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Differential Associations of Childhood Abuse and Neglect with Adult Autonomic Regulation  
and Psychopathology

THESIS

Submitted in partial satisfaction of the requirements

for the degree of

MASTER OF ARTS

In Social Ecology

By

Sarah Katheryn Stevens

Thesis Committee:  
Associate Professor Alyson K. Zalta, Chair  
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Distinguished Professor Julian F. Thayer

2022



## TABLE OF CONTENTS

	Page
LIST OF FIGURES	iii
LIST OF TABLES	iv
ACKNOWLEDGEMENTS	v
ABSTRACT OF THE THESIS	vi
INTRODUCTION	1
METHODS	7
RESULTS	18
DISCUSSION	20
REFERENCES	25

## LIST OF FIGURES

		Page
Figure 1	Path Model for Baseline, Reactivity, and Recovery Epoch	33

## LIST OF TABLES

		Page
Table 1	Results from final structural analyses of path models	34
Table 2	Means, Standard Deviations, and Correlations for CTQ Subscales	35
Table 3	Means, Standard Deviations, and Correlations for MASQ Subscales	36

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## **ABSTRACT OF THE THESIS**

Differential Associations of Childhood Abuse and Neglect with Adult Autonomic Regulation  
and Psychopathology

By

Sarah Katheryn Stevens

Master of Arts in Social Ecology

University of California, Irvine, 2022

Associate Professor Alyson Zalta, Chair

This study assessed whether different types of childhood maltreatment (CM) (i.e., abuse vs. neglect) had differential relationships with heart rate variability (HRV) and baroreflex sensitivity (BRS), two established markers of autonomic functioning. Additionally, this study used a moderated mediation model to investigate the potentially mediating role of HRV in the relationship between CM subtypes and adult psychopathology, and whether these relationships differed in those with high vs. low resting HRV. Secondary analysis was performed using the MIDUS II Biomarker Project dataset. Structural equation modeling was used to assess the relationships between key variables at baseline, as well as reactivity and recovery to stressor tasks. Baseline pathways from abuse and neglect to BRS were nonsignificant, as was the pathway from HRV to psychopathology. Notably, greater abuse was significantly predictive of lower HRV (standardized  $\beta = -.42, p < .01$ ) while greater neglect was significantly predictive of higher HRV (standardized  $\beta = .32, p < .05$ ). Additionally, higher childhood abuse was significantly predictive of greater adult psychopathology (standardized  $\beta = .39, p < .001$ ), but

childhood neglect was not found to be related to adult psychopathology in this sample. Significant relationships between target variables were only found in those with low HRV. Our findings suggest that greater differentiation between abuse and neglect are appropriate in investigations of the impact of CM on adult physical and mental health outcomes. Additionally, our findings contribute further evidence that low HRV may be a transdiagnostic endophenotype for mood-related pathology.

## INTRODUCTION

Childhood maltreatment (CM) is a significant public health concern, with the Center for Disease Control (2022) reporting that 1 in 7 children in the United States have experienced abuse and neglect within the past year alone. Exposure to maltreatment during childhood can influence both biological and psychological development and has been shown to be associated with a wide range of maladaptive physical and mental health outcomes in adulthood (DeBellis & Zisk, 2014), particularly mood disorders such as anxiety and depression (Li et al., 2015). CM is posited to contribute to these later health concerns through a “biological embedding” of the maltreatment, which likely occurs through developmental alterations to the biological systems responsible for responding to stress (Berens, Jenson, & Nelson, 2017; Holochwost et al., 2020). The experience of CM can occur through several different types of exposures such as physical, sexual, and emotional abuse, as well as physical and emotional neglect (Matsumoto et al., 2021). While research attempting to assess the biological and psychological influences of different CM exposures such as abuse and neglect have been extensively performed with children and adolescents (McLaughlin, Weissman, & Bitran, 2019), few studies have directly assessed how biological embedding of these subtypes of CM may play out into specific psychological health outcomes in adulthood (McLaughlin et al., 2019). A better understanding of these processes could assist clinicians in early detection for those at risk for later sequelae and potentially lead to improved treatment of disorders such as anxiety and depression in survivors of CM.

The autonomic nervous system (ANS) is the body’s first line of defense against a detected stressor and is comprised of two branches: the sympathetic nervous system (SNS) (commonly known as the “flight-or-flight” system) and the parasympathetic nervous system

(PNS) (sometimes referred to as the “rest-and-digest” system). The PNS regulates the activity of the SNS through tonic inhibitory control; in response to a stressor, the PNS withdraws and allows the SNS to alter metabolic functioning to meet the demands of an acute stressor through adrenaline and noradrenaline signaling, which quickly increases cardiovascular and respiratory activity as well as blood flow while decreasing the activity of restorative systems such as digestion (Rotenberg & McGrath, 2016). The tonic inhibitory control of the PNS over the SNS is governed primarily by the vagus nerve, which acts as a bidirectional pathway between the brain and most major organs including the lungs, viscera, and heart (Smith et al., 2017; Thayer & Lane, 2000). Because of the regulatory role of the vagus over the body’s first response to stress, measurement of ANS activity has largely centered on cardiovascular markers representing vagal activity, such as heart rate variability (HRV) (Smith et al., 2017) and baroreflex sensitivity (BRS) (LaRovere et al., 2008). HRV is an index capturing beat to beat variation in heart rate (HR) and is widely used as a measure of effective vagal control over the heart (Laborde, Mosley, & Thayer, 2017). Higher HRV is largely known as a marker for a healthier and more adaptive ANS, while lower HRV has been associated with a range of both physical and psychological disorders (Heiss et al., 2021; Jung, Jang, & Lee, 2019; Nas et al., 2020). Baroreflex sensitivity (BRS) measures the activity of the baroreflex, a vagally-regulated process which maintains blood pressure levels through a dynamic feedback loop which lowers heart rate in response to increasing blood pressure (Parati et al., 2001). Impairment in BRS has been observed in patients with depression and anxiety (Rozanski et al., 2005), and has shown to be predictive of cardiovascular disease later in life (Norcliffe-Kaufmann, 2022).

