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Experiential Avoidance and the Relationship between Child Maltreatment and PTSD Symptoms: Preliminary Evidence

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Abstract

Objective—Not every adolescent exposed to child maltreatment develops symptoms of post-traumatic stress disorder (PTSD), emphasizing the need to identify variables that explain how some maltreated children come to develop these symptoms. This study tested whether a set of variables, respiratory sinus arrhythmia (RSA) and cortisol reactivity as well as experiential avoidance, explained the relationship between child maltreatment and PTSD symptoms.

Methods—Adolescent females ($N = 110$; $n = 51$ maltreated) 14–19 years of age completed interviews, questionnaires, and a stressor paradigm. A multiple mediator model was used to assess the effect for the set of variables while identifying specific indirect effects for each variable.

Results—Results indicated that the set of variables mediated the relationship between child maltreatment and PTSD symptoms. However, only experiential avoidance contributed significantly to this effect when simultaneously estimating all other variables. The indirect effect for experiential avoidance was also significantly stronger than the effects of RSA and cortisol reactivity.

Conclusions—Data support the examination of experiential avoidance in understanding how adolescents who have been maltreated develop PTSD symptoms with implications for prevention and intervention.

Keywords

Child maltreatment; experiential avoidance; cortisol; respiratory sinus arrhythmia; PTSD

Child maltreatment, including physical abuse, sexual abuse, and neglect, is one of the most consistent predictors of posttraumatic stress disorder (PTSD) symptoms. Literature reviews (Brown, 2003; Kendall-Tackett, Williams, & Finkelhor, 1993), meta-analyses (Chen, et al., 2010; Paolucci, Genuis, & Violato, 2001), as well as epidemiological (Duncan, Saunders, Kilpatrick, Hanson, & Resnick, 1996; Kilpatrick, et al., 2003) and cross-cultural studies (Kessler, et al., 2010) have all shown a direct link between child maltreatment and PTSD. However, not every maltreated child develops PTSD or PTSD symptoms (Copeland, Keeler, Angold, & Costello, 2007), suggesting that additional mediating variables may explain how maltreatment leads to PTSD symptoms.

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Toward this end, researchers have given considerable attention to identifying biomarkers of risk that would explain the link between child maltreatment and PTSD. The autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis have been targeted predominantly because they are the two main physiological systems activated during environmental stress. Briefly, in healthy individuals the sympathetic branch of the ANS is activated through the locus coeruleus which elevates production of norepinephrine and stimulates the adrenal medulla to release epinephrine. Catecholamines such as norepinephrine and epinephrine are ultimately responsible for increasing blood glucose, heart rate, and blood pressure that aid the body in resolving a stressor (Cacioppo, 1994). Parasympathetic influence over cardiac activity, which is responsible for modulating increased sympathetic activity through the hypothalamus and amygdala, often withdraws during stressor situations to facilitate the sympathetic response and promote resolution of the stressor (Porges, 2003). Activation of the HPA axis begins when a stressor stimulates the corticotrophin-releasing hormone (CRH) in the hypothalamus, leading to secretion of the adrenocorticotrophic hormone (ACTH) in the anterior pituitary, and resulting in increased concentrations of cortisol produced by the adrenal cortex (Chrousos & Gold, 1992). When the stressor is resolved, the hypothalamus and anterior pituitary regulate cortisol concentrations by suppressing production of CRH and ACTH in a process known as the negative-feedback loop (Munck, Guyre, & Holbrook, 1984). Despite being separate arms of the body's stress response, the ANS and HPA axis are both designed to assist the individual under stressful conditions, are influenced by similar brain structures and contain regulatory capacities that modulate heightened physiological activity.

