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Journal

Psychology and Aging, 38(3)

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Publication Date

2023-05-01

DOI

10.1037/pag0000738

Peer reviewed

HHS Public Access

Author manuscript

Psychol Aging. Author manuscript; available in PMC 2024 May 01.

Published in final edited form as:

Psychol Aging. 2023 May ; 38(3): 247–262. doi:10.1037/pag0000738.

Resilience to Stress Across the Lifespan: Childhood Maltreatment, Heart Rate Variability, and Bereavement

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Abstract

Following a stressful life event, there is considerable variation in how individuals respond and adapt. Multiple models of risk and resilience show that adverse childhood experiences may be associated with an individual's response to stress later in life. While there is considerable support that early adversity can sensitize the stress response system and lead to adverse outcomes later in life, there is mounting evidence that in adolescence and young adulthood, certain biological predispositions to stress may be associated with resilience in the context of subsequent stressors. In this study, we evaluated how individual differences in vagally mediated heart rate variability moderated the relationship between childhood maltreatment and grief among a sample of individuals experiencing a stressful life event (i.e., spousal bereavement) over time. Data were collected at approximately 3, 4.5, and 6.5 months after the death of a spouse (n = 130). Heart rate variability moderated the relationship between childhood maltreatment and grief symptoms over time ($b = -0.03$, $p < .001$), such that among individuals with more severe experiences of childhood maltreatment, those with higher heart rate variability had a faster recovery from grief than those with low heart rate variability. This research highlights an overall pattern of resilience among older adult's following spousal bereavement, as well as the relationships between childhood maltreatment, heart rate variability, and differential responses to grief following the loss of a spouse.

Public Significance Statement: Childhood maltreatment and heart rate variability may be related to differential responses to grief among bereaved spouses over time. Among bereaved spouses with a history of childhood maltreatment, those with higher heart rate variability experienced faster recovery from grief symptoms than bereaved spouses with lower heart rate

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variability. These findings suggest a pathway to intervention may include increased heart rate variability to mitigate the relationship between adverse childhood experiences and adjustment to stressors later in life, particularly among older adults.

Keywords

childhood maltreatment; heart rate variability; grief; spousal bereavement

Introduction

On average, older adults are confronted with multiple transitions and stressors. Stressful life events in older adulthood are associated with adverse health outcomes, such as depressive disorders, morbidity, and mortality (Dura et al., 1990; Goldman et al., 2005; Kiecolt-Glaser, 1999). Thus, it is important to understand how common stressors among aging populations may impact an individual's health and well-being outcomes. Notably, the death of a spouse is a common, notable stressor among older populations, with 58% of women and 28% of men older than 75 years becoming widowed in the United States (U.S. Census Bureau, 2021). Spousal bereavement is associated with adverse health outcomes, including increased risk of acute cardiovascular events and excess mortality within the first six months following the loss of a spouse (Carey et al., 2014; Charlton et al., 2001; Ennis & Majid; 2019; Moon et al., 2011, 2014; Neimeyer & Holland, 2015; Siegel & Kuykendall, 1990; Zisook et al., 1997). Spousal bereavement can lead to varying levels of grief symptomology, which is characterized by "longing, sadness, and preoccupations with thoughts, recollections, and images of the spouse" (Stroebe & Stroebe, 1987). Importantly, not all widow(er)s experience the same degree of grief symptomology and adverse outcomes (Bonanno et al., 2002, 2004; Wortman & Boerner, 2011; Wortman & Silver, 1989). While many older adults are remarkably resilient after the death of a spouse, others have considerable and prolonged difficulty following a life stressor (Bonanno et al., 2002, 2004). Given the prevalence of spousal bereavement in older adulthood, it is important to understand who may be at an increased risk for severe and prolonged mental health difficulties to intervene effectively among older adults facing loss.

There are multiple models of risk and resilience that suggest a relationship between adverse childhood experiences and adaptation to stressful life events (Andrews et al., 1993; Hammen et al., 2000; Harkness et al., 2006; Kendler et al., 2004; Lyons & Parker, 2007; McLaughlin et al., 2010; Oldehinkel et al., 2014; Rudolph & Flynn, 2007; Rutter, 2006; Seery et al., 2010). However, these models have been primarily tested in adolescence and early adulthood; data are needed to determine how adverse early life experiences interact with stressors that occur in older adulthood, given the length of time that has elapsed from childhood to older adulthood. Furthermore, models of how adverse early life experiences may be associated with subsequent reactions to stressors do not always come to the same conclusions. While the most prominent model (i.e., stress sensitization) contends adverse childhood experiences are associated with vulnerability to subsequent stressful events (Hammen et al., 2000; Harkness et al., 2006), competing models (e.g., the stress inoculation hypothesis) support the opposite (Andrews et al., 1993, Lyons & Parker, 2007; Oldehinkel et al., 2014; Rutter, 2006; Seery et al., 2010). There is some

empirical support for each model; null findings also exist. Recently, our group found a notable interaction between childhood maltreatment and spousal bereavement, such that the association between childhood maltreatment and depressive symptoms was stronger among those who were widowed than among matched comparisons who were not widowed (Chen et al., 2019). Given this interaction between childhood maltreatment and spousal bereavement, as well as the prevalence of spousal bereavement in older adulthood, it is important to understand the relationship between childhood maltreatment and adaptation to spousal bereavement among older adults over time. Thus, the current study attempts to elucidate what factors may contribute to the relationship between adverse early life experiences and adjustment to stressful life events in a population of older adults who recently experienced a life stressor requiring considerable adaptation (i.e., the death of a spouse).

The stress sensitization hypothesis asserts that those who experience significant adverse childhood events have a lower threshold to stressors across the lifespan than those who did not experience the same degree of early adversity (Hammen et al., 2000; Harkness et al., 2006; Hazel et al., 2008; Kendler et al., 2004; Monroe & Simons, 1991). Thus, even decades after an early life stressor has occurred, less stress is necessary to trigger adverse mental health outcomes in the face of subsequent stressors in older adulthood, in part because stressful physiological and social experiences early in life can augment future emotional and physiological reactivity across the lifespan (Ellis et al., 2005; Gunnar et al., 2001, 2003; Hammen et al., 2000; Harkness et al., 2006; Hazel et al., 2008; Kendler et al., 2004; Monroe & Simons, 1991; Pine & Charney, 2002; Post, 1994; Zeidner & Kampler, 2020). The stress-sensitization hypothesis, and research on childhood maltreatment broadly, has been increasingly utilized in the aging literature given evidence that even into older adulthood, early adversity is associated with mental and physical health outcomes, poor quality of life, and disease in older adulthood (Fagundes et al., 2013; Gershon et al., 2013; Gouin et al., 2012; Kiecolt-Glaser et al., 2011; Lähdepuro et al., 2019; Luo et al., 2021; McEniry, 2013; Ye et al., 2021). Thus, there has been increasing interest in how early life stressors may impact individuals as they age (i.e., in older adulthood). For example, adults with a history of childhood abuse exhibited lower levels of perceived closeness with their abusive parents later in life than adults without a history of childhood abuse (Kong & Martire, 2019). Additionally, adults who experienced childhood neglect reported less emotional closeness, less frequent contact, and fewer exchanges of support with their mothers and less emotional closeness with their father, compared to adults who were not neglected as children (Kong & Martire, 2019).