While developmental researchers have repeatedly observed that exposure to childhood abuse and neglect is associated with alterations to ANS functioning, findings have not always

been consistent. The current literature largely supports that those adolescents with a history of CM display increased SNS activity at rest and hyporeactivity in response to acute stress (Gooding et al., 2016; Holochwost et al., 2021; Leitzke, Hilt, & Pollak, 2015; McLaughlin et al., 2014; McLaughlin et al., 2015). However, differences have not always been observed between maltreated youth and healthy controls (Ford et al., 2010; Gordis et al., 2010) and some have even observed hyperreactivity in certain samples (Rinnewitz et al., 2018; Trickett, 2010). One of the reasons for these contradictory findings may be due to meaningful differences in the neurobiological effects of different CM exposure types. Studies using cumulative CM scores have less consistently found differences in ANS functioning (Bakema et al., 2020; Winzeler, K. et al., 2017), and several studies have observed that a history of childhood abuse and neglect result in different biobehavioral outcomes. McLaughlin and colleagues (2019) recently completed a systematic review of the literature surrounding CM and neural development and found that current evidence largely supports a dimensional model of childhood adversity, which posits that early threat-related maltreatment exposures (such as physical, sexual, and emotional abuse) and early deprivation-related exposures (such as physical and emotional neglect) differ in terms of their neurobiological consequences during development. For example, threat-related exposures were found to be more consistently related to decreased limbic structure volumes and increased amygdala reactivity/decreased prefrontal cortex (PFC) activity in response to acute stress, whereas deprivation-related exposures were more likely to be associated with reduced volume and thickness of frontoparietal structures such as the dorsolateral PFC and superior parietal cortex. Given that the vagus nerve acts as a bidirectional messenger relaying information between emotional neurocircuitry and physiological systems which mobilize the body to respond

to a stressor, it may be that these observed structural differences manifest into differential autonomic patterns both at rest and in response to stress.

Investigations of the relationship between CM and autonomic regulation in adulthood are similarly complicated. Evidence widely supports a connection between a history of CM and lower resting HRV in adulthood which is indicative of higher baseline SNS activity (Meyer et al., 2016), as well as impaired vagal regulation in response to acute stress (Dale et al., 2018; Heim et al., 2000). However, most studies have assessed this relationship using clinical samples. To our knowledge, only three studies have investigated how the relationship between CM and adult autonomic regulation may play out in non-clinical samples. A study by Buisman et al. (2018) found meaningful differences in how parents with a history of childhood abuse and neglect responded to resolving conflict with their offspring. Those parents with history of neglect displayed patterns of greater SNS reactivity in response to their child, which were not observed in parents with a history of childhood abuse. A recent population-based study (Bakema et al., 2020) found that cumulative CM exposure scores were associated with impaired BRS and lower resting HRV, but the relationship was no longer significant after adjusting for socioeconomic and demographic characteristics. Another recent study (Bielharz, 2020) using a community sample observed that those with a history of CM had higher resting heart rate (HR) and took longer to return to their resting HR in response to an acute stress task than those with no such history. Notably, no differences in resting HRV were found between those with a history of CM and those without, however an analysis of exposure subtypes showed that those with a history of physical abuse had higher HR and HRV during sleep, which may suggest a prolonged sympathetic response in those with this type of exposure.

Understanding how patterns of autonomic regulation may be dysregulated in those with a history of CM may shed a light on pathways by which vulnerability to psychopathology in adulthood is conferred. Autonomic functioning is known to be associated with affective states, as HRV is widely considered to be representative of emotion regulation and dysregulation (Thayer & Lane, 2000). Specifically, those with low HRV have demonstrated greater difficulties in emotion regulation and higher risk for mood-related psychopathology such as anxiety and depression. As survivors of CM are known to be at risk for the development of such mood disorders later in life, it is important to identify the biological patterns which may contribute to this vulnerability. Adolescent studies have investigated ANS markers as a potential mediator of internalizing and externalizing symptoms, with contradictory results (Kahle et al., 2018), but to our knowledge no studies to date have directly examined the potentially mediating role of ANS dysregulation in the relationship between exposure to maltreatment in childhood and the presence of mood symptomatology in adulthood. As emotion regulation, an affective index so closely associated with ANS functioning, has been observed to mediate the relationship between cumulative CM exposures and adult psychopathology (Cloitre et al., 2018), it becomes even more relevant to clarify the specific role of the ANS to this relationship.

Mounting evidence suggests that low HRV may be an endophenotype for a variety of psychological disorders (Thayer and Lane, 2000), which suggests the possibility of a vulnerability threshold whereby HRV may promote risk for or resilience to mood-related symptomatology. Additionally, a recent study by Fantini-Hauwel and colleagues (2020) observed that difficulties in emotion regulation were only related to depression symptoms in women with low HRV. This raises the possibility of a non-linear relationship between HRV and psychological distress, such that those with low HRV may be at greater risk for the development

of psychological disorders than those with high HRV. While much of the literature continues to investigate these relationships using linear models, examining meaningful differences in those with low HRV and high HRV may aid in better understanding the nature of the relationship between autonomic functioning and psychological distress.

