children.

Findings involving the role of parenting as a potential mediator are particularly promising given that they provide an opportunity for a possible point of prevention. The age-old adage that "prevention is better than cure" certainly applies to the development of anxiety in childhood and suggests that the emphasis in clinical research on anxiety should be on how to prevent these disorders, rather than the more common approach of researching effective treatments of these disorders. It has been estimated that anxiety disorders, because they are one of the most common forms of mental illness in the United States, cost the country more than $42 billion per year, nearly one third the national bill for all mental-health related costs. This tremendous cost to the country and to individuals suffering from anxiety disorders necessitates our better understanding of the ways in which these disorders might be prevented. Several methods of preventing the development of mental health disorders have involved parenting, and anxiety disorders are no exception. Moreover, parenting might serve as an especially effective means of preventing the development of anxiety disorders, particularly given the large role that parents play in their children's development at this stage. Parent-focused prevention programs targeting child anxiety have included such approaches as having parents model appropriate coping behavior, encourage

children to expose themselves to anxiety-provoking situations, help children to distinguish between threatening and nonthreatening stimuli, and reduce overprotective and critical childrearing responses (Donovan & Spence, 2000). The latter of these strategies is particularly relevant to the current findings given that negative engagement was one of the most consistently relevant factors in our mediation models. By training trauma-exposed mothers in how to minimize negative engagement and increase positive engagement in interactions with their children, there may be vast and long-lasting benefits to children who would have developed anxiety disorders without having received this intervention.

Our hypothesis that cortisol reactivity would mediate the relationship between maternal trauma exposure and child anxiety was not supported. This suggests that one of the more commonly hypothesized biological mechanisms in transgenerational risks, namely that of cortisol reactivity, may not actually explain the relationship between maternal trauma and child outcomes. Interestingly, cortisol reactivity was found to be negatively related to maternal sexual abuse. Notably, this finding complements results found within a previous study conducted by this research group in which maternal trauma exposure was found to predict both maternal and child cortisol levels (Brand et al., 2013). In this earlier study, maternal trauma exposure was found to predict *hypo*reactivity in maternal cortisol response after a stressor task, but was found to predict *hyper*reactivity among infants in the study. Researchers suggested that those patterns that were observed in infants may later develop to more closely resemble the neuroendocrinological profiles of their mothers, and according to our results, this may indeed be the case. The findings presented in the current paper suggest that, despite their *hyper*reactivity in infancy, children of trauma-exposed mothers may be *hypo*reactive to stressor tasks compared to children of non-trauma-exposed mothers by the time they reach preschool age. This

developmental trajectory of cortisol patterns from hyper- to hyporeactivity may serve as an important marker of a particularly unique group of at-risk children, and future research should explore what outcomes may be associated with this profile.

Relatedly, it is important to note that lower cortisol reactivity has been found to be associated with aggressive and antisocial behaviors (e.g., Alink, van IJzendoorn, Bakersmans-Kranenburg, Mesman, Juffer, & Koot, 2008; McBurnett, Lahey, Rathouz, & Loeber, 2000). Therefore, our non-findings between this potential mediator and the outcome variable of interest (child anxiety) may reflect too narrow a focus on the type of child outcome related to this neuroendocrinological profile. Future studies should not only explore other mechanisms by which the relationship between maternal trauma and child anxiety might exist, but should also explore whether cortisol hyporeactivity might be a mechanism explaining the relationship between maternal trauma exposure and other child outcomes, such as aggression.

Limitations

The current study is not without limitations. Trauma measures within the current study, while allowing us to look at the effects of maternal trauma exposure from several angles, could have enabled us to address our hypotheses with greater specificity. In the future, studies should explore measures of trauma that provide more detailed information about the time or age at which a trauma was experienced. This would give us a clearer understanding of the effects of timing of trauma exposure on parenting behaviors, cortisol reactivity, and child outcomes. Additionally, other ways of assessing the effects of different types of trauma and the cumulative experience or frequency of several types of trauma should be considered.

