

EMOTIONAL ABUSE AND LATER DATING INTERPERSONAL VIOLENCE: THE  
MODERATING ROLE OF CORTISOL REACTIVITY

by

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(Under the Direction of Anne Shaffer)

ABSTRACT

Interpersonal violence perpetration and victimization have been linked to past experiences of childhood maltreatment; however, little research has explored how biological stress reactivity functions within these relationships. The present study investigated the moderating effects of stress reactivity on associations between reports of childhood emotional abuse and later interpersonal violence in young adult romantic relationships. The current sample consisted of 57 young adult romantic partners. Salivary cortisol samples were collected before and after a stress task to measure stress reactivity. In line with previous literature, childhood emotional abuse was related to later dating victimization, but was not significantly related to young adult dating aggression. The relations between childhood emotional abuse and dating victimization and aggression varied depending on cortisol reactivity, such that for those who experienced low levels of cortisol reactivity, the relation between childhood emotional abuse and dating violence increased.

INDEX WORDS: Emotional abuse, psychological aggression, romantic relationships,  
cortisol reactivity

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## CHAPTER 1

### INTRODUCTION AND LITERATURE REVIEW

Childhood emotional abuse (CEA) have been linked to deleterious outcomes throughout the lifespan (Mullen, Martin, Anderson, Romans, & Herbison, 1996; Teicher, Samson, Polcari, & McGreenery, 2006; Wright, 2007). According to the American Professional Society on the Abuse of Children, emotional abuse is defined as the communication to a child that he or she is “worthless, flawed, unloved, endangered, or only valuable in meeting someone else’s needs” (APSAC, 2013, p. 127). This definition further includes subcategories that are used to elucidate the different ways in which a child may experience emotional abuse. These categories include terrorizing, isolating, denying emotional responsiveness, spurning, and exploiting/corrupting a child.

CEA has been shown to have particularly impactful consequences in young adulthood, including a heightened risk for depression, anxiety, and somatic symptoms (Allen, 2008; Wright, Crawford, & Del Castillo, 2009). Extant literature has also indicated that there is an association between CEA and later emotion regulation difficulties in young adulthood (e.g., Burns, Jackson, & Harding, 2010). In regards to personality, CEA has been associated with schizotypal, avoidant, and borderline personality symptomatology (e.g., Allen, 2008; Gibb, Wheeler, Alloy, & Abramson, 2001). Thus, it is evident that CEA is a pervasive, adverse experience, shown to negatively impact several domains of young adult functioning.

Current literature has also suggested that negative romantic relationship outcomes in adulthood have been associated with childhood maltreatment histories, including difficulties

maintaining intimate relationships, which may consequently lead to marital problems and divorce (Coleman & Widom, 2004; Mullen, Martin, Anderson, Romans, & Herbison, 1996). In a review by Reyome (2010), CEA was shown to be associated with several detrimental outcomes in adult romantic and marital relationships, including lower relationship quality, lower trust, a greater fear of intimacy, codependency, and greater distance in relationships. Davis and colleagues (2001) also found that CEA was a significant predictor of relationship quality, above and beyond the impact of childhood experiences of physical and sexual abuse. To better elucidate the relationship between CEA and later interpersonal violence in romantic relationships, research has explored potential factors to explain this relationship. Specifically, maladaptive schemas (e.g., schemas of mistrust, emotional inhibition) have been shown to mediate the relation between childhood experiences of emotional maltreatment and later romantic relational aggression as well as victimization (Crawford & Wright, 2007).

Beyond outcomes in adult romantic and marital relationships, there is growing evidence that these associations can be found among young adult dating couples as well. Young adulthood has been identified as a developmental period characterized by the exploration of romantic relationships as well as emotional and physical intimacy (Arnett, 1998 & 2000). Thus, it may be particularly important to examine how the effects of a history of emotional abuse may lead to adverse outcomes in dating relationships during this highly salient phase. For example, DiLillo, Lewis, and Loreto-Colgan (2007) found that young adult women with a history of childhood maltreatment had significantly greater difficulties in dating conflict resolution. In regards to emotional forms of maltreatment, interpersonal conflict in a sample of young adult women was linked to experiences of CEA, above and beyond other parenting behaviors (Messman-Moore & Coates, 2008). Young adult males and females tend to report approximately the same level of

