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History of Childhood Emotional Abuse Predicts Lower Resting-State High-Frequency Heart Rate Variability in Depressed Women

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Abstract

Childhood emotional abuse impairs emotion regulation and increases risk for major depressive disorder in adulthood. Mounting evidence suggests that decreased resting-state high-frequency heart rate variability, an index of parasympathetic function, represents a transdiagnostic biomarker of emotion dysregulation. We propose that adults with histories of major depressive disorder *and* childhood emotional abuse represent a subpopulation at particularly high risk to exhibit deficits in parasympathetic control. The current report compared resting-state high-frequency heart rate variability across three groups: (1) depressed women who endorsed childhood emotional abuse (N=11); (2) depressed women without childhood emotional abuse (N=19), and (3) never-depressed women without childhood emotional abuse (N=22).Participants completed childhood trauma self-reports and assessment of resting-state high-frequency heart rate variability. ANCOVAs comparing the three groups after controlling for health-related, psychiatric, and respiratory factors were significant. Depressed women with childhood emotional abuse exhibited lower high-frequency heart rate variability than both groups without childhood emotional abuse (*d*'s ranging from 0.81—0.92). Surprisingly, psychiatric factors were non-significant predictors, indicating that childhood emotional abuse may have a unique impact on autonomic functioning. Future research on larger samples is needed to disentangle the relative and synergistic burdens of depression and childhood trauma on physiologic indicators of emotion dysregulation.

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Keywords

depression; childhood emotional abuse; high-frequency heart rate variability

I. INTRODUCTION

The experience of trauma in childhood is thought to impair development of adaptive emotion regulation, and to underlie risk for psychopathology in adulthood (Heim and Nemeroff, 2001). Emotional abuse is by far the most prevalent form of child trauma (Vachon et al., 2015), and has been specifically implicated in heightened stress reactivity (Shapero et al., 2014) and increased risk for major depressive disorder (Gibb et al., 2003; Gibb et al., 2007). A parallel literature has found that depressed adults exhibit deficits in stress-related emotion regulation (Gross and Muñnoz, 1995) and alterations in markers of parasympathetic nervous system function, such as decreased resting-state levels of high-frequency heart rate variability (Rottenberg, 2007). We propose that among depressed adults, the experience of childhood emotional abuse may help to identify a sub-sample of clients with alterations in high-frequency heart rate variability. Given the high prevalence of childhood emotional abuse among depressed adults and its negative impact on depression treatment outcome, clarifying this link has implications for advancing theoretical models regarding the nature of emotion regulation deficits among depressed adults and personalizing depression interventions.

Mounting evidence supports the contention that decreased resting-state high-frequency heart rate variability represents a biomarker of emotion dysregulation as well as psychopathology risk (Beauchaine, 2015). High-frequency heart rate variability, a measure of variation in time between heart beats, indexes parasympathetic input on cardiac function through the vagus nerve. The vagus nerve connects brain regions implicated in emotional reactivity and regulation (Thayer et al., 2012) with peripheral organs, including the heart. As a counterpart to sympathetic excitatory inputs, the parasympathetic ‘vagal brake’ has an inhibitory effect on heart rate. In the absence of stressors, when parasympathetic input dominates, higher resting-state high-frequency heart rate variability indicates greater perceived safety and emotional flexibility. In contrast, lower high-frequency heart rate variability is hypothesized to reflect decreased parasympathetic activation and control, which may lead to more rigid or exaggerated responses to future stressors and diminished emotion regulation capacity (Porges, 2007).

Childhood trauma exerts lasting effects on the central nervous system, impairing the body’s capacity to flexibly regulate emotional responses to environmental demands (Heim and Nemeroff, 2001). The experience of trauma has been associated with decreased high-frequency heart rate variability among adults with posttraumatic stress disorder (PTSD) - indicating that these populations indeed have lower parasympathetic flexibility and control (Cohen et al., 1997; Dale et al., 2009). Initial evidence examining the impact of *childhood* trauma supports similar decreases in high-frequency heart rate variability among adolescent girls (Miskovic et al., 2009). Given that the experience of childhood trauma predicts poor adjustment in adolescence (Mills et al., 2013), risk for adult psychopathology in general

(Kessler et al., 2010; MacMillan et al., 2001), increased risk for adult depression in particular (Gibb et al., 2007), and poorer depression prognosis (including worse treatment response and greater recurrence; Nanni et al., 2012), identifying enduring physiological effects of childhood abuse that may impair adaptive emotion regulation among adults with a history of depression is warranted.