Notably, cortisol levels within the current study were only assessed for children and not for mothers participating in the study. Given the aforementioned literature finding a relationship

between maternal trauma exposure and maternal cortisol levels, it is important that future research in this area explore the role of maternal cortisol regulation as a potential mediator explaining the development of child anxiety. Including only child cortisol levels in these analyses does not allow exploration of the fetal programming hypothesis, namely that children's cortisol levels and risk for later anxiety result from cortisol conditions when in utero. Inclusion of maternal cortisol levels would shed more light on whether offspring of trauma-exposed mothers may actually have experienced a riskier prenatal environment during pregnancy.

Additionally, our use of cortisol reactivity as a marker of cortisol regulation may not have been ideal. Some research has suggested that given the considerable variation in the timing of cortisol responses to stress, obtaining only one post-stressor cortisol sample may not be sufficient (Raysay & Lewis, 2003). Other studies suggest that measuring diurnal cortisol curves from awakening to midnight and perhaps over a period of several days may be necessary to adequately assess cortisol patterns (see Golden, Wand, Malhotra, Kamel, & Horton, 2011, for a review). Future studies should explore other ways of measuring cortisol to determine whether these methods might yield different results. Additionally, given the general mixed nature of cortisol findings in the literature, this mediation model should be examined within larger samples and within independent samples to determine whether our findings can be replicated.

Observations of mother-child interactions were used within the current study to assess parenting behaviors. However, it may be necessary to take a more comprehensive approach to understanding the health of the relationship between mother and child. Factors like attachment or parental stress sensitivity may need to be considered in understanding the intergenerational transmission of trauma effects. Furthermore, it is important that future studies examine parenting behaviors displayed by mothers, not only in a lab context, but also at home. There are

many measures that exist that can be used to assess parenting behaviors. For example, the Alabama Parenting Questionnaire (APQ; Frick, 1991) provides information concerning several types of parenting behaviors (both positive and negative) that may be rated by both mothers and children. Children's perceptions of their mothers' parenting behaviors may be particularly important to assess in understanding the impact of parenting behaviors. Much of the impact resulting from maternal trauma exposure likely depends on children's subjective experiences of living with and being parented by mothers with trauma histories. Therefore, it is critical that their perspectives of their mothers' parenting behaviors be understood, particularly in this age range as they are developing schemas for understanding themselves, others, and their environments.

Notably, the current study design is not sensitive to genetic influences that may be involved in the aforementioned observed relationships. Some research, particularly in the animal literature, suggests that there might be genetic or epigenetic transmission of trauma effects across generations. For example, Kilpatrick and colleagues (2007) demonstrated that a polymorphism in the serotonin transporter gene may be related to post-traumatic symptomatology. This may place offspring at genetic risk for development of anxiety and other emotional and behavioral problems. Another study demonstrated that when mice encounter chronic and unpredictable stress as pups, they go on to develop depressive-like symptoms in adulthood, and their offspring later develop similar behavioral alterations (Franklin & colleagues, 2010). These researchers suggest that this intergenerational transmission of effects can be attributed to changes in the profile of DNA methylation of several candidate genes, which are then passed on to subsequent offspring. These studies and others like them suggest that the effects of parental exposure to trauma might be transmitted to offspring genetically or epigenetically. While the current study

design precludes our ability to investigate genetic or epigenetic influences, future studies should explore this potential mechanism of intergenerational transmission of trauma-related risks.

