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Maternal Emotion Dysregulation, Parenting Stress, and Child Physiological Anxiety during Dark-Enhanced Startle

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Abstract

Maternal emotion dysregulation (ED) plays a crucial role in the development of psychopathology in children. The current study aimed to investigate parenting stress as a mediator of the relationship between maternal emotion dysregulation and child startle potentiation, with child sex as a moderator. Mothers were interviewed to obtain self-report of maternal ED and parenting stress and child's dark-enhanced startle (DES) response was measured using electromyographic recordings of the eye-blink muscle during the delivery of acoustic probes. We found that maternal ED was positively correlated with both her parenting stress and her child's DES. A bootstrap analysis yielded a full mediation of the association between ED and child DES via parenting stress. Child sex was not a significant moderator of these relationships. These results suggest that maternal ED has important consequences for the intergenerational transmission of risk and also highlight the interaction of behavioral and biological mechanisms of risk.

Keywords

emotion dysregulation; parenting stress; child anxiety; startle potentiation

Emotion regulation is a multi-dimensional process by which individuals control not only which emotions they have, but also the timing, subjective experience, and expression of those emotions (Gross, 1998b). The role of emotion regulation in maintaining healthy psychological functioning has been well established (Gross & Muñoz, 1995). Emotion dysregulation (ED) reflects deficits in several key areas including awareness and acceptance of emotions, maintaining goal-directed behavior in the presence of intense negative emotions, and use of effective emotion regulation strategies (Gratz & Roemer, 2004). ED is a trans-diagnostic phenomenon that is both a correlate and an antecedent of impaired

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functioning and psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011).

Given the importance of emotion regulation, and consequent adverse effects of dysregulation on adaptive functioning, research in child development has attempted to explore the consequences of emotion dysregulation on parenting and including its role in the intergenerational transmission of risk for maladjustment or anxious behavior in children. Empirical research supports the association between parental emotion dysregulation and children's negative adjustment, and this relationship is likely mediated by problematic parenting behaviors (Buckholdt, Parra, & Jobe-Shields, 2014; Crandall, Ghazarian, Day, & Riley, 2015). Indeed, there is substantial evidence indicating that the interaction of parenting behaviors, family emotional climate, as well as child and parent characteristics is instrumental in shaping children's emotional development (Cunningham, Kliever, & Garner, 2009; Morris, Silk, Steinberg, Myers, & Robinson, 2007; Ramsden & Hubbard, 2002; Shipman & Zeman, 2001). Studies that use parenting behaviors as mediators of the relationship between parental emotion dysregulation and child outcomes often do not account for contextual variables that may dictate if and how particular behaviors are reported (i.e., differences in expectations of appropriate and non-appropriate parenting behaviors) (Julian, McKenry, & McKelvey, 1994; Morris et al., 2007; Pinderhughes, Dodge, Bates, Pettit, & Zelli, 2000). Another construct that could serve as a potential mediator between parental ED and child outcomes is parenting stress, since it is highly correlated with parenting behaviors and valid across differing contexts of race, culture, and socioeconomic status (Anderson, 2008; Cardoso, Padilla, & Sampson, 2010). Parenting stress is defined as "the aversive psychological reactions to the demands of parenting ... experienced as negative feelings towards the self and towards the child or children" (Deater-Deckard, 1998). Although a certain level of parenting stress is common, research suggests high levels of parenting stress are related to negative psychological outcomes in children (Deater-Deckard, 1996; Deater-Deckard, Ivy, & Petrill, 2006). Deficits in emotion regulation contribute to maladaptive patterns of stress response by enabling over-reliance on and ineffective use of *emotion-focused coping* (Lazarus, 1993). Indeed, one study found that parents who experience emotion dysregulation may have greater difficulty accessing adaptive emotion regulation strategies and may be more prone to negative cognitive appraisals (Barnett, Hall, & Bramlett, 1990). The use of certain emotion-focused coping strategies such as avoidance, taking responsibility, and self-control, has also been associated with increased parenting stress (Miller, Gordon, Daniele, & Diller, 1992). Thus, emotion dysregulation likely augments parenting stress by impairing parents' ability to effectively cope with negative emotions that arise from the demands of parenthood.