The experience of childhood emotional abuse is common (prevalence estimate 36%) compared with childhood physical abuse 8% and sexual abuse 2% and neglect 4% (Kessler et al., 2010; Stoltenborgh et al., 2012). Additionally, mounting evidence indicates that depression is more strongly associated with the experience of childhood emotional abuse as compared with these other types of childhood trauma (e.g., Gibb et al., 2003; Gibb et al., 2007; for recent metaanalysis see Infurna et al., 2016). Because childhood emotional abuse involves family members directly expressing hurtful statements that make the child feel unloved or unwanted (Bernstein et al., 2003), the experience of childhood emotional abuse may specifically contribute to the development of insecure attachments as well as aspects of cognitive vulnerability implicated in depression risk (Rose and Abramson, 1992). Teasing apart these childhood risk factors is made difficult, however, by the fact that emotional abuse is often comorbid with other less common forms of childhood abuse or neglect (Dong et al., 2004). Regardless, the experience of childhood emotional abuse alone is sufficient to predict broad and potent effects on child psychiatric and behavioral outcomes (Vachon et al., 2015). These data highlight the importance of evaluating the impact that childhood emotional abuse may have on stress-related physiology in depression, such as indicators of parasympathetic activity indexed by measures of high-frequency heart rate variability.

Research on the link between childhood emotional abuse and alterations in high-frequency heart rate variability in depressed populations is limited. Although meta-analytic findings support a significant association between major depressive disorder and decreased high-frequency heart rate variability (Kemp et al., 2010; Koenig et al., 2016; Rottenberg, 2007), the impact of childhood emotional abuse has not been emphasized. One study linked global reports of lifetime trauma with decreased high-frequency heart rate variability among depressed women, compared to non-depressed women and depressed women without a lifetime trauma history (Cyranowski et al., 2011). Further, a history of childhood emotional abuse specifically predicts impairments in stress-related emotional and cardiovascular reactivity among depressed mothers (Cyranowski et al., 2009). The current report seeks to extend these findings by evaluating whether a history of childhood emotional abuse is associated with specific deficits in emotion regulation (decreased high-frequency heart rate variability), in a sample of women with or without a history of major depressive disorder.

We hypothesized that women with a history of depression *and* childhood emotional abuse would exhibit decreased resting-state high-frequency heart rate variability, relative to women with a history of major depressive disorder but no childhood emotional abuse. For comparison, we also included a group of never-depressed women without childhood emotional abuse. Models were run controlling for the known effects of health-related factors (age, body mass index, and smoking status) on high-frequency heart rate variability (Antelmi et al., 2004; Koenig et al., 2014). We also ran additional tests to examine the

robustness of associations after controlling for the influence of psychiatric and respiratory factors.

2. METHODS

2.1. Participants

The initial study included 41 women with a history of major depressive disorder, who were recruited from a larger NIMH-funded randomized controlled trial testing brief psychotherapeutic treatments for depressed mothers with psychiatrically-ill children (Swartz et al., 2016). For a comparison group, we also obtained data from a sample of 25 age-matched mothers without a history of major depressive disorder or other psychiatric disorders ("controls"). Depressed mothers were recruited from child psychiatric treatment clinics and community advertisements; controls were recruited via community advertisements. Participants were excluded for current substance abuse, history of psychotic or bipolar disorders, unstable medical conditions, and active suicidal ideation. Women taking medications known to influence high-frequency heart rate variability ($n = 10$) and those with abnormal ECG data were removed from the current analyses ($n = 3$: arrhythmia, premature ventricular contraction, and one extreme outlier, $z > 2.5$). Because only one of the remaining 23 controls reported a history of childhood emotional abuse, this participant was also excluded, leaving a final analysis sample of 22 controls without childhood emotional abuse, 19 depressed women without childhood emotional abuse, and 11 depressed women with a history of childhood emotional abuse. See Table 2. The University of Pittsburgh Institutional Review Board approved all study procedures.

