The Association between Early-Life SES, Childhood Trauma Exposures, and Cardiovascular Responses to Daily Life Stressors in Middle-Aged Adults

by

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Objective: Dysregulation in physiological responses to stress may provide a mechanism through which childhood socioeconomic adversity negatively impacts health. While the evidence linking early life SES to dysregulated physiological reactivity is unclear, exposure to childhood trauma may be an important source of heterogeneity. The present study examined whether early life SES and childhood trauma interact to predict cardiovascular stress reactivity to daily life stressors.

Methods: A sample of 361 healthy, middle-aged adults (60% female, 80% White, 64% BA or greater, mean age of 52.58) completed a 4-day ecological momentary assessment protocol that measured task strain, social conflict, and blood pressure at hourly intervals throughout the day. Early life SES and childhood trauma exposure were measured at baseline.

Results: Multilevel models controlling for both momentary influences on blood pressure and age, sex, and race provided inconsistent evidence that early life SES and childhood trauma may interact in predicting cardiovascular reactivity. A three-way interaction emerged for DBP reactivity to social strain, where individuals who grew up in middle SES environments showed exaggerated blood pressure reactivity in the absence of trauma, and blunted reactivity when having experienced trauma ($\gamma_{14} =$ -1.02, p = .006). While the three-way interaction did not reach significance, results also demonstrated that low SES individuals with a history of trauma demonstrate blunted SBP reactivity to task strain compared to low SES individuals without a

history of trauma (γ_{13} = -2.41, p = .036). There was no significant SES-trauma interaction in predicting SBP reactivity to social conflict and DBP reactivity to task strain. Exploratory analyses explore how race and type of trauma impact these relationships.

Conclusion: This study suggests that early life SES and trauma exposure may interact under some circumstances, but results are not entirely consistent with the hypothesized pattern.

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Preface

The completion of this document would not have been possible without the support of many people in my personal and academic lives. I want to thank my parents, sister, friends, and cohort for their love, support, and patience on my academic journey. I want to thank my mentor, Dr. Thomas Kamarck, for his unyielding and expert guidance in developing a line of scientific inquiry and clearly and concisely communicating this inquiry. Lastly, I want to thank my committee members, Dr. Karen Matthews and Dr. Elizabeth Votruba-Drzal, for their expert advice and guidance throughout this process.

1.0 Introduction

Disparities in health are widespread throughout the world and SES is a particularly salient predictor of health inequalities. In the US, people of lower SES, defined by occupation, education, income, or subjective social status, are at risk for greater all cause morbidity and mortality (Chetty et al., 2016; Williams, Mohammed, Leavell, & Collins, 2010). Additionally, people with lower education, less income, and lower social status than others are at higher risk for Cardiovascular Disease (CVD), hypertension, and stroke at earlier ages (Hostinar, Ross, Chen, & Miller, 2017; Kerr et al., 2011; Leng, Jin, Li, Chen, & Jin, 2015). SES- driven differences in health are of great societal importance, as the magnitude of SES disparities in health continues to grow(Bosworth, 2018).

Accumulating evidence suggests that children raised in lower SES conditions show greater risk for mortality and morbidity as adults, regardless of adult socioeconomic status (Adler & Stewart, 2010; Ferraro, Schafer, & Wilkinson, 2016). In a large review of the literature, Galobardes and colleagues found that individuals of lower early life socioeconomic status were at greater risk of premature mortality than their higher SES peers, regardless of their socioeconomic position in adulthood (Galobardes, Lynch, & Smith, 2008). Although this premature mortality was not linked with any specific disorder as the larger cause of mortality, cardiovascular disease deaths were more common in the low childhood SES group relative to their higher SES counterparts. Similarly, a review by Pollitt et al demonstrated that low SES in childhood, independent of adult SES, placed individuals at a modestly increased risk for adult CVD (Pollitt, Rose, & Kaufman, 2005).

There are several hypothesized mechanisms through which SES may "get under the skin" to affect health, including associations with limited access to health resources, detrimental environmental agents, and increased psychological stress. From a resource/environmental

viewpoint, people of low childhood SES are more likely to live and grow up in impoverished areas, which are associated with higher levels of environmental toxins, greater noise levels, higher rates of crime, and less access to health resources such as affordable healthcare, groceries, and public parks than more advantaged neighborhoods (Adler & Snibbe, 2003; Cohen, Janicki-Deverts, Chen, & Matthews, 2010), all of which have the potential to negatively impact health (Montez, Bromberger, Harlow, Kravitz, & Matthews, 2016). In addition to these physical environment factors, psychosocial factors associated with low SES may be health-impairing, and thus may serve as possible pathways through which SES impacts health. Low SES environments are associated with greater risks of familial conflict, harsh and inconsistent parenting, neighborhood crime and violence, and socioeconomic uncertainty and worry (Cohen et al., 2010). These psychosocial stressors may also negatively impact physiological development. The "life stress hypothesis" postulates that socioeconomic status impacts health through differential exposure and vulnerability to life stressors (Baum, Garofalo, & Yali, 1999). In terms of exposure, the hypothesis argues that individuals of lower socioeconomic status confront more social, psychological, and physical stressors than individuals of higher socioeconomic status. Over the course of a lifetime, this heavy environmental and psychological burden may accelerate the progression of disease.

Moreover, the volume of early life psychosocial stressors experienced by individuals of low SES can promote dysregulation of physiological systems, which may in turn increase the later-life burden of disease. Generally, many physiological systems are still relatively plastic in early life, and exposure to psychosocial stressors during this period may alter nervous, endocrine, and immune systems in persistent and permanent ways (Repetti, Taylor, & Seeman, 2002). From an evolutionary and developmental standpoint, the form that these alterations take may vary as a function of early life environmental exposure.

Building on existing theory in the developmental and evolutionary psychological literature, Ellis and colleagues have proposed that individual differences in the patterning of physiological stress responding forms as an adaption to the social and physical conditions present during development. This theory, termed the Adaptive Calibration Model (ACM), builds off of two developmental theories: conditional adaption and life history. The notion of conditional adaption refers to the idea that an organism will modify its own developmental trajectory in a manner that would represent the most adaptive pattern of response to the expected future environment. For example, conditional adaption theory suggests that when individuals are faced with stressful environments in early life, their development is directed towards strategies that are adaptive under stressful conditions (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). Life history theory posits that humans are fundamentally concerned with the optimal allocation of time and energy to two competing life purposes – somatic efforts (bodily maintenance and growth) and reproductive efforts. Due to limits in available time and energy, life history theory asserts that, as humans, we are constantly looking to optimize our ability to function in each of these areas. The optimization specifics, however, are dependent on the resources, threat levels, and stability in the environments in which we are reared. For example, an individual reared under high levels of extrinsic mortality-morbidity is better served to develop faster life history strategies (i.e. insecure social attachments, early pubertal development) that stress current maintenance rather than long-term reproductive efforts (Belsky, Steinberg, & Draper, 1991). In contrast, an individual reared under stable environments with low levels of extrinsic mortality-morbidity will place greater energy into longer-term reproductive efforts (i.e. investment in stable social relationships, later pubertal development) and slower life history strategies. Both life history and conditional

adaptation theories emphasize the role of early life environments as a source of information facilitating the development of adaptive social and physiological behaviors.

The ACM combines the theories of life history and conditional adaption to posit, in part, that individuals adapt optimal stress response systems dependent on the safety and predictability of their childhood environments. For an individual reared in a relatively safe and supportive environment, a moderately active physiological reactivity profile would allow the individual to respond adequately to emergencies, while also preserving energy for long-term fitness. However, in accordance with this model, a dangerous or unpredictable environment may cause an individual to adopt a highly reactive stress response profile, as this would increase the individual's ability to more effectively respond to short-term danger. In this environment, an individual may be better served placing resources into short-term growth and maintenance activities, as longer-term reproductive fitness is uncertain. In extremely unpredictable or dangerous environments, however, low reactivity may be favored. According to the ACM, individuals reared in very traumatic environments may adopt an unresponsive approach towards acute stressors, as constant activation of the stress response system may incur greater physiological costs with few short-term benefits. In these cases, a profile characterized by behavioral and physiological withdrawal, although it may impede the individual's ability to respond to acute social and physical insecurities, may be the optimal strategy because of the frequency and severity of expected stressors and the resource economization associated with withdrawal (Del Giudice, Ellis, & Shirtcliff, 2011). The ACM asserts that physiological stress reactivity develops as a direct consequence of the stability and safety of one's early life environment as the competing need for both acute adaptation and long-term fitness.

As alluded to in the above model, one metric of physiological dysregulation is the level of (either exaggerated or blunted) cardiovascular response to stress. The reactivity hypothesis asserts that prolonged or exaggerated cardiovascular reactivity to psychological stress promotes the development of CVD, in part, due to structural and functional changes in the coronary arteries that may accompany frequent or prolonged hemodynamic responses (Krantz, Manuck, & Wing, 1986; Obrist, 1981). Individual differences in the patterning and magnitude of CVR to stressors have been shown to be reliable over time (Manuck, Kamarck, Kasprowicz, & Waldstein, 1993). Empirically, exaggerated and prolonged cardiovascular responses to acute laboratory stressors have been linked with endothelial dysfunction (Ghiadoni et al., 2000), elevated fibrinogen and interleukin 6 (Steptoe & Marmot, 2006), and hypertension and coronary heart disease (Carroll et al., 2012; Chida & Steptoe, 2010; Panaite, Salomon, Jin, & Rottenberg, 2015; Treiber et al., 2003). While early life SES has been consistently linked with disease, the connection between SES and cardiovascular stress responding is inconclusive.

Lower childhood SES, typically measured through parental education and income, has been associated with both exaggerated and blunted cardiovascular reactivity to stress. To our knowledge, ten total studies have investigated childhood socioeconomic status in relation to cardiovascular reactivity (see Table 1) (Boylan, Jennings, & Matthews, 2016; Chen, Langer, Raphaelson, & Matthews, 2004; Chen & Matthews, 2001; G. W. Evans, Exner-Cortens, Kim, & Bartholomew, 2013; G. W. Evans & Kim, 2007; Gump, Matthews, & Räikkönen, 1999; Jackson, Treiber, Turner, Davis, & Strong, 1999; Kapuku, Treiber, & Davis, 2002; Musante et al., 2000; Wilson, Kliewer, Plybon, & Sica, 2000). Of these, five reported an inverse relationship, or exaggerated cardiovascular reactivity in relation to lower socioeconomic status (Chen et al., 2004; Chen & Matthews, 2001; Gump et al., 1999; Kapuku et al., 2002; Wilson et al., 2000). For instance,

one study performed by Chen and colleagues found that adolescents reared in lower SES environments showed heightened reactivity to videos of negative and ambiguous life situations when compared to adolescents of higher SES (Chen et al., 2004). In contrast, two studies identified reduced reactivity in relation to lower early life socioeconomic status (G. W. Evans et al., 2013; G. W. Evans & Kim, 2007; Musante et al., 2000). The remaining three studies found either null or contradictory effects (Boylan et al., 2016; G. W. Evans et al., 2013; Jackson et al., 1999). Of these, two studies demonstrated no association between childhood SES and cardiovascular reactivity (Boylan et al., 2016; G. W. Evans et al., 2013), while one study showed that SES interacted with race, such that White adolescents demonstrated exaggerated reactivity and Black adolescents demonstrated blunted reactivity (Jackson et al., 1999). In this way, the literature linking childhood SES and cardiovascular reactivity is decidedly mixed. A recent meta-analysis investigating the relationship between general SES (whether that be adult or child) and cardiovascular reactivity concluded that were is no overall relationship between the two, although they did find evidence for prolonged recovery times for lower SES individuals (Boylan, Cundiff, & Matthews, 2018). While it is entirely possible that the mixture of results linking SES and reactivity suggests no overall relationship, it is also possible that this relationship is moderated by a third variable, such as exposure to adverse childhood experiences, as outlined below.

In addition to the literature on childhood SES and cardiovascular reactivity, a parallel line of research in children investigates associations between adverse childhood events, defined as exposure to traumatic experiences such as abuse or neglect, and cardiovascular reactivity to laboratory stressors. Unlike the literature on childhood SES and reactivity, findings in this area tend to be more consistent. To our knowledge, eight total studies have investigated the association between childhood adversity and cardiovascular reactivity (B. E. Evans et al., 2015; Gooding,

Milliren, Austin, Sheridan, & McLaughlin, 2015; Heim et al., 2000; Lovallo, Farag, Sorocco, Cohoon, & Vincent, 2012; Luecken, Kraft, Appelhans, & Enders, 2009; McLaughlin et al., 2015; Murali & Chen, 2005; Voellmin et al., 2015), although more have investigated HPA reactivity. Of these, six studies demonstrate that children with increased exposure to adverse life events demonstrate lower cardiovascular reactivity to acute stressors than their peers with lower exposure to adverse experiences (Gooding et al., 2015; Lovallo et al., 2012; McLaughlin et al., 2015; Murali & Chen, 2005; Voellmin et al., 2015). One study demonstrated an positive relationship between childhood trauma and reactivity (Heim et al., 2000) and another demonstrated no overall relationship (Luecken et al., 2009). In the former investigation, 23 women with major depression disorder were compared to 26 matched controls (Heim et al., 2000). As Lovallo has previously suggested (Lovallo, 2013), individuals with a mental illness may respond to stressors differently from healthy people, accounting for these results. The latter study by Luecken and colleagues recruited 43 undergraduate students from bereaved (loss of a parent before the age of 17) and 48 from non-bereaved families, and investigated their emotional and blood pressure responses to self-report stressors every 30 minutes over a 24-hour ambulatory period (Luecken et al., 2009). While the Luecken et al., (2009) study demonstrated no difference in cardiovascular reactivity to stress across the two samples, the short day-long ambulatory measurement period may have been insufficient to allow sampling of stressor exposure, and the small sample sizes recruited for this study may decrease its generalizability as well. Although not entirely conclusive, the evidence from this literature generally suggests (6/8 studies) an inverse relationship between adversity and cardiovascular response to stress, where greater childhood adversity associates with blunted cardiovascular response to acute stress.

