

Early Life Adversity and the Stress Response System: Integrating Dimensions of Biological and
Psychological Responses to Stress in Adolescence

By

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This dissertation is dedicated to my family, who listened to my goal of pursuing a career in childhood adverse when I was young and have been paramount in making my dream become a reality every day since.

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CHAPTER I

BACKGROUND

Early life adversity (ELA) represents a particularly detrimental form of stress exposure that is linked to a host of psychiatric problems, including both internalizing and externalizing disorders (Kavanaugh, Dupont-Frechette, Jerskey, & Holler, 2017; Keyes et al., 2012). Indeed, evidence suggests that over 25% of psychological disorders in adolescence and up to 45% of psychological disorders in childhood are linked to ELA (Green et al., 2010; McLaughlin et al., 2012). Disruptions in the development of physiological reactivity to stress, coping strategies, and emotion regulation have been documented in adolescents with ELA (De Bellis & Zisk, 2014; Gruhn & Compas, 2020; Milojevich, Levine, Cathcart, & Quas, 2018) and have etiologic and exacerbating roles in psychopathology (Compas et al., 2017; Doom & Gunnar, 2013), highlighting stress responses as a candidate risk factor in developing psychosocial problems. However, specificity in the association between ELA and physiologic and psychological stress responses remains poorly understood, which hinders attempts to develop novel treatments and preventive interventions. The current study integrates dimensions of automatic, physiological reactivity with controlled, cognitive-behavioral efforts to cope with stress or regulate emotions to understand (a) how adverse stressful experiences early in life impact stress responses later in adolescence, (b) the relation between physiological and cognitive-behavioral response processes with one another, and (c) associations between ELA, stress responses, and symptoms of psychopathology in adolescence.

Biological Stress Response: Autonomic Nervous System Activity

The autonomic nervous system (ANS) is key in maintaining the body's homeostasis and allostasis through influences on multiple organs, including the heart, lungs, and kidneys, and plays a crucial role in automatic physiological stress responding (Gunnar & Quevedo, 2007). During times of perceived stress or threat, the ANS mediates physiological responses via interactions of the sympathetic nervous system (SNS), which works to mobilize physiological resources to respond to environmental demands, and the parasympathetic nervous system (PNS), which works reciprocally to inhibit sympathetic activation and facilitate a return to homeostasis (Porges, 2007).

Skin Conductance Level. The SNS is activated during times of stress, equipping the body for a fight-or-flight response by increasing heart rate and oxygen flow throughout the body (Boucsein, 2012). Skin conductance level (SCL) refers to electrodermal activity caused by the activity of sweat glands which are innervated solely by the SNS component, highlighting SCL an optimal indicator of SNS system functioning (Fowles, Kochanska, & Murray, 2000). Prior research underscores the importance of assessing SNS activity by observing baseline/resting SCL levels (SCL-B) as well as changes in SCL from baseline to stress/challenge conditions, referred to as SCL reactivity (SCL-R), as these assessments provide information on an individual's sympathetic response system (El-Sheikh, Keiley, Erath, & Dyer, 2013; Van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & Van Engeland, 2000). SCL-B is generally conceptualized as an indication of an individual's typical SNS activity, with low SCL-B suggesting low SNS-arousal and high SCL-B suggesting high arousal in the absence of stress. SCL-R provides additional information on the degree to which an individual is physiologically activated when a stressor is introduced (El-Sheikh et al., 2009), and indices of SCL-B and SCL-R each hold

implications for psychosocial functioning and psychopathology in youth. First, greater resting-state SCL (SCL-B) and heightened SCL-R in response to mildly challenging or frightening stimuli (i.e., larger increase from baseline to stressor) are associated with self-reported symptoms of internalizing problems, particularly anxiety, as well as character traits of shyness and inhibition in adolescents (El-Sheikh, 2005; Kagan, Reznick, & Snidman, 1987; Weems, Zakem, Costa, Cannon, & Watts, 2005). While these findings represent generally consistent associations for SCL and internalizing problems, the literature linking SCL with externalizing behaviors has been mixed (Scarpa & Raine, 1997). Higher reactivity (SCL-R) has been associated with aggressive behavior and externalizing problems in youth in some studies (e.g., El-Sheikh, 2005; Hubbard et al., 2002), while others have linked lower levels of SCL-B and SCL-R to externalizing problems (e.g., Fung et al., 2005; Sabine C. Herpertz et al., 2005). Further explication of relations between electrodermal reactivity and maladjustment is needed, including attention to environmental (e.g., ELA) processes (Raine, 2002)

Respiratory Sinus Arrhythmia. The PNS provides counter-regulation to SNS processes, and is involved in regulating (i.e., slowing) heart rate via the innervation of the sinoatrial node of the heart by the vagus nerve in order to return the body to a resting/homeostatic state (Koss & Gunnar, 2018). Respiratory sinus arrhythmia (RSA) is an established parasympathetic marker of self-regulatory capacity and stress responsivity. It is defined as the periodic oscillation in the sinus rhythm occurring at the frequency of respiration and typically displayed as an increase in heart rate with inspiration and a decrease during expiration (Porges, 2007). RSA during resting/baseline conditions (RSA-B) and reactivity of RSA to stress/challenge (RSA-R) are commonly used measures of PNS functioning (Bornstein & Suess, 2000; Calkins, Graziano, & Keane, 2007).