2.2. Measures

Current and lifetime psychiatric diagnoses were assessed using the Structured Clinical Interview for DSM-IV (First et al., 1995). Demographic information, lifestyle characteristics (smoking status, body mass index) and antidepressant use were also assessed during this interview. Participants reported on their current anxiety and depressive symptoms during the laboratory assessment via the Patient-Reported Outcomes Measurement Information System, 7-item anxiety scale (PROMIS, $\alpha = .92$; Pilkonis et al., 2011) and the 16-item Quick Inventory of Depressive Symptomatology (QIDS, $\alpha = .86$; Rush et al., 2003).

Participants reported their history of childhood abuse and neglect via the 28-item Childhood Trauma Questionnaire (CTQ, Bernstein et al., 2003). Each item is rated on a 5-point, Likert-type scale: 1= *never true,* 2 = *rarely true,* 3 = *sometimes true,* 4 = *often true,* 5 = *very often true.* Subscale scores were calculated by summing responses; established thresholds reflecting moderate abuse were used to create five dichotomous variables: presence versus absence of moderate emotional abuse (≥13), physical abuse (≥10), sexual abuse (≥8), emotional neglect (≥15) or physical neglect (≥10). Internal reliability was strong: $\alpha = .92$, $\alpha = .75$, $\alpha = .96$, $\alpha = .90$, and $\alpha = .86$ respectively. The CTQ has demonstrated excellent psychometric properties in clinical and nonclinical samples, including high levels of criterion-related validity with therapists' ratings of abuse (Bernstein et al., 2003).

The MindWare BioNex and HRV 2.16 software (MindWare Technologies, Gahanna, OH) were used to measure and process ECG and respiratory data. High-frequency heart rate variability was assessed via ECG signals sampled continuously at 1000 Hz with three spot electrodes placed in a Lead II configuration (right clavicle, right and left lower rib-cage). R-wave markers in the ECG signal were processed with artifact detection algorithms, with suspected artifacts corrected manually. Each 1 -minute segment was inspected visually to ensure accurate program R-wave detection. On rare occasions, suspected artifacts were corrected manually (which occurred in < 1% of all beats). Data segments with multiple irregularities that called into question identification of beats (such as those associated with movement artifacts) were removed from analyses (this also occurred in < 1% of available data). After data cleaning was complete, an artifact corrected IBI series was generated, processed using interpolation at 33hz and then detrended (Berntson et al., 1995). A frequency-domain procedure was used to calculate heart rate variability. More specifically, in a frequency-domain approach, the heart rate time series is segregated into separate components (e.g., high-frequency vs. low frequency) according to non-overlapping bandwidths. Thus, frequency is represented on the x-axis, heart rate variability on the y-axis. Heart rate variability was measured within the 0.12 - 0.40 Hz bandwidth in the current study. Fast Fourier transformations (in ms^2/Hz) were then applied to the residuals to derive the spectral distribution. Finally, spectral-power analysis was used to identify power values in the 0.12 - 0.40 Hz spectral bandwidth (ms^2) to indicate high-frequency heart rate variability values per minute (see Allen et al., 2007; Stein and Kleiger, 1999). Respiration was simultaneously assessed with a continuous transducer sampled at 5 Hz with the HRV 2.16 software. Breaths per minute were calculated by the software and visually confirmed. High-frequency heart rate variability and breaths per each of the six, one-minute segments were calculated individually, and then averaged to provide a measure of resting-state high-frequency heart rate variability (see Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology, 1996). The decision to assess heart rate variability in one-minute segments was based on two opposing concerns. One the one hand, one-minute epochs are not large enough to risk violating the stationary assumption of spectral analysis. Conversely, one-minute segments are large enough to adequately capture adults' typical respiration rate (Berntson et al., 1997), which is necessary as heart rate variability is intrinsically linked with respiratory cycle. Indeed, accurately controlling for the impact of respiration is critical given their inverse relation (respiration increases heart rate variability decreases) (Cooke et al., 1998; Stark et al., 2000). We calculated the impact of respiration by regressing respiration on high-frequency heart rate variability separately for each of the six one- minute segments, then taking the average of the six predicted residuals.

2.3. Procedures

Participants were instructed to refrain from caffeine and tobacco for three hours and alcohol for 48 hours prior to the laboratory visit. First, informed consent was obtained. Clinical interviews were completed prior to the physiological assessment, during which participants were seated in comfortable chairs and physiological sensors were placed. After completing questionnaires during a 10-minute habituation period, participants were instructed to rest quietly (without speaking) for six minutes, providing a measure of resting-state high-frequency heart rate variability.