dating aggression and perpetration within the extant literature, with parent relationship quality (e.g., communication, alienation) and childhood maltreatment significantly predicting aggression in romantic relationships (Fang & Corso, 2007; Linder, Crick, & Collins, 2002). More specific to CEA, Zurbriggen and colleagues (2010) also found that a history of CEA significantly predicted young adult sexual violence (both perpetration and victimization), above and beyond other forms of abuse. There has been additional research to support that CEA is associated with aggression and victimization in young adult dating relationships (Crawford & Wright, 2007; Sappington, Pharr, Tunstall, & Rickert, 1997). The extant literature also suggests that CEA is a stronger predictor than other forms of maltreatment on later dating aggression among young adults (e.g., Berzenski & Yates, 2010; Zurbriggen et al., 2010); however, additional research is needed to replicate and expand upon these findings.

One underlying theory that may aid in elucidating the relationship between experiences of CEA and later dating violence in young adult dyads is attachment theory. Within the extant literature, the role of attachment has been widely documented as a mechanism that exerts influence on later dating relationships (see Hazan & Shaver, 1994 for a review). Attachment theory is conceptualized as working models that are formed during early childhood interactions with primary caregivers that inform the perception and responses to others throughout the lifespan (see Bowlby, 1982). Building upon Bowlby's attachment theory, Ainsworth and colleagues developed the Strange Situation to better understand response patterns in infants that were temporarily separated from their caregiver (Ainsworth & Bell, 1970; Ainsworth, 1979). Through this groundbreaking research, three infant attachment styles were identified: secure, anxious-ambivalent, and avoidant. These infant attachment styles have also been shown to be an extension of later romantic attachment styles that are seen in adulthood (Hazan & Shaver, 1987).

Thus, all adult relationships are influenced, at least in part, by the (in)secure working model that formed during the caregiver-infant relationship (Waters & Cummings, 2000). However, childhood experiences of abuse have been shown to negatively impact both child and adult attachment styles, such that those with a history of childhood abuse are more likely to demonstrate insecure attachment styles (Styron & Janoff-Bulman, 1997). Further, in a sample of adult women, McCarthy and Taylor (1999) found that childhood experiences of abuse were related to increased difficulties in romantic relationships. More specifically, these authors found that avoidant attachment style mediated the association between childhood experiences of abuse and later adult romantic relationship difficulties. Other research has shown that anxious and avoidant attachment styles are associated with more deleterious outcomes (e.g., higher levels of negative emotionality within the dating relationship) than those who had secure attachment styles for both men and women (Simpson, 1990).

To better elucidate the relationship between CEA and later aggression and victimization in young adult dating relationships, research has moved towards examining factors that may mediate and moderate these associations. With the development of physiological measurements, researchers have begun to explore how physiological stress reactivity may contribute to, and exacerbate, deleterious outcomes. Cortisol, a hormone that is produced by the hypothalamic-pituitary-adrenocortical (HPA) axis, can be used as a biological marker of stress and can be utilized to assess stress reactivity (see Clements, 2013). Specifically, when a stressor is encountered the production of cortisol is amplified in the HPA-axis to prepare the body for the stressor. The activation and subsequent release of cortisol in the HPA-axis follows a negative feedback cycle, such that elevated levels of cortisol in the bloodstream suppress any further release (see Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009). Following the conclusion

of a stressor, cortisol levels in the body typically decrease back to baseline over a gradual period extending from minutes to hours (see Clements, 2013). However, when exposed to a prolonged stressor, the HPA-axis may become hyper- or hyposensitive, resulting in differing stress reactivity to future stressors (Miller, Chen, & Zhou, 2007). Additionally, psychiatric disorders have been associated with the dysregulation of the HPA-axis, such that posttraumatic stress disorder has been linked to a hyposensitive HPA-axis (Miller et al., 2007) and major depression in females has been linked to hyperactivity of the HPA-axis (see Shea, Walsh, MacMillan, & Steiner, 2005). Further, Gunnar and Vazquez (2006) postulate that hyperresponsivity of the HPA-axis may be a risk factor for adverse outcomes and maladaptation. Of note, there has been additional research to suggest that for some individuals, there is no heightened or blunted cortisol response to stressors (Chida & Steptoe, 2009). Thus, it appears that individual differences exist in whether and how individuals respond to specific stressors, including contextual factors such as early life stress exposure (Goldman-Mellor, Hamer, & Steptoe, 2012; Miller et al., 2007).