At rest, RSA is thought to represent an individual's flexibility of responses to changing environmental demands (Fortunato, Gatzke-Kopp, & Ram, 2013) and has been associated with the ability to regulate stress, emotional arousal, and attention (Bornstein & Suess, 2000). High RSA at rest (high RSA-B) is indicative of a relatively large difference in heart rate during inspiration as compared with expiration, and suggests a greater capacity for self-regulation (Beauchaine, 2001). Low RSA at rest (low RSA-B) is indicative of a smaller difference in heart rate during inspiration vs. expiration, and suggests atypical PNS functioning and a reduced capacity to enact effective responses to situational events (Beauchaine, 2001). In the context of stress, a greater decrease in RSA (low RSA-R) indicates greater vagal withdrawal, a process that increases the mobilization of physiological and attentional resources to engage in a stress response via PNS inhibition. Vagal augmentation (high RSA-R), indicates increasing PNS activation and dampening of SNS processes (Bornstein & Suess, 2000; Porges, 2007), which may indicate a failure to activate physiological resources that promote optimal engagement with environmental demands (Porges, 2007).

The majority of past work has conceptualized high levels of RSA at rest (RSA-B) and RSA withdrawal (i.e., negative RSA reactivity) as an adaptive physiological response to stress, while the inverse (i.e., low RSA-B; RSA augmentation) is indicative of a maladaptive response. This pattern has been supported by empirical work. For example, low resting RSA has been noted in children and adolescents with clinical levels of internalizing and externalizing behavior problems (Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007), and decreased reactivity to challenge has been noted in children with internalizing and externalizing problems (Calkins, Blandon, Williford, & Keane, 2007) and clinical levels of disruptive behaviors (Degnan, Calkins, Keane, & Hill-Soderlund, 2008). Though most researchers tend to view

greater vagal tone and greater vagal withdrawal during challenge as associated with more adaptive outcomes, contradictory evidence has found that these are associated with negative outcomes. For example, greater baseline RSA has been associated with greater levels of externalizing problems (Calkins, Graziano, et al., 2007; Dietrich et al., 2007). High RSA reactivity during a challenge has been associated with externalizing symptoms in middle childhood to early adolescence (Boyce et al., 2001; Calkins & Dedmon, 2000; Crowell et al., 2006). However, there is also evidence that excessive RSA withdrawal, particularly in response to emotionally charged tasks, is associated with increased internalizing and externalizing symptoms in children, and these relations may be impacted by early-life experiences (Beauchaine, 2015).

ELA and the Autonomic Nervous System. Importantly, the SNS and PNS are heavily dependent on environmental input early in life, and early experiences of trauma, including experiences of deprivation (e.g., neglect) and threat (e.g., neighborhood violence) may disrupt the development of corticolimbic pathways and compromise healthy SNS/PNS functioning (McLaughlin, Sheridan, Alves, & Mendes, 2014; Obradović, 2012). Many questions about the plasticity of the PNS and SNS remain unanswered, but a growing body of literature suggests that interactions with the environment program these systems during sensitive periods of development (e.g., Porges & Furman, 2011). Animal studies highlight links between experiences of acute stressors, long-term social or maternal separation, and disrupted caregiving with alterations in SNS and PNS functioning (Loria, Brands, Pollock, & Pollock, 2013; Trombini et al., 2012). Animals exposed to adverse events (e.g., repeated shocks) have been found to exhibit increased sympathetic nervous system activity and decreased vagal tone when at rest as well as increased SNS reactivity, particularly for stressors that are chronic and higher in severity

(Carnevali et al., 2011; Grippo, Lamb, Carter, & Porges, 2007a, 2007b; Grippo, Trahanas, Zimmerman, Porges, & Carter, 2009; Loria et al., 2013).

Studies with humans have echoed findings in animal studies, such that adolescents with experiences of early-life adversity have consistently been found to exhibit dysregulated physiological stress response patterns (Loman & Gunnar, 2010). Additionally, limited evidence suggests that SCL and RSA may have unique roles in understanding later outcomes in relation to ELA. For example, larger increases in sympathetic activity indexed by skin conductance from baseline to stress tasks (high SCL-R) has been found to increase the link between parent-related stress (observing marital conflict) on child externalizing behaviors (El-Sheikh, Keller, & Erath, 2007). Additionally, high resting RSA has been found to buffer against the negative impact of parent-related stressors (e.g., marital conflict) while low resting RSA has been found to exacerbate the link between parent-related stress and children's behavior problems (Blandon, Calkins, Keane, & O'Brien, 2008; El-Sheikh, 2005).