A number of studies have suggested a physiological basis for emotion regulation (Porges, 1984; Thayer & Lane, 2000). Previous research suggests the conscious use of emotion regulation strategies can alter physiological arousal to pleasant and unpleasant stimuli (Driscoll, Tranel, & Anderson, 2009; Gross, 1998a; Gross & Levenson, 1993; Jackson, Malmstadt, Larson, & Davidson, 2000; Lang, 1995; Lang, Bradley, & Cuthbert, 1990). Furthermore, a study from our lab found increased startle reactivity in adult women with a history of childhood sexual abuse (Jovanovic et al., 2009), which has been associated with numerous impairments in emotional functioning (Cloitre, Miranda, Stovall-McClough, &

Han, 2005; Kolk, 2005). Risk factors for ED, such as childhood maltreatment, have also been found to produce altered neurobiological functioning in children (Cicchetti, 2002).

Fewer studies have looked at physiological outcomes in children with regards to parental risk factors, however. Previous work in our laboratory found heightened acoustic startle reactivity and increased heart-rate variability in children of mothers with a history of childhood abuse (Jovanovic et al., 2011). Grillon, Dierker, and Merikangas (1997) also found that children of parents with anxiety disorders and/or alcoholism had higher startle magnitude than did children of control parents. This intergenerational effect was further supported in another study which found increased startle magnitude in children as well as grandchildren of probands with major depressive disorder compared to those of healthy control probands (Grillon et al., 2005).

The acoustic startle reflex, which consists of the involuntary contraction of skeletal muscles in response to unexpected and intense acoustic stimuli, has been used as a biomarker of anxiety using animal and human models (Davis, 2001; Grillon, Ameli, Woods, Merikangas, & Davis, 1991; Jovanovic et al., 2005; Landis & Hunt, 1939). The reflex occurs via a three-neuron circuit that is structurally and functionally similar in rodents and humans (Davis, 1992). Under fearful conditions, limbic structures such as the nuclei in the extended amygdala can modulate the intensity of the acoustic startle response (Walker & Davis, 1996). For instance, darkness is naturally anxiogenic for humans and has been shown to potentiate startle reactivity to acoustic probes, so that the magnitude of the acoustic startle response in the dark is greater than the original response observed in the light (Grillon, Pellowski, Merikangas, & Davis, 1997). This potentiation serves as non-invasive, objective physiological marker of the state of anxiety induced by the darkness (Grillon, Pellowski, et al., 1997; Jovanovic et al., 2011; Kamkwalala et al., 2012).

There is evidence that parental risk factors may differentially affect children based on gender (Birnbaum & Croll, 1984; Chaplin, Cole, & Zahn-Waxler, 2005). In an intergenerational study of psychophysiological transmission of depression, female grandchildren of probands with major depression exhibited high levels of startle magnitude throughout an acoustic startle task, whereas male grandchildren did not (Grillon et al., 2005). In our laboratory, we found that maternal childhood sexual abuse predicted lower maternal warmth towards daughters, but not sons (Cross et al., in press). These findings suggest that generally girls may be more adversely affected than boys by parental experiences and behaviors. Moreover, a longitudinal study found that parenting stress and discord were associated with internalizing symptoms in girls by mid-adolescence, but not in boys, whereas no sex differences were detected in early adolescence (Crawford, Cohen, Midlarsky, & Brook, 2001). This suggests that girls may be more adversely affected by parenting stress in particular when compared to boys. Furthermore, because these findings did not emerge before adolescence, it is possible that psychophysiological changes due to pubertal development during this period might contribute to sex-specific patterns of risk.