Child maltreatment has been linked to disrupted profiles of ANS and HPA axis activity, including significantly higher and lower estimates at rest and during stress reactivity paradigms, when compared to non-maltreated controls. For instance, child maltreatment is related to disruptions in the respiratory sinus arrhythmia (RSA), an index of parasympathetic control, at rest (Hopper, Spinazzola, Simpson, & van der Kolk, 2006; Miskovic, Schmidt, Georgiades, Boyle, & MacMillan, 2009) and under stress (Dale, et al., 2009) with disruptions in RSA predictive of PTSD symptoms (Blechert, Michael, Grossman, Lajtmán, & Wilhelm, 2007; Keary, Hughes, & Palmieri, 2009). Child maltreatment has also been linked to disruptions in cortisol activity, an indicator of HPA axis functioning, at rest (Cicchetti & Rogosch, 2001; King, Mandansky, King, Fletcher, & Brewer, 2001) and during chemical or laboratory stress paradigms (Carpenter, et al., 2007; Hart, Gunnar, & Cicchetti, 1995; Heim, et al., 2000), each of which have been linked to PTSD symptoms (Carrion, et al., 2002; Santa Ana, et al., 2006). However, research on disruptions in biomarker profiles in maltreated samples varies across developmental stages (Trickett, Noll, Susman, Shenk, & Putnam, 2010). Thus, studying a single developmental stage while controlling for confounding variables such as how recent the abuse occurred and the time of day biomarkers are sampled will strengthen inferences made about the role of physiological processes in eliciting PTSD symptoms.

Psychological processes represent mediational pathways that explain how an event, such as child maltreatment, exerts its effect on a particular psychological outcome while also identifying key targets for clinical intervention. One such process, experiential avoidance, has gained considerable attention recently as it has been linked to a number of different psychological outcomes (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Experiential avoidance involves an unwillingness to experience painful or aversive private events, such as unwanted thoughts, emotions, memories, and physiology, with attempts to control, suppress or inhibit the form or frequency of these private events and the contexts that occasion them (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). This process may play a particularly important role in promoting the development of PTSD symptoms in children who have been maltreated. Maltreatment, like other forms of trauma, can prompt painful

private events, such as re-occurring images or recollections of the event, heightened physiological states, and increased fear and anxiety. Coming into contact with these events may generate attempts to alter, suppress or otherwise control their form or frequency so that the aversiveness of the experience can be reduced. This can establish the use of avoidance strategies, a key symptom cluster of PTSD, while actually maintaining or increasing other PTSD symptoms (Cameron, Palm, & Follette, 2010; Rosenthal, Cheavens, Lynch, & Follette, 2006). Maltreated children commonly using avoidance strategies, including avoiding conversations about the abuse or subsequent private events, may actually prevent the experiencing and processing of the abuse that is central to the recovery from and treatment of child trauma (Cohen, Mannarino, & Deblinger, 2006) and PTSD (Foa & Rothbaum, 1998).

Child maltreatment has indeed been related to higher levels of experiential avoidance (Gratz, Bornova, Delany-Brumsey, Nick, & Lejuez, 2007; Sullivan, Meese, Swan, Mazure, & Snow, 2005) which in turn has predicted the development of PTSD symptoms above and beyond initial symptom severity (Plumb, Orsillo, & Luterek, 2004). Moreover, experiential avoidance has demonstrated mediation between child maltreatment and several different psychological outcomes (Marx & Sloan, 2002; Polusny, Rosenthal, Aban, & Follette, 2004). Thus, avoidance may play a key role in PTSD symptom development for maltreated individuals and explain how some of these individuals eventually develop PTSD symptoms when others do not. However, most of the research to date has been conducted with adult samples and an important area for future research is to extend this model of experiential avoidance to adolescents who have recently been maltreated and are experiencing PTSD symptoms.

Recent efforts explicating biobehavioral pathways to psychopathology have called for the simultaneous assessment of multiple physiological systems as opposed to a single system approach (Bauer, Quas, & Boyce, 2002). Furthermore, physiological and psychological processes co-occur and no study to date has tested whether a set of these processes mediates the relationship between child maltreatment and PTSD symptoms in the same statistical model. Thus, this study aims to test: 1) whether RSA reactivity, HPA axis reactivity and experiential avoidance mediates the relationship between child maltreatment and PTSD symptoms, 2) whether any specific indirect effect contributes significantly to a total indirect effect, and 3) whether the indirect effects of any one variable is superior when compared to the indirect effects of the other variables in the model. It was hypothesized that the set of variables would mediate the relationship between child maltreatment and PTSD and that each indirect pathway would contribute uniquely to the model. A sample of adolescent females was selected to test the study aims as older children experiencing trauma are most likely to develop PTSD symptoms (Copeland, et al., 2007) with females more likely to experience the different forms of child abuse (Gaudiosi, 2010) and more likely to develop PTSD symptoms when compared to males (Breslau, Wilcox, Storr, Lucia, & Anthony, 2004).