This study had three primary aims: firstly, to investigate whether threat-related CM exposures (such as physical, sexual, and emotional abuse) and deprivation-related CM exposures (such as physical neglect, emotional neglect) were associated with differential autonomic patterns in adulthood both at rest and in response to an acute stressor. Secondly, we sought to investigate the potentially mediating role of vagal tone as measured through HRV in the relationship between CM exposure subtypes and the severity of psychopathology in adulthood. Thirdly, we aimed to assess whether the relationships between these variables differed in those with clinically high HRV and those with clinically low HRV. In order to do this, secondary data analysis was conducted from the second wave of the Midlife Development in the U.S. (MIDUS) study Biomarker Project, which took place at the University of Wisconsin, the University of California Los Angeles, and Georgetown University between 2004 and 2009. We hypothesized that greater severity of abuse-related exposures would be associated with lower HRV and BRS, both at rest and in response to an acute stressor, than neglect related exposures. Additionally, we hypothesized that HRV activity at rest and in response to an acute stressor would mediate the association of CM subtypes with anxiety and depression symptoms in adulthood. Lastly, we hypothesized that the relationships between CM subtypes, HRV, and psychopathology would be stronger in magnitude in those with low HRV than in those with high HRV.

## METHOD

### Procedure

IRB approval was gained at each of the three MIDUS study sites, and informed consent was obtained from all participants prior to study activities. Following the consent process, participants completed demographic information, and were then connected to the electrocardiogram (ECG) for physiological data collection. Two consecutive baseline epochs of five minutes each were first collected while the participant was asked to sit quietly in a chair. Following baseline measurement collection of HRV and BRS, participants were counterbalanced to complete one of two cognitive stressor tasks: the MATH or the Stroop tasks. The MATH task is a mental arithmetic task designed for use in laboratory studies of cardiovascular stress and reactivity. The task involves trials of three different stimuli presentations: a math problem appearing for 2.0 sec, the word “Equals” for 1.5 sec, and a solution to the problem which appears for 1.0 sec. The participant is then asked to press one of two keys on a keypad to indicate whether the presented solution was correct. Participants must answer within the 1.0 sec period, or their response is recorded as incorrect. There are five levels of difficulty for this task, and all participants begin at level 3. Correct initial responses will prompt a step up in difficulty for the remaining presentations, while incorrect initial responses will result in a step down in difficulty. The number of trials varied by the participant’s response times, with all participants completing the task within 4-6 minutes. The Stroop task on the other hand presents the words of four different color names (blue, green, yellow, or red) in a font color that either matches or does not match the name of the color. The name of the color appeared on the screen, and participants were asked to press one of four keys in front of them, which corresponded to the color of the letters of the word, rather than the color name). The rate of stimuli presentation varied as a function of task

performance to standardize the stressfulness of the task among all participants. Those who initially answer accurately were then presented with a more rapid stimuli presentation rate, and those who answered less accurately had a slower rate of stimuli presentation. Participants completed their first cognitive stressor, and afterwards were asked to rest for five minutes quietly, in order to collect recovery measurements. After a five-minute recovery epoch, participants completed the second cognitive stressor task. Following the second cognitive stressor task there was a final five-minute resting period while recovery measurements were collected from the participants. After physiological data collection, participants completed the self-report questionnaires. Following completion of study tasks, participants were compensated for their time. All data collected from this study is publicly available at the following link: <https://midus.colectica.org/>.

## **Participants**

Adults over 18 years of age were recruited from areas surrounding three large universities in the United States, located in Madison, WI, Washington, D.C., and Los Angeles, CA. A total of 1059 participants were enrolled and completed some portion of study activities. For our secondary analysis, participants endorsing a history of cardiovascular disease, heart attack, or currently using a pacemaker were excluded from analysis, as these conditions highly influence autonomic patterns both at rest and in response to acute stress (Laborde et al, 2017). This resulted in a final sample of 967 participants who completed at least some portion of the psychophysiological protocol. Our final sample was 58.4% female ( $n = 564$ ) and 41.6% male ( $n = 403$ ), with a mean age of 55 years ( $SD = 10.64$ ). Our sample identified themselves as 75.9% White ( $n = 734$ ), 2.3% African American ( $n = 22$ ), 0.7% Asian or Pacific Islander ( $n = 7$ ), and

3.3% identifying as ethnically Hispanic ( $n = 32$ ), with the remainder of the sample declining to self-report ( $n = 172$ ).

## **Measures**

**Childhood Trauma Questionnaire (CTQ).** The CTQ is a 25-item self-report questionnaire measuring severity of exposure to childhood maltreatment across five domains (emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect). Participants were asked to report how often a particular statement was true for them during childhood (Ex: “I didn’t have enough to eat”, “I believe that I was physically abused.”) on a scale from 1 to 5, corresponding to descriptive statements ranging from “Never true” to “Very often true”. Each subscale contained five statements, resulting in the potential for scores to range from 5-25 for each of the five domains. Subscales were totaled in accordance with previously established guidelines (Bernstein and Fink, 1998) to assess the severity of exposure to abuse and neglect maltreatment subtypes in childhood.

**Mood and Anxiety Symptom Questionnaire (MASQ).** The MASQ is a 64-item self-report questionnaire measuring the presence of depression and anxiety symptoms over the past week. Four subscales were used to assess anxious symptoms, anxious arousal, depressive symptoms, and anhedonic depression. Participants were asked to endorse how much a particular symptom had bothered them that week from 1 to 5, with the scale corresponding to descriptive ranging from “Not at all” to “Extremely”. The four subscales were totaled in accordance with previously established methods (Watson et al., 1995) to assess the severity of ongoing symptomatology in each domain.