The present study is the first to examine the relationship between maternal ED on children's startle potentiation during a dark-enhanced startle task. Based on the previous literature demonstrating the impact of ED on parenting stress, and subsequent consequences on child

outcomes, we hypothesized that parenting stress would mediate the relationship between maternal ED and child startle potentiation. Further, we hypothesized that child sex would moderate both (a) the effect of parenting stress on child startle potentiation and (b) the indirect effect of maternal ED on child startle potentiation through parenting stress, such that startle in girls would be more strongly affected than that in boys.

Method

Participants

The sample consisted of 84 African American mother-child dyads (see Table 1 for descriptive details). Participants were recruited from the primary care, obstetrics and gynecology, diabetes, and pharmacy waiting rooms of a public, not-for profit hospital treating primarily underserved, low socioeconomic status minorities in urban Atlanta, GA. Exclusion criteria for both mothers and children included intellectual developmental disorder, active psychotic disorder, and autism spectrum disorder. Participants were initially enrolled in a larger NIMH-funded study of risk factors for civilian PTSD (Gillespie et al., 2009), and a subset of participants who were legal guardians of a child 8–12 years old were invited to further participate in an intergenerational study of trauma and parenting. In order to create comparison groups, participants were not required to have experienced trauma to be eligible for the study. The age range of the children was specified to include the period when pubertal development typically begins in children. Informed consent for self and child participation was obtained from mothers and assent was obtained from children. All study procedures were approved by the institutional review boards of Emory University School of Medicine and Grady Memorial Hospital in Atlanta, Georgia.

Psychological Assessment

Mothers completed a series of self-report measures, four of which were used in the present study. The Parenting Stress Index – Short Form (PSI-SF) is a well-validated 36 item self-report questionnaire examining the magnitude of stress in the parent-child system using three subscales: Parenting Distress, Dysfunctional Parent-Child Interaction, and Difficult Child (Abidin, 1995). The PSI-SF has been validated against other self-report measures and rater-observed measures of parenting stress (Haskett, Ahern, Ward, & Allaire, 2006). The PSI-SF has also been psychometrically validated in a low-income, minority population (Reitman, Currier, & Stickle, 2002). The Difficulties in Emotion Regulation Scale (Driscoll et al.) is a 36-item self-report questionnaire of deficiencies in emotion regulation with six subscales: Non-acceptance, Goals, Impulse, Awareness, Strategies, and Clarity (Gratz & Roemer, 2004). The DERS has high internal consistency and construct validity with the Negative Mood Regulation scale (Gratz & Roemer, 2004). The Childhood Trauma Questionnaire (CTQ) is a self-reported history of childhood physical, sexual, and emotional abuse and neglect. Categorical values (high and low abuse) for the CTQ were obtained based on data presented on previous research by Jovanovic and colleagues (Jovanovic et al., 2011). Finally, child report of trauma exposure was assessed using the Violence Exposure Scale for Children-Revised (VEX-R) (Fox & Leavitt, 1995). The VEX-R is a 22-item cartoon-based self-report interview of children's lifetime exposure to violence that has been validated in an African American urban population (Cross et al., 2017).

Startle Data Acquisition

The startle data were acquired using Biopac MP150 for Windows (Biopac Systems, Inc., Aero Camino, CA). The acquired data were filtered, rectified, and smoothed using MindWare software (MindWare Technologies, Ltd., Gahanna, OH), and exported for statistical analyses. Startle data were collected via recording of the eye-blink muscle contraction using the electromyography (EMG) module of the Biopac system. The startle potentiation was recorded with two 5-mm Ag/AgCl electrodes; one was placed on the orbicularis oculi muscle 1cm below the pupil and the other 1cm lateral to the first electrode. A common ground electrode was placed on the neck below the right earlobe. Impedance levels were less than 6 kilo-ohms for each participant. The startle probe was a 108-dB(A) SPL, 40ms burst of broadband noise delivered through headphones (Maico, TDH-39-P). The maximum amplitude of the eyeblink muscle contraction 20–200 ms after presentation of the startle probe was used as a measure of startle magnitude.