Method

Sample

A total of 110 females 14-19 years of age participated in this study. Substantiation of child maltreatment was determined by a Child Protective Service (CPS) agency investigation of physical neglect or contact physical or sexual abuse. All maltreated females ($n = 51$) experienced substantiated maltreatment within the 12 months prior to study participation. Thus, the recency of abuse was confined to those experiencing maltreatment in the past year. Of the 51 maltreated participants, 49% experienced sexual abuse, 45% experienced physical abuse, and 16% experienced physical neglect with 10% experiencing more than one form of

abuse. Comparison females ($n = 59$) were recruited from a local teen health center. Comparison females were screened and excluded from the current study if they had a substantiated case of maltreatment within the 12 months prior to study entry. A dummy-coded variable, 'Child maltreatment' was created to distinguish between participants with substantiated maltreatment (Child maltreatment = 1) from those participants without (Child maltreatment = 0) in subsequent statistical analyses.

Procedure

All procedures were approved by the local Institutional Review Board prior to beginning the study. Participants responding to recruitment were provided with initial information about the study and scheduled for a laboratory assessment. Upon arrival to their appointment, complete information about the study and study procedures was provided to all adolescent participants and to their non-abusing caregiver when the adolescent was under the age of 18. Once questions and concerns about participation were addressed, signed consent and child assent when applicable was obtained. Once consent was obtained, participants completed a general interview about current health habits related to study variables, self-report questionnaires, and a semi-structured interview of PTSD symptoms. All appointments were scheduled between 11 a.m. and 5 p.m. to minimize sampling time variability in the diurnal rhythms of RSA and HPA axis activity.

Participants completed a five-minute resting condition prior to a stressor paradigm assessing physiological responses to performance and interpersonal challenge. The resting paradigm involved each participant sitting comfortably in a chair while listening to soft music and watching slow-moving images on a computer screen. A combined performance and interpersonal stressor was chosen given varying HPA axis and ANS responses to different stressor types (Stroud, et al., 2009). The performance aspect of the stressor paradigm involved each participant completing a series of timed, affect recognition tasks (Porges, Cohn, Bal, & Lamb, 2007). Each task required participants to view a face that changed from a neutral expression into one of six emotions: anger, sadness, happiness, fear, surprise, and disgust. Participant's responses were timed and each participant was asked to identify the emotion as quickly as they could while not making any mistakes before the time elapsed. The average length of time to complete the series of affect recognition tasks was 7.45 minutes ($SD = 1.19$). The interpersonal stressor involved participants viewing a series of video-clips of parent-adolescent conflict used in previous research with observed changes in physiological activity (Gordis, Granger, Susman, & Trickett, 2008). The length of time required to view all videos for each participant was 8 minutes.

Measures

General demographics and health habits form—Demographic information was assessed via self and caregiver report and included age, race, family income and family constellation (single-caregiver vs. dual-caregiver homes). Health habits assessed were: pregnancy status, use of steroids (topical, oral and inhaled), cigarettes, over-the counter (aspirin, ibuprofen) and prescription drugs (psychotropic, seasonal allergies, oral contraceptive), whether participants ate anything one hour prior to study participation, and whether participants drank caffeine the day of study participation. Participants who reported being pregnant ($n = 6$) were excluded from statistical analyses.

RSA—Inter-beat intervals (IBI's), the time measured in milliseconds between heart contractions, were recorded via electrocardiogram (ECG) using disposable Ag/AgCl electrodes placed on the chest and abdomen of each participant. IBI's were detected in real-time using a QRS peak detection algorithm applied to the ECG data and stored on the Biolog 3991x/2-EIR system manufactured by UFI Inc. IBI's were subsequently transferred