In contrast to the stress sensitization hypothesis, there are multiple models suggesting that adverse childhood experiences sometimes are associated with resilience to stressful events across the lifespan (Andrews et al., 1993, Collishaw et al., 2007; Hong et al., 2018; Lyons & Parker, 2007; Rutter, 2006; Seery et al., 2010; Wekerle et al., 2012). The stress inoculation hypothesis purports that experiences of early life stress may lessen the relationship between stressors across the lifespan and poor outcomes in cases where stressors are mild to moderate (Oldehinkel et al., 2014). In a related model, “the theory of psychophysiological toughness,” stressful life events can have a “toughening effect” if proceeded by successful recovery because future stressful events are appraised more positively (Dienstbier, 1989).

While the stress inoculation model focuses more on the severity of the stressor, the theory of psychophysiological toughness contends that how one adapts is the determining factor (Dienstbier, 1989; Oldehinkel et al., 2014). One interpretation of this model is that a person with an underlying ability to deal with stressors may gain awareness of their capacity for resilience when stressful events arise. This may be particularly relevant in older adults who potentially have greater awareness of their capacity for resilience across the lifespan compared to younger populations (Benight, 2004; Bukvic et al., 2018; Cherry, 2009).

Examples of discrepancies between the stress sensitization hypothesis and theories of stress inoculation have appeared in the disaster literature (Cherry et al., 2021). For instance, the stress sensitization hypothesis posits that prior loss can exacerbate an individual's response to future disaster-related distress (Smid et al., 2012; Zvolensky et al., 2015). Related to this hypothesis, Gargano et al. (2019) found that PTSD symptoms associated with the terrorist attacks of September 11, 2001 (9/11) in New York City became worse for some participants after Hurricane Sandy (also in New York City), which suggests that experiencing a natural disaster further exacerbated already existing PTSD symptoms. Thus, prior traumatic events may be associated with poorer adjustment to future disasters (Cherry et al., 2021). Conversely, evidence for the inoculation hypothesis shows that previous exposure to disaster can protect older individuals from distress when facing subsequent disasters (Eysenck, 1983; Ferraro, 2003; Knight et al., 2000; Norris & Murrell, 1988; Rafiey et al., 2016; Shrira et al., 2014). For example, Shrira et al. (2014) found that, unlike younger adults, older adults did not experience a positive relationship between high exposure to 9/11 and current PTSD symptoms following exposure to Hurricane Sandy. Recently, Cherry et al. (2021) expanded on this line of research by examining individuals who had experienced both Hurricane Katrina/Rita in 2005 and flooding in Baton Rouge, Louisiana in 2016, compared to individuals who had only experienced the flooding in 2016. They found that individuals in both groups did not differ in mental health outcomes; however, older adults were less prone to mental health symptoms than younger adults. The authors proposed several explanations for these findings, including that compared to younger adults, older adults may be more knowledgeable when facing a natural disaster (Bukvic et al., 2018; Cherry, 2009), older adults may have more substantial disaster coping self-efficacy perceptions (Benight, 2004), and older adults may cope differentially to cumulative disaster-related stressors. In sum, these studies highlight how individuals, particularly older adults, may exhibit differential responses to stressful life events and potential factors contributing to older adults' response to stress (e.g., knowledge, coping self-efficacy perceptions).

Relevant to the current study, there is accumulating data in the child and adolescent literature demonstrating that specific attributes of physiological functioning can partially explain individual differences in vulnerability to environmental stressors (Fletcher et al., 2017; Koss & Gunnar, 2018; Somers et al., 2017). Heart rate variability, or the beat-to-beat variability in heart rate, can be used as a non-invasive marker of parasympathetic tone as it captures the vagus nerve's inhibitory influence on the heart's sinoatrial node. Furthermore, heart rate variability can serve as a non-invasive index of self-regulation, a multidimensional construct that encompasses physiological and psychological control of behavior, emotion, and cognition (Bridgett et al., 2013). Individual differences in parasympathetic nervous system functioning may be associated with basic self-regulatory capacities that interact

with an individual's relational history when adapting to a stressful life event (Fagundes et al., 2012; Sbarra & Borelli, 2013). Mechanistically, heart rate variability can indicate an individual's response to stressors as it serves as a marker of parasympathetic tone, a key component of the autonomic nervous system. Several studies show that autonomic balance favors energy conservation at rest via parasympathetic influences rather than sympathetic influences (Thayer et al., 2009). However, under stressful conditions, the body's regulatory systems may experience autonomic imbalance, or a hyperactive sympathetic system and a hypoactive parasympathetic system (i.e., decreased heart rate variability), placing excessive energy demands on the body that subsequently lead to disease (Thayer et al., 2009). Furthermore, recent models link autonomic imbalance and vagal function to cardiovascular disease, with decreased vagal function (i.e., low heart rate variability) associated with cardiovascular disease risk factors and increased vagal function (i.e., high heart rate variability) associated with improved risk profiles (Jarczok et al., 2019; Kivimäki & Steptoe, 2018; Thayer et al., 2009; Thayer et al., 2010).

Thayer and Lane's neurovisceral integration model suggests that individual differences in heart rate variability represent a flexible neural network that can interact with stress and aging across the lifespan (Thayer et al., 2009, 2021; Thayer & Lane, 2000). Several studies have indicated the relationship between heart rate variability and age-related changes in autonomic function (Antelmi et al., 2004; Zulfiqar et al., 2010). While studies suggest a steady decline in heart rate variability with each decade of age, it is interesting to note that individuals above age 80 had relative parasympathetic dominance and higher heart rate variability (Antelmi et al., 2004; Zulfiqar et al., 2010). Given this individual variation in heart rate variability levels among older adults, heart rate variability may be relevant in understanding the relationship between early adversity and mental health outcomes following a stressful life event among older adults.

Previous studies have indicated that heart rate variability may interact with the relationship between troubled childhood experiences and mental health outcomes. For instance, low heart rate variability exacerbated the relationship between childhood maltreatment and alcohol problems in adolescence and young adults (Oshri et al., 2018). This research is in line with other studies indicating heart rate variability as a moderator between early adversity and internalizing problems in youth (McLaughlin et al., 2014), as well as harsh parenting and adjustment issues in youth (El-Sheikh et al., 2001). Specifically, El Sheikh et al. (2001) found that the relationship between witnessing marital conflict as children and developing health problems related to internalizing and externalizing behavior patterns among 8–12-year-olds was moderated by high heart rate variability. This line of research suggests that individuals with higher heart rate variability may be able to moderate their arousal when facing stress, reducing health problems associated with hyperactivation of the sympathetic nervous system (El-Sheikh et al., 2001; El-Sheikh & Harger, 2001; Falkner & Ragonesi, 1986). Thus, heart rate variability may interact with the relationship between early adversity and mental health outcomes following a stressful life event later in life.