**Heart Rate Variability (HRV).** HRV was collected via a three-lead electrocardiograph (ECG) at a sampling rate of 500 Hz, and the captured high frequency RR intervals (bandwidth 0.15-0.40 Hz, msec<sup>2</sup> units) were used to calculate all indices of HRV. ECG sensors were placed on the left and right shoulders, and on the left lower side of the torso. Root mean square of successive differences (RMSSD) was calculated through measurement of time differences between RR intervals in milliseconds. These values are then squared, the result is averaged, and finally the square root of the total is calculated. RMSSD values were then log transformed to improve normality of distribution. Baseline HRV values were collected during the two five-minute epochs of rest prior to the first cognitive stressor tasks. In order to measure HRV reactivity to the two counterbalanced cognitive stressor tasks, two change scores were created: one by subtracting the participant's baseline RMSSD from their RMSSD following the MATH stressor task (representing the change in RMSSD from baseline to the MATH task), and the other by subtracting their baseline RMSSD from their RMSSD following the Stroop stressor task (representing the change in RMSSD from baseline to the Stroop task). Measurement of HRV recovery following the stressor tasks was calculated similarly, with two change scores created: one by subtracting the participant's RMSSD following the MATH stressor task from the RMSSD of the recovery period following the MATH task (representing the change in RMSSD from the MATH epoch to the recovery epoch), and the other by subtracting their RMSSD following the Stroop stressor task from the RMSSD of the recovery period following the Stroop task (representing the change in RMSSD from the Stroop epoch to the recovery epoch).

**Baroreflex Sensitivity (BRS).** To observe the engagement of the baroreflex in the alteration of blood pressure (BP) through changes in heart rate (HR), both BP and HR were measured during baseline resting, reactivity to acute stress, and recovery from acute stress. HR

was measured as mentioned above using high frequency ECG recording at a sampling rate of 500 Hz, with RR-intervals converted to beats per minute (BPM) units. BP was measured using a Finometer blood pressure cuff which was placed on the participant's middle finger of their non-dominant hand, as well as a cuff placed on the upper arm on the same side as the finger cuff. Indices of BRS were measured using a sequence method approach. Three or more BP values which increase or decrease consistently increasing or decreasing are considered a "sequence" which represents engagement of the baroreflex. These sequences are then used to calculate BRS during each of the collection periods. BRS is calculated by first identifying the corresponding BP and ECG data during each period and subtracting 250ms from all BP times to adjust for the known delay in the Nexfin BP recording monitor. The diastolic records were then removed, the RR-intervals computed, and data occurring during Bad Intervals from both BPs and RRs are removed. Sequences of three or more consecutive BP increases, or decreases were then identified, and the corresponding RR-intervals matched with the BP sequences. Linear regression was then used to estimate the slope of each sequence, and unwanted sequences (those with more than one BP reading within 1 RR interval, any RR interval greater than two seconds) are removed. The mean slope was then created from the remaining sequences. One representative mean slope was created for each epoch: one for the baseline period, one for each of the two cognitive stressor tasks (MATH and Stroop), and one for each of the two recovery periods following the stressor tasks.

### **Statistical Analysis**

The primary goals of this study were to assess whether meaningful differences in abuse and neglect related CM exposures influenced adult autonomic functioning, as well as whether HRV mediated the relationship between severity of CM exposure subtypes and adult

psychopathology. Additionally, this study sought to assess whether those relationships may differ among those with high HRV and those with low HRV. As such, a three separate multiple group structural equation models were performed using R statistical software (R Core Team, 2021) version 4.1.1 “Kick Things” (2021-08-10). These models examined the relationship between abuse and neglect severity at three separate epochs of physiological functioning: at rest using baseline measurements of HRV and BRS (baseline model), in response to the MATH and Stroop cognitive stressors (reactivity model), and during the recovery period following the cognitive stressor tasks (recovery model). In order to examine these relationships among those with high and low HRV, two groups were created using an established transdiagnostic RMSSD value threshold of 25 (Jarczok et al., 2019), with the models structured the same way for both the low and high HRV groups. Age, gender, and hypertensive medication use were assessed as potential covariates of HRV and BRS at the bivariate level and were found to be associated with both HRV and BRS functioning at all time points. These were then included as covariates in all three models as direct predictors of the HRV and BRS latent variables. Full information maximum likelihood was used to estimate values missing within the dataset for all three models. Measurement and structural details for each of the models are presented below. Structural paths for the three models are illustrated in Figure 1. Code for all analyses performed are available upon request.

### *Baseline Model*

In line with McLaughlin and colleagues’ (2019) work supporting a dimensional model of childhood adversity, this study used subscales from the Childhood Trauma Questionnaire (CTQ) to create two latent variables, Abuse (representing severity of childhood threat-related exposures) and Neglect (representing severity of childhood deprivation-related exposure types). The

physical abuse, sexual abuse, and emotional abuse subscales served as the three indicators for latent variable Abuse, and the physical neglect and emotional neglect subscales served as two indicators for latent variable Neglect. As the experience of CM is known to increase the risk for other CM-related exposures and bivariate analyses revealed that subscales of our CTQ measure were highly correlated with one another across our conceptual abuse and neglect domains (see Table 1), we correlated the errors of the two latent variables to account for this relationship.

In considering the physiological variables in our model, we created a latent variable for Baseline HRV which was comprised of the two resting epoch measurements taken at the beginning of the psychophysiological protocol. As only one measurement of BRS was taken during this period, we used this measurement as an observed variable and no latent variable was created. Structural paths from latent variables Abuse and Neglect to physiological markers HRV and BRS were created to assess the relationship between severity of these exposure subtypes and HRV and BRS activity at rest. As measurements for Baseline HRV and BRS are highly related, the errors of these variables were correlated to account for this relationship. Additionally, age, gender, and medication use were set as direct predictors of latent variable Baseline HRV and observed variable baseline BRS in order to adjust for the influence of these potential covariates in the final model.