The current sample is characterized by an overrepresentation of psychopathology (and particularly depression) amongst mothers as a result of our sampling strategy. Consequently, these findings may not be generalizable to non-clinical populations. The decision was made not to control for mothers' current depressive symptoms or psychopathology in our analyses for two reasons. First, we believed that, conceptually, adding maternal depressive symptoms as a covariate might eliminate some of the variance in relationships that we were interested in examining. For example, if a mother's ineffective parenting behaviors are related to depressive symptoms resulting from her lifetime trauma exposure, controlling for these symptoms would eliminate some of the variance in maternal behaviors that we were attempting to explain. Second, it is often the case in the child literature that maternal depressive symptoms are included as covariates to ensure that subjective ratings provided by mothers do not reflect a general negative affective bias on constructs of interest. However, given that our measures include ratings from multiple reporters (maternal ratings, alternate caregiver ratings, and observational data collected by researchers in a lab setting) we did not feel that controlling for depressive symptoms to eliminate maternal report bias was necessary.

This study's sample may also not be representative of the general population given the high incomes seen across mothers enrolled. Despite this socioeconomic advantage, there remain some risk conferring properties of the sample given the prevalence of trauma exposure and psychopathology amongst mothers within the sample. Nevertheless, considering the uniqueness of this sample, future studies should explore these relationships within populations that are socioeconomically, ethnically, and psychologically more diverse than ours to ensure greater

generalizability.

It is important to note that there were many analyses conducted within the current study. As is often the case when examining relationships among several potential predictor variables, mediators, moderators, and outcomes, there is a possibility of attaining significant results solely by chance or Type I error. There does appear to be some consistency among findings, however, that would suggest that some confidence can be had in our results. Namely, there was a relationship between maternal trauma exposure and child anxiety as reported by both mothers and alternate caregivers. Moreover, mediation analyses were consistently found to support the roles of positive engagement and negative engagement as mediators, but not the roles of positive reinforcement or cortisol reactivity. Given the consistent trends seen throughout our results, we believe that these findings are meaningful and shed light on the broader possibility of intergenerational transmission of risk and of potential etiological pathways in the development of child anxiety. However, these findings should be replicated within independent samples to assure that our results are valid and reliable.

Conclusion

The current study addresses gaps in the literature that are very much in need of attention. Given the prevalence of trauma exposure in our country and the possible effects of that exposure on both mothers and offspring, it is essential that we understand whether maternal trauma exposure confers risks upon children, and if so, what mechanisms might be responsible for such transmission of risk. The current study suggests that there is a relationship between maternal exposure to trauma and child anxiety. This further suggests that risks for child anxiety may be missed when we limit ourselves to measuring only the elements found within a child's own lifetime and experiences, rather than also considering his/her mothers' life experiences.

Demonstration of this relationship in an at-risk sample of mothers and children shows that transgenerational effects of trauma need not occur only in cases where parents (and in much of the literature, fathers) experience traumatic events in the context of war or disaster, but in the context of mothers' exposure to other forms of trauma as well.

Lastly, our mediation hypotheses were only partially supported. Parenting was found to serve as a mediator in the relationship between maternal trauma and child anxiety; however, cortisol reactivity was not. Given that there is relatively limited knowledge of the mechanisms by which trauma might have intergenerational effects on offspring, much work remains to be done in understanding why and under what conditions we might see this relationship between maternal trauma experience and child outcomes. Only by doing this can effective preventative measures be taken in minimizing this potential risk to offspring of mothers with histories of trauma exposure.

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Tables

Table 1
Pattern Matrix Component of Parenting Behaviors

	Rotated Factor Loadings		
Items	1	2	3
Negative Talk	--	**0.897**	--
Direct Command	--	**0.972**	--
Indirect Command	**0.447**	0.306	--
Indirect Question	**0.829**	--	--
Direct Question	**0.839**	--	--
Neutral Talk	**0.729**	--	--
Reflective Statement	**0.584**	--	--
Unlabeled Praise	--	--	**0.590**
Behavioral Descriptions	--	--	**0.535**
Labeled Praise	--	--	**0.896**

Note. Factor loading > 0.40 are in boldface.