Study Objective

The purpose of this study was to identify how heart rate variability, a biological index of self-regulation, moderated the relationship between adverse childhood experiences and an individual's responses to spousal bereavement, a common stressful life event in older adulthood, over time. Thus, we examined whether heart rate variability moderated the relationship between childhood maltreatment and grief symptoms over time among bereaved spouses using data collected from each participant at 3-time points: approximately 3 months, 4.5 months, and 6.5 months following the death of the spouse, with time assessed as a continuous variable (i.e., months post-loss). In post hoc analysis, we examined this relationship while controlling for depressive symptoms.

Method

Transparency and Openness

The authors affirm that the de-identified data on which the study conclusions are based, the analytic code needed to reproduce analyses, and the materials for this study are available at https://osf.io/w4m9q/. Additionally, the authors affirm that the study design, hypotheses, and analytic plan were not preregistered; however, these items were each proposed as part of a master's thesis prior to the analyses. The authors also affirm that we report how we determined our sample size, all data exclusions, all manipulations, and all measures in our manuscript.

Participants

We recruited individuals who recently experienced the death of their spouse (n = 130) from obituaries, support groups, flyers, online postings, and community events. The sample size was determined as part of a larger biobehavioral study on bereavement and cardiovascular risk. Bereaved individuals must have experienced the death of their spouse no more than 14 weeks before their visit. Exclusion criteria included being non-English speakers, having significant visual or auditory impairment, being pregnant or nursing, having an autoimmune or inflammatory disease, having experienced the loss of another loved one in the last year (in addition to the spouse), and being married for less than 3 years before the loss. All data was collected between 2016 and 2018 (i.e., prior to the COVID-19 pandemic) in Houston, TX. All study procedures were approved by the Rice University Institutional Review Board (Study Number: *IRB-FY2016-813*; Study Title: *742029-20 Project Heart: Biobehavioral effects on Cardiovascular Risk for Bereaved Spouses*). All participants provided informed consent before beginning the study.

Participants completed self-report demographic and clinical questionnaires; research assistants collected measurements including weight, height, waist circumference, and heart rate variability. The sample included 88 (67.7%) females and 42 (32.3%) males, as well as 117 (90.0%) White participants, 7 (5.4%) Black or African American participants, 3 (2.3%) Asian participants, 2 (1.5%) Hispanic or Mexican American participants, and 1 (0.77%) participant who identified as "Other".

For this analysis, each predictor (i.e., childhood maltreatment, heart rate variability, and all covariates) was measured at baseline (3 months following the spouse's death). Our outcome of interest, grief symptoms, was collected over three time points: approximately 3, 4.5, and 6.5 months after the death of a spouse. Time was operationalized as months post-loss and assessed continuously. While data was collected at approximately 3, 4.5 and 6.5 months post-loss; however, there was a window period in which participants were able to reschedule their appointment in the event of a scheduling conflict. Including the window, the ranges of each time point were approximately 1-4 months post-loss at the first time point (M = 2.77, SD = 0.58; actual range: 1.22 - 4.08), 3-6 months post-loss at the second time point (M = 4.55, SD = 0.55; actual range: 3.29 - 5.95), and 5-8 months post-loss at the third time point (M = 6.39, SD = 0.41; actual range: 5.33 - 7.73). To assess whether there were outliers related to time, we examined the data two different ways. First, we assessed whether any data fell outside of the ranges listed above (i.e., first time point: 1-4 months post-loss, second time point: 3-6 months post-loss, and third time point: 5-8 months post-loss). Second, we assessed whether any time points were 3 standard deviations below or above the mean of each time point (i.e; first time point: $M = 2.77$, $SD = 0.58$, second time point: $M = 4.55$, $SD = 0.55$; third time point: $M = 6.39$, $SD = 0.41$). In both methods to assess outliers, only one outlier emerged. Specifically, there was one participant whose second time point was outside of the range using both methods. Thus, we removed this data point from our analysis; the actual range at the second time point was reported with this outlier removed.

Measures

Demographics.—Participants answered questions about their age, gender, and educational attainment. To designate educational attainment, which has been a reliable estimation of socioeconomic status, participants checked their “highest grade completed” with 4-8 indicating years of junior high, 9-12 indicating high school, 13-16 indicating college, and 17-20+ indicating graduate or professional school. Educational level was used to assess socioeconomic status because some individuals were retired or never worked outside of the home. In addition, education is less vulnerable to fluctuations in current income and job status (Gorman & Sivaganesan, 2007; Marmot et al., 1998; Winkleby et al., 1992). Descriptive information for the categorical variables of the sample demographics can be found in Table 1. Descriptive statistics and correlations for continuous variables can be found in Table 2.

Childhood maltreatment.—The Childhood Trauma Questionnaire (CTQ) provides data on early events that may be related to vulnerabilities in adulthood. Widely used, the CTQ has excellent normative data for its five scales: physical abuse, emotional abuse, sexual abuse, physical neglect, and emotional neglect (Bernstein & Fink, 1998). Higher scores are associated with greater distress among adults and greater risk for PTSD and depression. Each subscale is comprised of five items, and a five-point Likert scale is used: 1 = *never true*, 2 = *rarely true*, 3 = *sometimes true*, 4 = *often true*, 5 = *very often true*. Each subscale score ranges from 5 (no history of abuse and neglect) to 25 (very extreme history of abuse and neglect). A total childhood maltreatment score was taken by averaging the five subscales.

Both the CTQ as a whole and subscales of the questionnaire have been validated and demonstrated reliability via multiple studies. In the current sample, Cronbach's α was calculated for the following subscales: emotional abuse (α = .80), physical abuse (α = .62), sexual abuse (α = .88), emotional neglect (α = .90), and physical neglect (α = .70). Initial studies including the CTQ exhibited convergent and discriminant validity with a structured trauma interview, excellent test-retest reliability over a 2-to 6-month interval, and an intraclass correlation of 0.88 (Bernstein et al., 1994, Fink et al., 1995). Supporting the CTQ's criterion-related validity, the CTQ has shown good sensitivity and satisfactory or better specificity after comparing therapists' trauma ratings about the patient and corroborating independent evidence, including information from referring clinicians, agencies, and other informants (Bernstein et al., 1997).