Exposure to a materially disadvantaged socioeconomic background is often seen as one specific type of childhood adversity. While increased childhood trauma, also a form of adversity, is more prevalent among low SES environments, not all low SES environments are associated with high rates of childhood trauma (Goodman, Miller, & West-Olatunji, 2012). Variability in childhood trauma exposure may be an important source of variability in stress-related responding among low SES individuals, the effects of which remain to be explored. In line with the Adaptive Celebration Model previously mentioned, severe childhood traumas may be associated with blunted physiological reactivity to stress, whereas mild adversity (for example, that which is present in low SES environments with low physical resources but moderately stable psychosocial environments) could be linked to exaggerated physiological reactivity to stress (Del Giudice et al., 2011). In this way, it may be that the category of low SES individuals includes two separate clusters: low SES individuals with a history of childhood trauma who demonstrate blunted cardiovascular reactivity and low SES individuals with no history of childhood trauma who demonstrate exaggerated cardiovascular reactivity relative to their high SES counterparts.

The purpose of the present study is to assess whether individuals raised in low SES environments show dysregulated cardiovascular reactivity profiles as adults. In line with research in the childhood and adolescent literature, the study also seeks to uncover whether severity of childhood exposures, as operationalized through presence or absence of childhood traumatic events, moderates the effect of SES on reactivity.

Most of the previous work on this topic has centered on reactivity defined in the laboratory. In contrast, the current study proposes use of ecological momentary assessment (EMA), a behavioral and psychosocial data collection method in which information on an individual's current state is gathered as it occurs in the natural environment, typically through use of portable

devices programmed for recurrent data collection (Shiffman, Stone, & Hufford, 2008). Methodologically, there are three advantages of using EMA to investigate SES differences in physiological stress reactivity. First, momentary measures of stress may provide a more representative depiction of daily life experiences than global self-report assessments. Second, if we assume that low SES individuals may be less comfortable than their more affluent counterparts in a formal laboratory setting, it is possible that ambulatory assessments may permit us to assess stress reactivity in a manner than is more equivalent across the SES gradient. Third, psychosocial factors and health risks measured through EMA protocols may be more strongly associated with biological assessments (Conner & Barrett, 2012) and markers of disease than those measured using global retrospective self reports, again, potentially because EMA measures provide a more representative 'slice of life' (Kamarck et al., 2002; Kamarck, Muldoon, Shiffman, & Sutton-Tyrrell, 2007; Pickering, Harshfield, Devereux, & Laragh, 1985). In their 2007 study, Kamarck and colleagues found that EMA measures of job strain predicted intima-medial thickness, a measure of preclinical vascular disease, at baseline and at a three year follow up while traditional global self-report measures of job strain did not (Kamarck et al., 2007). They repeated this finding in their six-year follow-up of the same population (Kamarck, Shiffman, Sutton-Tyrrell, Muldoon, & Tepper, 2012). Again, EMA measures of job strain predicted intima-medial thickness at six years, while traditional self-report measures of job strain did not. Use of an EMA protocol in the current study may allow us to link stress with psychological responding in the natural environment while avoiding potential SES-driven confounds when examining the contextual association between SES, childhood trauma, and cardiovascular response to stress.

Altogether, previous research has suggested that individuals raised in lower socioeconomic environments may experience a variety of environmental, physical, and physiological stressors

which, in turn, lead to physiological dysregulation and increased health burden. While previous research has shown mixed evidence for childhood SES differences in cardiovascular reactivity to stressors, it may be that differential exposure to childhood adversity may account for these mixed findings. Preliminary theoretical and empirical work suggests that exposure to mild adversity in childhood, such as that associated with low SES, may lead to exaggerated cardiovascular response to stressors. Simultaneously, exposure to severe adversity in childhood, such as that associated with traumatic experiences, may lead to blunted cardiovascular response to stressors. To our knowledge, this interaction between SES and childhood trauma on cardiovascular reactivity has yet to be examined empirically in a single study. Furthermore, while these associations have been examined in the laboratory, they have yet to be assessed in daily life. Knowledge of the specific mechanisms through which socioeconomic variation in response to daily life stressors may unfold can help us in crafting interventions focused on mitigating disparities in health. The present report assesses whether childhood SES associates with the effect of daily life stress on concurrently assessed blood pressure as measured through ambulatory assessment, and whether childhood adversity moderates this association.

2.0 Methods

2.1 Participants

Participants were drawn from the Study of Health and Interactions in the Natural Environment (SHINE) conducted through the Behavioral Medicine Research Group at the University of Pittsburgh. This study recruited a diverse sample of healthy adults from the Pittsburgh area. SHINE participants were recruited between the years of 2014 and 2018. Eligibility criteria for the SHINE study required participants to be between the ages of 40 and 65, and excluded those with history of cardiovascular disease, schizophrenia or bipolar disease, chronic hepatitis, renal failure, neurological disorder, lung disease requiring drug treatment, or stage 2 hypertension, those using insulin, glucocorticoid, antiarrhythmic, antihypertensive, lipid-lowering, psychotropic, autonomically active, or prescription weight loss medications, and those reporting current pregnancy. The study was approved by the University of Pittsburgh Institutional Review Board, and all participants were administered informed consent before enrollment. Participants were compensated between $350 - $400 for their participation.

2.2 Procedure

Individuals completed a total of four laboratory visits. In visit 1, participants provided demographic and medical information and verified their eligibility for the study. At visit 2, participants completed a variety of questionnaires not relevant to this study. At visit 3, participants were trained for all aspects of the ambulatory phase of the study. They were instructed on use of an automated blood pressure cuff (Oscar 2 oscillometric monitor, Suntech Medical Inc, Morrisville, NC), an accelerometry-based physical activity device (Sensewear Pro3, Body Media, Pittsburgh, PA), and an electronic diary. Following the training, the participants completed a 7-10-day ambulatory monitoring protocol. Four of these days constituted “full monitoring” days in

which the participants responded to hourly surveys, gathered physical activity information, and obtained hourly blood pressure measurements. On each of the other, "partial monitoring days," participants responded to morning and evening questionnaires. Following the ambulatory monitoring period, participants returned to the laboratory for a debriefing. During this fourth and final visit, participants also completed various questionnaires including the Childhood Trauma Questionnaire.

2.3 Measures

2.3.1 Socioeconomic Status

Participants completed a self-report demographic questionnaire which included questions regarding their parents' occupation and education level, current household income, household size (i.e., number of people living in home), and education level. Parental education was separately assessed for each individual's mother and father, and was scored on a 12-point scale (i.e., 1 = less than eighth grade, 2 = eighth grade, 3 = Junior high school (9th grade), 4 = Partial high school (11th or 12th grade), 5 = GED, 6= high school diploma, 7 = some technical training, 8 = some college, no degree, 9 = associates degree, 10 = bachelor's degree, 11 = master's degree, 12 = MD, PhD, JD, PharmD). Early life SES was conceptualized in the present study as the highest level of education level achieved by the most-educated parent. Based on this, individuals were divided into three dummy-coded groups: low early life SES (most-educated parent had a high school degree or less), middle early life SES (most-educated parent had some college experience but no four-year degree), and high early life SES (most-educated parent had at least a college degree). 35% of the sample grew up in a household in which the most-educated parent had a high school degree or less, 28% were from a household in which the most-educated parent completed some

college or an associate's degree and 35% were from a household in which the most-educated parent had a bachelor's degree or higher.

2.3.2 Childhood Trauma

During visit 4, participants completed the Childhood Trauma Questionnaire- Short Form (CTQ) (Bernstein et al., 2003). This survey consists of a total of 28 questions each rated on a 1-5 Likert scale, and it assesses the presence and severity of childhood trauma. An example question is "People in my family hit me so hard that it left me with bruises or marks," rated from 1 (never) to 5 (very often true). The scale provides five sub-scores: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. The CTQ subscales can be considered as continuous scales or dichotomized occurring to clinical cut points. In this study, the Walter et al (1999) scores were used for each of the scales to designate the presence of trauma exposure as follows: emotional abuse ≥ 10, physical abuse ≥ 8, sexual abuse = 8, emotional neglect = 15, physical neglect = 8 (Walker et al., 1999). Presence of childhood trauma was operationalized dichotomously as the exposure to or absence of one or more of these subdomains of trauma. This measure has been found to have good criterion-related validity (Bernstein et al., 2003).

2.3.3 Ambulatory Blood Pressure

Ambulatory blood pressure (ABP) was assessed at consistent, hourly intervals across the waking period of each monitoring day. Both systolic (SBP) and diastolic (DBP) readings were gathered. All ABP observations associated with an error code (e.g: leaky cuff, low battery) were deleted prior to analyses. In accordance with criteria put forth by Marler, Jacob, Lehoczky, and Shapiro (1988), ABP readings were removed as invalid whenever SBP < 70 mmHg or > 250 mmHg, DBP < 45 mmHg or > 150 mmHg, or SBP/DBP < [1.065 + (.00125 X DBP)] or > 3 (Marler, Jacob, Lehoczky, & Shapiro, 1988).

2.3.4 Daily Life Stressors

After each ABP measurement, participants completed an ambulatory diary questionnaire on an electronic diary. The diary presented about 50 multiple choice questions regarding recent daily life stressors and social interactions. Using these diary questions, the two types of life stressors were obtained.

In this study, two types of life stressors were measured: task strain and social conflict. The Karasek job strain model posits that environments high in psychological demands but low in decision control or latitude are associated with elevated cardiovascular risk (Karasek Jr, 1979). Empirically, work from this lab has shown that individuals who rate their daily activities, through use of EMA assessments, as high in demand and low in control show elevated risk for subclinical atherosclerosis (Kamarck, Li, Wright, Muldoon, & Manuck, 2018; Kamarck et al., 2004; Kamarck et al., 2007; Kamarck et al., 2012). Social conflict, another well-studied daily life stressor, has likewise been associated with elevated risk of atherosclerosis and coronary heart disease (De Vogli, Chandola, & Marmot, 2007; Gallo, Bogart, Vranceanu, & Walt, 2004; Joseph, Kamarck, Muldoon, & Manuck, 2014; Smith, Uchino, Berg, & Florsheim, 2012). In a previous study out of our lab, frequent exposure to social conflict combined with increased cardiovascular reactivity to EMA-assessed social conflict associated positively with intima-medial thickness(Kamarck et al., 2018).

2.3.4.1 *Task Strain.*

On each interview, participants were asked to "think about mental and physical activity in the past 10 minutes," then to respond to three items measuring the degree of psychological demand associated with this activity, and two items measuring relative control in the same context. Demand was captured through asking whether the activity required "working hard?", "working fast?", and

whether the individual “juggled several tasks at once?”. Control was ascertained through items asking if the participant felt “in command?” and if they “handled [themselves] well?”. Of note, this assessment of control is distinct from that previously utilized in our lab (Kamarck et al., 2018; Kamarck et al., 1998). Items were scored on a 1-10 Likert scale. In accordance with the job strain model, situations with “task strain” are those high in demand and low in control. In this sample, situations rated above the sample median in demand and below or equal to the sample median in control were coded “1” as instances of task strain. All other situations (those low in demand and those high in control) were coded “0” as instances absent of “task strain”.

2.3.4.2 *Social Conflict.*

Participants responded to three items regarding their most recent social interaction, and whether that interaction was marked by conflict. Specifically, the study asked if “someone was insensitive to you,” “someone interfered with your efforts,” and if “someone made you tense.” These items were again scored on a 10-point Likert scale. Only interactions which occurred within the 10 minutes preceding blood pressure measurement were included in the analyses. Social conflict was operationalized as the mean of these three items.

2.4 Covariates

2.4.1 Time-Dependent Covariates

Several factors known to influence blood pressure were measured and included as time-dependent covariates. In the hourly interview ratings, participants were asked about their current posture (standing, sitting, or lying down), perceived level of temperature comfort (comfortable, too hot, or too cold), speaking status (speaking or not speaking), self-report physical activity (limited, light, moderate, or vigorous), and recent consumption of food, alcohol, or caffeine (yes or no). These variables have previously been shown to be associated with ABP changes (Kamarck

et al., 1998), and were included in the analyses involving stress-related changes in ambulatory blood pressure.