There is evidence in the extant literature to suggest that stress reactivity is influenced by a history of maltreatment and childhood stress (see Gunnar & Quevedo, 2007). It has been demonstrated that there is a relationship between childhood maltreatment and cortisol reactivity, in that adults maltreated as children tend to have a higher responses to stressors (Tarullo & Gunnar, 2006). However, extant literature also suggests that blunted HPA reactivity is possible in adults who experienced childhood maltreatment (Carpenter et al., 2007). Specific to emotional maltreatment, research has shown that CEA predicts a lower stress response in adulthood independent of other maltreatment experiences and participant age (Carpenter, Tyrka, Ross, Khoury, Anderson, & Price, 2009). These varied results are difficult to reconcile at this point, but

certainly suggest that not everyone who experiences maltreatment will have similar alterations, if any, to their HPA reactivity.

Thus, it is worth considering how differences in stress reactivity might amplify or buffer against the influence of other developmental risks. Some studies have addressed these questions by testing stress reactivity, broadly defined, as a moderator variable. Research conducted by Obradović and colleagues (2010) revealed that stress reactivity, as measured by respiratory sinus arrhythmia and salivary cortisol, was a significant moderator in the relationship between child family adversity and child adaptive functioning. Specifically, these authors found that highly reactive children had worse adaptive functioning (e.g., more externalizing symptoms, less prosocial behaviors, less school engagement, and less academic competence) in the context of high adversity. Equally reactive children in settings of low adversity showed high levels of adaptation. The same study additionally found evidence to suggest that high cortisol reactivity is associated with higher levels of externalizing behaviors for those in high adversity settings and high levels of prosocial behaviors in the context of low adversity. Moreover, low cortisol reactivity has been shown to moderate the relationship between testosterone and overt aggression and violent criminality in delinquent adolescent males (Dabbs, Jurkovic, & Frady, 1991; Popma et al., 2007). Taken together, these findings suggest that dysregulated cortisol reactivity (e.g., heightened or blunted) may exacerbate the risk for aggression and violence. However, extant literature has not yet examined how cortisol stress reactivity may moderate experiences of childhood abuse, and specifically CEA, to later dating aggression or victimization.

The goal of the present study was to investigate how cortisol reactivity may influence the relationship between CEA and later interpersonal violence in a sample of young adult dating dyads. In line with previous literature, we expected that individuals with a history of CEA would

be at greater risk for perpetrating later dating aggression in young adult dyads. We further hypothesized that the relationship between CEA and later aggression would vary as a consequence of individual physiological stress reactivity, as measured by cortisol reactivity. Specifically, we expected the relations between a history of CEA and current interpersonal aggression would be strongest for those who show dysregulated cortisol reactivity (e.g., reactive or blunted) during a stress-inducing task. We also investigated the parallel associations between CEA and interpersonal victimization. To better elucidate this potential relationship, we used cortisol reactivity as a moderator. However, due to contradictory evidence that CEA predicts later romantic relational victimization, these analyses are considered exploratory.

## CHAPTER 2

### METHOD

#### Participants

Participants in the current study were a part of a larger investigation designed to examine predictors of romantic relationship outcomes. Participants were recruited from a southeastern U.S. university through the Department of Psychology's Research Participant Pool and flyers that were distributed around campus. Participants were required to be at least 18 years old and in a romantic relationship of at least one month's duration. The sample included 57 individuals (46 females, 11 males) between the ages of 18 and 24 ( $M$  age = 19.47,  $SD$  = 1.39) who were in a heterosexual romantic relationship. Current relationship duration ranged from 1 to 66 months, with a median length of 13 months ( $M$  = 18.55,  $SD$  = 15.98). The ethnic composition was as follows: 67.2% of participants were Caucasian, 13.8% Asian, 8.6% Hispanic, 1.7% African American, 6.9% mixed race, and 1.7% identified as "other." The University of Georgia's Institutional Review Board approved the present study and informed consent was obtained from all participants.

#### Measures

*The Child Trauma Questionnaire (CTQ; Bernstein & Fink, 1998)*. The CTQ is a self-report measure used to assess retrospective histories of childhood maltreatment. The questionnaire is comprised of 28 items and includes five subscales of maltreatment (physical abuse, sexual abuse, emotional abuse, physical neglect, and emotional neglect). The item responses are rated on a Likert scale ranging from 1 (never true) to 5 (very often true). For the

present study, the emotional abuse subscale was utilized. This measure has been used extensively in the literature with acceptable reliability (e.g., Bradbury & Shaffer, 2012; Perry, DiLillo, & Peugh, 2007). The internal consistency of the total scale and emotional abuse subscale was measured using Cronbach's alpha (total-score  $\alpha = .54$  and emotional abuse subscale  $\alpha = .68$ ). Childhood emotional abuse scores were used as the independent variable in the present analyses.