Heart rate variability.—Vagally mediated heart rate variability was continuously measured non-invasively with the Polar s810 wristwatch and Wearlink 31 belt band; the 1000 Hz sampling rate provides valid and reliable ECG data (Gamelin et al., 2006; Nunan et al., 2009). All participants were in a sitting position during a 5-minute relaxation period while heart rate variability was measured. During these 5 minutes, participants were told to relax and not to do anything with their phone or other distracting objects. During this time, they were in a relaxing environment with low light and no music. Vagally mediated heart rate variability measurements were taken at a 5-minute epoch to form an overall indicator of baseline heart rate variability. Before analyzing heart rate variability, we preprocessed the raw interbeat intervals for artifacts using KUBIOS heart rate variability analysis software (Tarvainen et al., 2009). The KUBIOS software produced values for vagally mediated (parasympathetic) heart rate variability using the time-domain method, square root of mean successive differences (RMSSD) between R-Waves, with units in milliseconds (Malik et al., 1996; Stein et al., 1994). RMSSD is highly correlated with spectral derived measures of heart rate variability and is less affected by respiration and other artifacts than spectral indices of heart rate variability (Penttila et al., 2001). All procedures followed the recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology (Malik, 1996).

This assessment of heart rate variability has been shown to be an effective indicator of the participant's overall trait heart rate variability levels in daily life. Bertsch et al. (2012) found that trait specificity (i.e., the variance proportion due to a consistent factor across the multiple measurement conditions) explained about 70% of individual differences in resting/spontaneous heart rate variability. Furthermore, Li et al. (2009) demonstrated the stability of resting heart rate variability by indicating that baseline resting heart rate variability predicted baseline resting heart rate variability levels 18 months later ($p < .01$). Specifically, levels of RMSSD at rest showed high tracking stabilities, with a partial correlation coefficient of 0.62 (Li et al., 2009). Similarly, Goedhart et al. (2007) found very high correlation coefficients of sitting heart rate variability (0.7 for RMSSD) over 3 years.

Grief Symptoms.—The Inventory of Complicated Grief (ICG) measures symptoms of grief following bereavement that are associated with long-term impairments (Prigerson et al., 1995). The ICG measures 19 different grief-related symptoms and has excellent test-

retest reliability (Cronbach's α for the current sample = .917). All questions are answered on a 5-point Likert scale that ranges from *0 - never* to *4 - always*. Sample items include "Memories of the person who died upset me," "I feel I cannot accept the death of the person who died," and "I feel myself longing for the person who died." The ICG scores range from a minimum value of 0 to a maximum of 76. Symptoms of grief were analyzed with a continuous approach using the total ICG score.

Depressive Symptoms.—The Center for Epidemiological Studies Depression Scale (CES- D) has been used extensively as a brief measure of depressive symptoms (Basco et al., 1997; Radloff, 1977). The measure asks participants to rate how often they have experienced depression-related symptoms (i.e., restless sleep, poor appetite, and loneliness) in the past week. Each of the twenty items is measured on a scale from 0 to 3: 0 = *rarely or none of the time*, 1 = *some or a little of the time*, 2 = *moderately or much of the time*, 3 = *most or almost all the time*. Scores range from 0 to 60 with higher scores indicating greater depressive symptoms. Studies have shown acceptable test-retest reliability and excellent construct validity; the Cronbach's α for the current sample was .920. (Basco et al., 1997).

Covariates.—At the first time point, participants provided self-reports of their age, gender, education, months since the passing of the spouse at baseline, nicotine use, and beta-blocker use, which were each included as covariates in the adjusted model. Nicotine and beta-blocker use were included as covariates as previous studies have shown that both substances may be associated with differences in heart rate variability (Barutcu et al., 2005; Niemela et al., 1994). In post-hoc analysis, depressive symptoms (at the first time point) were also included as a covariate.

Statistical Analysis

All analyses were conducted in *R* (version 1.1.463; R Core Team, 2019). We utilized the *nlme* package for the analyses using a linear mixed-effects model (Pinheiro et al., 2019) and the *ggeffects* package for data visualization (Wickham, 2016). The *bagImpute* method in the *caret* package was used to impute missing values across all predictors. This method produces unbiased parameter estimates to reflect the true variability of missing data in a less biased manner than listwise deletion (Enders, 2017, Kuhn, 2020). Missing data comprised less than 2.25% of the sample. Before imputation, childhood maltreatment and heart rate variability were grand mean centered. Preliminary statistical analysis included descriptive statistics, assessment of normality of distributions, and examination for skewness and kurtosis. We also examined residuals to confirm normal distribution, examined assumptions of linearity and homoscedasticity, and evaluated collinearity statistics. In order to examine grief symptoms, we created a grief symptoms panel with all 3 time points using a linear mixed-effects model. Zero-order correlations assessed relationships between childhood maltreatment, heart rate variability, grief symptoms, and all continuous covariates.

For the primary analysis, grief symptoms were modeled as a function of the interaction between time post-loss and baseline measures of childhood maltreatment and heart rate variability, controlling for all constituent lower-order interactions and predictors, with random effects to account for correlated observations. We calculated childhood maltreatment

and heart rate variability as continuous variables in our analysis. To calculate simple slopes and plot our interaction, we determined levels of heart rate variability by calculating one standard deviation above and below the mean to evaluate high and low levels of the moderator; we assessed childhood maltreatment at the 1st quartile, median, and 3rd quartile to account for positive skew in the childhood maltreatment variable. For each analysis, we ran both unadjusted and adjusted regression models.

Mixed-Effects Model Fit

First, we tested whether there was statistically significant variability in the intercepts across participants, whereby the level 1 variable was the time point, and the level 2 variable was the participant. We investigated whether there was significant variability in intercepts across participants by first estimating an unconditional means model, where the intercept for each null model represented the mean level of grief symptoms across individuals. According to the null model, which contained only the random intercept variance term to allow the intercepts to differ across individuals, 85.04% of the variance in grief symptoms could be explained by the participant. Further, the intraclass correlation (ICC2) was over .93, indicating that participants could be reliably differentiated in terms of their grief symptoms. When assessing model fit, −2 log likelihood results indicated that the model including the random intercept (of person) fit the data better than the model without the random intercept. Finally, testing for slope variability indicated that the model including the fixed slope over time fit the data best (Bozdogan, 1987; Vrieze, 2012); thus, the models with the random intercepts and fixed slopes were used for this analysis.

Results

Preliminary Analyses

Demographic information for categorical variables and time-varying continuous variables can be found in Table 1. For our categorical variables, we ran a t-test and ANOVA to assess whether gender and education level, respectively, were associated with differences in grief symptoms. Gender was not associated with differences in grief symptoms, $t(356) = 1.33$, $p = 1.83$, while differences in education level were associated with differences in grief symptoms, $F(4,353) = 3.04$, $p = .017$.

Descriptive statistics and correlations between the variables of interest can be found in Table 2. Prior to grand-mean centering childhood maltreatment and heart rate variability, we examined the descriptive statistics for the variables of interest: childhood maltreatment score after averaging the 5 subscales ($M = 6.75$, $SD = 1.88$) and heart rate variability as indexed by RMSSD (milliseconds) ($M = 23.16$, $SD = 18.58$). Childhood maltreatment and heart rate variability were grand mean centered before calculating the correlations and running the analysis.