Experimental Design

The experimental paradigm began with a 2-minute acclimation period during which no startle probes were delivered, followed by a startle habituation segment, and a dark-enhanced startle segment that occurred without interruption. The total session duration was 8 minutes long (2 minutes acclimation, 2 minutes habituation, and 4 minutes dark-enhanced segment). The acclimation period involved the participant sitting quietly in the startle booth in the light for two minutes, without administering any startle probes. The startle habituation segment consisted of eight startle probes delivered at randomized intervals. Immediately following habituation, participants underwent the dark-enhanced segment consisting of two blocks each with eight startle probes. In each block, four startle probes were delivered in the dark phase and four were delivered in the light phase. The light and dark phases alternated, with each phase lasting one minute. The order of light and dark was counterbalanced across subjects. Across the entire experimental session, inter-trial intervals ranged from 9 to 22 seconds.

Statistical Analyses

The dependent variable in the analyses was child dark-enhanced startle measured as percent startle potentiation in the dark compared to the light. This was calculated as the average startle magnitude in the dark phase minus the average startle magnitude in the light phase divided by the startle magnitude in the light phase and multiplied by 100. A univariate ANOVA was conducted to compare mean percent potentiation during DES for boys vs. girls to determine whether there was a main effect of sex on startle potentiation. Initial analyses included bivariate correlations examining associations between continuous total scores for maternal ED and Parenting stress (PSI) and average percent child startle potentiation during DES. Bonferroni-corrected alpha value = 0.017 was used to correct for multiple comparisons.

PROCESS (Hayes, 2013a; Preacher & Hayes, 2008) for SPSS was used to formally test for the mediation of parenting stress on ED and child DES. This allowed for the generation of 5000 bootstrap samples with a 95% bias-corrected confidence interval of the indirect effect $a \times b$. In our simple mediation analysis (model 4 in PROCESS, Hayes, 2013b), path a

represented the path between maternal ED and parenting stress, and path b represented the impact of the mediator, parenting stress, on child startle potentiation. Path c represented the total effect of maternal ED on child startle potentiation DES, and path c' represented the direct effect of maternal ED on child startle potentiation when controlling for parenting stress. Additionally, in order to control for the potential effect of maternal childhood trauma on ED, categorical values (high and low abuse) for maternal childhood trauma were entered as a covariate in analyses based on data presented by Jovanovic et al. (2011).

Next, we planned a moderated mediation analysis (model 14 in PROCESS, Hayes, 2013b), again controlling for maternal childhood trauma, to examine the moderating effect of child sex on path b based on previous research suggesting that parenting stress may differentially affect children based on gender (Crawford et al., 2001; Cross et al., in press). The index of the moderated mediation is calculated as $(a_1b_2)\delta$, where δ is the difference between two values of a dichotomous moderator, in our case 0 = male and 1 = female, thus providing a test of the equality of the two conditional indirect effects (Hayes, 2014). Additionally, in order to test the effect of child sex on the relationship between parenting stress and child startle potentiation outside of the mediation context, we ran a simple moderation analysis (model 1 in PROCESS, Hayes, 2013b).

Results

Startle was potentiated in the dark relative to the light, $F(1,83)=18.04$, $p<0.001$, however, there was no sex difference in startle potentiation, $p>.10$ (see Figure 1). Age was not correlated with startle potentiation in the dark $r=-.075$, $p>.10$. Further, trauma exposure in children was not correlated with their startle potentiation $r=-.08$, $p>.10$. However, with respect to maternal variables, correlation analyses showed maternal ED was significantly positively associated with parenting stress, $r=.424$, $p<.001$ (Figure 2A). Child startle potentiation was significantly positively associated with both parenting stress, $r=.317$, $p=.003$, and maternal ED, $r=.267$, $p=.01$ (see Figures 2B and 2C). Examination of the of the separate sub-scales of the PSI-SF in a follow-up analysis showed that the total parental stress score correlation with child startle was driven primarily by Parental Distress, $r=.372$, $p<0.001$, whereas the Difficult Child and Parent-Child Dysfunctional Interactions sub-scales were not significantly associated with startle ($r=.18$ and $r=.16$, respectively, both $p>.10$).