to a computer via USB for later inspection. CardioEdit and CardioBatch software (Brain-Body Center, University of Illinois at Chicago) were used to visually inspect and edit IBI streams for artifacts. Editing consisted of integer arithmetic such as dividing intervals when detections are missed or adding intervals when spuriously invalid detections occur. After editing of IBI data streams, estimates of RSA were calculated consistent with procedures developed by Porges (Porges & Byrne, 1992). Estimates of RSA were obtained in 30-second epochs and averaged across the resting, RSA_{Rest} , and stressor conditions, RSA_{Stress} . Reliable ECG recordings were not obtained on three participants and therefore their data are omitted from analyses. A manipulation check was performed to assess if the stressor paradigm significantly changed values of RSA from resting to stressor conditions. As expected, a paired sample t -test indicated that RSA estimates declined significantly from resting to stressor conditions, $t(105) = -3.52, p < .001$.

HPA axis—Cortisol was selected as a measure of HPA axis activity and was assessed across five samples collected at strategic times throughout the study to detect resting and stress responses. Each participant was instructed not to eat or drink 1 hour prior to participation. Upon arrival, each participant was asked to swish their mouth with water prior to beginning their appointment. The first sample, $Cortisol_{Rest}$, was collected approximately 25 minutes ($M = 25.30, SD = .24$) after participants began their study appointment to give them time to acclimate to the research environment and procedures. There was no significant difference between the maltreated and comparison groups as to the time of day their first sample was collected, $t(108) = .82, p = .41$. Samples two through five were collected 5-, 10-, 20- and 30-minutes post stressor to detect the maximum cortisol response to stress, $Cortisol_{Stress}$. Saliva was obtained through passive drooling into 2 mL polypropylene vials and stored at $-80^{\circ}C$ until assayed. Samples were assayed in duplicate using a highly-sensitive enzyme immunoassay from Salimetrics, LLC. The test has a lower limit sensitivity of $<.003 \mu g /dl$ and average intra-and inter-assay coefficients of variation 3.35% - 3.65% and 3.75% - 6.41%, respectively. A manipulation check was also performed to assess if cortisol concentrations changed significantly from resting to stressor conditions. A paired sample t -test indicated that cortisol concentrations, on average, did not change significantly from the resting to the stressor condition for the total sample, $t(108) = .71, p = .48$.

Acceptance and Action Questionnaire (AAQ; Hayes, et al., 2004)—Experiential avoidance was measured using the 22-item version of the AAQ. Items are rated on a 7-point Likert scale ranging from “Never True” to “Always True” with higher scores representing higher levels of experiential avoidance. Example items include, “I’m not afraid of my feelings (reverse scored).”, “Anxiety is bad.”, and “I try hard to suppress thoughts and feelings that I don’t like by just not thinking about them.” The AAQ has demonstrated reliability ($\alpha = .70 - .79$) and convergent validity with measures of anxiety ($r = .59, p < .01$), depression ($r = .75, p < .01$) and child trauma ($r = .18, p < .01$). The reliability of the AAQ in the current study is $\alpha = .72$.

PTSD symptoms—The Comprehensive Trauma Interview (CTI; Barnes, Noll, Putnam, & Trickett, 2009) is a semi-structured interview assessing a wide variety of information following an instance of child maltreatment. The CTI has demonstrated good inter-rater reliability with information collected from CPS investigations ($\kappa = .70 - .87$). The CTI has a specific section devoted to the assessment of PTSD symptoms consistent with the Diagnostic and Statistical Manual of Mental Disorders-IV-Text Revision (APA, 2000). Examples of questions asked are: “Have you ever had painful images, memories or thoughts of what happened?”, “Have you ever avoided doing things or getting into situations that reminded you of what happened?”, and “Have you ever been jumpy, on edge, or easily

startled because of what happened?” Responses to questions about PTSD symptoms are coded (0= No, 1=Yes) and a summary score of all responses is then calculated to reflect cumulative levels of PTSD symptoms. Reliability of the items used to derive the summary score for PTSD symptoms in the current sample was excellent, $\alpha = .89$.