Primary Analysis

In each analysis, we utilized GLMM (generalized linear mixed models) to longitudinally model grief symptoms as a function of time post-loss. Grief symptoms were measured at approximately 3, 4.5, and 6.5 months following the spouse's death. Our primary

analysis indicated a significant 3-way interaction between childhood maltreatment, heart rate variability, and time post-loss predicting grief symptoms in unadjusted and adjusted models (unadjusted: $b = -0.03$, $p < .001$; adjusted: $b = -0.03$, $p < .001$; Figure 1). When conducting our simple slope analyses, we first found that among participants with low childhood maltreatment (i.e., left panel of Figure 1), those with low baseline heart rate variability exhibited a decrease in grief symptoms over time (unadjusted: $b = -1.20$, $p < .001$; adjusted: $b = -1.19$, $p < .001$), while those with high baseline heart rate variability did not experience changes in grief symptoms over time (unadjusted; $b = -0.12$, $p = .728$; adjusted: $b = -0.12$, $p = .733$). Second, among participants with average levels of childhood maltreatment (i.e., middle panel of Figure 1), grief symptoms decreased over time, irrespective of heart rate variability levels (among individuals with low heart rate variability: unadjusted: $b = -0.80$; $p < .001$; adjusted: $b = -0.79$, $p < .001$; among individuals with high heart rate variability: unadjusted $b = -0.60$; $p = .020$: adjusted: $b = -0.60$; $p = .021$). Finally, among participants with high childhood maltreatment (i.e., the right panel of Figure 1), those with high baseline heart rate variability exhibited a decrease in grief symptoms over time (unadjusted: $b = -1.09$, $p < .001$; adjusted: $b = -1.10$, $p < .001$) while those with low baseline heart rate variability did not experience changes in grief symptoms over time (unadjusted: $b = -0.38$, $p = .110$; adjusted: $b = -0.37$, $p = .123$). Table 3 provides results from the unadjusted and adjusted model for the 3-way interaction, its constituent lower-order interactions, lower-order predictors, and covariates.

To confirm that our findings were not driven by outliers, we conducted two sensitivity analyses with outliers removed to confirm our findings. For our first sensitivity analysis, we Z-scored all of the outcome variables and removed the 3 data points where the outcomes Z score was above 3 or below −3. We then ran a sensitivity analysis on the dataset with outliers removed and found that the interaction and simple slopes all remained significant ($b = -0.03$, $p < .001$; Figure S1). To address any remaining skew on the outcome variable in our second sensitivity analysis, we square root transformed grief symptoms, z-scored the square root transformed grief symptoms, and removed the 1 data point where the z-scores were above 3 or below −3. After running our analysis on this dataset, we also found that the interaction and simple slopes remained significant ($b = -0.03$, $p < .001$; Figure S2).

In post-hoc analyses, we found that the significant interaction between childhood maltreatment, heart rate variability, and time post-loss predicting grief symptoms held when also controlling for depressive symptoms ($b = -0.02$, $p < .001$) (Table 3). Similar to our primary analysis, participants with low childhood maltreatment and low baseline heart rate variability had a decrease in grief symptoms over time ($b = -1.13$, $p < .001$), participants with average levels of childhood maltreatment had a decrease in grief symptoms, irrespective of heart rate variability (among individuals with low heart rate variability: $b = -0.76$; $p = .001$; among individuals with high heart rate variability: $b = -0.62$; $p = .016$), and participants with high childhood maltreatment and high baseline heart rate variability had a decrease in grief symptoms over time ($b = -1.11$, $p < .001$). Given that several studies have indicated the relationship between heart rate variability and age-related changes in autonomic function (Antelmi et al., 2004; Zulfiqar et al., 2010), we assessed whether childhood maltreatment, heart rate variability, and age interacted to predict grief

symptoms, adjusting for time, and found a non-significant 3-way interaction in unadjusted (b = 0.01; p = .271) and adjusted models (b < 0.01; p = .142).

Discussion

Based on work suggesting that adverse childhood experiences may be associated with differences in mental health across the lifespan, we investigated the interaction between childhood maltreatment, baseline heart rate variability, and time post-loss, with grief symptoms among older adults following the loss of a spouse, a common stressor in aging. We found a significant 3-way interaction between childhood maltreatment, heart rate variability, and time in our unadjusted model and adjusted models, as well as in post-hoc analyses that also included depressive symptoms in the model. Each of these models found a similar pattern. First, grief symptoms decreased over time among participants with average levels of childhood maltreatment (i.e., the middle panel of Figure 1), irrespective of heart rate variability. Second, for participants with below-average levels of childhood maltreatment (the left panel of Figure 1), those with higher heart rate variability did not exhibit a change in grief symptoms over time, but those with lower heart rate variability exhibited decreases in grief symptoms over time. Finally, for participants with above-average levels of childhood maltreatment (the right panel of Figure 1), lower heart rate variability was related to no change in grief symptoms over time, whereas higher baseline heart rate variability was related to greater decreases in grief symptoms over time. While our findings did not support our initial assumption regarding the stress inoculation hypothesis, our findings corroborated previous research noting different grief trajectories of older adults facing the death of a spouse (Bonanno et al., 2002, 2004). Indeed, while grief trajectories differed as a function of the interaction of childhood maltreatment and heart rate variability, the prevailing pattern across these trajectories was in line with previous findings that many older adults exhibit resilience after losing a spouse (Bonanno et al., 2002, 2004). Examining grief trajectories from these findings can provide insight into factors (i.e., childhood maltreatment and heart rate variability) that may impact grief trajectories over time; however, future research with data beyond the 3-6.5 month range examined in this study is needed to make further conclusions about these patterns. These findings also add to the aging literature regarding the relationship between early-life stress and spousal bereavement in older adulthood. Indeed, the interaction between adverse childhood relational experiences and patterns of autonomic self-regulatory capacity does not appear to be less relevant as people age physiologically.

Multiple theoretical models have proposed that heart rate variability may play a role in modifying an individual's response to stressors following early life stress (Carnevali et al., 2018; Fletcher et al., 2017; Heim et al., 2000; Hinnant and El-Sheikh, 2009; Koss & Gunnar, 2018; Somers et al., 2017). Individual differences in the maturation of the parasympathetic system, indexed by heart rate variability, may modulate self-regulation abilities throughout the lifespan, which may be especially relevant when experiencing a stressful life event (Calkins & Keane, 2004; Porges et al., 1994). For example, there may be an autonomic predisposition for how the nervous system develops due to stress, resulting in high heart rate variability in some and low heart rate variability in others. As a result, heart rate variability can function as an index of how an individual self-regulates or responds to stress later in life.