2.4. Data Analysis

Three analysis of covariance (ANCOVA) models were run to test the primary hypothesis: that women with a history of both major depressive disorder and childhood emotional abuse (n=11) would exhibit the lowest levels of resting-state high-frequency heart rate variability, compared to both depressed women (n =19) and controls (n =22) without a childhood history of emotional abuse. First, a one-way ANCOVA compared resting-state high-frequency heart rate variability among the three groups after controlling for key health-related factors identified in the broader high-frequency heart period variability literature (namely, age, body mass index, and smoking status). A second ANCOVA controlled for psychiatric variables identified in the literature (lifetime history of PTSD, current depressive symptoms, current anxiety symptoms, on/off antidepressants). Finally, a third model examined whether results were maintained after accounting for variance in high-frequency heart rate variability due to respiration rate (utilizing residual scores). Post-hoc comparisons of group means were conducted via Tukey's HSD (honest significance difference) tests.

3. RESULTS

Descriptive physiological data related to average heart rate variability, heart rate and respiration rate by group are provided in Table 1. Intra-class correlations of high-frequency heart rate variability across the six 1-minute measures was high (ICC = .966), as were those for heart rate (ICC = .995) and respiration rate (ICC = .959).

Demographic and clinical characteristics across the three groups are presented in Table 2. ANOVAs and Chi-square tests were run to test for group differences in demographic, health-related, and psychiatric variables with controls as the reference group. The three groups did not differ on race, education level, marital status, body mass index, or history of smoking cigarettes. As would be expected, the depressed groups reported higher depressive and anxiety symptoms than controls, as well as higher rates of antidepressant use. There was a significant difference in age, with control women being older than depressed women with a history of childhood emotional abuse. Finally, there was a group difference in annual income, with control women being the highest earners (median household annual income between $75,000-$99,000) followed by depressed women without childhood abuse (median $40,000-$49,999) and then depressed women with a history of emotional abuse (median $10,000-$19,999). However, because income did not represent an a priori control and income variability was not independently associated with high-frequency heart rate variability, $F(1, 51) = 0.43$, $p = .52$, $\eta_p^2 < .01$, income was not included as a covariate.

The ANCOVA model controlling for established health-related predictors of high-frequency heart rate variability (age, body mass index and smoking), was significant, $F(5, 51) = 4.04$, $p = .004$, $\eta_p^2 = .31$. Group was a significant predictor, $F(2, 51) = 3.381$, $p = .043$, $\eta_p^2 = .13$. As anticipated, depressed women with childhood emotional abuse exhibited decreased high-frequency heart rate variability ($M = 4.92$, $SE = 0.34$) compared to depressed women without childhood emotional abuse ($M = 5.95$, $SE = 0.28$; Tukey's HSD $p < .001$; Cohen's $d = 0.92$), or controls ($M = 5.89$, $SE = 0.27$; Tukey's HSD $p = .001$; $d = 0.81$). In contrast, the two groups of women without a history of childhood emotional abuse did not significantly differ (Tukey's HSD $p = .824$). See Figure 1 for a scatterplot of these results. Of the covariates in

the model, body mass index [$F(1, 51) = 10.17$, $p = .003$, $\eta_p^2 = .18$] and age [$F(1, 51) = 4.50$, $p = .04$, $\eta_p^2 = .09$] had significant effects, while smoking status did not [$F(1, 51) = 1.78$, $p = .189$, $\eta_p^2 = .03$].

The follow-up ANCOVA controlling for both health-related factors *and* psychiatric factors maintained significance [$F(9, 51) = 2.24$, $p = .039$, $\eta_p^2 = .33$]. Group continued to predict unique variance in high-frequency heart rate variability [$F(2, 51) = 3.24$, $p = .049$, $\eta_p^2 = .14$], with comparisons yielding an identical pattern of results. Specifically, depressed women with childhood emotional abuse displayed lower high-frequency heart rate variability than the other two groups. None of the psychiatric predictors had significant independent effects (lowest $p = .295$).