Data Analytic Strategy

A multiple mediator model was employed to identify the total and specific indirect effects of RSA_{Stress}, Cortisol_{Stress} and AAQ scores when explaining the relationship between child maltreatment and PTSD symptoms (see Figure 1). The multiple mediator model in the present study was performed using PASW v.18 and a supplemental macro (see Preacher & Hayes, 2008). This method of testing mediation is particularly useful in the current study because of its use of bootstrapping to derive estimates of indirect effects. Bootstrapping is a non-parametric, re-sampling procedure for estimating indirect effects and their standard errors. By randomly sampling from n observations and estimating indirect effects k times with replacement of observations, a probability distribution of the estimates of the total and specific indirect effects, their standard errors and confidence intervals can be obtained. The ratio of point estimates and their respective standard errors, Z , can be calculated for a particular indirect effect with corresponding p values easily obtained. There is also the opportunity to enter variables into the multiple mediator model as statistical covariates. Results of the current multiple mediator model are based on $k = 5000$ bootstrap samples with bias-corrected and accelerated 95% confidence intervals (BCa 95% CI).

Results

Demographic Information and Data Screening

The mean age of the total sample was 17.00 years ($SD=1.17$), 58% of the adolescents were from single-caregiver homes, the median family income level was \$20,000 to \$29,000, and the sample was 42% Caucasian, 51% African-American, 1% Hispanic and 6% Multi-racial. Demographic information is presented by group membership in Table 1. Chi-square and multivariate analysis of variance (MANOVA) tested for differences between maltreatment and comparison groups on key demographic, health and study-related variables. Results indicated significant between-group differences on the use of prescribed steroid medication (Steroid use = 1; No steroid use = 0), $\chi^2(1) = 5.27$, $p = .02$, $\phi = .23$, and cigarette use (Cigarette use = 1; No cigarette use = 0), $\chi^2(1) = 7.81$, $p = .01$, $\phi = .28$, with the maltreated group using steroid medication and cigarettes more often than the comparison group (see Table 1). No other health-related variable differed significantly between groups. The omnibus MANOVA yielded a significant effect for group membership, Wilk's $\gamma = .70$, $p < .001$, $\eta^2 = .30$, with post-hoc tests indicating that the maltreated group was significantly younger, had more PTSD symptoms, were more likely to avoid painful private events, and had marginally higher levels of RSA at rest and during stress (see Table 2 for full results). No other significant group differences were found.

Because age, steroid and cigarette use were sample characteristics that differed significantly between groups, these variables were used as covariates in all subsequent analyses. To ensure that experiential avoidance was not highly correlated with any one cluster of PTSD symptoms, specifically the avoidance cluster, partial correlations were performed controlling for age, steroid and cigarette use. Results indicated that AAQ scores were only moderately correlated with all three PTSD symptom clusters, re-experiencing: $r = .32$, hyperarousal: $r = .36$, and avoidance: $r = .33$, with no significant differences in the strength of the relationships. There were no significant differences between maltreated and non-maltreated groups on time of sampling for RSA or cortisol and therefore these variables were not included as covariates in subsequent analyses. A partial correlation matrix of variables used

in the multiple mediator analysis is presented in Table 3. Results of the partial correlation matrix controlling for age, steroid use and cigarette use indicate that maltreatment was significantly associated with more experiential avoidance, blunted $\text{Cortisol}_{\text{Stress}}$ estimates, and a higher number of PTSD symptoms.

Multiple Mediator Analysis

In addition to age, steroid use and cigarette use, RSA_{Rest} and $\text{Cortisol}_{\text{Rest}}$ were added as covariates in the multiple mediator model to control for the influence of initial values on stress estimates of physiological variables. The total (c) and direct (c') effects for the relationship between child maltreatment and PTSD symptoms were $B = 4.82$, $p < .001$, and $B = 3.89$, $p < .001$, respectively, indicating that maltreatment was related to more PTSD symptoms. Because the difference between the total and direct effects is the total indirect effect exerted through the set of mediators, a point estimate of $B = .93$ was obtained. As can be seen in Table 4, the corresponding BCa 95% confidence interval (CI) does not contain zero, indicating that the total indirect effect for $\text{RSA}_{\text{Stress}}$, $\text{Cortisol}_{\text{Stress}}$ and AAQ as a set mediated the relationship between child maltreatment and PTSD symptoms.