*The Conflict Tactics Scale-Revised (CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996).* The CTS2 is a 78-item self-report questionnaire that contains five subscales for victimization and aggression (physical assault, psychological aggression, negotiation, injury, and sexual coercion) to examine various types of conflict in current romantic relationships. Item responses are on an 8-point scale that ranges from 0 (no frequency, has never happened), 6 (high frequency, more than 20 times in the past year), to 7 (not in the past year, but it has happened). This measure has been used extensively in the literature to assess interpersonal violence within romantic relationships (e.g., Lilly & Graham-Bermann, 2010). Internal reliability for victimization and aggression subscales was computed (dating aggression  $\alpha = .79$ ; dating victimization  $\alpha = .78$ ). A composite of interpersonal dating aggression within the participant's current relationship was created by rescored psychological aggression, physical aggression, sexual aggression, and injury items as either 0 (this behaviors has not occurred in my current dating relationship) or 1 (this behavior has occurred in my current dating relationship). Mirroring the composite for interpersonal dating aggression, a composite was used to assess interpersonal dating victimization using psychological victimization, physical victimization, sexual victimization, and injury. Thus, reports of dating aggression and victimization could range from 0 (no occurrence of behavior) to 32 (all aggressive/victimization behaviors occurred). This computation has been used successfully in the extant literature to assess interpersonal violence in

romantic relationships (Harding et al., 2013). The total composite scores from the dating aggression and victimization were used as the dependent variables in the analyses.

*Cortisol.* To measure stress reactivity of the participants, cortisol samples were collected following consent procedures and a brief introduction to the laboratory to establish a baseline. Cortisol samples were collected a second time to assess for stress reactivity 25 minutes following the start of the individual stressor task (described below) due to the extant literature that suggests that cortisol takes approximately 20 minutes to enter the saliva (Stansbury & Gunnar, 1994). Prior to presenting at the laboratory, participants were asked not to eat, drink, smoke, or brush their teeth for two hours before the assessment. When the participants arrived at the laboratory, they were asked if they had engaged in any of the restricted activities or were on any medications that may affect their cortisol levels. If participants endorsed any of the above items, they were rescheduled for another date. Participants were given the instructions to drool through a straw that was put inside a plastic vial. The container was then put into a freezer for appropriate storage. Cortisol reactivity, as measured by area under the curve (AUC; see below), was calculated and used as the proposed moderator variable. This computational technique has been used in the extant literature and has demonstrated utility in measuring stress reactivity (Pruessner et al., 2003).

### Procedure

Respondents were instructed to complete an online survey using Survey Monkey, a website that meets the U.S. Department of Commerce's Safe Harbor Privacy Standards. The survey took approximately two hours to complete and all items included a "prefer not to answer" answer selection. Participants were assigned a personal identification number to ensure confidentiality and informed consent was given prior to the start of the online survey. Research

credit for those in a psychology course was used as an incentive for participation. For participants who were recruited via flyers, entry into a raffle for a \$50 gift card was an incentive for participation. Participants who endorsed being in a current romantic relationship were asked to complete an additional 2.5-hour assessment in a laboratory setting with their current partners, including additional measures not utilized in the current study. For this second phase, participants were given the choice of research credit hours or \$20 as an incentive to participate. All partners of the participants received \$20 for their participation.

In the laboratory assessment, participants and their significant others were given informed consent and introduced to the laboratory. Following this procedure, baseline cortisol samples were collected from both partners. Dyads were then separated to prepare and complete a speech task, modeled after the Trier Social Stress Task (Kirschbaum, Pirke, & Hellhammer, 1993), regarding the strengths and weaknesses of their current relationship. Given the variability in cortisol reactivity during stress-inducing laboratory tasks noted in the extant literature, it has been suggested that measured stress reactivity depends on the nature of the stressor, with support that performance tasks that include a social evaluation component significantly elevate stress reactivity (see Dickerson & Kemeny, 2004). Therefore, participants were told that their speeches were to be watched live by a research assistant and would be evaluated by relationship experts at a later time. Fifteen minutes following the speech task (i.e., 25 minutes following the beginning of the 10-minute stressor), cortisol samples were obtained from participants.