Despite promising evidence on the role of the autonomic nervous system in the risk pathway between ELA and later adjustment, it is important to note that findings of ANS functioning following early life adversity have been mixed. ELA has been linked to both hyper- (e.g., high SCL-B, high SCL-R) and blunted- (e.g., low SCL-B, low SCL-R) patterns of ANS response (see Loman & Gunnar, 2010 for a review). One possible explanation for mixed findings is the tendency for studies to investigate only one index of SNS (e.g., SCL) or PNS (e.g., RSA) activity as an indicator of all ANS reactivity. Given that the two branches of the ANS have dynamic and complementary responses, assessing the pattern of activity across systems by including measurement of both the SNS and PNS within a single study paradigm represents an important step in understanding relations between ELA and physiological stress responses.

Furthermore, research suggests that the SNS and PNS exhibit different patterns of reactivity across varying contexts (Bush, Alkon, & Obradovic, 2011), and that variable response patterns are partly due to the impact of early experiences (Fries, Shirtcliff, & Pollak, 2008; Oosterman, De Schipper, Fisher, Dozier, & Schuengel, 2010; Rudd & Yates, 2018; Saltzman, Holden, & Holahan, 2005). Thus, the current study investigated PNS and SNS reactivity across ecologically valid nonsocial and social stress tasks.

Psychological Stress Response: Coping, Emotion Regulation

In addition to automatic, physiological responses to stress, individuals intentionally employ cognitive and behavioral resources in reaction to stress. Coping has been defined as conscious, volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stress (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001), whereas emotion regulation (ER) has been defined as the process by which individuals influence the occurrence, timing, nature, experience, and expression of their emotions (Gross, 2013). Although closely related, these constructs differ in that coping exclusively refers to controlled responses to stress, whereas emotion regulation includes efforts to manage emotions under a wider range of situations, and includes both controlled and automatic processes (Compas et al., 2014; Compas et al., 2017; Gross, 2013).

Notably, the current study is focused only on the *controlled* aspects of coping and emotion regulation in response to stress for several reasons. First, controlled processes reflect cognitive and behavioral strategies that are purposefully employed; thus, they may be more accessible to conscious awareness, improving the validity of questionnaire measures (e.g., Compas et al., 2017). Second, controlled processes are less likely than automatic processes to be

confounded with symptoms of psychopathology (e.g., automatic anger responses and aggression symptoms; Achenbach & Rescorla, 2001). Third, controlled processes of coping and emotion regulation can be more readily changed through interventions compared to unintentional regulation processes, increasing the clinical relevance of the current findings (e.g., Compas et al., 2010; Tein, Sandler, Ayers, & Wolchik, 2006; Tein, Sandler, MacKinnon, & Wolchik, 2004). Finally, automatic emotion regulation processes may overlap with measures of automatic stress reactivity patterns, which could impede attempts to delineate relations between coping/ER and physiological reactivity.

This study is guided by a control-based model, which organizes coping and ER strategies into three factors: primary control (i.e., changing the stressor or one's emotions via strategies of problem solving, emotion modulation, or emotion expression), secondary control (i.e., adapting to the stressor via acceptance cognitive reappraisal, or distraction), and disengagement (i.e., avoiding the stressor via denial, wishful thinking, or avoidance) (Compas et al., 2001; Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000). These factors have been differentially related to symptoms of psychopathology across multiple samples, such that primary and secondary control strategies are generally lower levels of internalizing and externalizing symptoms while disengagement strategies are linked to higher levels of symptoms (see Compas et al., 2017, for a meta-analytic review). Effective prevention efforts have increased secondary and primary control strategies and decreased disengagement strategies (Compas et al., 2010; Spence, Sheffield, & Donovan, 2003), highlighting the relevance of assessing specific regulation strategies to inform intervention efforts in populations of maltreated youth.

ELA and Coping/ER. Families provide one of the most powerful and salient contexts of stress, coping, and emotion regulation, placing maltreated youth at an extreme disadvantage for

acquiring adaptive self-regulation strategies. Conceptual models (Morris, Silk, Steinberg, Myers, & Robinson, 2007) and an emerging body of empirical work (Abaied & Rudolph, 2010; Monti, Rudolph, & Abaied, 2014; Watson et al., 2014) suggest that the acquisition of coping and regulation strategies is learned through interpersonal interactions, such as direct communication, modeling, and expressions of support and warmth. Victims of childhood trauma, including parental maltreatment, may therefore fail to acquire adaptive coping and emotion regulation strategies due to decreased exposure to healthy examples of coping during interpersonal interactions and/or increased exposure to maladaptive stress response processes (Kim & Cicchetti, 2010). Further, children may be most sensitive to coping socialization efforts during times of significant challenge, suggesting that maltreated youth may not only be failing to acquire optimal regulation strategies, but also learning maladaptive strategies (Abaied & Rudolph, 2011).