2.4.2 Between-Subject Covariates

In addition to the momentary covariates, self-reported race, sex, and age were included as between-person covariates. These were all assessed in the demographic questionnaire obtained in visit 1. Race (white vs black, indigenous, people of color (BIPOC)) and sex (male vs female) were dichotomized. Age was added as a continuous variable.

2.5 Plan of Analysis

Analyses were performed utilizing R Statistical Software(Team, 2018). Multilevel modeling (MLM) procedures were used to examine Aims 1 and 2. MLM models correct for nested data structures, allowing for representation of within-person changes over multiple time points and modeling of how individuals differ in these changes from one another. All analyses were run using the "nlme" statistical package for linear and nonlinear mixed effects models(Pinheiro et al., 2017) in R software 3.5.1. All models utilized log-likelihood methods, or "ML", for estimating fixed and random effects simultaneously. Likewise, all models were run with a continuous AR1 autocorrelation structure that adjusts for error by characterizing time between samples as continuous. As recommended by the multivariate literature, task strain and social conflict were both person-centered prior to analyses(Enders & Tofighi, 2007). This process created two variables for each stress measure: within-person measures of strain added at level 1 and between-person measures of strain added at level 2. All independent variables, including covariates, were centered at either the person (level-1 variables) or group (level-2 variables) levels prior to statistical modeling.

Analyses were performed separately for each measure of strain and each of the two types of blood pressure outcome such that a total of four models were fit to assess the impact of early-life SES and childhood trauma on: SBP reactivity to task strain, SBP reactivity to social conflict, DBP reactivity to task strain, and DBP reactivity to social conflict. For each of the aforementioned models, analyses followed five steps. First, unconditional multilevel models were estimated to inspect the variance in ABP accounted for by within and between-person differences. Second, level-1 predictors (both person-centered stress and time-varying covariates) were added as fixed effects to the original unconditional model in order to test the acute effect of stress on ABP in this sample. A random effect of person-centered stress (either task strain or social conflict) was also added to allow associations between stress and ABP to vary between people. Third, all level-1 predictors and covariates were added as main effects and childhood trauma and early life SES were added as cross-level interactions in order to examine the main effects of childhood trauma and early life SES on ABP and ABP reactivity. Fourth, the two-way interactions between early life SES and childhood trauma on blood pressure were added as fixed effects. This fourth model step was done in order to provide a comparison to test significance for the fifth model step. In a fifth step, the three-way cross-level interaction terms between SES, childhood trauma, and the within-person effect of stress were added in order to examine for the presence of a three-way interaction (see Figure 1 for complete model). Model comparisons occurred between each step, and were based on likelihood ratio tests (LRTs).

The results of the third step were used to test study aim 1 and the results of the fifth step were interpreted to test study aims 2. These fully-identified models included (a) level 1 analyses with all fixed person-centered time-dependent covariates (posture, temperature comfort, speaking status, self-report physical activity, food/caffeine/alcohol consumption), (b) person-centered stress

predictors added as fixed and random effects, (c) level-2 fixed effects of early life SES, childhood trauma, and their interaction terms, and (d) all between-person covariates. In models where the 3-way interaction terms significantly increased variance accounted for in the model (significant LRT between steps 4 and 5), simple slopes were investigated by re-centering the reference groups for the childhood education and childhood abuse variables and examining the relevant 2-way interactions, following recommendations by Aiken and West (1991) (Aiken & Stephen, 1991)).

In order to investigate whether associations between social conflict and ABP are independent from the simultaneous influence of task strain, two follow-up MLMs were run. In these, the between-person and within-person measures of social conflict and task strain were jointly included in models. As before, between-person social conflict and task strain were included as level-2 fixed effects and within-person social conflict and task strain were included as fixed and random effects at level 1. All covariates, early life SES, and childhood trauma were added as in previous models. All cross-level interactions between early life SES, childhood trauma, and social conflict/task strain were included as fixed effects.

2.5.1 Exploratory Analyses

In models in which a significant three-way interaction appeared between childhood trauma, early life SES, and cardiovascular reactivity, exploratory analyses were conducted to examine whether the types of childhood trauma exposure interact differently with daily stressor exposure and SES to predict cardiovascular reactivity. Exploratory analysis involved fitting an MLM almost equivalent to that of the analogous step 5 MLM. Here, the "childhood trauma" variable was instead divided into four distinct groups: history of abuse only, history of neglect only, history of both abuse and neglect, or history of neither abuse nor neglect.

3.0 Results

From the original sample of 391, 24 individuals were removed due to missing trauma history data and 5 were removed for incomplete information on their parental education. One additional participant was removed for having less than two ABP observations. The final analytic sample was composed of 361 healthy, employed adults (60% female, 80.0% white, 64% bachelor's degree or greater, mean age of 52.6). In total, 41% of the sample reported exposure to some form of childhood trauma. See table 3 for more demographic information. Each individual completed an average of 46 ambulatory measurements over the course of the four days (range: 9 - 67), and, on average, 26 of these ambulatory measurements occurred within ten minutes of a social interaction (range: 2 - 57). In total, task strain analyses included 16,491 observations, and social conflict analyses included 9,519 observations.

In depth output for each step for all four models is depicted in Tables 4 – 7. The simple unconditional models (step 1) demonstrated that 60.3% of the variance in SBP and 54.1% of the variance in DBP occurs at the between-person level. There is a significant nested structure in ABP values in this dataset and the use of MLM is warranted.

3.1 SBP Reactivity to Task Strain

When compared to the unconditional model, the addition of level-1 predictors led to a significant increase in SBP variance, indicating significant influence of momentary factors on SBP (L.ratio = 1104.33, $p < .001$). This second step demonstrated a significant fixed effect of within-person task strain emerged such that, after controlling for the time-varying covariates, a one point increase in task strain associated with a 1.38 mmHg increase in SBP, on average ($\gamma_{10} = 1.38$, $p < .001$). There was also a significant random effect of task strain on SBP ($\tau_{11} = 3.36$, 95% CI: [0.57, 19.75]), demonstrating that people vary in their SBP response to task strain.

Level-2 covariates and two-way cross level interactions were then added to the model in a third step which explained additional variance (L.ratio = 58.25, $p < .001$). That being said, no significant cross-level interactions emerged. In step four, two way interactions between early life SES and childhood trauma on SBP were added. This step did not explain additional variance from step 3, and was completed as a comparison for step 5. Three-way interactions were then added to the model in step 5. While no overall three-way interaction emerged (L.ratio = 2.59, $p = .27$), a significant simple effect of childhood trauma appeared for the low SES group (γ_{13} = -2.41, $p = .036$). More specifically, individuals raised in a low SES environment with a history of childhood trauma showed reduced SBP reactivity to task strain than their low SES counterparts without a history of childhood trauma (see Figure 2 & Table 4).

3.2 DBP Reactivity to Task Strain

When compared to the unconditional model, the addition of level-1 predictors led to a significant increase in variance explained, indicating significant influence of momentary factors on DBP (L.ratio = 1590.19, $p < .001$). A significant fixed effect of within-person task strain emerged such that, after controlling for the time-varying covariates, a one point increase in task strain was associated with a 0.85 mmHg increase in DBP, on average ($\gamma_{10} = 0.85$, $p < .001$). Likewise, there was a significant random effect of task strain on DBP (τ_{11} = 2.89, 95% CI: [1.42, 5.87]).

The addition of level-2 covariates and two-way cross-level interactions in step three led to a significant increase in variance explained (L.ratio = 53.99, $p < .001$). That being said, no significant cross-level interactions emerged between either early life SES or childhood trauma and within-person task strain. Step 4 was modeled and compared to step 5. No significant three-way

interaction between early life SES, childhood trauma, and DBP reactivity emerged in step 5 (L.ratio = 0.44, p = .80; see Figure 3).

3.3 SBP Reactivity to Social Conflict

When compared to the unconditional model, the addition of level-1 predictors led to a significant increase in SBP variance explained, indicating a significant influence of momentary factors on SBP (L.ratio = 506.84, p < .001). A significant fixed effect of within-person social conflict emerged such that, after controlling for the time-varying covariates, a one point increase in social conflict was associated with a 0.42 mmHg increase in SBP, on average ($\gamma_{10} = 0.40$, p < .001). A significant random effect of social conflict on SBP (τ_{11} = 0.38, 95% CI: [0.12,1.20]) indicated that individuals vary in terms of their within-person relationship between social conflict and SBP.

The addition of level-2 covariates and two-way cross-level interactions in step three led to a significant increase in variance explained (L.ratio = 50.89, p < .001). That being said, no significant cross-level interactions emerged between either early life SES or childhood trauma and within-person social strain. Two way interactions between early life SES and childhood trauma were added in a forth step in order to set up a comparison for step five. There was no further increase in variance explained when the three-way interactions were added to the model in step five (L.ratio = 0.19, p = .91; see Figure 4).

3.4 DBP Reactivity to Social Conflict

In the second step of modeling, level-1 predictors were added as fixed effects and within-person social conflict was added as a random effect. There was a significant increase in DBP variance explained in this model, indicating significant influence of momentary factors on DBP (L.ratio = 738.11, p < .001). Specifically, a significant fixed effect of within-person social conflict

emerged such that, after controlling for the time-varying covariates, a one point increase in social conflict was associated with a 0.24 mmHg increase in DBP, on average ($\gamma_{10} = 0.24$, $p < .001$). Likewise, there was a significant random effect of social conflict on DBP ($\tau_{11} = 0.25$, 95% CI: [0.09,0.67]). In other words, individuals varied in terms of the within-person relationship between social conflict and DBP.

In step 3, all level-2 predictors and covariates were then added to the model as main effects and two-way cross level interactions (L.ratio = 53.72, $p < .001$). No significant two-way cross level interaction emerged between early life SES or childhood trauma and DBP reactivity. Step four was modeled as a comparison to step 5. The addition of three-way interactions in step five caused a significant increase in variance explained, indicating a significant three-way interaction between early life SES, childhood trauma, and DBP reactivity to social conflict (L.ratio = 7.62, p =.021; see Figure 5). The model showed, specifically, a three-way interaction term between childhood trauma, middle early life SES (compared to low SES), and the influence of social conflict on DBP ($\gamma_{14} = -1.02$, $p = .006$).

Analysis of simple slopes indicated that there were significant differences in DBP reactivity for individuals who grew up in a middle SES environment, where those without a history of childhood trauma demonstrated significantly greater reactivity than those with a history of childhood trauma ($\gamma = -0.80$, $p = .003$). There were no childhood trauma differences in DBP reactivity for the individuals who grew up in high SES or low SES environments ($\gamma = -0.23$, p = .32; $\gamma = 0.17$, $p = .49$, respectively). Likewise, for all individuals without a history of childhood trauma, those who grew up in a middle SES environment showed higher DBP reactivity than those who grew up in a low SES or high SES environment ($\gamma = 0.69$, $p = .004$; $\gamma = 0.48$, p =.047,

respectively). Among the individuals exposed to childhood trauma, there were no significant SES-driven differences in DBP reactivity.

3.5 Exploratory Analyses

The above DBP reactivity to social conflict results deviated from our expectations. While the expected 3-way interaction was observed, we did not expect an exaggerated reactivity profile for the middle SES group compared to the low and high SES groups. In order to understand the results observed, we examined how the characteristics of the middle SES group differed from the other groups. We found that the racial composition and childhood trauma type differed between SES groups, where the middle SES group had a greater proportion of BIPOC individuals and individuals with a history of both abuse and neglect than the other two SES groups (see Table 3). Likewise, we wondered whether heterogeneity in the middle SES group itself (which includes parents with an associates/ technical degree and those who initiated but did not complete college) may manifest in different childhood socioeconomic environments and reactivity profiles. These sources of heterogeneity were examined through exploratory analyses investigating the impact of type of trauma exposure on reactivity results and how race and middle SES subgroups influence ABP reactivity to social conflict.

As initially proposed, the model for which a three-way interaction emerged between early life SES, childhood trauma, and ABP reactivity to a stressor was broken down by type of childhood trauma (abuse only, neglect only, both abuse and neglect, neither abuse nor neglect). One exploratory MLM was run on DBP reactivity to social conflict. The MLM revealed a significant three-way interaction term between social conflict, middle SES, and a history of both childhood neglect and abuse, (γ = -1.31, p = .004; see Table 8). None of the other three-way interaction terms were significant. Results suggest that the observed SES and trauma-contingent differences in DBP

reactivity to social conflict manifest only for the individuals exposed to both childhood neglect and trauma.

The influence of racial identity was investigated in combination with early life SES, childhood trauma, and within-person reactivity to social conflict to investigate whether interactive effects observed vary by race. These interaction models mirrored the full MLMs assessing DBP and SBP reactivity to social conflict except for the addition of a four-way interaction term between race (white versus BIPOC), early life SES, childhood trauma, and within-person reactivity and all relative lower-order interaction terms. Results demonstrated no significant four-way interaction in predicting either DBP or SBP (L.ratio = 8.69, p = .34; L.ratio = 6.67, p = .57, respectively). The effect of early life SES and childhood trauma does not appear to differ by racial identity.