In the simple mediation analysis of parenting stress on the relationship between maternal ED and child startle potentiation while controlling for maternal childhood trauma, we found a positive and significant indirect effect ($a \times b$), $\beta = .247$, $SE = .127$, 95% CI [.066, .595], a positive and significant total effect (c), $\beta = .840$, $SE = .391$, 95% CI [.062, 1.62], and a non-significant direct effect (c'), $\beta = .593$, $SE = .429$, 95% CI [-.200, 1.39], demonstrating a full mediation (Zhao, Lynch, & Chen, 2010) (see Fig. 3). Because the cross-sectional design of our study cannot directly address the temporal relationship between maternal ED and parenting stress, we ran a follow-up mediation analysis using parenting stress as the independent variable and maternal ED as the mediator to predict child startle potentiation. We found that this model did not yield a significant indirect effect of parenting stress on child startle through maternal ED, supporting our original model of maternal ED predicting stress.

We followed this analysis with a moderated mediation analysis with child sex (see Fig. 4). In path b_2 , we did not find a significant interaction of parenting stress and child sex in predicting child startle potentiation. The index of moderated mediation was $\beta = .235$, $SE = .213$, 95% CI $[-.093, .771]$. To confirm whether an effect of child sex existed outside of the mediation, we ran an follow-up simple moderation analysis of the effect of child sex on the relationship between parenting stress and child startle potentiation and found that the interaction of child sex and parenting stress was also not significant, $\beta = 1.43$, $SE = 1.13$, 95% CI $[-.833, 3.69]$.

Discussion

In the present study, we examined the association between of maternal ED and children's startle potentiation, a physiological marker of anxiety. We found that maternal parenting stress fully mediated the relationship between maternal ED and child startle potentiation during a dark-enhanced startle task. This effect was significant even after controlling for maternal childhood trauma. Contrary to previous research however, we found that child sex was not a significant moderator of the relationship between parenting stress and child startle potentiation.

Previous research suggests that potentiation of the acoustic startle response during DES tasks is a physiological marker of the emotional state of anxiety (Davis, Walker, & Lee, 1997; Kamkwalala et al., 2012). The finding that higher maternal ED predicted greater dark-enhanced startle potentiation in children suggests that maternal ED confers risk for children in the form of a general heightened state of anxiety under aversive conditions (e.g., darkness). Research suggests that high state anxiety interferes with the executive functions required to engage in cognitive components of emotion regulation (Pedersen & Larson, 2016; Robinson, Vytal, Cornwell, & Grillon, 2013). There is also evidence that clinical, state, and trait anxiety share similar patterns of cognitive tendencies related to attentional bias and inhibitory control of anxious responding (Robinson et al., 2013). Thus, high state anxiety may indicate risk for maladaptive anxiety.

Intergenerational studies of startle have found increased risk in children of clinically depressed and anxious individuals. Grillon, Dierker, and Merikangas (1998) found that the magnitude of the acoustic startle reflex has been found to be greater in children of clinically anxious individuals compared to children of healthy controls. In a different study, the same researchers also found greater startle responses in children and grandchildren of clinically depressed probands (Grillon et al., 2005). Rodent and human models of DES have pointed to anxiety/fear as the primary emotions behind the startle response, rather than sadness (Davis et al., 1997; Kamkwalala et al., 2012), and longitudinal research has also pointed to anxiety symptoms as a risk factor for the development of depression (Batterham, Christensen, & Calear, 2013). Grillon et al. (2005) proposed that contextual anxiety as indexed by greater startle response to aversive conditions may indicate a broad risk factor for depression and anxiety disorders; this was supported in a retrospective analysis showing that the children and grandchildren of depressed probands were at greater risk for anxiety disorders than the descendants of healthy controls. Thus, together with previous findings from intergenerational studies of startle reactivity, the finding that maternal ED was associated