Previous work has established broad deficits in coping and emotion regulation as a result of ELA, and cited coping/ER deficits as an important mediator in the relation between ELA and child/adolescent psychopathology (Jennissen, Holl, Mai, Wolff, & Barnow, 2016). In an effort to more clearly delineate the specific relation between ELA and coping/ER, a recent meta-analysis was conducted on studies reporting an effect in the ELA-coping/ER relationship (Gruhn & Compas, 2020). Results included 35 studies ($N = 11,344$ participants) quantifying ELA and multiple coping/ER categories in children and adolescents, ages 5-18. At the broadest level of coping/ER measurement, *emotion regulation* and *emotion dysregulation* were significantly associated with ELA, indicating decreased endorsement of emotion regulation and increased endorsement of emotion dysregulation were associated with higher levels (or presence compared to absence) of childhood adversity (Gruhn & Compas, 2020). At the more specific, strategy

level, *avoidance* ($k = 7$), *emotional suppression* ($k = 3$), and negative *emotional expression* ($k = 3$) were significantly positively related to adversity, such that ELA is associated with more endorsement of strategies geared toward avoiding stressors and suppressing emotions in relation to stress experiences, respectively (Gruhn & Compas, 2020).

Associations between Biological and Psychological Stress Responses

Understanding relations between coping, emotion regulation, and physiological reactivity to stress represents an important step in conceptualizing responses to stress as a risk factor. In the broad literature, physiology has demonstrated different patterns based on intentional efforts to cope with stress or regulate emotions. For example, disengagement strategies, such as avoidance, have been linked to increased physiological reactivity (e.g., Feldner, Zvolensky, Eifert, & Spira, 2003) while secondary control coping, such as acceptance, have been linked to decreased physiological reactivity (e.g., Connor-Smith & Compas, 2004; Lindsay, Young, Smyth, Brown, & Creswell, 2018).

Cortisol represents the most commonly used measure of physiological reactivity in past studies assessing links to coping/ER, necessitating further research on coping/ER in relation to ANS functioning. Studies focused on sympathetic reactivity have cited low baseline skin conductance levels (SCL-B) and low reactivity (SCL-R) to more effective ER strategies and high baseline (SCL-B) and reactivity (SCL-R) to emotion dysregulation. Parasympathetic nervous system activity has also been associated with to broad indices of ER, such that higher RSA at baseline (RSA-B) and lower RSA-R (i.e., greater decrease in RSA in response to stress) have been linked to adaptive ER strategies (e.g., Bandon et al., 2008; Calkins, 1997; Calkins & Keane, 2004; Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994; Gottman & Katz, 2002;

Hessler & Katz, 2007), and lower RSA-R (smaller decrease in RSA) is related to more maladaptive coping/ER (Santucci et al., 2008). Although this pattern of findings provides support for the premise that SCL and RSA at rest and in response to a stressor may be associated with coping and emotion regulation processes, the specific coping/ER strategies that contribute to “dysregulation” in relation to SNS and PNS functioning remain unknown.

ANS, Coping/ER, and ELA. Research has consistently cited the voluntary ways people cope with stress and regulate emotions (Compas et al., 2017) and the involuntary (e.g., physiological) ways people react to stress (Graziano & Derefinko, 2013; Lorber, 2004) as risk factors for psychopathology, and demonstrated links between ELA and impairments in each of these dimensions of stress responding following ELA (Bernard, Frost, Bennett, & Lindhiem, 2017; Gruhn & Compas, 2020)). However, little is known about the role of coping/ER in regulating physiological processes in the context of ELA. Further understanding of how specific strategies that individuals employ in response to stress relate to SCL and RSA may aid in explaining mixed findings in ANS patterns for youth with ELA.

In addition to influencing the type of coping and emotion regulation skills that children learn to use in response to stress, the effectiveness of a given coping strategy in decreasing vs. amplifying physiological reactivity may be altered. For example, despite links between acceptance and decreased physiological reactivity in the general population, repeated exposure to traumatic life events (e.g., witnessing domestic violence) may negate the effectiveness of this strategy in calming the body. Given the discrepancy between certain forms of ELA and a child’s cognitive and physical capacity to respond, disengagement strategies, such as avoidance, may actually prove *more* adaptive in regulating emotions and physiology, creating a coping/ER-physiology link that is different than that of youth without ELA. To date, however, virtually no

investigations have explicitly tested for moderation of coping/ER-ANS associations by childhood adversity, highlighting major gaps in our understanding of how ELA impacts the stress response system during adolescence.