To test the robustness of this finding, a third model was run after regressing out variance in high-frequency heart rate variability due to respiration. We continued to covary for health-related factors in this model but did not include the non-significant psychiatric covariates. The model was significant [$F(5, 51) = 4.24$, $p = .003$, $\eta_p^2 = .32$], and group status continued to predict the high-frequency heart rate variability residuals [$F(2, 51) = 3.51$, $p = .038$, $\eta_p^2 = .14$].

Depressed women with childhood emotional abuse exhibited significantly decreased high-frequency heart rate variability compared to depressed women without emotional abuse (Tukey's HSD $p = .004$, $d = 0.90$) or controls (Tukey's HSD $p = .005$, $d = 0.87$), even after controlling for both respiratory and health-related factors.

While childhood emotional abuse frequently co-occurred with other forms of abuse or neglect in the current sample (i.e., in 9 of 11 cases), the low incidence of the other individual trauma subtypes [physical abuse (n=4), sexual abuse (n=5), emotional neglect (n=10), and physical neglect (n=5)] prevented post-hoc analyses to test unique impacts of other forms of childhood trauma. However, we did conduct a post-hoc analysis to evaluate whether history of *any* childhood trauma would impact women's resting-state high-frequency heart rate variability. Resting-state high-frequency heart rate variability levels were compared among women with a history of depression and *any* childhood trauma (n= 13), women with a history of depression but not childhood trauma (n =17) and control women with no childhood trauma history (n =18). (Given the small cell size, 4 control women with a history of *any* childhood trauma were removed from this analysis.) Re-running the original model covarying for health-related predictors, we found that while the overall ANCOVA model was significant [$F(5,48) = 2.90$, p =.024, $\eta_p^2 = .26$], the group effect of *any* childhood trauma history on high-frequency heart rate variability did not reach statistical significance [$F(2,48) = 1.32$, p =.28, $\eta_p^2 = .06$].

4. DISCUSSION

We examined whether a history of both major depressive disorder and childhood emotional abuse would account for variance in resting-state high-frequency heart rate variability, a global indicator of parasympathetic function with potential impacts on emotion regulation. As anticipated, women with a history of *both* depression and childhood emotional abuse

exhibited significantly decreased resting-state high-frequency heart rate variability. Importantly, psychiatric factors including current depression and anxiety severity and diagnosis of PTSD were *not* significant predictors of high-frequency heart rate variability in the model. This pattern indicates that childhood emotional abuse may be a stronger predictor of parasympathetic control. Further, this finding aligns with our previous research, that found that a lifetime trauma history was associated with decreased high-frequency heart rate variability among depressed women (Cyranowski et al., 2011), and that depressed mothers with a history of childhood emotional abuse show exaggerated emotional and blood pressure responses to child-focused stressors (Cyranowski et al., 2009).

The impact of childhood emotional abuse on depressed mothers' resting-state high-frequency heart rate variability is significant, as is the fact that the severity of *current* depression/anxiety symptoms did not significantly predict high-frequency heart rate variability in the current sample (which is consistent with Koenig et al, 2016, but not with Kemp et al, 2010). Prior work has also linked PTSD diagnosis among adults (indicated experience of trauma in adulthood) with lower heart rate variability (e.g., Cohen et al., 1997; Sack et al., 2004). Thus, the lack of utility of PTSD, depression and anxiety severity for predicting heart rate variability in the current model is striking and may indicate that the impact of emotional abuse *in childhood* is e particularly salient to emotion regulation in adulthood. Moreover, alterations in parasympathetic tone, indicating emotion dysregulation, also has implications for adult interpersonal functioning. That is, research has established that both depression (Beach et al., 1990) and trauma history (Reyome, 2010) negatively impact interpersonal functioning. Parallel work also supports the assertion that decreased high-frequency heart rate variability levels are linked with poorer interpersonal functioning, which may represent a key link across associations among lifetime depression, low social support, and risk for coronary disease (Smith et al., 2011; for review see Smith et al., 2004). These findings align with a growing body of research implicating the developmentally-potent consequences of early trauma across multiple physical and emotional adult health outcomes (Nusslock and Miller, 2016).

Maternal childhood trauma history has also been implicated in the intergenerational transmission of depression risk - particularly for women. Specifically, in a large, population-based Finnish study, *female offspring of mothers who had experienced childhood trauma* were significantly more likely to develop adult mood disorders (Santavirta et al., 2017). The current finding, indicating decreased parasympathetic function specific to mothers with a history of depression and childhood emotional abuse, may help to further elucidate potential mechanisms underlying the intergenerational transmission of depression risk. Future research is needed, however, regarding the nature and direction of relationships among maternal impairments in parasympathetic function, stress responsivity and parenting, and the impact these may have on child development and depression risk.