To investigate the presence of heterogeneity within the middle early life SES group, middle SES was broken down into two sub-categories corresponding to a completed associates/technical degree or some college. The full model investigating DBP reactivity to social conflict demonstrated three-way interactions for both the group with a parent who completed an associates/technical degree and the group with a parent who attended some college without a degree (γ = -1.07, p = .010; γ = -1.01, p =.059, respectively). Analysis of simple effects showed that both the middle SES associates degree group and middle SES some college group demonstrated trauma-based differences in reactivity, where group individuals without a history of trauma showed exaggerated reactivity when compared to those with a history of trauma (γ = -0.90. p = .007; γ = -0.83, p = .077, respectively). In the absence of trauma, only the middle SES associates degree group demonstrated exaggerated reactivity compared to their counterparts reared in high and low SES environments (γ = 0.73, p = .010; γ = 0.92, p = .001, respectively). This finding suggests that early life SES and childhood trauma interact with DBP reactivity to social

strain for both middle SES groups, although the specific manifestations of this interaction differ slightly.

3.6 Joint Strain Modeling

In order to evaluate the influence of social conflict on ABP above and beyond the influences of task strain, two joint models were run which simultaneously incorporated interactions with both task strain and social conflict. Both joint models resulted in interaction coefficients very similar to those observed in the social conflict only models. For example, the three-way interaction between momentary social conflict, childhood trauma, and early life SES on DBP in the joint model (γ = -1.03, p = .005, see Table 9) resembled that in the initial model just investigating social conflict (γ = -1.02, p = .006). In other words, the addition of early life SES and trauma effects on task strain do not appear to impact the effects of early life SES and trauma on social conflict.

4.0 Discussion

The present study sought to examine the impact of childhood SES and childhood trauma on cardiovascular reactivity to daily life social conflict and task strain in healthy mid-life adults. It was hypothesized that there would be no overall association between early life SES and cardiovascular reactivity, but instead an interactive effect between early life SES and exposure to childhood trauma. Results are in line with hypotheses and previous literature demonstrating no overall effect of SES on cardiovascular reactivity to stressors(Boylan et al., 2018). Interestingly, the present study differed from some previous work(Gooding et al., 2015; Lovallo et al., 2012; McLaughlin et al., 2015; Murali & Chen, 2005; Voellmin et al., 2015) in that we found no main effects of childhood trauma on cardiovascular reactivity to stress in daily life. Rather, as hypothesized, the present study found some evidence suggesting that exposures to childhood trauma may interact with childhood socioeconomic disadvantage to predict stress responsivity. This being said, the observed interactions were not entirely consistent with predictions. While we expected low SES individuals without a history of childhood trauma to show exaggerated reactivity compared to the high SES no trauma group, this simple effect did not materialize. Rather, one model demonstrated that the middle SES individuals without a history of childhood trauma demonstrated exaggerated cardiovascular reactivity compared to the other SES groups. Likewise, while we hypothesized that individuals who grew up in lower SES environments with history of childhood trauma would show attenuated cardiovascular reactivity to stressors than their low SES-no trauma peers and their high SES peers, there was only weak evidence to this effect. The interaction effects were generally inconsistent and suggested differences across cardiovascular measures and differences in reactivity to social and task stress.

4.1 Cardiovascular Reactivity to Social Conflict

In the models investigating cardiovascular reactivity to social conflict, the middle childhood SES group who had not experienced childhood trauma appeared to show greater CVR than their lower and higher SES peers (Figures 4 & 5). Specifically, a statistically-significant three-way interaction emerged when investigating DBP reactivity to social conflict. Analysis of simple effects demonstrated that, among individuals without a history of childhood trauma, the middle SES group was more reactive to social conflict than the low or high SES groups. Likewise, among the middle SES individuals, those with a history of childhood trauma showed significantly lower DBP reactivity to social conflict than their counterparts without a history of childhood trauma. No significant interactions appeared when investigating SBP reactivity to social conflict. This being said, some results trended in the same directions as above (see Figure 4). While results were not entirely consistent, there appear to be individual differences in cardiovascular reactivity to social conflict which are partially explained by the interaction of childhood SES and trauma history.

While the observed three-way interaction between early life SES, childhood trauma, and DBP reactivity aligns with hypotheses, the fact that the effect is most pronounced in the middle SES group departs from expectations. We had predicted that the low early life SES group would show marked trauma-dependent disparities in reactivity, rather than the middle early life SES group. In exploratory analyses, we tested whether this deviation from expectation was, in part, due to demographic differences between the SES groups. In this sample, the "middle" early life SES group had a greater number of people with exposure to both abuse and neglect and a higher proportion of BIPOC individuals than the other two SES groups. Planned exploratory analyses demonstrated that the three-way interaction between childhood trauma, early life SES, and DBP reactivity to social conflict was only independently significant for the group with a history of both

abuse and neglect. This finding is consistent with previous work demonstrating graded effects of childhood trauma exposure on physical and mental health indicators (Dube, Felitti, Dong, Giles, & Anda, 2003; Heim et al., 2009), and suggests that the interactive effect of socioeconomic and trauma stress on cardiovascular reactivity is stronger (or perhaps only appears) under more severe levels of childhood trauma. The possibility that race impacts the intersection of childhood trauma, early life SES, and cardiovascular reactivity to social conflict was tested through two four-way interaction models. While the models provided no evidence of race differences, they were also likely underpowered.

Alternatively, it is possible that these disparate findings may reflect nuance in our conceptualization of the "middle SES" group. In the present study, individuals were placed in this middle SES bracket if their highest educated parent had at least one year of higher education (but no four-year degree). This group is heterogeneous and includes parents with an associates or technical degree as well as those who initiated but did not complete college. The careers and stressors that accompany each of these two parental realities may manifest in very different childhood socioeconomic environments. It is possible, for instance, that initiating but not completing college indicates that greater underlying mental or socioeconomic adversities are present. When we tested this hypothesis by separating out the middle SES group into these two early life SES clusters, however, results indicated that the SES/trauma interaction effect remained present for both groups.

Assuming that the results reflect meaningful distinctions rather than chance occurrence, ACM theory may be applied to help interpret the results. It may be that individuals who grew up in these middle SES households faced moderate/fluctuating socioeconomic adversity growing up, while those in the low and high SES households faced severe/persistent and mild socioeconomic

adversity, respectively. If this were the case, ACM theory would expect that, in the absence of trauma, the development of a heighted response to stressors may have been adaptive (Del Giudice et al., 2011) in the face of intermittent but dramatic socioeconomic stress (middle SES). To the extent that individuals who grew up in a "low" SES background experienced more severe/persistent economic stressors, ACM theory would expect a blunted profile.

4.2 Cardiovascular Reactivity to Task Strain

Contrary to the social conflict models, neither model investigating ABP reactivity to task strain demonstrated a significant three-way interaction between childhood trauma, early life SES, and task strain reactivity. This being said, a group difference appeared for individuals who grew up in a low SES environment depending on their childhood trauma exposure. Those individuals who grew up in a low SES environment with a history of childhood trauma demonstrated significantly lower SBP reactivity to task strain than the low SES individuals without a history of childhood trauma (see Figure 2, Table 4). Given that the overall three-way interaction is not significant, we are hesitant to emphasize the interpretation of this group simple effect. However, the trend suggests two principles which may be important in future investigations. Firstly, the trend suggests that the present analyses may not have been sufficiently powered to detect smaller three-way interaction effects in fairly large and non-parsimonious models. Secondly, the trend provides some support for the notion that group differences in cardiovascular reactivity to task strain may be dependent on childhood socioeconomic and trauma environments in the hypothesized manner. Future work is needed to adequately test whether early life SES and trauma history significantly interact to predict cardiovascular reactivity to task strain, as well as how reactivity to task strain differs from social conflict.

The relationships between childhood socioeconomic disadvantage and trauma and cardiovascular reactivity in the present study differ by the type of daily life stressor encountered, with the effects more dramatic for responses involving social conflict. It may be that individuals react differently to different stressors, and that this is, in part, influenced by the early life environment. Some evidence suggests that individuals who perceive themselves to be of lower social standing show greater neural activity in the face of negative social evaluation than their peers of higher social standing (Muscatell et al., 2016), and are more likely to perceive social threat during ambiguous social situations (Chen et al., 2004). It has been suggested that individuals who develop in a lower SES environment may place more emphasis on social resources due to the decreased availability of physical resources (Kraus & Keltner, 2009; Piff, Kraus, Côté, Cheng, & Keltner, 2010), thus prompting greater reactivity to social conflict. When simultaneously exposed to the severe stress of childhood trauma, however, exaggerated reactivity to social conflict may no longer be advantageous and this relationship may attenuate. Conversely, an SES-driven greater sensitivity to social situations would not impact reactivity to task strain, and may explain the lack of exaggerated response observed in the task strain models.

4.3 The Nature of the Interaction Between Early Life SES and Childhood Trauma

The present study hypothesized that childhood trauma would serve as a source of heterogeneity in the relationship between early life SES and cardiovascular stress response. Specifically, under this theoretical framework we expected the low SES group to differ based on trauma exposure with those without a history of childhood trauma showing exaggerated responses to daily life stress and those with a history of trauma to show blunted responses. However, only one model provided any support of the SES-dependent exaggerated pattern. Rather, there was more consistent evidence demonstrating a trauma-dependent blunted response to stress for the

low/middle SES groups, suggesting that the relationship between childhood trauma and cardiovascular reactivity to stress may be affected by heterogeneity in early life SES. This nuance in the interaction effect is meaningful given the growing literature linking childhood trauma with blunted reactivity to stress (Gooding et al., 2015; Lovallo et al., 2012; McLaughlin et al., 2015; Murali & Chen, 2005; Voellmin et al., 2015). Many of the key studies demonstrating this relationship utilized a predominantly lower SES sample (Gooding et al., 2015; Lovallo et al., 2012; McLaughlin et al., 2015), and therefore may neglect a key distinction in the relationship. While the present literature provides some early, albeit inconsistent, evidence for an interaction effect, results seem to suggest that early life SES may moderate the relationship between childhood trauma and stress reactivity.

4.4 Physiological Etiology and Consequences of Exaggerated and Blunted Reactivity

In this study, we suggest that childhood stress exposures may lead to either an exaggerated or unreactive stress response profile, depending on the type of early life experience. Considering that early life SES and childhood trauma are associated with negative long-term health outcomes such as cardiovascular disease (Basu, McLaughlin, Misra, & Koenen, 2017; Galobardes et al., 2008) and that only exaggerated reactivity is traditionally considered a risk factor for cardiovascular disease, the disparate associations with cardiovascular reactivity at first appear paradoxical. However, recent work has suggested that exaggerated vs blunted reactivity represent distinct pathways to pathology.

Much of the work investigating cardiovascular reactivity to stress has been guided by the cardiovascular reactivity hypothesis (Krantz et al., 1986; Obrist, 1981). As such, a large and consistent evidence base links heightened cardiovascular reactions to laboratory and daily life stress exposures with the development of hypertension, endothelial dysfunction, elevated

fibrinogen and interleukin 6, and coronary heart disease (Carroll et al., 2012; Chida & Steptoe, 2010; Ghiadoni et al., 2000; Panaite et al., 2015; Steptoe & Marmot, 2006; Treiber et al., 2003). More recently, blunted cardiovascular responses have also been related to negative health outcomes. Specifically, a non-reactive cardiovascular response profile to laboratory stress has been associated with depression, obesity, bulimia, and addiction (Carroll, Ginty, Whittaker, Lovallo, & de Rooij, 2017).

While the pathways through which blunted responses manifest and associate with negative health outcomes are not completely understood, Carroll and colleagues have suggested that a blunted cardiovascular response profile serves as a marker for poor functioning in the brain areas essential for motivation and behavioral regulation (Carroll et al., 2017). They argue that these deficits in motivation and behavioral regulation may then play a role in eliciting negative physiological outcomes, including obesity and addictive behaviors. Alternatively, Gianaros and Jennings (2020) have suggested that both blunted and exaggerated reactivity profiles are the manifestation of cognitive prediction errors (in part, due to faulty neurological systems). In both cases, the cardiovascular reaction is misaligned with the actual metabolic needs of a situation. An exaggerated reactivity profile, they argue, reflects an overestimation of required metabolic needs. Conversely, a blunted reactivity profile exposes an underestimation of the metabolic needs required to face the stressor. Gianaros and Jennings argue that these prediction errors in the mechanisms through which the brain couples stress appraisals with coordinated changes in behavior/peripheral physiology underlie the development of adverse disease states. While the Adaptive Calibration Model posits that blunted reactivity develops as an adaptive response to coping with stress in the short term, it also has maladaptive long-term outcomes.

4.5 Strengths, Limitations, and Future Directions

The present report is the first to investigate the intersection of early life socioeconomic environment and childhood trauma exposure in predicting cardiovascular reactivity to stress. The findings provide some initial evidence that the development of cardiovascular stress reactivity profiles is influenced by the childhood environment, specifically socioeconomic upbringing and exposure to childhood trauma. Interestingly, the present analyses suggest that there may be differences in how these early life factors influence cardiovascular reactivity to social stressors versus task (more cognitive) stressors.