Study Aims and Hypotheses

Continued exposure to stress during development following experiences of ELA amplifies risk for psychopathology, highlighting the way in which individuals *respond* to stress during adolescence as a potential protective or risk factor (Compas et al., 2017; Repetti, Taylor, & Seeman, 2002). Although previous findings highlight alterations in automatic physiological reactivity to stress, and controlled efforts to cope with stress and regulate emotions following ELA, relatively little research has examined the specific types of coping/ER that are associated with ELA, nor have studies focused on the interplay between physiological reactivity and coping/ER in response to current stressful events for youth who have experienced ELA. Notably, empirical evidence suggests that deficits in regulation strategies can be rectified via teaching adaptive coping/ER skills (e.g., Compas et al., 2010), and emerging evidence suggests that physiological responses may be changeable as well (Cicchetti, Rogosch, Toth, & Sturge-Apple, 2011), making these systems a crucial research target to inform interventions.

The current study examined automatic, physiologic and controlled, cognitive-behavioral dimensions of the stress response system in youth, ELA, and symptoms of internalizing and externalizing problems to allow for a comprehensive assessment of stress-related risk during adolescence. Examining these processes during adolescence is particularly valuable, as this developmental period is characterized by marked increases in both stress exposure (Clark, Rodgers, Caldwell, Power, & Stansfeld, 2007) and psychopathology (Costello, Copeland, &

Angold, 2011). Specifically, observational paradigms, SNS and PNS measurement, multi-informant reports, and semi-structured interviews were used to assess links from ELA to later automatic, physiologic (SNS/PNS) and controlled (coping/ER) stress responses in adolescence. The following aims provide the focus of this research: (1) examine relations between ELA and stress responses, (2) assess associations between automatic, physiologic processes and controlled, cognitive-behavioral responses to stress, and (3) analyze the association between ELA, ANS activity, and coping/ER. Relations between ELA, stress responses, and internalizing and externalizing problems in youth were examined in a fourth, supplementary aim in order to better understand links between these risk pathways and current psychosocial functioning.

First, disruptions in ANS activity (reflected in higher SCL-B and SCL-R), decreased vagal tone (reflected in lower RSA-B) and blunted RSA withdrawal (reflected in higher RSA-R), are hypothesized to emerge for adolescents with higher ELA severity (*Aim 1: Hypothesis 1A*). In analyses of intentional, psychological stress responses, ELA is expected to be related to lower levels of primary control coping (e.g., problem solving), secondary control coping (e.g., acceptance), and cognitive reappraisal, and higher use of disengagement (e.g., avoidance) and expressive suppression. (*Aim 2: Hypothesis 1B*). Second, relations between ANS activity and coping/ER are expected to emerge such that disengagement coping and expressive suppression are related to higher SCL-B, SCL-R, and RSA-R and lower RSA-B, while primary control, secondary control, and cognitive reappraisal are related to lower SCL-B, SCL-R, and RSA-R and higher RSA-B (*Aim 2: Hypothesis 2*). Third, associations between coping and emotion regulation with SCL-B, SCL-R, RSA-B, and RSA-R will be examined at varied levels of ELA (Aim 3). Main effects of ELA, disengagement coping, and expressive suppression are expected to remain associated with ANS in regression analyses in the same direction as hypothesized for correlation

analyses (*Hypothesis 3*). Given the paucity of work assessing the impact of ELA on the coping-physiology relationship, hypotheses are not made regarding which coping/ER strategies will be moderated by ELA, nor whether interactions will more strongly predict SCL or RSA. Last, although not a primary aim of the present study, relations between ELA, stress responses, and internalizing and externalizing problems in youth in order to better understand possible links between these risk pathways to psychosocial problems. Higher levels of ELA, disengagement coping, expressive suppression, SCL-B, SCL-R, and RSA-R are expected to be linked to higher internalizing and externalizing problems in youth. (*Hypothesis 4*).

Supplementary analyses will be conducted to assess whether specific strategies that comprise each response factor uniquely drive associations between coping/ER and ANS activity using at-home survey and in-lab self-reports of coping. The direction of effects is expected to parallel that of the higher-order coping/ER factor, but no hypotheses of which specific strategies will significantly interact with ELA are proposed.

CHAPTER II

METHOD

Participants

Participants from an ongoing study include 56 young adolescents (53.6% female, 64.3% Caucasian) between 10-15 years-old ($M = 12.30$) with a range of early adverse experiences. Participants were excluded if they had a pre-existing neurodevelopmental condition, pervasive developmental disorder, or exhibit evidence of current or past psychosis. Adolescents and their current caregiver were eligible for study participation if the adolescent had resided in the home for at least six months to promote reliability of caregiver-reports on questionnaire measures and increase the relevance of the caregiver-child conflict discussion task. Recruitment for this sample was interrupted by closure of the research laboratory due to the COVID-19 pandemic. An additional 20 families had been recruited for the study and were scheduled for participation but data collection was halted with these additional families due to the closure. Recruitment of families will resume upon the reopening of the research laboratory and analyses with the full sample will be conducted but these additional data are not available for the current analyses.