The current results have several potential implications for clinicians. As noted above, decreased levels of parasympathetic activity displayed among depressed women with histories of childhood emotional abuse likely relate to observed impairments in regulating physical and emotional responses to stress (Cyranowski et al, 2009). Decreased high-frequency heart rate variability, as an index of emotion regulatory capacity, may not only

contribute to depressive episode recurrence, but also undermine positive interpersonal functioning, support-seeking and problem-solving, which may thereby contribute to further "stress generation" observed among women with depression histories (Hammen, 2016). Unfortunately, our small sample size limited the ability to examine how depressed mothers with lower levels of high-frequency heart rate variability faired in response to interpersonal therapy or brief supportive therapy (see Swartz et al., 2016 for primary study outcomes). Childhood trauma has, however, previously been associated with poorer depression prognosis. For example, women with a childhood trauma history are more likely to experience chronic or recurrent episodes that are more treatment-resistant (for review see: Nanni et al., 2012). Taken together, the current results highlight the need for future research that tests whether specific treatments are more effective for this depressed sub-population, and to explore ways to 'personalize' depression treatments.

To facilitate such personalized treatment approaches, clinicians would be wise to consider incorporating routine assessment questions (or surveys) regarding the experience of childhood emotional abuse into standard intake protocols. In general, childhood histories of physical and sexual abuse are more widely assessed and more easily identified by clinicians and clients alike. The insidious nature of childhood emotional abuse may make it easier for clinicians to overlook, and far less likely for clients to spontaneously report, at intake assessment. Thus, use of a standardized self-report scales and/or direct assessment of childhood experiences of emotional abuse (such as parents/guardians repeatedly calling one names, saying hurtful things, or making one feel hated or unwanted) may be particularly important to include in routine assessments of clients with a history of major depressive disorder.

Our findings also introduce interesting theoretical questions regarding the temporal association between high-frequency heart rate variability and depression. Decreased high-frequency heart rate variability is now considered a transdiagnostic biomarker of emotion dysregulation as well as psychopathology (Beauchaine, 2015). Meta-analyses support that among adults and adolescents, decreased vagal activity is at least a correlate of ongoing depression (Kemp et al., 2010; Koenig et al., 2016; Rottenberg, 2007), with some research to suggest that lower levels of high-frequency heart rate variability may precede depression onset (Jandackova et al, 2016). The cross-sectional and retrospective nature of the current study limits our capacity to examine these critical questions. Thus, goals for future research will be to clarify the extent to which decreases in high-frequency heart rate variability may develop in response to childhood emotional abuse, and whether decreases in high frequency heart rate variability are implicated in depression etiology and/or represent a 'scar' or residual feature of a particular subtype of depression (e.g., Bylsma et al., 2014).

Limitations of the current study include the small sample size and absence of a group of never-depressed women who experienced of childhood emotional abuse. We would note that the select nature of our control condition (which excluded individuals with *any* history of mood or anxiety disorders) likely contributed to this lack of variability among controls, and undermined our ability to utilize alternate statistical approaches, such as factorial ANOVA designs that could directly assess potential Depression Group x Childhood Emotional Abuse interactions. Future studies that incorporate larger, more representative samples of non-

depressed participants (who vary in their experience of childhood emotional abuse) will be needed to replicate and extend this report. We also note the potential limitations inherent to the reliance on retrospective reports of childhood trauma (Monroe, 2008), particularly with the goal of establishing temporal precedence and directional effects between childhood trauma, lifetime depression, and high-frequency heart rate variability. Future longitudinal work will be needed to clarify the temporal dynamics of these potentially interacting risk factors. Further, although we covaried for multiple health, psychiatric and respiratory factors implicated in prior research, our models did not account for other potential sources of influence (such menstrual cycle, physical activity level, assessment time, other non-psychiatric medication intake, or attachment style). Future research examining these additional factors would be warranted. Finally, depressed women included in the current report were mothers of school-aged children (who also met criteria for at least one lifetime internalizing disorder). Therefore, the findings may not generalize to other populations. Future studies will be needed to determine whether the associations found extend to other females or depressed male samples, and to further elucidate potential mechanisms underlying these relationships.