All four of the MASQ subscales were used as indicators for a single latent variable Psychopathology. As all four subscales demonstrate strong, positive correlations significant at the  $p < .001$  with one another (see Table 2), this approach is supported statistically. A structural path from HRV to latent variable Psychopathology was created in order to assess the potentially mediating role of HRV in the relationship between CM exposure subtype severity and psychopathology in adulthood. CM's relationship with psychopathology is well-established

(DeBellis & Zisk, 2014; Cloitre et al., 2018) therefore we also added direct paths from Abuse and Neglect to our endogenous outcome variable Psychopathology. This model was assessed and found to meet both rank and order conditions proposed by Bollen (1989) and is therefore identified. No further constraints were placed on the model in order to assess the freely estimated parameters of all paths.

As with linear regression models, assumptions are made regarding normality of endogenous variables in the estimation of structural equation model parameter estimates. Because the severity of each of these exposure subtypes are unlikely to take a normal gaussian distribution in the population and our sample included both CM exposed and unexposed individuals, normality of the CTQ subscales was assessed. All CTQ variables were found to be highly right skewed (with values of skewness ranging from .92 - 3.02). Additionally, we anticipated that mood symptomatology was unlikely to assume normal distribution in our sample and skewness was once again assessed for each of these indicators. Once again, our variables were found to be highly right skewed, with values ranging from 1.3-2.15. Skew present within endogenous latent variables is known to influence parameter estimates such that it increases the risk of making a Type I error (Curran, et al., 1996). In order to address this issue, maximum likelihood robust to skew (MLR) estimation was used to assess this model. MLR corrects the inflated standard error values that are known to occur in models which violate normality assumptions by using the Yuan-Bentler method, which scales a constant representing the amount of average multivariate kurtosis present in the model. This constant then serves as a correction factor, which the traditional goodness-of-fit indices are divided by in order to obtain corrected values (Yuan & Bentler, 1994). HRV group assignments were entered among estimation commands with the high HRV group comprised of those with a resting RMSSD of 25 or greater,

and the low HRV group comprised of those with an RMSSD of 24.99 or lower. Pathways were assessed according to the same specifications in each of the two HRV groups.

### *Reactivity Model*

Measurement considerations for the creation of exogenous latent variables Abuse and Neglect, as well as endogenous outcome variable Psychopathology were performed consistently with the baseline mediation model discussed above. The two reactivity change scores served as indicators for the latent variable HRV Reactivity. BRS measurements for the MATH and Stroop epochs were used as indicators for the creation of latent variable Stressor BRS. The errors of latent variables HRV Reactivity and Stressor BRS were correlated to account for the relationship between them. As with the baseline model, age, gender, and medication use were set as direct predictors of latent variables HRV Reactivity and Stressor BRS in order to adjust for the influence of these variables. Structural paths for this model were consistent with the baseline model, with direct paths from Abuse and Neglect to both HRV and BRS, as well as paths from Abuse, Neglect, and HRV Reactivity to Psychopathology. As with the baseline model, MLR estimation was used to assess this model and the model was found to meet both rank and order conditions. No further constraints were placed on the model in order to assess the freely estimated parameters of all paths. HRV group assignments were entered among estimation commands in accordance with the baseline model.

### *Recovery Model*

As with the reactivity model, measurement considerations for the creation of latent variables Abuse, Neglect, and Psychopathology were made in accordance with the baseline model. The two recovery change scores served as indicators for the latent variable HRV

Recovery. BRS measurements were taken during the recovery periods following both the MATH and Stroop tasks, and the measurements for these epochs were used as indicators for the creation of latent variable BRS Recovery. The errors of latent variables HRV Recovery and BRS Recovery were correlated to account for the relationship between them. As with the other models, age, gender, and medication use were set as direct predictors of latent variables HRV Recovery and BRS Recovery in order to adjust for the influence of these variables. Structural paths for this model were consistent with baseline, with paths from Abuse and Neglect to both HRV Recovery and BRS Recovery, as well as paths from Abuse, Neglect, and HRV Recovery to Psychopathology being assessed. As with the baseline and reactivity model, this model was found to meet both rank and order conditions and is therefore identified. No further constraints were placed on the model in order to assess the freely estimated parameters of all paths. MLR estimation was used to assess this model, consistent with baseline and recovery models. HRV group assignments were entered among estimation commands in accordance with the baseline model.

#### *Testing Indices of Model Fit*

In order to assess whether the data supported exploring these relationships in those with high vs. low HRV, the three models were first run both with and without the multiple groups specification and model fit indices were compared. A lower Bayesian information criterion (BIC) value indicates a more suitable fit for the data (Lin et al., 2017) when comparing models, and the three multiple groups models were found to yield lower BICs when compared with their non-multiple group alternative models (baseline BIC of 59052.46 compared with 61624.56, reactivity BIC of 61146.31 compared with 63141.35, and recovery BIC of 61590.05 compared with 63460.46). As such, the multiple groups path analysis was used for all three models.

In order to assess overall fit for the baseline, reactivity, and recovery models, MLR adjusted indices of fit were used, such as the Root Mean Square Error of Approximation (RMSEA), the Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI). While benchmarks for these fit indices are all highly contested (Hu & Bentler, 1999), this study followed the suggested values of a .08 threshold for an acceptable RMSEA value, and a .90 threshold value for the CFI and TLI values as proposed by Browne et al. (1993).