with startle response in children suggests the possibility that there may be an intergenerational transmission of risk for maladaptive anxiety. Furthermore, although ED and anxiety are distinct constructs, the former has been implicated in etiological models of maladaptive anxiety (Suveg, Morelen, Brewer, & Thomassin, 2010). It is therefore possible that in addition to risk for maladaptive anxiety specifically, maternal ED may also confer risk for ED in children more broadly (Zeman, Cassano, Perry-Parrish, & Stegall, 2006). Because we did not explicitly measure ED in the children, however, future studies should include these measures in order to more fully elaborate on the relationship between these constructs.

The finding that parenting stress mediates the relationship between maternal ED and her child's startle potentiation illuminates a possible mechanism through which maternal emotion dysregulation might indirectly confer risk for children. An important component of the parenting stress construct includes the use of coping mechanisms to regulate emotional responses to stressors (Deater-Deckard, 1998). There is evidence for several ways in which ED augments the stress response by creating over-reliance on ineffective emotion-focused coping skills (Barnett et al., 1990; Lazarus, 1993; Miller et al., 1992). As such, ED likely compounds parenting stress by impairing the ability to cope with stressors brought on by parental responsibilities. Increased parenting stress in turn contributes to the likelihood of problematic parenting behaviors (Deater-Deckard, 1998) and likely reduces parents' capacity to adaptively participate in their children's emotional development, including teaching children how to manage anxious feelings (Deater-Deckard, 1998; Morris et al., 2007; Suveg et al., 2008). Furthermore, the significance of these associations even after controlling for maternal childhood trauma suggests that the contributions of ED to parenting stress are unique beyond the effects of trauma.

The finding that child gender did not moderate the relationship between parenting stress and child startle is surprising given previous research (Cross et al., in press; Grillon et al., 2005). One reason may be that our analyses were statistically under-powered due to a relatively small sample size. Another reason may be that it is too early in the developmental stage to detect significant sex differences (Crawford et al., 2001). Although the age range of the children in the current study includes the period during which psychophysiological changes typically begin to occur due to pubertal development, sex differences may not be apparent until mid- to late adolescence. Future studies should aim to expand these findings in an adolescent population to ascertain whether sex differences become more apparent further along in development.

A considerable strength of this study is the use of physiological markers to assess anxiety in children, where most have focused on behavioral indices of anxiety using self-report measures. Previous literature supports startle potentiation as an important biomarker of anxiety (Davis, 2001; Grillon, 2002; Grillon et al., 1991), and data from our lab show correlations between DES and self-reported measures of behavioral anxiety (Jovanovic et al., 2011). Using startle reactivity to complement self-report measures of behavioral anxiety could have useful clinical implications; however, additional longitudinal research is needed to determine whether startle reactivity, specifically darkness-potentiated startle, could reliably predict non-clinical anxiety as well as anxious psychopathology. Furthermore, our

findings suggest that the intergenerational transmission of risk can be observed at both the biological and behavioral level (Cicchetti, 2016; Meaney, 2001; Stein, Schork, & Gelernter, 2008).

Limitations of this study include the relatively small sample size, use of self-report measures of both maternal ED and parenting stress, and the lack of self-report measures of child anxiety. With regards to the self-reporting of maternal variables, it is possible that individual biases may have resulted in either under- or over-reporting on either measure. However, since parenting stress and ED are inherently subjective experiences and both the DERS and PSI have shown good convergent validity with other measures of ED and parenting stress, respectively (Gratz & Roemer, 2004; Haskett et al., 2006), these are likely valid measures of our constructs.