Table 2
Descriptions of coded parenting behaviors

Factor	Description
Positive Engagement	
Reflective Statement	Statement that reflects upon and has the same meaning as previous verbalizations made by the child. For example, in response to a child saying, "this game is fun," a reflective statement might be "you like playing this game!"
Neutral Talk	A statement made to the child that is non-descriptive and non-evaluative of the child or the child's actions (e.g., "I want to draw with you.")
Indirect Command	Parent suggests that their child perform a particular vocal or motor behavior; suggestion can be made in the form of a question or statement (e.g., "I'd like you to finish the picture," "Will you tell me what color this is?")
Information Question	Parent asks a question to which they expect an informative response (e.g., "What time is it?")
Descriptive Question	A descriptive statement expressed in question form with the expectation that the child give a brief (often yes/no) response (e.g., "That was fun, wasn't it?")
Negative Engagement	
Negative Talk	Parent expresses disapproval of the child or the child's activities and choices (e.g., "I think you are being careless.)
Direct Command	Declarative statement made by the parent that orders or directs the child to engage in certain behaviors (e.g., "hurry up," "put these away")
Positive Reinforcement	
Labeled Praise	Positive evaluation of the child or the child's behaviors and activities (e.g., "you are a good builder," "your picture is pretty")
Unlabeled Praise	Parent expresses non-specific approval of the child or child's behavior (e.g., "good job," "I like that")
Behavioral Description	Declarative statement made about the child's actions (e.g., "you are singing a song," "you and I are playing")

Table 3
Covariates For Each Proposed Mediator or Outcome Variable

Variable	Covariates
Mother-Reported PROMIS	None
Mother-Reported CBCL-Anxiety	None
Alternate Caregiver-Reported CBCL-Anxiety	Mother's total number of children
Cortisol Reactivity	Child having eaten before lab visit
	Child having a loose tooth at lab visit
Positive Engagement	Maternal age
	Maternal education level
	Child's minority status
Negative Engagement	Maternal age
	Mother's total number of children
Positive Reinforcement	None

Table 4
Trauma Variables: Sample Characteristics

Trauma Type/Variable	*N*	%
Childhood Trauma (sexual or physical abuse)	48	31.4
Lifetime Trauma	52	34.6
Cumulative Childhood Trauma (sexual and physical abuse)	11	7.3%
Childhood Sexual Abuse Only	34	22.6
Childhood Physical Abuse Only	24	16.1

Table 5
Descriptive Statistics for Variables of Interest

Variable	*M*	*SD*
Childhood Trauma (0, 1)	.314	.466
Lifetime Trauma (0, 1)	.350	.477
Cumulative Trauma (0, 1, 2)	.387	.621
Childhood Sexual Abuse Only (0, 1)	.226	.420
Childhood Physical Abuse Only (0, 1)	.161	.368
Cortisol Reactivity	-.056	.291
Positive Engagement	49.804	9.813
Negative Engagement	13.454	5.245
Positive Reinforcement	4.492	2.524
PROMIS-Anxiety (Mother Report)	9.236	9.538
CBCL-Anxiety (Mother Report)	1.919	2.474
CBCL-Anxiety (Alternate Caregiver Report)	1.961	2.541

Note. In the cases of Childhood Trauma, Lifetime Trauma, Childhood Sexual Abuse Only, and Childhood Physical Abuse Only, variables were coded for presence or absence of the assessed type of trauma. In the case of Cumulative Trauma, possible values were 0 (no childhood trauma), 1 (either childhood physical or sexual trauma), or 2 (both childhood physical and sexual trauma).