## RESULTS

Results from the baseline model yielded a scaled chi-squared test that was statistically significant:  $\chi^2(55) = 493.14, p < .001$ . While this does indicate that the model may not be the best fit for the data, the chi-square test is known to be sensitive to inflation by large sample sizes (Barrett, 2007). The tests for other indices of fit yielded more acceptable values, with the RMSEA showing an adjusted value of .07, and the CFI and TLI showing values of .94 and .92 respectively. Parameter estimates for the direct structural paths can be found in Table 1. No significant relationships were found between variables in the group with high HRV. In those with low HRV, the paths from latent variables Abuse and Neglect to BRS were not significant, however the pathway from Abuse (standardized  $\beta = -.42, p < .01$ ) and Neglect (standardized  $\beta = .32, p < .05$ ) to baseline HRV were both found to be significant. HRV was not found to be significantly related to Psychopathology. Similarly, Neglect was not found to be predictive of Psychopathology, however the path from Abuse to Psychopathology was significant (standardized  $\beta = .39, p < .001$ ). Indirect effects were additionally assessed, but no significant indirect effects were observed among our variables ( $p > .05$ ).

Results of the scaled chi-square test for the reactivity model were similarly poor, with  $\chi^2(88) = 575.46, p < .001$ . Other overall model fit indices were within acceptable ranges however, with an adjusted RMSEA of .07, a CFI of .93, and a TLI of .91. As with the baseline model, no significant relationships were found between variables in the group with high HRV. In those with low HRV, direct structural paths from latent variables Abuse and Neglect to HRV Reactivity were not significant ( $p > .05$ ), nor was the path from HRV Reactivity to Psychopathology. The path from Neglect to Stressor BRS was not found to be significant, however Abuse was significantly associated with Stressor BRS (standardized  $\beta = -.23, p < .05$ ).

The path from Neglect to Psychopathology was nonsignificant, however the path from Abuse to Psychopathology was once again statistically significant (standardized  $\beta = .35, p < .001$ ). Indirect effects were similarly nonsignificant ( $p > .05$ ).

Our recovery model scaled chi-square test yielded a value of  $X^2(88) = 533.56, p < .001$ . As with baseline and reactivity models, the other overall model fit indices were below conventional benchmarks indicating adequate model fit, with an adjusted RMSEA of .07, a CFI of .94, and a TLI of .92. As with baseline and reactivity, no significant relationships were observed between the target variables in those with high HRV. In those with low HRV, the direct structural paths for the recovery model from latent variables Abuse and Neglect to HRV Recovery and BRS Recovery were not significant ( $p > .05$ ), nor was the path from HRV Recovery to latent variable Psychopathology. The direct path from Neglect to Psychopathology was similarly nonsignificant, however the direct path from Abuse to Psychopathology was statistically significant (standardized  $\beta = .35, p < .001$ ). As with the baseline and reactivity models, indirect effects were nonsignificant ( $p > .05$ ).

## DISCUSSION

The primary aim of the current study was to assess whether severity of threat-related CM exposures (such as physical, emotional, and sexual abuse) and deprivation-related exposures (such as physical and emotional neglect) were differentially associated with impairments in autonomic reactivity both at rest and in response to acute stress. Second, this study aimed to assess the potentially mediating role of HRV functioning to the relationship between CM exposures subtypes and severity of psychopathology in adulthood. Lastly, we sought to assess whether the relationships between CM exposures, autonomic functioning, and adult psychopathology differed in those with high vs. low HRV. Notably, no significant relationships were found between target variables in those with high HRV, including between severity of CM exposure subtypes and adult psychopathology. CM is a well-established risk factor for the development of psychopathology in adulthood (Li et al., 2015); the fact that no relationship was observed in this group implicates autonomic functioning as a potential moderator in the vulnerability of CM survivors to adult psychopathology. High HRV may in fact serve as a protective factor against adult psychopathology in this population, however further research is needed in order to determine the full nature of the role that HRV may play in the risk for or resilience to psychopathology.

Results within the low HRV group displayed differential associations in the relationship between CM exposure subtypes and autonomic functioning. At baseline, no relationship was found between CM exposures and BRS functioning, however severity of abuse (standardized  $\beta = -.42, p < .01$ ) and severity of neglect (standardized  $\beta = .32, p < .05$ ) were both found to be associated with baseline resting HRV. Notably, these two pathways differ in terms of direction, indicating that higher severity of abuse-related exposures are associated with lower HRV and

higher severity of neglect-related exposures is associated with higher HRV in this sample. These findings are consistent with the dimensional model of childhood adversity, which states that threat-related exposures and deprivation-related exposures have differential neurobiological effects. Additionally, threat-related exposures have been shown to lead to a more hyperreactive emotional neurocircuitry (McLaughlin et al., 2019), which may lead to higher baseline relative sympathetic activity due to the bidirectional nature of the vagus nerve in responding to threat (Thayer & Lane, 2000). Higher sympathetic activity at rest results in lower baseline HRV (a less efficient and responsive vagal control of sympathetic activity), which may then contribute to a blunted autonomic reactivity in response to threat. While deprivation-related exposures have not been observed to promote these alterations in the emotional neurocircuitry responsible for responding to threat, the positive relationship observed in this sample is surprising. Higher HRV is typically associated with greater emotion regulation; it may be that neglect-related exposures promoted greater regulation strategies within our sample. The pathways between exposure subtypes and HRV were not significant within the stress reactivity or recovery epochs; however, it is interesting to note that the direction of the nonsignificant coefficients was consistent across all three models. While we did not observe relationships between CM exposure subtypes and BRS at baseline, we did observe a significant relationship during the stressor task epoch such that those with higher severity of abuse displayed lower BRS in those with low HRV. This further implicates abuse-related exposures as being a specific risk factor for impairment in autonomic functioning. These findings provide support for greater differentiation of CM exposure subtypes in those investigating the biological underpinnings of CM survivors' vulnerability to adult pathology.