The present study has several strengths worth mentioning. First of all, as the first investigation to synthesize the literatures linking childhood trauma and SES to cardiovascular reactivity, novel results suggest that the two may play an interactive role in predicting cardiovascular reactivity and should be considered together in future work. Additionally, the present analysis utilized an EMA design to investigate stress reactivity in the natural environment. This momentary assessment of stress provided for more frequent and real-time depictions of daily life experiences than global self-report assessments and the assessment of stress reactivity in a manner potentially more equivalent across the SES gradient.

Despite the many strengths of the present study design, key limitations are also worth noting. Firstly, given the complexity of the models, the present analyses may have been underpowered to detect small three-way interaction effects. Additionally, the present study recruited a fairly specific sample of healthy, middle-aged adults from the Pittsburgh area who were higher SES than the national average and predominantly white. These factors limit the variability of the sample, inhibit the generalizability of the study, and contribute to a growing body of literature normed on white individuals. Second, while a key strength of the study involves the use

of EMA methods, the study does not have comparable laboratory measures of stress reactivity. As such, no conclusions can be drawn as to whether SES associations with stress reactivity in the natural environment differ from those in the laboratory on the basis of findings in this sample, although significant associations between blood pressure responses to laboratory stressors and ambulatory measures of the type used here have previously been reported in a different sample (Kamarck, Schwartz, Janicki, Shiffman, & Raynor, 2003). Likewise, the present report investigates the impact of childhood environments on cardiovascular reactivity in adulthood and ignores all intermediate life events which may impact the development of cardiovascular reactivity.

Future work should address the limitations of the present study and build upon the principles introduced. More specifically, future work should first replicate the present analyses in a larger and more socioeconomically and racially diverse sample of individuals. Ideally, follow-up study would also compare daily life stress reactivity to the more traditional measures of laboratory reactivity. Likewise, follow-up work should adopt a lifestyle perspective when investigating these hypotheses. Specifically, upcoming studies should investigate the trajectory of cardiovascular reactivity development to better understand the plasticity of exaggerated/ blunted reactivity profiles across development. The age-related inquiries should also examine the advancement of differences in stress response to task strain versus social conflict, and the factors impacting each. Additionally, future investigations should examine the impact of childhood trauma and early life SES in predicting later experiences of trauma and stress in adulthood. Cardiovascular reactivity profiles may be influenced by the extent to which early life factors predict later life stress exposures. Lastly, future studies should continue to examine the extent to which "blunted" and

"exaggerated" reactivity states serve as direct causes of pathology versus markers of underlying neurological dysregulation in cognitive appraisal and emotion regulation.

5.0 Conclusion

Overall, the present thesis integrates literatures on early life SES and childhood trauma with respect to their joint influence on physiological reactivity. The findings provide some inconsistent evidence that childhood trauma and socioeconomic standing may interact to predict later-life cardiovascular responses to daily life stress. However, the findings are divergent from expectations. Specifically, results suggest that individuals who grow up in a middle SES childhood environment without a history of childhood trauma may show exaggerated reactivity to social conflict compared to lower and higher SES peers without a history of childhood trauma, and individuals who grow up in a middle SES environment with history of childhood trauma may show attenuated (blunted) reactivity to social conflict. Some results also suggest that low SES individuals with a history of trauma may have blunted cardiovascular reactivity to task strain compared to low SES individuals without a history of trauma. Results may have implications for understanding the underlying mechanisms through which childhood disadvantage contributes to physical and mental disease. To the extent that childhood trauma serves as a source of heterogeneity in the functioning of stress response systems for individuals raised in low SES environments, results may also have implications for triage in early intervention efforts.

Appendix A: Tables of Previous Studies

Table 1 Studies on Early-Life SES and Cardiovascular Reactivity

Study	Early Life SES Measurement	Covariates	Acute Stress Paradigm	Cardiovascular Outcomes	Nature of Relationship	Study Participants
Boylan et al, 2016	Parent SES (Hollingshead)	Age, race, marital status, BMI, smoking status, current SES, psychosocial resources	Mental arithmetic, Mirror tracing, Anger recall speech task	SBP, DBP, HR	Null	246 adult men (mean age 32.1)
Chen & Matthews, 2001	Parent SES (Hollingshead's four-factor index of social status)	Resting SBP/HR	Mirror Tracing, Cold Forehead	SBP, DBP, HR, CO, TPR (calculated)	Inverse, CO: 1, β = .23, t(193) = 3.25, p < .01 TPR: β = –.17, t(191) = 2.41, p < .025	90 male and female adolescents (mean age 13.6)
Chen, Langer, Raphaelson & Matthews, 2004	Parent SES (Hollingshead's four-factor index of social status)	Race	CAUSE videos	SBP, DBP, HR	Inverse, HR: β = -.32, *t* = 2.92, *p* < .01 DBP: β = -.38, *t* = 3.55, *p* < .01	101 male and female adolescents (mean age 17.3)
Evans & Kim, 2007	Income: Needs ratio	--	Mental arithmetic	DBP, SBP	Positive, DBP: *b* = −1.62, *p* < .03 SBP: *b* = −3.62, *p* < .01	207 male and female adolescents (mean age 13.4)
Evans, Exner-Cortens, Kim & Bartholomew, 2013(G. W. Evans et al., 2013)	Income: needs ratio	Age, sex, race	Mental arithmetic	DBP, SBP	Null	185 male and female adolescents (mean age 17)
Gump, Matthews & Raikkonen, 1999	Neighborhood SES & Parent SES (Hollingshead)	Age, sex	Mirror-tracing task Cold forehead task	SBP, DBP, HR	Inverse	147 male and female adolescents
Jackson, Treiber, Turner, Davis	Neighborhood SES	--	Verbal conflict with parent	SBP, DBP, HR	Mixed (Inverse for Whites,	272 male and female adolescents

& Strong, 1999					Positive for Blacks) SBP: $F = 7.8$, $p < 0.001$	(mean age 13.5)
Kapuku, Treiber & Davis, 2002	Neighborhood SES	--	Video game stressor	SBP, DBP	Inverse DBP: $r = -.41$, $p < .05$	24 Black males (mean age 18.8)
Wilson, Kliewer, Plybon & Sica, 2000	Neighborhood SES, Parent Education	BMI, family history of hypertension	Video game stressor	SBP, DBP	Inverse DBP: 767 (high income low SES neighborhood) versus 1268 mm Hg (low income, high SES neighborhood) , $p < .05$	76 adolescents (mean age 14)
Musante et al., 2000	Parent SES (Hollingshead's four-factor index of social status)	Age, sex, BMI, neighborhood SES, ethnicity, number of life events	Car driving simulation, Social Competence interview	SBP, DBP, CO, HR, PEP	Positive, SBP, DBP, CO, HR: all p values < .05	483 male and female adolescents (mean age 16.7)

Note. The literature investigating the link between early life SES and cardiovascular reactivity to a stressor is mixed. SBP refers to systolic blood pressure, DBP refers to diastolic blood pressure, HR refers to heart rate, CO refers to cardiac output, TPR refers to total peripheral resistance, PEP refers to pre-ejection period, SES refers to socioeconomic status. An "Inverse" relationship describes an association where lower SES is associated with increased cardiovascular reactivity to stress. A "positive" relationship describes an association where lower SES is associated with decreased cardiovascular reactivity to stress.

Table 2 Studies on Childhood Trauma and Cardiovascular Reactivity

Study	Childhood Adversity Measurement	Covariates	Acute Stress Paradigm	Cardiovascular Outcomes	Nature of Relationship	Study Participants
Lovallo, Farag, Sorocco, Cohoon & Vincent, 2012	Number of Adverse Life Events	--	Public speaking, Mental arithmetic	HR	Inverse, $F(2,340) = 8.11$, $p < .0004$	354 healthy men and women participants
Voellmin et al., 2015	ETI-SR	BMI, oral contraceptives	MIST	HR	Inverse, $F(1.98, 168.32) = 5.86$, $p < .01$; $\eta_p^2 = .07$	104 healthy young women (mean age 21.7)
Gooding, Milliren, Bryn Austin, Sheridan & McLaughlin, 2015	Childhood Trauma Questionnaire (CTQ)	Age, sex, race/ethnicity, parent education, BMI, smoking status	TSST	DBP, SBP	Inverse, SBP: (20.66 mmHg versus 28.97 mmHg), $p = 003$ DBP: (18.19 mmHg versus 32.4 mmHg), $p < 001$	145 healthy male and female adolescents (mean age 14.9)
Murali & Chen, 2006	Experienced, Observed, & Subjective Violence	--	Debate, Verbal puzzle	SBP, DBP, HR	Inverse, SBP: $\beta = -.13$, $p = .05$ HR: $\beta = -.21$, $p < .001$	115 healthy black and white adolescents
McLaughlin, Sheridan, Tibu, Fox, Zeanah & Nelson, 2015	Groups, raised in foster care or in an orphanage	Sex	TSST, Non-social frustration task	HR, SBP, DBP, PEP	Inverse HR: $F = 7.65$, $p < .0001$ SBP: $F = 7.13$, $p < .0001$ DBP: $F = 7.65$, $p < .0001$ PEP: $F = 11.58$, $p < .0001$	136 children in Romania (mean age 12.9)
Heim et al., 2000	Early Trauma Inventory (ETI)	Ethnicity	TSST	HR	Positive, Difference between ETI/MDD group versus controls (89.7beats/min vs 78.4 beats/min), $p < .01$	49 women between the ages 18-45

Luecken, Kraft, Appelhans & Enders, 2009	Death of parent	1) posture, substance use, location 2) Age, gender, BMI, medication use	Ambulatory, yes or no to stress events and emotions from PANAS	SBP, DBP	Null	91 undergraduate students, 43 from bereaved families
Evans, Greaves - Lord, Euser, Koning, Tulen, Franken & Huizink, 2015	Parent with a substance abuse disorder, or not	Age, sex, BMI, parent occupation, ubanicity	TSST	HR	Null for reactivity, Inverse for recovery, β = 0.13, t = 2.72	75 children of parents with substance use disorder & 363 matched controls (mean age 15)

Note. Description of the literature investigating the link between childhood trauma and cardiovascular reactivity to a stressor. SBP refers to systolic blood pressure, DBP refers to diastolic blood pressure, HR refers to heart rate, PEP refers to pre-ejection period, SES refers to socioeconomic status. An "Inverse" relationship describes an association where increased childhood trauma is associated with decreased cardiovascular reactivity to stress. A "positive" relationship describes an association where increased childhood trauma is associated with increased cardiovascular reactivity to stress.

Appendix B: Tables of Study Results

Table 3 Descriptive Characteristics

Variable	Low SES (*n*= 123)	Middle SES (*n*= 101)	High SES (*n*= 137)	Total Sample (*n*= 361)
Age	53.78 ± 6.68	51.88 ± 7.42	52.02 ± 7.08	52.58 ± 7.08
Sex (female)	65%	60%	56%	60%
Race				
White	79%	70%	86%	80%
Black	19%	30%	10%	19%
Asian	2%	2%	4%	3%
American Indian	1%	1%	0%	1%
Childhood Trauma				
No History	63%	51%	60%	59%
Abuse Only	11%	20%	15%	15%
Neglect Only	7%	6%	7%	7%
Abuse & Neglect	18%	23%	18%	19%
(Adult) Education				
High School	10%	5%	4%	6%
Some College	40%	32%	18%	29%
College Degree	28%	33%	36%	32%
Advanced Degree	22%	31%	41%	32%
(Adult) Income	7.84 ± 3.92	7.38 ± 3.47	8.47 ± 4.04	7.95 ± 3.86

Note. Values reflect the mean ± standard deviation or %. An adult household income score of 7 corresponds with $65,000 - 79,999/year, a score of 8 corresponds with $80,000 - 94,999/year, and a score of 9 corresponds with $95,000 - 109,999/year.