Procedures

Adolescents and their current caregivers were recruited through multiple sources for study participation through email advertisements, adoption service agencies, and psychiatric outpatient clinics. Interested caregivers completed a brief phone screen to assess eligibility. Caregivers who endorse that adolescents (a) are outside of the age-range of 10-15 years, (b) do

not primarily reside with that caregiver (i.e., at least 50% of the time), (c) have resided with the caregiver for less than six months, or (d) have a pre-existing neurodevelopmental condition, pervasive developmental disorder, or psychosis were excluded from the study. Prior to the initial lab visit, eligible caregivers and adolescents completed a set of questionnaires, including the Emotion Regulation Questionnaire (Gross & John, 2003), Responses to Stress Questionnaire (Connor-Smith et al., 2000), Childhood Behavior Checklist (Achenbach & Rescorla, 2001), and Youth Self Report (Achenbach & Rescorla, 2001), using a secure, web-based data capture system (REDCap; Harris et al., 2009). Caregivers and their adolescent children provided informed assent and consent for the questionnaire portion of the protocol via REDCap in compliance with the Vanderbilt Internal Review Board (IRB). Participants who endorsed clinically significant behaviors and were not in treatment received personalized referrals to mental health centers.

Following completion of at-home questionnaires, in-person assessments were conducted in a private laboratory space in Jesup Hall at Vanderbilt University. Upon arrival, caregivers and youth provided written informed consent and assent for continued study participation. Children completed confidential questionnaires about previous ELA experiences via a computerized interview survey about past and current stressful life events (Stress and Adversity Inventory; Slavich & Epel, 2010). This assessment was offered as an interview for adolescents with reading levels below the fourth grade or who request assistance. In cases in which the participant endorsed an item of safety-concern (e.g., ongoing abuse or neglect, suicidal ideation) on any questionnaire measure, research assistants consulted with clinical supervisors, met with families to discuss the concern as appropriate, and mandated reporting procedures were followed.

Next, adolescents and caregivers participated in a physiological assessment to measure

respiratory sinus arrhythmia (RSA) and skin conductance level (SCL) during resting conditions, two lab challenge tasks, and recovery periods. Physiological sensors (electrodes) were placed on the adolescent with the caregiver present. Adolescents were reminded that all portions of the assessment are voluntary, and were given the option of placing sensors themselves, having the caregiver assist in placing sensors, or having sensors placed by the research assistant. After sensors are placed, the adolescents were given an additional 3 minutes to acclimate, followed by a 3-minute baseline, during which he/she were instructed to sit quietly and breathe normally. A board was then be placed across from the participant's chair and a sheet of paper with a picture of three stars of varying sizes were provided (star-tracing task). Stars were blocked from direct view of the adolescent, but visible through a mirror, and adolescents were asked to trace the stars in descending size order using only the mirror image as a visual guide. After the time period (3 minutes) has ended, a recovery period (3 minutes) ensued. Adolescents then engaged in a 3-minute speaking baseline, where they counted aloud from a list of numbers. Next, caregiver-adolescent dyads were instructed to talk about an issue that they currently engage in conflict about for 10 minutes. Discussions should include how they each feel about it, why it has become a source of conflict, and reach an agreeable resolution.

After completion of the conflict discussion task, the research assistant returned to the room, and adolescents were instructed to engage in a final recovery period (3 minutes) and resting baseline (3 minutes). Sensors was then be removed, and the caregiver and adolescent were debriefed about the research study. During this time, participants were given an opportunity to discuss what happened during the procedures, what feelings or thoughts about ELA or current stress may have surfaced, how each participant felt about the discussion task, and raise any remaining questions they may have. Families received \$100 compensation (\$50 for child, \$50 for

caregiver) for study participation.

Measures

Multiple methods were utilized to assess ELA, stress responses, and symptoms of internalizing and externalizing problems in youth in order to provide a complete representation of these complex constructs (Cole, Martin, Powers, & Truglio, 1996; Greenwald & Farnham, 2000). Table 1 lists each core measure by study, construct, methodology, and respondent. Demographic questionnaires were given to participants to assess age, education, ethnicity, caregiver occupation and marital/partner status.