Specific indirect effects were then assessed to determine whether each of the variables contributed significantly to the total indirect effect when simultaneously considering the indirect effects of the other two variables. Results demonstrated that the BCa 95% CI around the point estimate for the specific indirect effect of the AAQ did not include zero, indicating that the AAQ accounts for a significant proportion of the mediated effect when estimating simultaneously the contributions of $\text{RSA}_{\text{Stress}}$ and $\text{Cortisol}_{\text{Stress}}$, $Z = 2.34$, $p = .01$. The unstandardized parameter estimates indicated that child maltreatment was significantly related to higher levels of experiential avoidance, $B = 8.74$, $p = .01$, with higher levels of experiential avoidance significantly related to more PTSD symptoms, $B = .10$, $p < .01$. Neither of the specific indirect effects for $\text{RSA}_{\text{Stress}}$ or $\text{Cortisol}_{\text{Stress}}$ contributed significantly to the total indirect effect in this model. Unstandardized parameter estimates for all paths were not significantly different from zero for both $\text{RSA}_{\text{Stress}}$ and $\text{Cortisol}_{\text{Stress}}$.

Pairwise contrasts comparing the strength of a specific indirect effect relative to another indirect effect in the model were estimated for each variable. Results demonstrated that the BCa 95% CI around indirect effect for AAQ did not contain zero in any planned comparison, indicating that the indirect effect for AAQ was significantly stronger in comparison to both $\text{RSA}_{\text{Stress}}$, $Z = 2.17$, $p = .02$ and $\text{Cortisol}_{\text{Stress}}$, $Z = 2.18$, $p = .01$. There was no difference in the magnitude of the indirect effects between $\text{RSA}_{\text{Stress}}$ and $\text{Cortisol}_{\text{Stress}}$.

Discussion

The current study tested a set of variables that would account for the relationship between child maltreatment and PTSD symptoms. To do so, a multiple mediator model was tested using three variables, RSA reactivity, cortisol reactivity and experiential avoidance, with prior empirical support to explain the relationship between child maltreatment and PTSD symptoms. The set of variables did indeed mediate the relationship between child maltreatment and PTSD symptoms, suggesting that these variables play a key role in explaining how maltreated children develop PTSD symptoms. However, experiential avoidance was the only variable to contribute significantly to the mediational model, exerting a significantly stronger indirect effect when compared to the indirect effects of RSA and cortisol reactivity. This indicates that the more maltreated participants avoided painful thoughts, emotions, memories, and physiology, the more PTSD symptoms they reported. Moreover, experiential avoidance was positively related to all three PTSD symptom clusters, suggesting that it is associated with a range of PTSD symptoms not one cluster in particular.

These results raise the possibility that avoidance plays a role in the development of PTSD symptoms while preventing maltreated children from experiencing and processing the abuse in an effective manner. Alternatively, maltreated children who do not commonly use strategies to avoid painful private events, or who engage in experiencing private events with awareness and acceptance, may be more resilient to the trauma and less likely to develop PTSD symptoms. Thus, focusing on the extent to which adolescents are willing to experience painful private events appears to have considerable utility in research and intervention models.

A particular advantage to studying psychological processes is that they have direct implications for psychological interventions. For instance, secondary prevention programs can incorporate a focus on disrupting experiential avoidance as part of an overall program assisting adolescents recovering from child maltreatment. This method of prevention could potentially disrupt the pathway from maltreatment to PTSD symptoms and thereby prevent or mitigate the development of PTSD symptoms. Intervention models could also be adapted to disrupt experiential avoidance as an alternative means for achieving treatment goals when maltreated adolescents are experiencing PTSD symptoms. For example, by promoting a willingness to experience difficult thoughts, emotions and memories adolescents may be able to counteract patterns of avoidance and promote the experiencing and processing of the abuse to achieve recovery. Moreover, experiential avoidance may have broader implications beyond PTSD symptoms. Experiential avoidance is an approach to all painful private experiences, not just those that are related to traumatic events or PTSD symptoms. Interventions that promote acceptance of private events in conjunction with behavioral change strategies, such as dialectical behavior therapy (Linehan, 1993) and acceptance and commitment therapy (Hayes, Strosahl, & Wilson, 1999), have a growing empirical base. Thus, the extension of these treatments to PTSD symptoms may be an avenue to explore in future research.