## CHAPTER 3

### RESULTS

#### Data Preparation

The original sample of participants in romantic relationships was 62. A total of five participants were excluded from the present analyses due to age restrictions on young adulthood ( $n = 2$ ), relationship status ( $n = 1$ ), and missing cortisol data ( $n = 2$ ). Thus, the final sample included 57 heterosexual participants in a current romantic relationship. To assess sample size with the present analyses, a power analysis was computed and indicated a sample of 74 participants would be ideal; however, this dataset was collected prior to the development of the current study and thus it is acknowledged that some analyses may be underpowered.

In order to assess stress reactivity to the speech task, cortisol samples collected prior to the task and the sample taken fifteen minutes following the conclusion of the speech task were used in the present analyses. The Shapiro-Wilk test was used in SPSS version 22 to determine if cortisol reactivity scores fell within a normal distribution. It was determined that the scores were skewed (Cortisol sample baseline  $S-W = .51, p < .001$ ; Cortisol sample following stressor  $S-W = .62, p < .001$ ). Therefore, cortisol reactivity scores were first log-transformed to correct for non-normality. Area under the curve (AUC) computation was completed via Microsoft Excel. AUC is formulated from the area of a trapezoid and has been utilized extensively in endocrinology research (Pruessner, Kirschbaum, Meinlschmind, & Hellhammer, 2003).

### Primary Analyses

Possible covariates were examined in the present study. Independent samples *t*-tests were conducted to assess potential gender differences within the variables of interest; results revealed that there was a significant gender difference in childhood experiences of emotional abuse (Levene's Test for Equality of Variance;  $F = 4.88, p = .03$ ;  $t(28.25) = 2.02, p = .05$ ). One-way ANOVAs were examined to determine if there were racial differences within the variables of interest; no significant differences emerged. One-way ANOVAs were additionally used to determine if there were differences within the variables of interest due to current dating relationship length. Results indicated that experiences of CEA differed significantly by the current dating relationship length [ $F(25, 31) = 2.23, p = .02$ ]. Therefore, dating relationship duration and gender were included as covariates in subsequent regression analyses.

To assess the relationship between CEA and self-reported dating aggression and victimization, bivariate correlations were examined. Childhood experiences of emotional abuse were positively correlated to later dating victimization in young adulthood ( $r = .262, p = .047$ ). Although experiences of childhood emotional abuse were not significantly related to later aggression in dating relationships ( $r = .222, p = .094$ ), the results were in the expected direction. However, when the aforementioned covariate variables were included, partial correlations indicated that experiences of CEA were not correlated to later dating victimization in young adulthood ( $r = .18, p = .18$ ) or later dating aggression ( $r = .134, p = .33$ ). Young adult experiences of victimization and aggression in dating relationships were significantly correlated ( $r = .81, p < .001$ ) in the partial correlation analyses. Cortisol reactivity was not significantly related to any of the aforementioned variables (emotional abuse  $r = -.08$ ; dating victimization  $r = -.16$ ; dating aggression  $r = -.10$ ). Although results indicated that experiences of CEA were not correlated with later dating aggression or victimization in young adulthood after controlling for

relationship duration and gender, it was still plausible that cortisol stress reactivity moderated these relationships. Specifically, and in line with the hypotheses of the current study, for those that exhibited either high or low cortisol stress reactivity, the relationship would be significant. Therefore, moderation results were conducted.

Moderation analyses were completed through the PROCESS macro (Preacher & Hayes, 2008) in SPSS version 22. Moderation analyses via the PROCESS macro use bootstrapping methods to determine interactions. Significant interactions are indicated when the 95% confidence intervals (CI) do not include zero. All variables were centered prior to analyses, and participant gender and relationship duration were included as covariates. The PROCESS macro interprets significant interactions at one standard deviation above and below the mean for the moderator variable, so both hypoactive and hyperactive cortisol reactivity in the sample were assessed as potential moderating effects. For brevity, these levels are referred to as high (1 SD above mean), average (mean), and low (1 SD below mean) levels of cortisol reactivity.