ELA. Early life adversity was assessed via youth self-report on the Adolescent version of the Stress and Adversity Inventory (Adolescent STRAIN; George M. Slavich, Stewart, Esposito, Shields, & Auerbach, 2019). The STRAIN (Slavich & Epel, 2010) is a NIMH/RDoC recommended online, interview-based system designed to flexibly and reliably assess the occurrence of major life stressors over time. This measure includes comprehensive coverage of all primary life domains assessed by gold-standard life stress interviews (e.g., the Life Events and Difficulties Schedule; Brown & Harris, 1978), and covers major life domains. For each stressor endorsed, participants are asked a series of tailored follow-up questions to determine stressor severity, frequency, timing, and duration. The predictive validity of the STRAIN has been demonstrated in predicting metabolic, mental, and physical health (Kurtzman et al., 2012; Slavich & Epel, 2013), and the reliability and validity of the core questions and interviewing procedures have been well established (for reviews, see Dohrenwend, 2006; Hammen, 2005; Monroe, 2008).

The version of the Adolescent STRAIN employed in the current study (version 1.1)

assesses the severity, frequency, timing, and duration of 12 primary life domains (i.e. Housing, Education, Work, Treatment/Health, Marital/Partner, Reproduction, Financial, Legal/Crime, Other Relationships, Parent/Guardian, Death, Life-Threatening Situations) and five social-psychological characteristics (i.e. Interpersonal Loss, Physical Danger, Humiliation, Entrapment, Role Change/Disruption). The Adolescent STRAIN codes each potential stressor as either present (1) or absent (0), and data on follow-up questions ascertaining severity, frequency, timing, and duration of endorsed (1) stressors is acquired. Consistent with previous work assessing ELA via the STRAIN, the severity index of cumulative adverse life events was chosen as an index of ELA (e.g., Shields, Moons, & Slavich, 2017). Higher scores indicate greater stress exposure and associated severity.

Physiological Reactivity. Adolescents' physiological responses (RSA and SCL) were assessed using the MindWare MW100A Acquisition System Following standard guidelines (Berntson, Cacioppo, & Quigley, 1991; Scerbo, Freedman, Raine, Dawson, & Venables, 1992). Sensors were placed on the rib cage (2 sensors, centered in the right and left rib), collarbone (2 sensors 10-15 cm below each armpit), and chest center (1 at base of throat, 1 at the end of the xyphoid process) to assess RSA, and non-dominant hand (2 sensors on the thenar and hypothenar eminences) to assess SCL. Physiological data was acquired during resting conditions (baseline) and in the context of two lab challenge tasks (star-tracing and caregiver-child conflict task).

The star-tracing task is a well-established, cognitively challenging stressor (Lafayette Instrument Company) that consistently induces RSA and SCL reactivity, which in turn has been associated family conflict and internalizing and externalizing problems in youth (El-Sheikh, 2005; El-Sheikh et al., 2007). The conflict interaction task was a 10-minute videotaped interaction during which adolescent-caregiver dyads discussed a commonly occurring source of

conflict. Conflict topics were chosen via the Issues Checklist (Prinz, Foster, Kent, & O’Leary, 1979), which assesses how often each of 44 topics (e.g., “cleaning up bedroom,” “bothering child when he/she wants to be left alone”) was discussed during the last 2 weeks and instructs participants to rate the intensity level of each conversation on a 5-point Likert scale ranging from 1 (calm) to 5 (angry). Caregiver-adolescent dyads were instructed to discuss the topic endorsed as most ‘emotionally charged’ by both raters for 10 minutes and attempt to come to a resolution. Conflict tasks using a similar protocol have consistently produced ANS activation (Beijersbergen, Bakermans-Kranenburg, Van Ijzendoorn, & Juffer, 2008).

Examination of adolescents’ responses to both non-social (star tracing) and social (conflict interaction) stressors is optimal to characterize physiological reactivity stability over time and provide information about the specificity vs. generality of the role of psychophysiological responses (Chen, Matthews, Salomon, & Ewart, 2002; El-Sheikh, 2005). Because the focus of this study is on individual differences in interpersonal stress responses rather than differential responding to tasks, and because the star-tracing tends to be frustrating and may serve as a prime for arousing negative affect prior to the caregiver-adolescent conflict task, a fixed task order were used (see El-Sheikh et al., 2013): Adaptation (3 min); Resting Baseline (3 min); Star-Tracing Task (3 min); Recovery (3 min); Speaking Baseline (3 min); Conflict Interaction Task (10 min); Recovery (3 min); Resting Baseline (3 min). Adaptation allows for acclimation to the lab and sensors, recovery facilitates the return of ANS activity to resting levels. RSA and SCL were examined continuously and data were synced with the MindWare BioNex system to examine simultaneous readings across ANS measures.