The lack of findings for cortisol and RSA were surprising. Once group differences on demographic and health-related behaviors were controlled, the maltreated group did display a blunted cortisol reaction to stress. This finding is consistent with previous research showing a relationship between child maltreatment and blunted cortisol reactions in stress paradigms (Hart, et al., 1995). However, contrary to expectations results of this study failed to find a relationship between cortisol reactivity and PTSD symptoms. While a relationship between cortisol reactivity and PTSD symptoms has been reported in adult samples (Heim, et al., 2000), this association is not always found with adolescent samples (MacMillan, et al., 2009). One explanation for this may come from longitudinal research showing that women who were sexually abused have higher resting cortisol concentrations during childhood, comparable estimates to non-sexually abused peers during adolescence, and significantly lower concentrations in young adulthood (Trickett, et al., 2010). Thus, an adjustment of the HPA axis may be occurring in maltreated samples across development with profiles assessed in younger and older developmental stages more strongly tied to PTSD symptoms. In addition, RSA estimates declined significantly from resting to stress conditions, however, these estimates did not vary significantly by group nor were they systematically related to PTSD symptoms. There may be several explanations for these findings. Like cortisol, there is some variation in studies examining the relationship between RSA and PTSD using adult (Sahar, Shalev, & Porges, 2001) and child or adolescent samples (Scheeringa, Zeanah, Myers, & Putnam, 2004). Thus, there may be a similar adjustment in parasympathetic control over stress occurring through development, although future research would need to demonstrate such a change. RSA estimates may also vary by type of trauma although again more research is needed to support such a claim.

Several limitations should be considered when interpreting the results of this study. First, these data are cross-sectional and therefore causal attributions are not appropriate despite the use of mediational modeling. Mediational modeling was used to examine the contributions of several empirically-tested variables in a single model in order to further inform the research literature and theoretical perspectives of maltreatment and PTSD. While this same model may hold in future research, longitudinal models will be needed to promote generalized causal inferences about the effects of RSA, cortisol, and experiential avoidance in mediating the relationship between child maltreatment and PTSD. Moreover, alternative models, such as ones that view cortisol and RSA as moderators of the relationship between child maltreatment and PTSD symptoms may yield additional explanatory value as opposed to viewing these variables as mediators of this relationship. Second, the sample for this study consists entirely of females. While the focus for the study was to examine an adolescent sample at highest risk for PTSD, results cannot necessarily generalize to maltreated males. Third, the effects of maltreatment in this sample are limited to those with a substantiated case of maltreatment, which tend to represent the more severe cases of abuse, in the past 12 months. Thus, results cannot be generalized to chronic cases of abuse per se, abuse that has occurred prior to the past 12 months, or other methods such as self-report. Fourth, while diurnal changes in cortisol stabilize as early as 11 a.m. (Kupper, et al., 2005; Wessa, Rohleder, Kirschbaum, & Flor, 2006), sampling cortisol at later times in the day may provide more reliable estimates as the diurnal rhythm continues to stabilize across the day. Finally, timing within the menstrual cycle was not assessed and can influence hormone concentrations, particularly cortisol. Future research should control the effect of menstrual cycling on cortisol concentrations. Despite these limitations, the results of this study suggest that experiential avoidance may be an important variable explaining how maltreated adolescents develop PTSD symptoms. This study provided a strong test of several potential mediators of the relationship between child maltreatment and PTSD symptoms and offers viable ideas for future prevention or intervention research of PTSD symptoms. The results suggest that when maltreated adolescents are willing to experience painful private events, they are more likely to experience fewer PTSD symptoms.

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Figure 1.
Conceptual diagram of proposed multiple mediator model.

Table 1

Demographic information for maltreated and comparison groups.