Table 4 Step-by-Step MLM Model of Task Strain on Systolic Blood Pressure

	Step 1	Step 2	Step 3	Step 4	Step 5
Fixed Effects					
Intercept, γ_{00}	138.0(0.92)***	138.0(0.92)***	135.5(1.81)***	136.5(2.04)***	136.7(2.04)***
Level 1 Variables					
Posture Standing, γ_{20}		3.54(0.25)***	3.54(0.25)***	3.54(0.25)***	3.54(0.25)***
Posture Laying,		-7.31(0.51)***	-7.31(0.51)***	-7.31(0.51)***	-7.31(0.51)***
Talking, γ_{30}		2.07(0.25)***	2.07(0.25)***	2.07(0.25)***	2.08(0.25)***
Physical Activity, γ_{40}		2.21(0.22)***	2.21(0.22)***	2.21(0.22)***	2.21(0.22)***
Recent Meal, γ_{50}		1.17(0.25)***	1.17(0.25)***	1.17(0.25)***	1.17(0.25)***
Caffeine, γ_{60}		0.04(0.31)	0.04(0.31)	0.04(0.31)	0.04(0.31)
Temperature Hot, γ_{70}		1.14(0.50)*	1.14(0.50)*	1.14(0.50)*	1.13(0.50)*
Temperature Cold		3.05(0.55)***	3.05(0.55)***	3.05(0.55)***	3.07(0.55)***
Alcohol,		-0.51(0.63)	-0.51(0.63)	-0.51(0.63)	-0.52(0.63)
Task Strain, γ_{10}		1.38(0.30)***	0.98(0.58)†	0.98(0.58)†	1.47(0.65)*
Level 2 Variables					
Child SES_{middle}, γ_{01}			0.55(2.21)	0.26(2.93)	0.26(2.93)
Child SES_{high}, γ_{02}			-0.20(2.05)	-2.52(2.56)	-2.52(2.56)
Childhood Trauma, γ_{03}			0.61(1.76)	-2.08(3.04)	-2.08(3.04)
Child SES_{middle}*Child Trauma, γ_{04}				1.34(4.44)	1.80(4.46)
Child SES_{high}*Child Trauma, γ_{05}				6.18(4.15)†	6.70(4.17)
Task Strain $_{(person\ means)}$, γ_{06}			7.87(3.23)*	7.45(3.23)*	7.46(3.23)*
Race, γ_{07}			-7.35(2.18)***	-7.34(2.18)***	-7.34(2.18)***
Age, γ_{08}			0.47(0.12)***	0.48(0.12)***	0.48(0.12)***
Gender, γ_{09}			-9.67(1.76)***	-9.46(1.77)***	-9.46(1.77)***
Cross-Level Interaction Variables					
Task Strain*Child SES_{middle} γ_{11}			0.71(0.79)	0.70(0.79)	1.05(1.02)
Task Strain*Child SES_{high}, γ_{12}			1.31(0.71)†	1.30(0.71)†	0.52(0.88)
Task Strain*Child Trauma, γ_{13}			-0.84(0.61)	-0.84(0.61)	-2.41(1.15)*
Task Strain*Child SES_{middle}*Child Trauma, γ_{14}					2.09(1.60)
Task Strain Child SES_{high}*Child Trauma, γ_{15}					2.25(1.47)
Random Effects					
Intercept, τ_{00}	298.25	298.92	257.57	255.50	255.49
Task Strain slope, τ_{11}		3.36	2.99	2.99	2.58
Level 1, Var within person, σ^2	196.39	184.31	184.26	184.26	184.31
LRT (compared to previous model)		L.ratio = 1104.33***	L.ratio = 58.25***	L.ratio = 0.29	L.ratio =2.59

Note. An † depicts $p < .10$, an * indicates $p < 0.05$, an ** indicates $p < .01$, an *** indicates $p < .001$. All random effects are significantly different from zero.

Table 5 Step-by-Step MLM Model of Task Strain on Diastolic Blood Pressure

	Step 1	Step 2	Step 3	Step 4	Step 5
Fixed Effects					
Intercept, γ_{00}	81.88(0.56)***	81.89(0.56)***	80.64(1.10)***	81.40(1.24)***	81.42(1.24)***
Level 1 Variables					
Posture Standing, γ_{20}		3.40(0.18)***	3.40(0.18)***	3.40(0.18)***	3.40(0.18)***
Posture Laying,		-7.66(0.36)***	-7.66(0.36)***	-7.66(0.36)***	-7.66(0.36)***
Talking, γ_{30}		1.89(0.17)***	1.89(0.17)***	1.89(0.17)***	1.89(0.17)***
Physical Activity, γ_{40}		1.06(0.15)***	1.06(0.15)***	1.06(0.15)***	1.06(0.15)***
Recent Meal, γ_{50}		-0.20(0.18)	-0.20(0.18)	-0.20(0.18)	-0.19(0.18)
Caffeine, γ_{60}		-0.05(0.21)	-0.05(0.21)	-0.05(0.21)	-0.05(0.21)
Temperature Hot, γ_{70}		0.04(0.35)	0.04(0.35)	0.04(0.35)	0.04(0.35)
Temperature Cold		1.39(0.38)***	1.39(0.38)***	1.39(0.38)***	1.40(0.38)***
Alcohol,		-0.62(0.43)	-0.62(0.43)	-0.62(0.43)	-0.63(0.43)
Task Strain, γ_{10}		0.85(0.22)***	0.78(0.43)†	0.78(0.43)†	0.90(0.48)†
Level 2 Variables					
Child SES_{middle}, γ_{01}			-0.50(1.35)	-0.60(1.78)	-0.61(1.78)
Child SES_{high}, γ_{02}			-0.09(1.24)	-1.82(1.56)	-1.86(1.56)
Childhood Trauma, γ_{03}			-0.30(1.07)	-2.23(1.85)	-2.28(1.85)
Child SES_{middle}*Child Trauma, γ_{04}				0.75(2.70)	0.79(2.70)
Child SES_{high}*Child Trauma, γ_{05}				4.60(2.53)†	4.60(2.53)†
Task Strain $_{(person\ means)}$, γ_{06}			5.44(1.97)**	5.15(1.96)**	5.15(1.96)**
Race, γ_{07}			-5.67(1.33)***	-5.64(1.33)***	-5.64(1.33)***
Age, γ_{08}			0.13(0.07)†	0.14(0.07)†	0.14(0.07)†
Gender, γ_{09}			-5.93(1.07)***	-5.76(1.07)***	-5.76(1.08)***
Cross-Level Interaction Variables					
Task Strain*Child SES_{middle} γ_{11}			0.37(0.58)	-0.37(0.58)	0.30(0.76)
Task Strain*Child SES_{high}, γ_{12}			0.45(0.53)	0.44(0.53)	0.19(0.66)
Task Strain*Child Trauma, γ_{13}			-0.54(0.45)	-0.54(0.45)	-0.92(0.85)
Task Strain*Child SES_{middle}*Child Trauma, γ_{14}					0.28(1.19)
Task Strain Child SES_{high}*Child Trauma, γ_{15}					0.71(1.10)
Random Effects					
Intercept, τ_{00}	109.65	110.06	94.91	93.84	93.84
Task Strain slope, τ_{11}		2.89	2.80	2.80	2.76
Level 1, Var within person, σ^2	93.05	83.77	83.76	83.76	83.77
LRT (compared to previous model)		L.ratio = 1590.19***	L.ratio = 53.99***	L.ratio = 3.81	L.ratio = 0.44

Note. An † depicts $p < .10$, an * indicates $p < 0.05$, an ** indicates $p < .01$, an *** indicates $p < .001$. All random effects are significantly different from zero.

Table 6 Step-by-Step MLM Model of Social Conflict on Systolic Blood Pressure

	Step 1	Step 2	Step 3	Step 4	Step 5
Fixed Effects					
Intercept, γ_{00}	139.1(0.93)***	139.1(0.93)***	138.0(1.84)***	139.4(2.07)***	139.3(2.08)***
Level 1 Variables					
Posture Standing, γ_{20}		3.48(0.33)***	3.48(0.33)***	3.48(0.33)***	3.48(0.33)***
Posture Laying,		-7.54(0.82)***	-7.53(0.82)***	-7.53(0.82)***	-7.53(0.82)***
Talking, γ_{30}		1.83(0.30)***	1.83(0.30)***	1.83(0.30)***	1.84(0.30)***
Physical Activity, γ_{40}		1.84(0.29)***	1.83(0.29)***	1.83(0.29)***	1.83(0.29)***
Recent Meal, γ_{50}		1.21(0.33)***	1.21(0.33)***	1.21(0.33)***	1.21(0.33)***
Caffeine, γ_{60}		-0.19(0.42)	-0.19(0.42)	-0.19(0.42)	-0.19(0.42)
Temperature Hot, γ_{70}		1.03(0.65)	1.02(0.65)	1.02(0.65)	1.02(0.65)
Temperature Cold		3.58(0.78)***	3.57(0.78)***	3.57(0.78)***	3.57(0.78)***
Alcohol,		0.52(0.79)	0.52(0.79)	0.52(0.79)	0.52(0.79)
Social Conflict, γ_{10}		0.42(0.10)***	0.40(0.18)*	0.40(0.18)*	0.37(0.21)†
Level 2 Variables					
Child SES_{middle}, γ_{01}			-0.01(2.26)	-0.98(2.99)	-0.90(2.99)
Child SES_{high}, γ_{02}			-0.40(2.08)	-3.13(2.61)	-3.11(2.61)
Childhood Trauma, γ_{03}			0.49(1.81)	-3.04(3.10)	-2.98(3.11)
Child SES_{middle}*Child Trauma, γ_{04}				2.90(4.51)	2.72(4.53)
Child SES_{high}*Child Trauma, γ_{05}				7.24(4.22)†	7.20(4.24)†
Social Conflict $_{(person\ means)}$, γ_{06}			0.98(0.82)	0.87(0.82)	0.87(0.82)
Race, γ_{07}			-8.13(2.21)***	-8.16(2.21)***	-8.15(2.21)***
Age, γ_{08}			0.48(0.13)***	0.50(0.12)***	0.50(0.12)***
Gender, γ_{09}			-8.90(1.80)***	-8.72(1.80)***	-8.72(1.80)***
Cross-Level Interaction Variables					
Social Conflict*Child SES_{middle} γ_{11}			0.43(0.26)†	0.43(0.26)†	0.52(0.34)
Social Conflict *Child SES_{high}, γ_{12}			-0.11(0.24)	-0.11(0.24)	-0.10(0.30)
Social Conflict *Child Trauma, γ_{13}			-0.16(0.21)	-0.16(0.21)	-0.08(0.35)
Social Conflict*Child SES_{middle}*Child Trauma,γ_{14}					-0.22(0.52)
Social Conflict* Child SES_{high}*Child Trauma, γ_{15}					-0.05(0.49)
Random Effects					
Intercept, τ_{00}	296.45	297.26	260.04	257.90	257.89
Social Conflict slope, τ_{11}		0.38	0.30	0.30	0.29
Level 1, Var within person, σ^2	199.14	188.81	188.81	188.82	188.83
LRT (compared to previous model)		L.ratio = 506.84***	L.ratio = 50.89***	L.ratio = 3.00	L.ratio = 0.19

Note. An † depicts $p < .10$, an * indicates $p < 0.05$, an ** indicates $p < .01$, an *** indicates $p < .001$. All random effects are significantly different from zero.

Table 7 Step-by-Step MLM Model of Social Conflict on Diastolic Blood Pressure

	Step 1	Step 2	Step 3	Step 4	Step 5
Fixed Effects					
Intercept, γ_{00}	82.74(0.57)***	82.76(0.56)***	82.20(1.12)***	83.08(1.26)***	83.07(1.26)***
Level 1 Variables					
Posture Standing, γ_{20}		3.36(0.23)***	3.36(0.23)***	3.36(0.23)***	3.36(0.23)***
Posture Laying,		-7.85(0.56)***	-7.84(0.56)***	-7.84(0.56)***	-7.82(0.56)***
Talking, γ_{30}		1.72(0.21)***	1.71(0.21)***	1.71(0.21)***	1.72(0.21)***
Physical Activity, γ_{40}		0.88(0.21)***	0.87(0.20)***	0.87(0.20)***	0.87(0.20)***
Recent Meal, γ_{50}		-0.07(0.23)	-0.07(0.23)	-0.07(0.23)	-0.07(0.23)
Caffeine, γ_{60}		0.04(0.29)	0.05(0.29)	0.05(0.29)	0.05(0.29)
Temperature Hot, γ_{70}		-0.25(0.45)	-0.25(0.45)	-0.25(0.45)	-0.24(0.45)
Temperature Cold		1.49(0.54)**	1.49(0.54)**	1.49(0.54)**	1.52(0.54)**
Alcohol,		0.48(0.54)	0.48(0.54)	0.48(0.54)	0.51(0.54)
Social Conflict, γ_{10}		0.24(0.07)***	0.24(0.13)†	0.24(0.13)†	0.09(0.15)
Level 2 Variables					
Child SES_{middle}, γ_{01}			-0.76(1.37)	-0.94(1.82)	-0.90(1.82)
Child SES_{high}, γ_{02}			-0.17(1.26)	-2.15(1.59)	-2.14(1.59)
Childhood Trauma, γ_{03}			-0.39(1.11)	-2.60(1.89)	-2.56(1.89)
Child SES_{middle}*Child Trauma, γ_{04}				0.95(2.76)	0.86(2.76)
Child SES_{high}*Child Trauma, γ_{05}				5.23(2.58)*	5.19(2.58)*
Social Conflict $_{(person\ means)}$, γ_{06}			0.91(0.50)†	0.83(0.50)†	0.83(0.50)†
Race, γ_{07}			-6.34(1.34)***	-6.31(1.34)***	-6.29(1.35)***
Age, γ_{08}			0.15(0.08)†	0.16(0.08)*	0.16(0.08)*
Gender, γ_{09}			-5.86(1.10)***	-5.66(1.10)***	-5.68(1.10)***
Cross-Level Interaction Variables					
Social Conflict*Child SES_{middle} γ_{11}			0.28(0.19)	0.28(0.19)	0.72(0.24)**
Social Conflict *Child SES_{high}, γ_{12}			0.07(0.17)	0.07(0.17)	0.19(0.21)
Social Conflict *Child Trauma, γ_{13}			-0.26(0.15)†	-0.26(0.15) †	0.17(0.25)
Social Conflict*Child SES_{middle}*Child Trauma,γ_{14}					-1.02 (0.37)**
Social Conflict* Child SES_{high}*Child Trauma, γ_{15}					-0.36(0.35)
Random Effects					
Intercept, τ_{00}	110.12	110.76	95.97	94.78	94.78
Social Conflict slope, τ_{11}		0.25	0.24	0.24	0.20
Level 1, Var within person, σ^2	93.14	85.08	85.04	85.04	85.04
LRT (compared to previous model)		L.ratio = 738.11***	L.ratio = 53.72***	L.ratio =	L.ratio = 7.62*

Note. An † depicts $p < .10$, an * indicates $p < 0.05$, an ** indicates $p < .01$, an *** indicates $p < .001$. All random effects are significantly different from zero.