While the associations between neglect and psychopathology were nonsignificant, the pathways from severity of abuse to severity of psychopathology was statistically significant in all three models. This suggests that threat-related exposures may provide a greater risk for psychopathology in adulthood than deprivation-related exposures in those with low HRV. This is an important consideration worthy of future research, as exposure type is rarely considered in investigations of the link between CM and adult psychopathology. In fact, cumulative exposure scores such as Adverse Childhood Experience (ACE) scores are one of the most common ways to assess this relationship (Cloitre et al., 2019). Our model provides some evidence that collapsing across CM exposure subtypes may be mitigating or even obscuring the very relationships these studies are attempting to assess.

Our findings may provide some insight into the inconsistency of results in previous investigations of the relationship between CM and ANS functioning in adulthood. Associations between exposure to CM and adult vagally-mediated cardiovascular processes such as HRV and BRS may in fact be non-linear in non-clinical samples; as previous research has exclusively examined these relationships using linear models, this may have contributed to the lack of findings by recent population-based studies (Bakema et al., 2020; Beilharz et al., 2020). Additionally, low HRV has repeatedly shown to be associated with poor emotion regulation and psychological distress (Thayer and Lane, 2000). As our study failed to observe a relationship between CM and psychopathology in those with high HRV, previous investigations of these relationships using clinical samples may have unknowingly been oversampling those with lower HRV than the general population.

In considering these results, it is important to keep several limitations in mind. First of all, the cross-sectional nature of this study prevents any casual links from being assessed.

Investigations into the biological underpinnings of the relationship between CM and adult psychopathology require extensive longitudinal methodology, something that this secondary data analysis cannot provide. Relatedly, the mean age of our sample was 55 years, which further complicates the attempt to investigate childhood exposures and adult outcomes; most of our participants would have had multiple intervening decades between exposure and assessment which could influence our findings. Additionally, as our study was conducted as a secondary data analysis of a large, national study, the self-report measures used may not be optimal for investigating differential influences of threat and deprivation related CM exposures on adult outcomes. Structured clinical interviews are the gold standard for assessing the presence or absence of psychopathology, our study used a self-report that focused only on severity of depression and anxiety symptomatology over the past month. Similarly, self-report measures may not be the best way to assess exposure to maltreatment as it relies on both accurate retrospective reporting and honest self-disclosure, two elements which are challenging to ensure in such a large sample. In general, respondents have been observed to under-report CM-related experiences on retrospective self-report measures (Saunders & Adams, 2014), which is likely to lessen the effectiveness of their use in investigations such as these, particularly in an older sample. Lastly, subscales of our CM measure were highly correlated with one another, even across our dimensional categories. A recent meta-analysis of the CTQ measure observed that a third “emotional exposures” relationship may be warranted, as emotional abuse and emotional neglect in particular are highly correlated in the population. Assessment of abuse, neglect, and emotional exposures may be an area for researchers to consider in future explorations of the influence of CM exposure subtypes on adult health outcomes.

While results from this study do not provide support for the role of adult HRV as a mediating link between CM exposure and adult psychopathology, the lack of an association between severity of CM and psychopathology in those with high HRV does implicate autonomic functioning as a potential moderator in this relationship. As increasing evidence supports low HRV as a transdiagnostic endophenotype for both physical and mental health disorders, understanding the nature of this potential moderation is highly important. Additionally, the observed differences in the relationships between abuse-related exposures and neglect-related exposures and both autonomic and psychological functioning highlight the need for greater differentiation in future studies seeking to understand the vulnerability of CM survivors to physical and mental disease. Understanding the differential contributions that these experiences may have both biologically and psychologically could assist clinicians in improving detection for and treatment of those most at risk for psychological sequelae following exposure to maltreatment in childhood.