Variable	Maltreated (<i>n</i> = 51)	Comparison (<i>n</i> = 59)
	<i>M</i> (<i>SD</i>) or <i>N</i> (%)	<i>M</i> (<i>SD</i>) or <i>N</i> (%)
Race		
African-American	24 (22%)	32 (29%)
Caucasian	24 (22%)	22 (20%)
Hispanic	0 (0%)	1 (1%)
Multi-racial	3 (3%)	3 (3%)
Income		
Under \$10,000	14 (13%)	14 (13%)
\$10,000 - \$19,000	8 (7%)	6 (6%)
\$20,000 - \$29,000	9 (8%)	9 (8%)
\$30,000 - \$39,000	5 (5%)	10 (9%)
>\$40,000	14 (13%)	19 (18%)
Family Environment		
Single-caregiver home	26 (25%)	33 (32%)
Dual-caregiver home	20 (20%)	23 (23%)
Pregnant	4 (4%)	2 (2%)
Steroid use	9 (8%) *	2 (2%)
Cigarette use	14 (13%) **	4 (4%)
Eat Time	18 (18%)	13 (13%)
Caffeine Use	9 (9%)	8 (8%)

** Note: = $p < .01$;

* $p < .05$.

Table 2

Means, standard deviations, and post-hoc results of multivariate analysis of variance.

Variable	Maltreated <i>M (SD)</i>	Comparison <i>M (SD)</i>	<i>F</i>	<i>df</i>	<i>Cohen's d</i>
Age	16.75 (1.10)	17.20 (1.20)	6.33**	1, 96	.39
RSA _{Rest}	7.58 (1.05)	7.20 (1.13)	3.72 [†]	1, 96	.35
RSA _{Stress}	7.40 (.98)	7.01 (1.13)	3.60 [†]	1, 96	.37
Cortisol _{Rest}	.12 (.10)	.15 (.13)	2.54	1, 96	.26
Cortisol _{Stress}	.13 (.12)	.16 (.11)	1.99	1, 96	.26
AAQ	85.02 (13.79)	78.29 (14.81)	5.95*	1, 96	.47
PTSD	8.47 (4.85)	3.73 (3.98)	28.21***	1, 96	1.07

****p* = .001;

***p* = .01;

**p* = .05;

[†]*p* = .06.

RSA_{Rest} = RSA assessed during rest; RSA_{Stress} = RSA assessed during stressor; Cortisol_{Rest} = Cortisol assessed during rest; Cortisol_{Stress} = Cortisol assessed during stress; AAQ = Acceptance and Action Questionnaire; PTSD = PTSD symptoms.

Table 3

Partial correlation matrix for variables used in the multiple mediator analysis.

Variable	1	2	3	4	5	6	7
1. Maltreatment	-						
2. RSA _{Rest}	.17 [†]	-					
3. RSA _{Stress}	.16	.88***	-				
4. Cortisol _{Rest}	-.13	-.05	-.02	-			
5. Cortisol _{Stress}	-.20*	-.05	-.08	.71***	-		
6. AAQ	.28**	-.03	.02	-.13	-.11	-	
7. PTSD	.46***	.11	.11	-.08	-.12	.39***	-

Note: = *p* .001;
**
= *p* .01;
*
= *p* .05;
[†]
= *p* .10.

Age, steroid use, and cigarette use were controlled in matrix. Maltreatment coded as 0 = Comparison, 1 = Maltreated; RSA_{Rest} = RSA assessed during rest; RSA_{Stress} = RSA assessed during stressor; Cortisol_{Rest} = Cortisol assessed during rest; Cortisol_{Stress} = Cortisol assessed during stress; AAQ = Acceptance and Action Questionnaire; PTSD = PTSD symptoms.

Table 4

Mediation of the relationship between child maltreatment and PTSD symptoms.

	Point		BCa 95% CI		
	Estimate	SE	Z	Lower	Upper
Indirect Effects					
Total	.93	.45	2.07*	.21	1.95
RSA _{Stress}	-.00	.12	-.02	-.29	.21
Cortisol _{Stress}	.04	.16	.25	-.19	.41
AAQ	.89	.38	2.34**	.28	1.81
Pairwise Contrasts					
AAQ vs. Cortisol _{Stress}	.85	.39	2.18**	.24	1.86
AAQ vs. RSA	.89	.41	2.17*	.24	1.87
Cortisol vs. RSA	.05	.20	.25	-.47	.37

** Note: = *p* .01;
* = *p* .05.

BCa 95% CI = bias corrected and accelerated 95% confidence intervals; Cortisol_{Stress} = Cortisol assessed during stress; RSA_{Stress} = RSA assessed during stress; AAQ = Acceptance and Action Questionnaire. Estimates based on 5000 bootstrap samples.