Furthermore, the mechanism by which a positive cardiac vagal tone development moderates the relationship between early stressful events and perceived physical well-being could be related to a better ability to cope with stressful events and adapt to stressful environments (Fabes et al., 1994; Fox, 1989).

In the current study, participants with high childhood maltreatment and high heart rate variability had a decrease in grief symptoms over time. Previous work indicates that individual differences related to the regulation of physiological responses to stress might indicate an individual's ability to cope and adapt to adversity (Carnevali et al., 2018; Hinnant and El-Sheikh, 2009). Other empirical evidence may provide insight into how childhood maltreatment and heart rate variability can interact to predict grief symptoms over time. For example, Patron et al. (2020) recently found that high heart rate variability acted as a protective factor among children who experienced a high number of early stressful events. Specifically, among children who experienced a higher number of early stressful events, a positive cardiac vagal tone development trajectory was associated with better physical well-being. In contrast, a negative or flat cardiac vagal tone development trajectory was associated with the lowest physical well-being. Another explanation may be related to heart rate variability's role on emotion regulation effort. For instance, higher heart rate variability may be associated with successful regulation of the negative emotions associated with the loss of a spouse, leading to decreases in grief symptoms (Williams et al., 2015). While the limited time-frame in the present study (i.e., lack of pre-loss data and data beyond 6.5 months) makes it difficult to draw definitive conclusions regarding our findings, we provide potential explanations while emphasizing the need for future studies to further examine underlying mechanisms. When facing the loss of a spouse, individuals with high self-regulation via heart rate variability may have been more capable of regulating their physiological arousal during the grieving process. Research on the grieving brain indicates that grieving individuals need time to disengage the brain network associated with psychological bonding with the deceased (O'Connor, 2019; O'Connor et al. 2022; O'Connor & McConnell, 2018). Individuals with high self-regulation via high heart rate variability may be better able to face the disengagement process and may experience more grief during this process initially. However, these individuals may be better able to regulate their physiological arousal when facing grief and in turn, experience an overall decrease in grief symptoms over time. In contrast, individuals with lower self-regulation ability via low heart rate variability may disengage with this process preemptively to potentially avoid being overwhelmed with grief, given their lower capacity to self-regulate their physiological arousal when facing bereavement.

The other significant finding in the present study indicated that participants with low childhood maltreatment and low heart rate variability had a decrease in grief symptoms over time (while individuals with low childhood maltreatment and high heart rate variability did not). We approach this counterintuitive finding with caution and emphasize the need for future studies to test further underlying mechanisms that may explain this result. Additionally, we acknowledge that the lack of pre-loss data and data beyond 6.5 months limits our interpretation of these findings. Nonetheless, we provide potential explanations that may account for our unexpected result. One reason for this may be related to baseline grief symptoms (i.e., at the first time point approximately 3 months post-loss), which

did not vary among individuals with low heart rate variability (regardless of childhood maltreatment levels). Given that grief symptoms at baseline did not differ among individuals with low heart rate variability (irrespective of childhood maltreatment levels), those with low childhood maltreatment and low heart rate variability may have experienced less vulnerability to stress, and in turn, a greater decrease in grief symptoms between 3-6.5 months post-loss compared to those with high childhood maltreatment and low heart rate variability. Additionally, we note that among those with low childhood maltreatment, those with low heart rate variability started with higher grief severity compared to those with high heart rate variability (Figure 1), which may, in part, explain why their change from approximately 3 to 6.5 months post-loss was most pronounced. We acknowledge that these findings may be a function of this limited window of time (i.e., between 3 to 6.5 months post-loss), and future research should examine these longitudinal effects over a more extended amount of time to better understand these patterns. Finally, we acknowledge recent work that has examined multiple mechanisms and factors (i.e., adult psychopathology, age) that may play differing roles in the relationship between childhood maltreatment and heart rate variability (Sigrist et al., 2021). For instance, heart rate variability mediated the relationship between cumulative exposure to stressful life events and mental health outcomes in combination with age-related decreases in heart rate variability (Liddell et al., 2016). Given the complexities in how childhood maltreatment, heart rate variability, current psychopathology, and age may interact, particularly among older adults facing stress, it is important for future research to examine further factors that may explain the pattern of results in this study. Specifically, future research should further examine the role of heart rate variability on individuals who have experienced low childhood maltreatment to better understand potential mechanisms underlying this finding. It is also important to emphasize in the context of these findings that childhood maltreatment is of high public health concern (Noll & Shenk, 2010); thus, these findings should not be interpreted as suggesting that childhood maltreatment is beneficial in any way.

Strengths, Limitations, and Future Directions

This study has several methodological strengths, including the prospective design, biobehavioral focus, and rigorous protocol. However, it is important to consider study limitations. We first continue to emphasize that the limited time-frame (i.e., the lack of pre-loss data and data beyond 6.5 months post-loss) limits the interpretation of the grief trajectories presented in the current study. Other limitations include a lack of heart rate variability data before the loss, pre-loss relationship quality data, and data assessing adversities other than childhood maltreatment. Pre-loss relationship quality may play an important role on bereavement outcomes (Abakoumkin et al., 2010; Hooghe et al., 2013); thus, future research including this data would provide greater insight into the factors impacting grief trajectories. Additionally, examining other adversities other than childhood maltreatment would also help to understand the mechanisms related to the role of early life stress on an individual's response to stress later in life. Given that childhood maltreatment only measures child abuse and neglect, the present study is limited by not accounting for other childhood stressors that may impact an individual's response to bereavement. We also acknowledge that our sample had an overrepresentation of White participants and participants with high socioeconomic status (as measured by education). It

is important to note that the overrepresentation of White individuals in our sample limits the generalizability of these findings. Additionally, given that cultural differences can impact the grieving process following the loss of the spouse, our sample is limited in its ability to address how cultural differences would potentially impact these relationships in a more diverse sample. Thus, replicating these findings with larger, more diverse samples would boost confidence in our findings.

To account for potential methodological issues, we carefully assessed factors such as variable skew and outliers to rule out potential methodological issues (Burros, 1951; West et al., 2004). One limitation of this study was the non-normal distribution of childhood maltreatment, such that childhood maltreatment was positively skewed, with more participants reporting a history of low maltreatment rather than high maltreatment. While we acknowledge that non-normal distribution (i.e, positively skewed) of childhood maltreatment is normative in the general population (Etain et al., 2010; Sonmez et al., 2021; Üçok, A., & Bikmaz, 2007), we acknowledge that a priori selection of our sample to have a normal distribution across childhood maltreatment could have avoided some of these interpretation problems. To account for this limitation, we graphed our findings and analyzed simple slopes using the 1st quartile, median, and 3rd quartile values of childhood maltreatment rather than the mean and one standard deviation above and below the mean. Furthermore, the interpretation of the results was influenced by a low representation of individuals with high levels of childhood maltreatment and high heart rate variability (i.e., at least 1 standard deviation above the mean) due to variable skew; however, we note that assumptions of linear regression were met despite variable skew.