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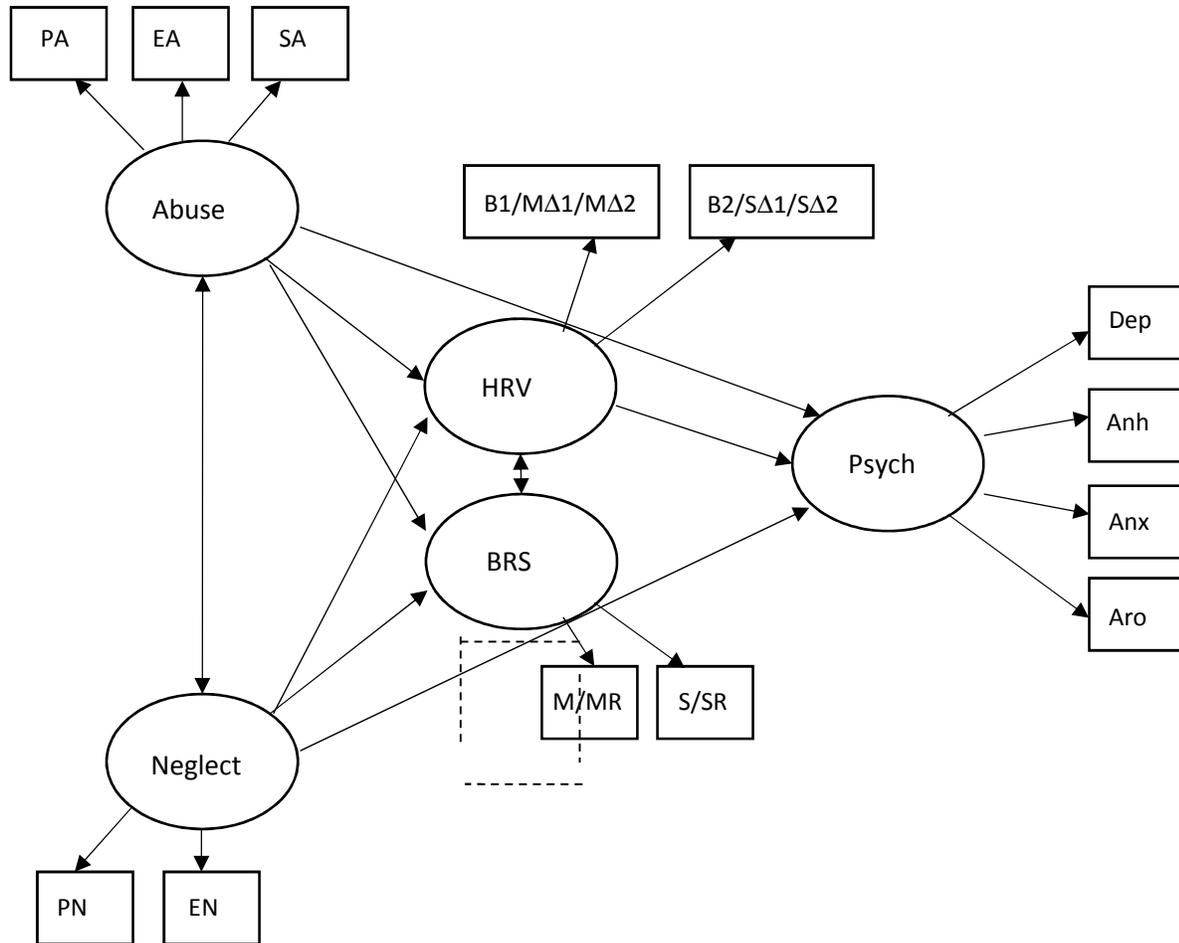
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**Figure 1.** Path Model for Baseline, Reactivity, and Recovery Epochs



*Figure 1.* Path model representing analyses performed, which assess the potentially mediating role of ANS dysregulation in the relationship between CM exposure types and psychopathology in adulthood during three different epochs of the psychophysiological protocol: baseline, reactivity (during cognitive stressor tasks), and recovery (at rest following the tasks). Parameter estimates are reported in Table 1. Structural paths remained the same throughout the three models, however BRS was entered as an observed variable in the baseline model and a latent variable comprised of two epochs in the reactivity and recovery models. Covariates age, gender, and medication use were adjusted for in each of the three models. Key: PA- physical abuse, SA- sexual abuse, EA- emotional abuse, PN- physical neglect, EN- emotional neglect, HRV- heart rate variability, BRS- baroreflex sensitivity, Psych- psychopathology, B1- baseline 1 RMSSD, B2- baseline 2 RMSSD, Dep- depression, Anh- Anhedonia, Anx- anxiety, Aro- arousal.

**Table 1.** Results from Final Structural Analyses of Path Models

Paths	Baseline		Reactivity		Recovery	
	$\beta$	SE	$\beta$	SE	$\beta$	SE
Abuse → HRV						
Low HRV	<b>-.42**</b>	.032	.04	.015	.12	.014
High HRV	-.08	.018	-.08	.022	.02	.017
Neglect → HRV						
Low HRV	<b>.32*</b>	.032	-.02	.016	-.16	.015
High HRV	.06	.023	.03	.027	.02	.021
Abuse → BRS						
Low HRV	-.32	.077	<b>-.23*</b>	.062	-.16	.07
High HRV	.10	.162	.18	.129	.01	.146
Neglect → BRS						
Low HRV	.17	.081	.13	.067	.05	.077
High HRV	-.15	.195	-.19	.169	-.07	.181
HRV → Psychopathology						
Low HRV	.03	.585	.03	.928	.01	1.43
High HRV	.05	1.53	.04	1.628	.01	1.96
Abuse → Psychopathology						
Low HRV	<b>.39*</b>	.427	<b>.35*</b>	.412	<b>.35*</b>	.413
High HRV	.30	.405	.30	.413	.30	.405
Neglect → Psychopathology						
Low HRV	.05	.419	.08	.404	.09	.403
High HRV	.04	.435	.04	.446	.04	.434

\* Standardized variables, significant at the 0.05 level (2-tailed), \*\* Standardized variables, significant at the 0.01 level (2-tailed). N=967. MLR was used for all parameters estimates.

**Table 2.** Means, Standard Deviations, and Correlations between CTQ Subscales

Variables	Mean	SD	1	2	3	4	5
1. CTQ-PA	6.99	3.11	-				
2. CTQ-SA	6.58	3.99	.40**	-			
3. CTQ-EA	8.08	4.26	.68**	.40**	-		
4. CTQ-PN	6.92	2.83	.53**	.28**	.68**	-	
5. CTQ-EN	9.79	4.61	.51**	.36**	.55**	.63**	-

\*\* . Correlation is significant at the 0.01 level (2-tailed). N=967.

**Table 3.** Means, Standard Deviations, and Correlations for MASQ Subscales

Variables	Mean	SD	1	2	3	4
1. Depression	18.42	6.33	-			
2. Anhedonia	11.92	3.99	.80**	-		
3. Anxiety	16.50	4.57	.69**	.64**	-	
4. Arousal	21.61	5.14	.54**	.58**	.65**	-

\*\* . Correlation is significant at the 0.01 level (2-tailed). N=967.