Table 6
Zero-Order Correlations Between Variables of Interest

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Childhood Trauma	---	.370**	.924**	.800**	.647**	-.096	-.016	.135	-.116	-.110	.159	-.117
2. Lifetime Trauma		---	.408**	.343**	.292**	.079	.174*	.306**	-.008	-.274**	.270**	-.016
3. Cumulative Childhood Trauma			---	.817**	.754**	.045	.079	.168	-.090	-.227**	.278**	-.166
4. Childhood Sexual Abuse Only				---	.239**	-.004	.032	.071	-.166	-.199*	.198*	-.026
5. Childhood Physical Abuse Only					---	.081	.096	.213	.037	-.163	.252**	-.256**
6. PROMIS-Anx (Mother Report)						---	.657**	.554**	.033	-.169	.222*	.004
7. CBCL-Anx (Mother Report)							---	.514**	-.088	-.177*	.197*	.045
8. CBCL-Anx (Alt.Cgv. Report)								---	.160	-.125	.129	.039
9. Cortisol Reactivity									---	-.055	.035	-.048
10. Positive Engagement										---	-.802**	-.145
11. Negative Engagement											---	-.342**
12. Positive Reinforcement												---

*$p<.05$. **$p<.01$.

Table 7

Results for Maternal Trauma Exposure Predicting Child Anxiety Levels

Variable	PROMIS-Anxiety (Mother Report)			CBCL-Anxiety (Mother Report)			CBCL-Anxiety (Alternate Caregiver Report)		
	B	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*
Childhood Trauma	-1.966	1.778	-.096	-.085	.459	-.016	.778	.630	.139
Lifetime Trauma	1.558	1.740	.079	.892*	.441*	.174*	1.488*	.608*	.269*
Cumulative Trauma	.684	1.33	.045	.313	.343	.079	.627	.439	.160
Childhood Sexual Abuse Only	-.089	1.964	-.004	.190	.508	.032	.659	.708	.106
Childhood Physical Abuse Only	2.119	2.269	.081	.640	.576	.096	1.123	.765	.167

**p<.05.*

Table 8
Cortisol Reactivity Indirect Effects Analyses

Indirect Effects Pathway	***Effect***	***SE***	***Lower Limit CI***	***Upper Limit CI***
Childhood Trauma→Cortisol Reactivity				
→Mother-reported PROMIS	-.035	.362	-1.389	.4702
→Mother reported CBCL	.042	.075	-.061	.273
→AltCgv-reported CBCL	-.070	.146	-.773	.048
Lifetime Trauma→ Cortisol Reactivity				
→Mother-reported PROMIS	-.038	.186	-.904	.168
→Mother-reported CBCL	.009	.043	-.050	.139
→AltCgv-reported CBCL	-.045	.078	-.355	.034
Cumulative Trauma→ Cortisol Reactivity				
→Mother-reported PROMIS	-.056	.249	-1.050	.199
→Mother-reported CBCL	.019	.049	-.040	.196
→AltCgv-reported CBCL	-.038	.120	-.541	.048
Childhood Sexual Trauma→Cortisol Reactivity				
→Mother-reported PROMIS	-.109	.449	-1.575	.511
→Mother-reported CBCL	.057	.106	-.117	.315
→AltCgv-reported CBCL	-.084	.177	-.926	.047
Childhood Physical Trauma→ Cortisol Reactivity				
→Mother-reported PROMIS	.002	.317	-.562	.846
→Mother-reported CBCL	-.010	.083	-.226	.125
→AltCgv-reported CBCL	-.014	.140	-.386	.236