In this study, we were interested in understanding the variability in how older adults adapt to a discrete stressful life event. We focused on the death of a spouse because it is generally regarded as a stressful life period in an older adult's life that is also ubiquitous in older adulthood (Hahn et al., 2014; Stroebe et al. 2007; Zettel & Rook, 2004). The combination of its ubiquity and ability to elicit challenging emotions make it one of the best contexts to study stressful life events in older adulthood. Indeed, we acknowledge other less common experiences could be perceived as more stressful (e.g., death of a child or grandchild); however, these experiences are far less common (Smith-Greenway et al., 2021).

We emphasize that future directions of this research should first aim to better understand the grief trajectories described in the current study while addressing the limitations of the current study. Further implications of this work, following replication studies addressing these limitations, include looking at interventions that may help increase heart rate variability to potentially mitigate the relationship between childhood maltreatment and adjustment to stressful life events in older adulthood. Interventions have shown that heart rate variability can be improved through a variety of mechanisms (Grässler et al., 2021; Makovac et al., 2017; Nesvold et al., 2012; Nolan et al., 2005; Tang et al., 2009; Thayer et al., 2021). Grässler et al. (2021) recently published a meta-analysis detailing how exercise and training regimens can increase heart rate variability. For example, they found multiple studies showing that endurance and resistance training was associated with higher heart rate variability in healthy adult populations. In addition, Nesvold et al., (2012) and Tang et al., (2009) showed that meditation can help individuals increase their heart rate variability and

vagal control. Nolan et al., (2005) also found that their intervention of paced breathing with heart rate variability biofeedback (i.e., showing participants how their heart rate variability responded) led to increased heart rate variability, as well as decreased stress and depressive symptoms. Finally, Makovac et al. (2017) ran a meta-analysis that showed brain stimulation of the prefrontal cortex (either through electrical or magnetic currents) was associated with increased heart rate variability. Future research should explore how interventions may improve heart rate variability to potentially mitigate the relationship between childhood maltreatment and an individual's response to stress later in life.

Conclusion

The present study aims to understand the relationship between childhood maltreatment, heart rate variability, and grief symptoms over time among older adults who recently experienced the death of a spouse. There was a significant interaction between childhood maltreatment, heart rate variability, and time in both unadjusted and adjusted models with grief symptoms as the outcome. Post-hoc analyses replicated these findings when also controlling for depressive symptoms. In each model, participants with high childhood maltreatment and high heart rate variability had a reduction in grief symptoms over time. Additionally, participants with low childhood maltreatment and low heart rate variability had a decrease in grief symptoms over time. Future studies can continue to explore mechanisms underlying how heart rate variability can provide further understanding into differences in how adverse childhood experiences may be associated with adjustment to stressful life events among older adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding for this project was provided by the National Institute of Health: National Institute on Aging (Grant No. 5F31AG069439-02; PI: Michelle Ai-Lien Chen) and National Heart, Lung, and Blood Institute (Grant No. 1F32HL164050-01; PI: Michelle Ai-Lien Chen and Grant No. 5R01HL127260-05; PI: Christopher Paul Fagundes). Prior dissemination of the ideas and data appearing in the manuscript include Ms. Chen's master thesis defense, a talk within the Rice University Psychological Sciences department research interest group, and a virtual poster session at the American Psychosomatic Society 2020 Virtual Meeting. While the study design, hypotheses, and analytic plan were not preregistered, these items were each proposed as part of a master's thesis, prior to the analyses and master's thesis defense. The de-identified data on which the study conclusions are based, the analytic code needed to reproduce analyses, and the materials for this study are available at https://osf.io/w4m9q/.

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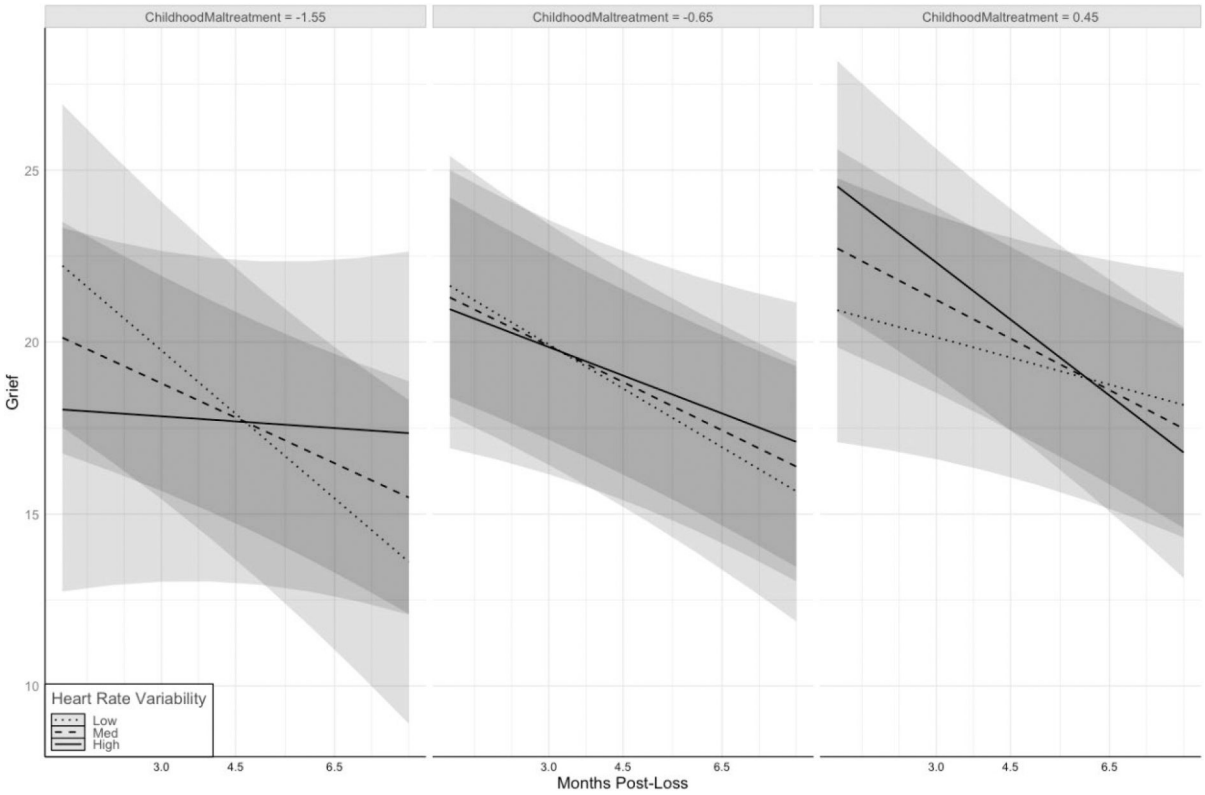

Figure 1. Adjusted Interaction of Childhood Maltreatment, Heart Rate Variability, and Time (Months Post-Loss) Predicting Grief Symptoms

Note: Data was collected at approximately 3, 4.5 and 6.5 months post-loss; however, there was a window period in which participants were able to reschedule their appointment in the event of a scheduling conflict. Including the window, the ranges of each time point were approximately 1-4 months post-loss at the first time point (M = 2.77, SD = 0.58; actual range: 1.22 - 4.08), 3-6 months post-loss at the second time point (M = 4.55, SD = 0.55; actual range: 3.29 - 5.95), and 5-8 months post-loss at the third time point (M = 6.39, SD = 0.41; actual range: 5.33 - 7.73). Time is reflected as a continuous measure of months post-loss, with axis denoting approximately where each data collection point took place.