Results indicated that although there was not a direct effect between CEA and later adult dating aggression ( $t = 1.19, p = .24, 95\% \text{ CI} = -.11, .45$ ), there was a crossover interaction (see Figure 2), as cortisol reactivity was a significant moderator in the association between CEA and later young adult dating aggression ( $F(5, 51) = 3.58; p = .008; R^2 = .26$ ). These results are presented in Table 2. In probing main effects, low cortisol reactivity accounted for a significant proportion of the variance in the relationship between emotional abuse in childhood and later young adult dating aggression ( $t = 2.88, p = .006, 95\% \text{ CI} = .22, .12$ ); effects were not significant at average ( $t = 1.19, p = .24, 95\% \text{ CI} = -.11, .45$ ) and high ( $t = -1.65, p = .10, 95\% \text{ CI} = -.85, .08$ ) levels of cortisol reactivity.

A similar pattern of results was found in predicting dating victimization. Specifically, results did not show a main effect of CEA ( $t = 1.49, p = .14, 95\% \text{ CI} = -.07, .46$ ), but revealed a significant moderating effect of cortisol reactivity in the relationship between CEA and later young adult dating victimization ( $F(5, 51) = 3.09, p = .02, R^2 = .23$ ), as shown in Table 3. Low levels of cortisol reactivity significantly moderated the relationship between experiences of CEA and later adult dating victimization ( $t = 2.57, p = .01, 95\% \text{ CI} = .13, 1.07$ ); this effect was not significant for high ( $t = -.97, p = .34, 95\% \text{ CI} = -.64, .23$ ) or average ( $t = 1.49, p = .14, 95\% \text{ CI} = -.07, .46$ ) levels of cortisol reactivity. As with dating aggression, these results were indicative of a crossover interaction (see Figure 3).

## CHAPTER 4

### DISCUSSION

The purpose of the present study was to assess the relation between experiences of childhood emotional abuse (CEA) and later young adult dating violence in the context of individual cortisol reactivity. The hypotheses of the current study were generally supported. The relationship between experiences of CEA and later young adult dating violence were in the expected directions, but were not significantly correlated. Although these results appear to be inconsistent with a large body of research that indicates experiences of CEA predicts later deleterious relationship outcomes in adulthood (see Reyome, 2010 for a review), moderation analyses (as discussed below) revealed that this relationship was significant for a subset of the sample. Within the child maltreatment and dating violence literature, associations of CEA and later young adult dating aggression as well as victimization have been widely reported (Crawford & Wright, 2007; Messman-Moore & Coates, 2008; Sappington, Pharr, Tunstall, & Rickert, 1997; Zurbriggen, Gobin, & Freyd, 2010). It is likely that childhood experiences of emotional abuse undermine the ability for a child to learn appropriate and healthy strategies to use in close relationships. The parent-child relationship may also serve as a model for how one engages in close relationships with significant others.

However, the partial correlations and main effect relations of CEA to dating violence were not strong in magnitude or consistently at the level of statistical significance in the current study. Moderation analyses indicated individuals' cortisol reactivity affected the relations of CEA to dating violence. Specifically, lower levels of cortisol reactivity, but not higher levels of

cortisol reactivity (relative to the mean level of cortisol reactivity), during a laboratory stress-induced speech task significantly moderated the relation between childhood experiences of CEA and later young adult dating aggression as well as victimization. In other words, the relationship between CEA and later dating aggression and victimization was significant for participants who had lower levels of cortisol reactivity. Therefore, it seems likely that lower levels of cortisol reactivity amplify the risk of later maladaptive relationship outcomes, and specifically dating violence, in young adulthood. It also may be that a lower stress response indicates that an individual does not recognize a stressor. If so, it could be that the individual's physiological stress system is not cuing the individual to a threat and/or danger (in the case of dating victimization) and that an individual's maladaptive behavioral responses in a relationship are less physiologically stimulating (in the case of dating aggression) when compared to individuals who exhibit mean levels or heightened cortisol stress reactivity. Given that the moderating role of cortisol reactivity in the relationship between CEA and later dating aggression as well as victimization had not been previously examined in the extant literature, these ideas warrant further exploration. Of note, it is important to recognize that not all children who experience early adversity, and specifically emotional abuse, go on to exhibit dysregulated cortisol reactivity and we are not able to make causal statements about the relations between CEA and cortisol reactivity in our sample.

Young adult dating victimization and aggression were correlated in the present sample. These findings are consistent with the extant literature that interpersonal violence often involves some instances of perpetration as well as victimization (Linder, Crick, & Collins, 2002). In addition, no gender differences were found in relation to experiences of young adult dating aggression and victimization. These results are also consistent with the growing body of

literature that supports that both genders perpetrate interpersonal violence and can be victimized by their romantic partners (Fang & Corso, 2007; Goldstein, Chesir-Teran, & McFaul, 2008).