History of childhood emotional abuse was a stronger predictor of high-frequency heart rate variability than history of *any* childhood trauma in this sample. The pattern of childhood trauma prevalence obtained in the current sample mirrors prior findings (Kessler et al., 2010; Stoltenborgh et al., 2012), in that childhood emotional abuse was most common but frequently co-occurred with other less prevalent trauma subtypes (such as sexual or physical abuse). The low rates of other abuse and neglect subtypes prevented us from comparing the unique individual (or additive) impacts of various childhood trauma subtypes. Taken together, however, the current results suggest that a self-reported history of childhood emotional abuse *is itself* a useful predictor of emotion dysregulation in adulthood. Given World Health Organization reports regarding the “universality” of childhood emotional abuse - which may be experienced by up to a third of adults globally (Kessler et al., 2010; Stoltenborgh et al., 2012) - the potentially wide spread impact of this risk factor is worthy of further study.

Results of the current report indicate that depressed women with a history of childhood emotional abuse display decreased resting-state high-frequency heart rate variability, an indicator of parasympathetic function that may be associated with emotion dysregulation. Future research replicating this finding is warranted to determine if childhood emotional abuse history accounts for previous reports of diminished high-frequency heart rate variability observed among depressed adults (Kemp et al., 2010; Rottenberg, 2007). Given its prevalence within depressed populations, clinicians should assess for indicators of childhood emotional abuse among adult clients with a history of major depressive disorder, as these clients may benefit from interventions designed to enhance emotion regulation.

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Highlights

- Childhood emotional abuse is common, and implicated in emotion regulation deficits
- Decreased high-frequency heart rate variability may represent a marker of emotion dysregulation
- Women with depression *and* child emotional abuse had low high-frequency heart rate variability
- Therapy for depressed women with childhood emotional abuse should target emotion dysregulation