Table 8 Social Conflict on Diastolic Blood Pressure by Type of Childhood Trauma

	Coefficient *(SE)*	95% CI
Fixed Effects		
Intercept	82.68(1.26)***	80.22 – 85.14
Level 1 Variables		
Posture Standing	3.37(0.23)***	-8.89 – -6.69
Posture Laying	-7.79(0.56)***	2.92 – 3.83
Talking	1.73(0.21)***	1.32 – 2.14
Physical Activity	0.87(0.20)***	0.47 – 1.26
Recent Meal	-0.06(0.23)	-0.51 – 0.40
Caffeine	0.04(0.29)	-0.51 – 0.60
Temperature Hot	-0.16(0.45)	-1.04 – 0.72
Temperature Cold	1.51(0.54)**	0.45 – 2.57
Alcohol	0.51(0.54)	-0.55 – 1.56
Social Conflict	0.09(0.15)	-0.19 – 0.38
Level 2 Variables		
Child SES_{middle}	-0.91(1.80)	-4.44 – 2.62
Child SES_{high}	-2.12(1.57)	-5.20 - 0.96
Childhood Trauma: Abuse Only	-3.24(2.89)	-8.91 – 2.44
Childhood Trauma: Neglect Only	-4.47(3.56)	-11.46 – 2.53
Childhood Trauma: Both Abuse & Neglect	-1.49(2.42)	-6.24 – 3.27
Child SES_{middle}*Child Trauma: Abuse Only	-1.76(3.91)	-9.44 – 5.92
Child SES_{middle}*Child Trauma: Neglect Only	10.10(5.56)†	-0.81 – 21.01
Child SES_{middle}*Child Trauma: Both Abuse & Neglect	0.35(3.47)	-6.47 – 7.17
Child SES_{high}*Child Trauma: Abuse Only	7.29(3.82)†	-0.21 – 14.79
Child SES_{high}*Child Trauma: Neglect Only	3.41(4.90)	-6.21 – 13.04
Child SES_{high}*Child Trauma: Both Abuse & Neglect	4.29(3.32)	-2.23 – 10.82
Social Conflict $_{(person\ means)}$	0.70(0.51)	-0.29 – 1.70
Race	-6.43(1.34)***	-9.07 – -3.79
Age	0.16(0.08)*	0.01 – 0.31
Gender	-5.61(1.09)***	-7.77 – -3.47
Cross-Level Interaction Variables		
Social Conflict*Child SES_{middle}	0.72(0.24)**	0.25 – 1.19
Social Conflict *Child SES_{high}	0.19(0.21)	-0.21 – 0.60
Social Conflict *Child Trauma: Abuse Only	-0.17(0.39)	-0.94 – 0.60
Social Conflict *Child Trauma: Neglect Only	-0.51(0.75)	-1.98 – 0.96
Social Conflict *Child Trauma: Both Abuse & Neglect	0.41(0.29)	-0.15 – 0.98
Social Conflict*Child SES_{middle}*Child Trauma: Abuse Only	-0.64(0.52)	-1.67 – 0.38
Social Conflict*Child SES_{middle}*Child Trauma: Neglect Only	-0.25(0.91)	-2.03 – 1.52
Social Conflict*Child SES_{middle}*Child Trauma: Both Abuse & Neglect	-1.31(0.45)**	-2.19 – -0.43
Social Conflict* Child SES_{high}*Child Trauma: Abuse Only	0.16(0.50)	-0.82 – 1.14
Social Conflict* Child SES_{high}*Child Trauma: Neglect Only	0.52(0.90)	-1.25 – 2.28
Social Conflict* Child SES_{high}*Child Trauma: Both Abuse & Neglect	-0.73(0.44)†	-1.59 – 0.12
Random Effects		
Intercept, τ_{00}	92.52	79.25 – 108.01
Social Conflict slope, τ_{11}	0.17	0.05 – 0.54
Level 1, Var within person, σ^2	84.71	82.13 – 87.37
log- Likelihood	-34940.09	

Note. An † depicts $p < .10$, an * indicates $p < 0.05$, an ** indicates $p < .01$, an *** indicates $p < .001$.

Table 9 Joint Models of Task Strain and Social Conflict on ABP

	DBP		SBP	
	Coefficient *(SE)*	95% CI	Coefficient *(SE)*	95% CI
Fixed Effects				
Intercept	82.52(1.31)***	79.97 – 85.09	138.4(2.15)***	134.2 – 142.6
Level 1 Variables				
Posture Standing	3.30(0.23)***	2.85 – 3.76	3.41(0.34)***	2.75 – 4.06
Posture Laying	-7.78(0.56)***	-8.88 – -6.68	-7.40(0.82)***	-9.00 - -5.80
Talking	1.71(0.21)***	1.30 – 2.12	1.82(0.30)***	1.23 – 2.41
Physical Activity	0.81(0.20)***	0.41 – 1.21	1.73(0.30)***	1.15 – 2.31
Recent Meal	-0.05(0.23)	-0.51 – 0.40	1.23(0.33)***	0.58 – 1.88
Caffeine	0.04(0.29)	-0.52 – 0.60	-0.20(0.42)	-1.01 – 0.62
Temperature Hot	-0.21(0.45)	-1.09 – 0.67	1.03(0.65)	-0.24 – 2.31
Temperature Cold	1.55(0.54)**	0.50 – 2.61	3.63(0.78)***	2.10 – 5.16
Alcohol	0.55(0.54)	-0.50 – 1.61	0.63(0.79)	-0.93 – 2.18
Social Conflict	0.07(0.15)	-0.22 – 0.36	0.33(0.21)	-0.08 – 0.73
Task Strain	0.63(0.63)	-0.62 – 1.88	1.45(0.89)	-0.30 – 3.19
Level 2 Variables				
Child SES_{middle}	-0.92(1.81)	-4.48 – 2.64	-0.92(2.98)	-6.78 – 4.93
Child SES_{high}	-2.20(1.58)	-5.31 - 0.91	-3.23(2.61)	-8.35 – 1.89
Child Trauma	-2.54(1.88)	-6.24 – 1.16	-2.93(3.10)	-9.02 – 3.15
Child SES_{middle}*Child Trauma	0.89(2.75)	-4.51– 6.29	2.78(4.52)	-6.10 – 11.66
Child SES_{high}*Child Trauma	5.17(2.57)*	0.12 – 10.21	7.17(4.23)†	-1.14 – 15.47
Social Conflict (person means)	0.47(0.56)	-0.64 – 1.57	0.21(0.92)	-1.61 – 2.02
Task Strain (person means)	3.16(2.17)	-1.10 – 7.44	5.67(3.56)	-1.31 – 12.66
Race	-6.01(1.35)***	-8.67 – -3.35	-7.62(2.22)***	-11.97 – -3.27
Age	0.16(0.08)*	0.01 – 0.31	0.50(0.12)***	0.26 – 0.75
Gender	-5.66(1.09)***	-7.81 – -3.51	-8.73(1.79)***	-12.25 – -5.21
Cross-Level Interaction Variables				
Social Conflict*Child SES_{middle}	0.70(0.24)**	0.22 – 1.18	0.51(0.34)	-0.16 – 1.17
Social Conflict *Child SES_{high}	0.16(0.21)	-0.25 – 0.58	-0.12(0.30)	-0.70 – 0.46
Social Conflict *Child Trauma	0.21(0.25)	-0.28 – 0.70	-0.01(0.35)	-0.69 – 0.67
Social Conflict*Child SES_{middle}*Child Trauma	-1.03(0.37)**	-1.76 – -0.31	-0.26(2.29)	-1.23 – 0.79
Social Conflict* Child SES_{high}*Child Trauma	-0.38(0.35)	-1.06 – 0.31	2.90(2.10)	-1.05 – 0.85
Task Strain*Child SES_{middle}	0.59(0.98)	-1.34 – 2.51	0.61(1.38)	-2.08 – 3.31
Task Strain*Child SES_{high}	0.32(0.85)	-1.35 – 1.99	-0.19(1.19)	-2.52 – 2.14
Task Strain*Child Trauma	-1.59(1.18)	-3.90 – 0.73	-1.87(1.67)	-5.14 – 1.41
Task Strain*Child SES_{middle}*Child Trauma	0.66(1.62)	-2.51 – 3.84	-0.26(2.29)	-4.75 – 4.23
Task Strain*Child SES_{high}*Child Trauma	1.72(1.49)	1.20 – 4.63	2.90(2.10)	-1.20 – 7.00
Random Effects				
Intercept, τ_{00}	94.26		256.28	
Social Conflict slope	0.19		0.24	
Task Strain slope	3.81		5.38	
Level 1, Var within person, σ^2	84.43		187.75	
log- Likelihood	-35035.59		-38655.27	

Note. An † depicts $p < .10$, an * indicates $p < 0.05$, an ** indicates $p < .01$, an *** indicates $p < .001$.

Appendix C: Models

Level 1:

$$ABP_{ij} = \beta_{0j} + \beta_{1j}(Stress(person-centered)_{ij}) + \beta_{2j}(Posture\ Standing_{ij}) + \beta_{3j}(Posture\ Laying_{ij}) + \beta_{4j}(Talking_{ij}) + \beta_{5j}(Physical\ Activity_{ij}) + \beta_{6j}(Recent\ Meal_{ij}) + \beta_{7j}(Caffeine_{ij}) + \beta_{8j}(Temperature\ Hot_{ij}) + \beta_{9j}(Temperature\ Cold_{ij}) + \beta_{10j}(Alcohol) + r_{ij}$$

Level 2:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(ChildSES\ dummy\ some\ college_j) + \gamma_{02}(ChildSES\ dummy\ college/adv_j) + \gamma_{03}(Childhood\ Trauma_j) + \gamma_{04}(ChildSES\ dummy\ some\ college_j)(Childhood\ Trauma_j) + \gamma_{05}(ChildSES\ dummy\ college/adv_j)(Childhood\ Trauma_j) + \gamma_{06}(Stress\ (person\ means)_j) + \gamma_{07}(Race_j) + \gamma_{08}(Age_j) + \gamma_{09}(Gender_j) + \upsilon_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(ChildSES\ dummy\ some\ college_j) + \gamma_{12}(ChildSES\ dummy\ college/adv_j) + \gamma_{13}(Childhood\ Trauma_j) + \gamma_{14}(ChildSES\ dummy\ some\ college_j)(Childhood\ Trauma_j) + \gamma_{15}(ChildSES\ dummy\ college/adv_j)(Childhood\ Trauma_j) + \upsilon_{1j}$$

$\beta_{2j} = \gamma_{20}$
$\beta_{3j} = \gamma_{30}$
$\beta_{4j} = \gamma_{40}$
$\beta_{5j} = \gamma_{50}$
$\beta_{6j} = \gamma_{60}$
$\beta_{7j} = \gamma_{70}$
$\beta_{8j} = \gamma_{80}$
$\beta_{9j} = \gamma_{90}$
$\beta_{10j} = \gamma_{100}$

Combined:

$$ABP_{ij} = \gamma_{00} + \gamma_{01}(ChildSES\ dummy\ some\ college_j) + \gamma_{02}(ChildSES\ dummy\ college/adv_j) + \gamma_{03} + \gamma_{04}(ChildSES\ dummy\ some\ college_j)(Childhood\ Trauma_j) + \gamma_{05}(ChildSES\ dummy\ college/adv_j)(Childhood\ Trauma_j) + \gamma_{06}(Stress\ (person\ means)_j) + \gamma_{07}(Race_j) + \gamma_{08}(Age_j) + \gamma_{09}(Gender_j) + \upsilon_{0j} + \gamma_{10}(Stress(person-centered)_{ij}) + \gamma_{11}(ChildSES\ dummy\ some\ college_j)(Stress(person-centered)_{ij}) + \gamma_{12}(ChildSES\ dummy\ college/adv_j)(Stress(person-centered)_{ij}) + \gamma_{13}(Childhood\ Trauma_j)(Stress(person-centered)_{ij}) + \gamma_{14}(ChildSES\ dummy\ some\ college_j)(Childhood\ Trauma_j)(Stress(person-centered)_{ij}) + \gamma_{15}(ChildSES\ dummy\ college/adv_j)(Childhood\ Trauma_j)(Stress(person-centered)_{ij}) + \upsilon_{1j}(Stress(person-centered)_{ij}) + \gamma_{20}(Posture\ Standing_{ij}) + \gamma_{30}(Posture\ Laying_{ij}) + \gamma_{40}(Talking_{ij}) + \gamma_{50}(Physical\ Activity_{ij}) + \gamma_{60}(Recent\ Meal_{ij}) + \gamma_{70}(Caffeine_{ij}) + \gamma_{80}(Temperature_{ij}) + \gamma_{90}(Temperature_{ij}) + \gamma_{100}(Temperature_{ij}) + r_{ij}$$