It is important to note that the current study had some limitations that may impact generalizability of the findings. The sample in the current study was small in size and the majority of participants were females. In addition, most of the current sample composition was Caucasian and participants were primarily drawn from a large university in the Southeast of the United States. Therefore, given the sample characteristics as well as the sample size, it will be important for future studies to replicate the current findings within a larger, more diverse sample. It will also be beneficial for future studies to utilize multi-informant reports of childhood emotional abuse and young adult dating violence.

Although previous literature has found evidence of hyperactive stress responses in relation to exposure to childhood adversity (Tarullo & Gunnar, 2006), the current study did not support these findings. Instead, the current findings are in line with previous research conducted by Carpenter and colleagues (2007) that indicated adults exhibit lower cortisol reactivity following a stressor in a childhood maltreatment sample. Given that previous research has primarily focused on childhood adversity instead of specific forms of maltreatment and that the participants in the present study did not endorse severe levels of CEA overall, it may be that the results of the current study differ from some of the previous results in the literature due to a different sample composition (e.g., childhood maltreatment vs. community samples). Regardless, a hyper- or hypoactive cortisol stress response is an individual difference that can be exhibited in individuals from various backgrounds, under numerous conditions, and the development of cortisol stress reactivity is beyond the scope of the current paper. Given that cortisol stress reactivity has not been examined as a moderator within the relationship between CEA and later

dating violence, it may be that hyperactive stress responses do not exert the same influence as lower stress responses in a moderation model. Alternatively, given the aforementioned limitations in the current study (e.g., sample size, sample characteristics), the moderating role of hyperactive stress responses may be particularly salient in other samples that include more variability. Thus, it will be important for future studies to replicate and expand on the current study by obtaining samples that will aid in the generalizability of the current findings (e.g., psychiatric samples, childhood maltreatment samples).

In conclusion, the current study provides evidence that that childhood emotional abuse exposure can increase the risk of interpersonal violence in young adult dating relationships for those that exhibit lower physiological responses to stressors. Given that young adulthood is a salient developmental period for romantic relationships (Arnett, 1998 & 2000), dysfunctional relationship processes during this period may continue or progress into further maladaptation within interpersonal relationships throughout adulthood. Further, although cortisol stress reactivity is not typically examined in clinical practice, an individual's stress response is important to take into consideration, as exposure to stressors throughout the lifespan can be cumulative and have long lasting consequences. The consequential "wear and tear" of the body due to repeated and chronic exposures to stressors, known as allostatic load, has been documented in the literature (McEwen & Seeman, 1999). This chronic physiological distress has been shown to lead to further physiological consequences, later health issues, and psychological impairment (Brody et al., 2013; McEwen & Seeman, 1999). Therefore, it is important to understand the underlying mechanisms, under what conditions, and for whom childhood adversity impacts later interpersonal functioning in young adulthood as well as throughout the lifespan.

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Table 1

*Descriptive statistics and partial correlations*

	<i>M</i>	<i>SD</i>	Emotional Abuse	Victimization	Perpetration
Emotional Abuse	7.59	2.73			
Victimization	2.60	2.78	.18		
Perpetration	3.26	3.02	.13	.81**	
Cortisol Reactivity	2.03	.58	-.08	-.16	-.10

Note. \* $p < .05$ ; \*\* $p < .01$

Table 2

*Moderation analyses of CEA and cortisol reactivity on dating aggression*

Model	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>	LLCI	ULCI
Constant	1.52	1.85	.82	.42	-2.21	5.24
Cortisol reactivity	-.82	.66	-1.25	.22	-2.14	.50
CEA	.17	.14	1.19	.24	-.11	.45
CEA X Cortisol Reactivity	-.96**	.34	-2.81	.01	-1.64	-.27
Model Summary	<i>R</i>	<i>R</i> <sup>2</sup>	<i>F</i>	df1	df2	<i>p</i>
	.51**	.26	3.58	5	51	.007
CEA X Cortisol Reactivity		<i>R</i> <sup>2</sup> Change	<i>F</i>	df1	df2	<i>p</i>
		.11**	7.91	1	51	.007