Another limitation is the cross-sectional design of the study, which does not allow us to draw causal conclusions about the relationship between maternal and child variables. Additionally, in tests of full mediation, a longitudinal design is preferred in order to establish temporal relationships between the independent and mediating variable. Since maternal ED and temporal precedence of ED to parenting stress cannot be established using the current study design, we ran an additional mediation analysis examining parenting stress as the independent variable and maternal ED as the mediator and found that the indirect effect was not significant, providing some support for our proposed design in which that parenting stress is the mediator of the relationship between maternal ED and child startle potentiation. Furthermore, ED may be conceptualized as the independent variable from a clinical perspective since quasi-experimental manipulation of ED is possible through clinical interventions aimed at teaching adaptive emotion regulation skills, whereas manipulation of individual experiences of parenting stress is less feasible.

Developmental psychopathology research also points to transactional exchanges between children and their environment, such that children's biologically based characteristics and learned behaviors can elicit interactions with their environment (Cicchetti, 1993; Meaney, 2010; Stein et al., 2008). It is therefore possible that children's propensity for anxiety (i.e., behavioral inhibition, difficult temperament, etc.) contributes to parenting stress and related parenting behaviors and vice versa (Degnan & Fox, 2007; Hirshfeld et al., 1992; Pérez-Edgar & Fox, 2005). Additionally, the development of anxious behavior in children could increase existing levels of parenting stress, creating a feedback loop. Future research should consider a longitudinal design in order to establish a clearer temporal relationship between parenting stress and child anxiety. Lastly, child development is, in general, a robust process, where the presence of some risk factors do not necessarily lead to negative outcomes (Cicchetti & Rogosch, 1996; Osofsky & Thompson, 2000), especially when protective factors like warmth are also present (Deater-Deckard et al., 2006; Patterson, Cohn, & Kao, 1989). Thus, the cumulative interactions of co-occurring risk and protective factors have significant implications for children's ultimate outcome and deserve further empirical examination.

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Key Points

- The current study found that parenting stress mediated the relationship between maternal emotion dysregulation (ED) and potentiation of the acoustic startle response in their children, even after controlling for maternal childhood trauma.
- The current study found that child sex did not moderate the relationship between parenting stress and child startle outcome.
- This is the first study to use psychophysiological markers to examine mechanisms of intergenerational transmission of risk for emotional maladjustment.

Dark Enhanced Startle in Boys and Girls

Figure 1.

A one-way ANOVA showed startle was potentiated in the dark relative to the light, $F(1,83)=18.04$, $p<0.001$, however, there was no sex difference in startle potentiation, $p>.10$.

Figure 2.

A. Scatter plot of the correlation between Maternal ED and Parenting Stress, $r = .424$, $p < .001$. $R^2 = .180$

B. Scatter plot of the correlation between Parenting Stress and Child Startle Potentiation during dark-enhanced startle, $r = .317$, $p = .01$. $R^2 = .100$

C. Scatter plot of the correlation between Maternal ED and Child Startle Potentiation during dark-enhanced startle, $r = .267$, $p < .001$. $R^2 = .071$

Figure 3.

Mediation of the relationship between maternal emotion dysregulation and child startle potentiation through parenting stress. Unstandardized coefficients are reported for each path. Path *c'* is the direct effect of maternal emotion dysregulation on child startle potentiation when accounting for parenting stress.

Figure 4. Proposed moderated mediation of the effect of child sex on path *b*. The interaction of parenting stress and child sex was not significant, $p=.21$.

Table 1

Demographic table for data sample ($n = 84$ mother-child pairs). All mothers were self-identified African-American females. Children were 8–12 years old.

Maternal Age, M (<i>SD</i>)	34.5 (<i>8.03</i>)
Currently employed, N (%)	31 (36.9)
Monthly Household Income, N (%)	
USD\$0–999	37 (44.6)
\$1000–1999	29 (34.9)
\$2000	17 (20.5)
Education, N (%)	
< 12 th Grade	20 (23.8)
High school diploma/GED	28 (33.3)
Some college/technical school	18 (21.4)
College/technical school degree	18 (21.4)
Child female sex	37 (44%)
Child age, M (<i>SD</i>)	9.7 (<i>1.50</i>)
Male	9.83 (<i>1.61</i>)
Female	9.46 (<i>1.50</i>)