Table 9
Positive Engagement Indirect Effects Analyses

Indirect Effects Pathway	***Effect***	***SE***	***Lower Limit CI***	***Upper Limit CI***
Childhood Trauma→Positive Engagement				
→Mother-reported PROMIS	.916	.804	-.190	3.093
→Mother reported CBCL	.156	.163	-.043	.683
→AltCgv-reported CBCL	.081	.321	-.443	.955
Lifetime Trauma→Positive Engagement				
→Mother-reported PROMIS	**1.356**	**.845**	**.198**	**3.932**
→Mother-reported CBCL	.207	.170	-.049	.622
→AltCgv-reported CBCL	-.065	.371	-.777	.723
Cumulative Trauma→Positive Engagement				
→Mother-reported PROMIS	**1.005**	**.600**	**.135**	**2.510**
→Mother-reported CBCL	**.189**	**.135**	**.001**	**.570**
→AltCgv-reported CBCL	.066	.243	-.341	.783
Childhood Sexual Trauma→Positive Engagement				
→Mother-reported PROMIS	**1.519**	**1.012**	**.046**	**4.2770**
→Mother-reported CBCL	**.298**	**.212**	**.008**	**.887**
→AltCgv-reported CBCL	.161	.379	-.473	1.168
Childhood Physical Trauma→Positive Engagement				
→Mother-reported PROMIS	1.092	1.008	-.208	3.997
→Mother-reported CBCL	.185	.205	-.050	.900
→AltCgv-reported CBCL	.052	.292	-.296	.996

Table 10
Negative Engagement Indirect Effects Analyses

Indirect Effects Pathway	***Effect***	***SE***	***Lower Limit CI***	***Upper Limit CI***
Childhood Trauma→Negative Engagement				
→Mother-reported PROMIS	**.991**	**.724**	**.026**	**3.079**
→Mother reported CBCL	.202	.189	-.011	.745.
→AltCgv-reported CBCL	.166	.342	-.466	.902
Lifetime Trauma→Negative Engagement				
→Mother-reported PROMIS	**1.285**	**.793**	**.191**	**3.698**
→Mother-reported CBCL	.253	.194	-.002	.779
→AltCgv-reported CBCL	.007	.256	-.469	.554
Cumulative Trauma→Negative Engagement				
→Mother-reported PROMIS	**1.131**	**.664**	**.149**	**2.950**
→Mother-reported CBCL	.242	.190	-.006	.774
→AltCgv-reported CBCL	.095	.270	-.335	.736
Childhood Sexual Trauma→Negative Engagement				
→Mother-reported PROMIS	**1.229**	**.841**	**.111**	**3.745**
→Mother-reported CBCL	.281	.239	-.003	.937
→AltCgv-reported CBCL	.272	.359	-.266	1.156
Childhood Physical Trauma→Negative Engagement				
→Mother-reported PROMIS	**1.670**	**1.070**	**.025**	**4.335**
→Mother-reported CBCL	.344	.281	-.017	1.115
→AltCgv-reported CBCL	.131	.371	-.442	1.021

Table 11
Positive Reinforcement Indirect Effects Analyses

Indirect Effects Pathway	***Effect***	***SE***	***Lower Limit CI***	***Upper Limit CI***
Childhood Trauma→Positive Reinforcement				
→Mother-reported PROMIS	.023	.282	-.450	.824
→Mother reported CBCL	-.028	.078	-.275	.068
→AltCgv-reported CBCL	-.203	.192	-.924	.022
Lifetime Trauma→ Positive Reinforcement				
→Mother-reported PROMIS	.011	.180	-.258	.512
→Mother-reported CBCL	-.001	.045	-.123	.077
→AltCgv-reported CBCL	.002	.097	-.162	.309
Cumulative Trauma→ Positive Reinforcement				
→Mother-reported PROMIS	-.029	.257	-.642	.492
→Mother-reported CBCL	-.039	.070	-.225	.055
→AltCgv-reported CBCL	-.138	.125	-.503	.030
Childhood Sexual Trauma→ Positive Reinforcement				
→Mother-reported PROMIS	-.002	.217	-.460	.487
→Mother-reported CBCL	-.007	.058	-.196	.068
→AltCgv-reported CBCL	-.086	.140	-.653	.063
Childhood Physical Trauma→ Positive Reinforcement				
→Mother-reported PROMIS	0.226	.634	-1.526	1.077
→Mother-reported CBCL	-.146	.172	-.657	.111
→AltCgv-reported CBCL	-.347	.324	-1.380	.017