Note. CEA = Childhood emotional abuse; \**p* < .05; \*\**p* < .01

Table 3

*Moderation analyses of CEA and cortisol reactivity on dating victimization*

Model	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>	LLCI	ULCI
Constant	1.41	1.73	.81	.42	-2.07	4.88
Cortisol reactivity	-.96	.61	-1.57	.12	-2.19	.27
CEA	.20	.13	1.49	.14	-.07	.46
CEA X Cortisol Reactivity	-.70*	.32	-2.21	.03	-1.34	-.06
Model Summary	<i>R</i>	<i>R</i> <sup>2</sup>	<i>F</i>	df1	df2	<i>p</i>
	.48*	.23	3.09	5	51	.02
CEA X Cortisol Reactivity		<i>R</i> <sup>2</sup> Change	<i>F</i>	df1	df2	<i>p</i>
		.07*	4.88	1	51	.03

Note. CEA = Childhood emotional abuse; \* $p < .05$ ; \*\* $p < .01$

Figure 1

*Low cortisol reactivity moderates the relationship between childhood emotional abuse and later dating aggression in young adulthood*

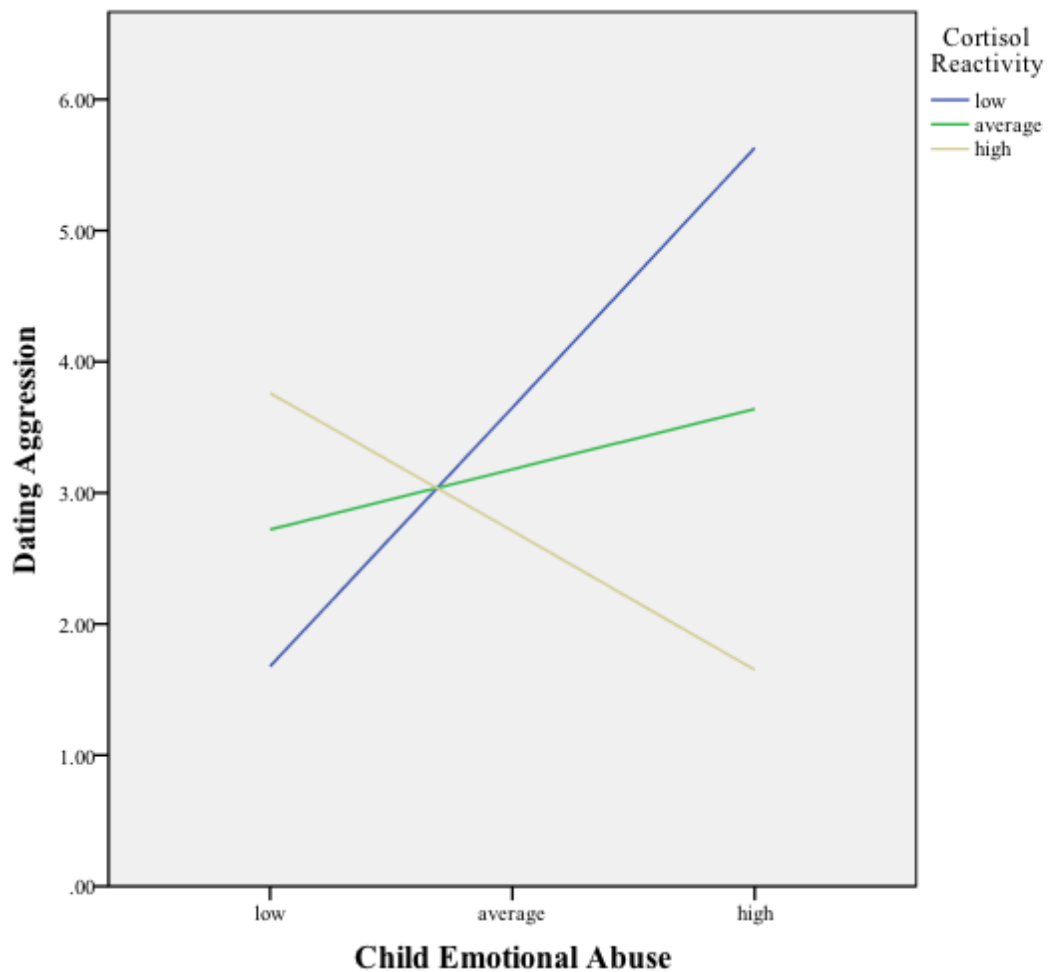


Figure 2

*Low cortisol reactivity moderates the relationship between childhood emotional abuse and later dating victimization in young adulthood*