Table 1

Descriptive Statistics for Categorical Variables and Time-Varying Continuous Variables

	N (%) or M (SD)
Gender	
Female	88 (67.7%)
Male	42 (32.3%)
Age	
35-39	1 (0.77%)
40-49	4 (3.08%)
50-59	18 (13.85%)
60-69	43 (33.08%)
70-79	52 (40.00%)
80-85	12 (9.23%)
Highest Level of Education	
Graduate/Professional	82 (63.08%)
3+ Years College	17 (13.08%)
Up to 3 Years College	13 (10.00%)
High School	17 (13.08%)
Race/Ethnicity	
White	117 (90.0%)
Black or African American	7 (5.4%)
Asian	3 (2.3%)
Hispanic or Mexican American	2 (1.5%)
Other	1 (0.77%)
Time (Months Post-Loss)*	
Time 1	2.77 (0.58)
Time 2	4.55(0.55)
Time 3	6.39(0.41)
Grief Symptoms	
Grief Symptoms at Time 1	22.42 (12.15)
Grief Symptoms at Time 2	20.63 (12.17)
Grief Symptoms at Time 3	19.53 (11.96)

Note: Data was collected at approximately 3, 4.5 and 6.5 months post-loss; however, there was a window period in which participants were able to reschedule their appointment in the event of a scheduling conflict. Including the window, the ranges of each time point were approximately 1-4 months post-loss at the first time point (M = 2.77, SD = 0.58; actual range: 1.22 - 4.08), 3-6 months post-loss at the second time point (M = 4.55, SD = 0.55; actual range: 3.29 - 5.95), and 5-8 months post-loss at the third time point (M = 6.39, SD = 0.41; actual range: 5.33 - 7.73).

Table 2

Means, Standard Deviations, and Correlations with Confidence Intervals for All Continuous Variables at Baseline

Variable	*M*	*SD*	1	2	3	4	5	6
1. Childhood Maltreatment	6.75	1.88						
2. Heart Rate Variability	23.16	18.58	.08 [−.02, .18]					
3. Age	68.19	9.22	−.25** [−.34, −.16]	.09 [−.01, .18]				
4. Education	0.74	1.09	.03 [−.07, .13]	.03 [−.07, .13]	.04 [−.06, .14]			
5. Months Since Passing	2.77	0.58	.04 [−.06, .14]	.18** [.08, .28]	.10* [.00, .20]	−.03 [−.13, .07]		
6. Baseline Grief Symptoms	22.41	12.13	.18** [.08, .27]	−.02 [−.12, .08]	−.23** [−.32, −.13]	−.09 [−.19, .01]	−.20** [−.29, −.10]	
7. Baseline Depressive Symptoms	18.06	11.26	.38** [.29, .47]	−.03 [−.13, .07]	−.30** [−.38, −.20]	−.08 [−.18, .02]	−.14** [−.23, −.04]	.64** [.57, .69]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014).

* indicates $p < .05$.

** indicates $p < .01$.

Table 3

Mixed-Model Analysis of Interaction of Childhood Maltreatment, Heart Rate Variability, and Time (Months Post-Loss) Predicting Grief Symptoms

	Unadjusted Model			Adjusted Model			Adjusted Model with Depressive Symptoms		
Predictors	***B***	***SE***	***CI***	***B***	***SE***	***CI***	***B***	***SE***	***CI***
(Intercept)	24.58***	1.23	[22.17, 27.00]	50.36***	10.11	[30.48, 70.25]	19.80*	8.40	[3.27, 36.32]
Childhood Maltreatment * Heart Rate Variability * Time (months post-loss)	−0.03***	0.01	[−0.04, −0.01]	−0.03***	0.01	[−0.04, −0.01]	−0.02***	0.01	[−0.04, −0.01]
Childhood Maltreatment * Heart Rate Variability	0.12*	0.06	[0.01, 0.23]	0.13*	0.05	[0.02, 0.24]	0.09	0.05	[−0.01, 0.18]
Childhood Maltreatment * Time (months post-loss)	−0.05	0.09	[−0.22, 0.12]	−0.05	0.09	[−0.22, 0.12]	−0.05	0.09	[−0.22, 0.12]
Heart Rate Variability * Time (months post-loss)	−0.01	0.01	[−0.03, 0.01]	−0.01	0.01	[−0.03, 0.01]	−0.01	0.01	[−0.03, 0.01]
Childhood Maltreatment	1.58*	0.66	[0.28, 2.88]	1.37*	0.67	[0.04, 2.70]	−0.17	0.59	[−1.34, 0.99]
Heart Rate Variability	0.03	0.07	[−0.11, 0.16]	0.06	0.07	[−0.08, 0.19]	0.05	0.06	[−0.06, 0.17]
Time (months post-loss)	−0.74***	0.16	[−1.05, −0.44]	−0.73***	0.16	[−1.04, −0.43]	−0.71***	0.15	[−1.02, −0.41]
Age				−0.22	0.12	[−0.45, 0.01]	−0.04	0.09	[−0.22, 0.14]
Gender				−1.48	2.19	[−5.78, 2.83]	−4.47***	1.70	[−7.82, −1.12]
Education				−0.70	0.92	[−2.51, 1.10]	−0.23	0.71	[−1.63, 1.16]
Months Since Passing				−3.83*	1.76	[−7.29, −0.37]	−1.37	1.38	[−4.07, 1.34]
Nicotine Use				0.53	5.34	[−9.98, 11.03]	1.45	4.09	[−6.59, 9.50]
Beta-Blockers				3.71	2.40	[−1.01, 8.43]	0.55	1.87	[−3.13, 4.24]
Depressive Symptoms							0.73***	0.08	[0.57, 0.88]
Random Effects									
σ^2	19.36			19.36			19.10		
τ_{00}	123.86 $_{\text{Subject}}$			119.15 $_{\text{Subject}}$			65.50 $_{\text{Subject}}$		
ICC	0.86			0.86			0.77		

Note:

* indicates $p < .05$

** indicates $p < .01$

*** indicates $p < .001$