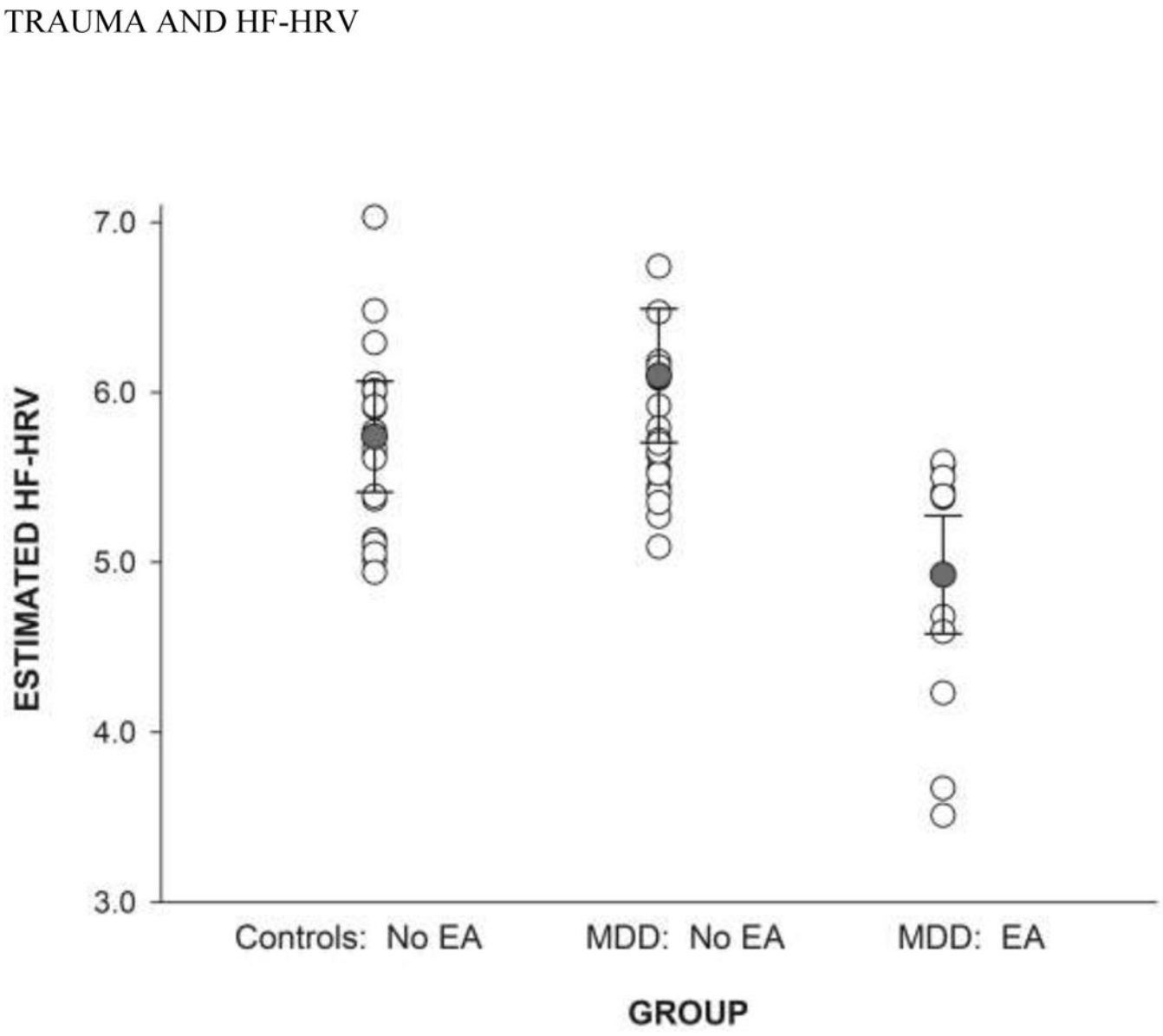

Figure 1.

History of childhood emotional abuse predicts lower resting-state HF-HRV. Estimated marginal means displayed according to group after covarying for health factors (age, BMI, smoking status). Error bars represent standard errors. MDD = lifetime history of major depressive disorder; EA= childhood emotional abuse.

Table 1:

Descriptive Statistics of ECG and Respiratory Data (n = *52)*

	Controls: No EA (n = 22)		MDD: No EA (n = 19)		MDD: EA (n = 11)	
Variable	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Heart Rate	69.71	12.49	72.13	10.19	78.12	8.35
Heart Rate Variability	5.63	1.23	5.78	0.95	4.87	1.26
Respiration	15.74	3.85	15.23	3.09	16.58	1.98

Note: EA: history of emotional abuse in childhood; MDD = women with a history of major depressive disorder

Table 2:

Demographics and Covariates According to Rates of Childhood Emotional Abuse

	Controls		History of Major Depression Disorder				
Variable	**No Emotional Abuse (n = 22)**		**No Emotional Abuse (n = 19)**		**Emotional Abuse (n = 11)**		**F(2, 52)**
Age: mean, *SD* [b]	47.2	4.5	44.4	7.1	40.2	6.7	5.03**
Race: White *n*, %	17	77%	15	80%	10	91%	0.73
Education ≥ Associates: *n*, %	15	68%	12	63%	7	64%	1.74
Married: *n*, %	15	68%	10	53%	4	36%	3.20
Income < $30,000: *n*, % [abc]	1	5%	6	32%	6	55%	12.72***
BMI: mean, *SD*	28.1	6.0	29.3	5.8	32.3	7.5	1.63
Smoke: *n*, %	3	14%	2	11%	4	36%	1.92
Antidepressants: *n*, % [ab]	0	0%	6	32%	3	27%	5.89*
Lifetime PTSD: *n*, %	0	0%	5	26%	0	0%	0.41
PROMIS anxiety: mean, *SD* [ab]	10.5	3.4	17.2	5.8	17.0	6.4	10.66***
QIDS, *n* (%) current MDD [ab]	0	0%	6	32%	3	27%	8.44***

Note: Emotional Abuse= reported history of childhood emotional abuse; Race: all other participants were African American, no-one reported Hispanic ethnicity. Education: achieved an Associates Degree or higher; BMI = Body Mass Index; PTSD = lifetime history of Posttraumatic Stress Disorder; PROMTS: current anxiety symptom levels; QIDS ≥ 9 as clinical threshold for current major depressive disorder (MDD).

[a] Significant group differences between controls and women with MDD history no emotional abuse

[b] Significant group differences between controls and women with MDD and emotional abuse history

[c] Significant group differences between the two MDD groups

* $p < 0.05$,

** $p \leq .01$,

*** $p \leq .001$