Figure 1. Complete Multilevel Model (Step 5)

Appendix D: Interaction Plots

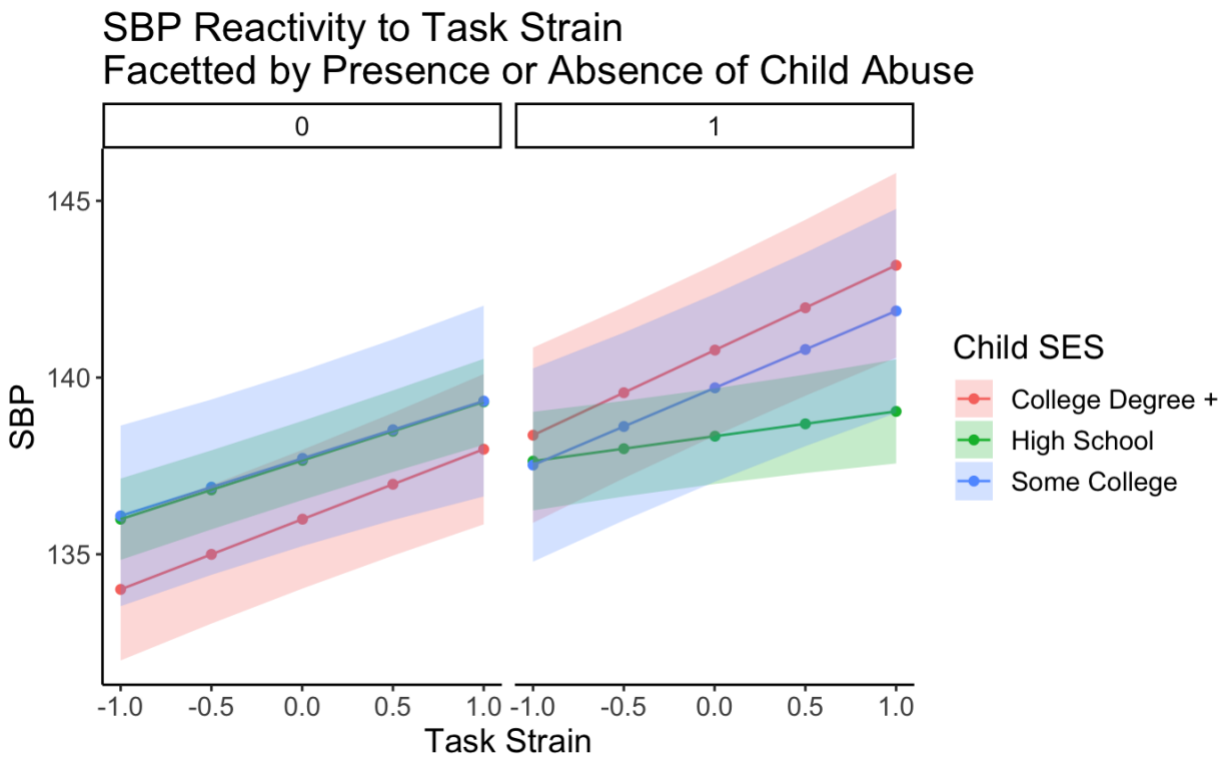

Figure 2. Interactions Between Early Life SES and Childhood Trauma in Predicting SBP Reactivity to Task Strain

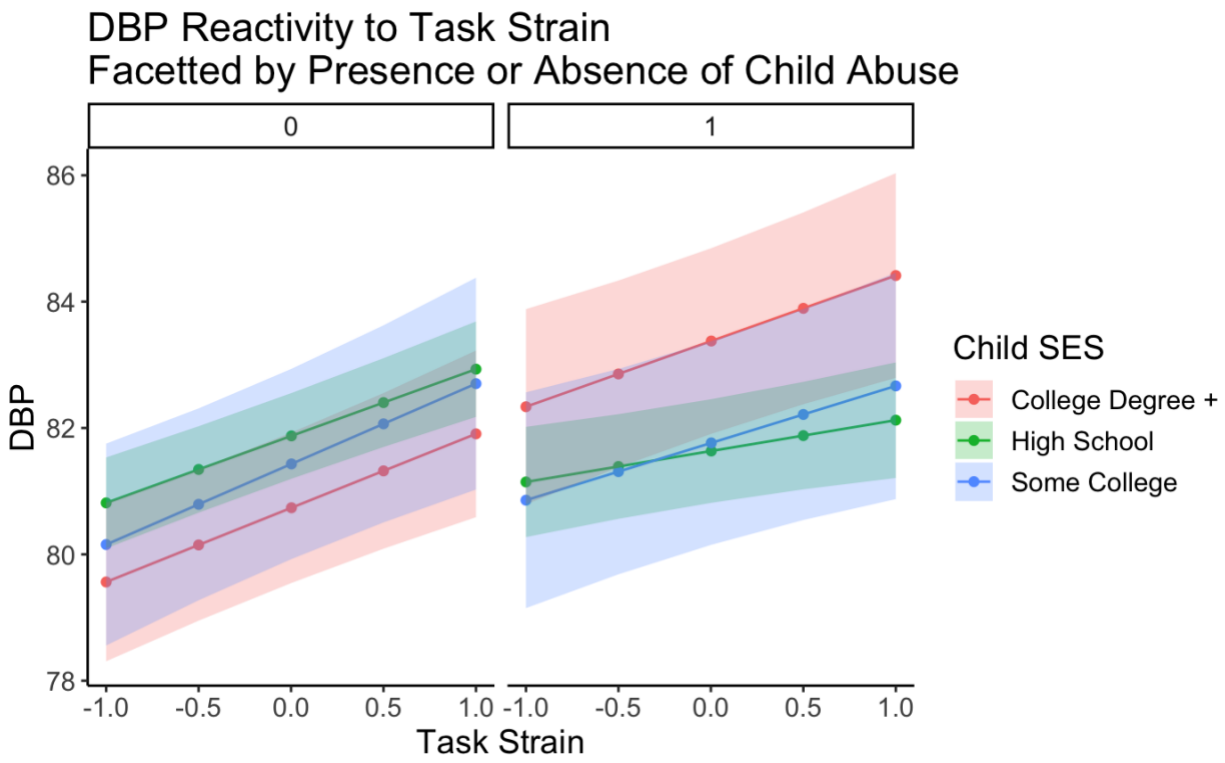

Figure 3. Interactions Between Early Life SES and Childhood Trauma in Predicting SBP Reactivity to Task Strain

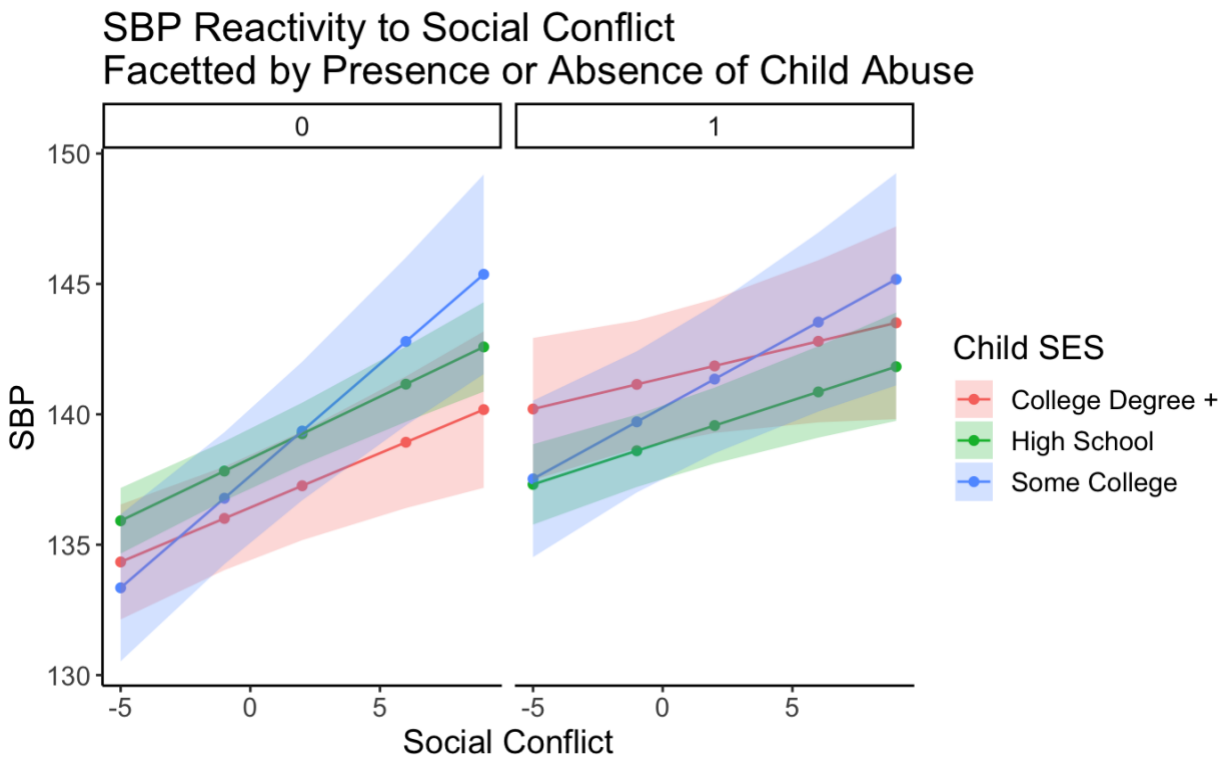

Figure 4. Interactions Between Early Life SES and Childhood Trauma in Predicting SBP Reactivity to Social Conflict

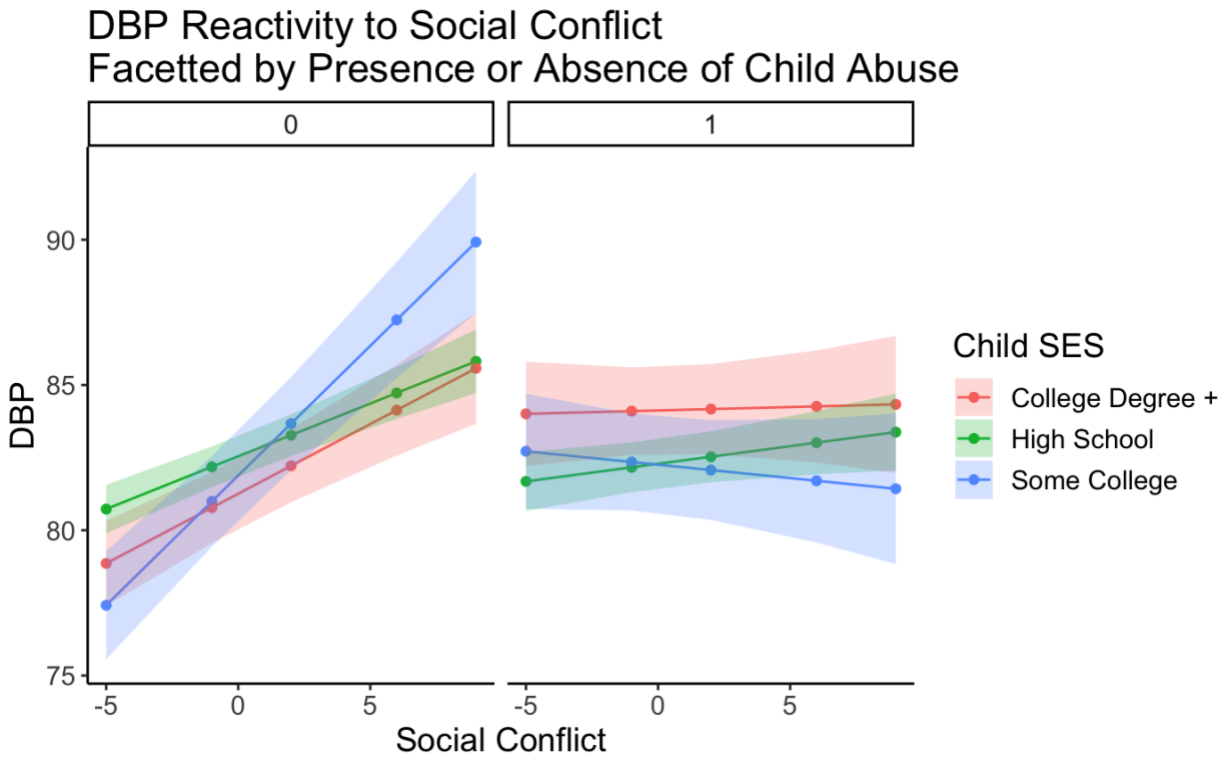

Figure 5. Interactions Between Early Life SES and Childhood Trauma in Predicting SBP Reactivity to Social Conflict

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