Coping and Emotion Regulation. The Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000) and the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) were

used to assess adolescents' typical coping and emotion regulation patterns in response to stress via self- and caregiver- reports. The RSQ consists of 57 items that represent a range of volitional coping and involuntary responses to stress characteristic of adolescence. Items are rated on a scale from 1 to 4 that indicates the degree to which or frequency with which each response was enacted by the individual from 1 (not at all) to 4 (a lot). Only items assessing volitional coping efforts are included in the present study. These items were selected to represent both cognitive and behavioral responses, and items for the involuntary response scales were selected to capture cognitive, behavioral, emotional, and physiological responses. Primary control coping parcels included problem solving (e.g., I try to think of different ways to change the problem or fix the situation, emotion modulation (e.g., I keep my feelings under control when I have to, then let them out when they won't make things worse), and emotional expression (e.g., I let someone or something know how I feel). Secondary control coping items included cognitive restructuring (e.g., I think about the things that I am learning from the situation, or something good that will come from it), acceptance (e.g., I just take things the way they are, I go with the flow), positive thinking (e.g., I tell myself that everything will be alright), distraction (e.g., I imagine something really fun or exciting happening in my life). Disengagement coping includes wishful thinking (e.g., I deal with the problem by wishing it would just go away, that everything would work itself out), avoidance (e.g., I try to stay away from people and things that make me feel upset or remind me of the problem), and denial (e.g., When something goes wrong with my family, I say to myself, "This isn't real"). The RSQ uses proportional scoring, which takes into account the total number of items endorsed when reporting the factor statistics (Connor-Smith et al., 2000).

The ERQ is a 10-item self-report measure of emotion regulation strategies pertaining to cognitive reappraisal and suppression of emotions (i.e., expressive suppression). Items assessing

cognitive reappraisal include statements such as “when I want to feel more positive emotion (such as joy or amusement), I change what I’m thinking about,” and items assessing expressive suppression included statements such as “I keep my emotions to myself.” Adolescents responded to each statement on a 7-point Likert-scale from 1 (strongly disagree) to 7 (strongly agree) Following standard procedures (Gross & John, 2003), the sum of endorsed cognitive reappraisal items were summed to create a composite of Emotion Regulation- Reappraisal (ERR) and the sum of endorsed expressive suppression items were summed to create a composite of Expressive Suppression (ES). Both the RSQ and ERQ have well-established factor structures (RSQ: primary control, secondary control, and disengagement; ERQ: cognitive reappraisal, expressive suppression) that have been replicated with diverse samples and a wide range of stressors (Andreotti et al., 2013; Balzarotti, John, & Gross, 2010; Compas et al., 2006; Wadsworth, Rieckmann, Benson, & Compas, 2004) and demonstrate good internal consistency, test-retest reliability, and convergent and construct validity (Andreotti et al., 2013; Compas et al., 2017).

Internalizing and Externalizing Problems. The Child Behavior Checklist (CBCL) and Youth Self Report (YSR) were given to the caregiver and adolescent, respectively, to assess adolescent internalizing and externalizing symptoms in adolescents over the past six months (Achenbach & Rescorla, 2001). The YSR and CBCL have demonstrated strong test-retest reliability, and criterion-related validity has been established (Achenbach & Rescorla, 2001). Following previous protocols, scores on the YSR and CBCL for internalizing problem index and externalizing problems index were standardized and averaged in order to create a composite of parent- and child-reported symptoms.

Data Analytic Approach

Preliminary Analyses. Resting levels of skin conductance (SCL-B) were obtained by averaging each 1-minute mean SCL response during baseline conditions, skin conductance levels during stress tasks were obtained by averaging responses during 60 second increments of each stress task (SCL-S), and SCL reactivity (SCL-R) was computed as within-participant difference (change) by subtracting pre-task SCL-B from SCL-S in the star tracing (SCL-R: ST) and conflict discussion (SCL-R: CT) tasks. Skin conductance level reactivity (SCL-R, RSA-R) was computed by subtracting the pre-task, baseline SCL from the mean response during the star tracing and conflict discussion tasks. SCL-R scores greater than zero indicate an increase as compared to baseline, whereas scores less than zero indicate a decrease compared to baseline.

RSA were assessed via rhythmic fluctuations in heart rate that are accompanied by phases of the respiratory cycle (Grossman, Karemaker, & Wieling, 1991) using the peak-to-valley method, which has been validated for the quantification of RSA (Berntson et al., 1997). Resting RSA levels (RSA-B) were computed by using the difference in Interbeat Interval readings from inspiration to expiration onset in baseline assessments. RSA reactivity (RSA-R) were computed as a within-participant difference (change) score of RSA-S relative to RSA-B. Lower (i.e., more negative) RSA-R scores indicate greater vagal withdrawal, while higher (i.e., more positive) RSA-R scores indicate greater vagal augmentation.

All physiological data were scored using MindWare analysis software. Preliminary analyses included paired-sample *t* tests to examine whether RSA and SCL at baseline significantly differed from RSA and SCL during stress tasks as an indication of whether the star tracing and conflict tasks successfully elicited physiologic responses. Based on conceptual models (Compas et al., 2001; Connor-Smith et al., 2000) and past empirical work (Andreotti et al., 2013), ERQ cognitive reappraisal is reported under the category of secondary control

strategies, and ERQ expressive suppression is reported under the category of disengagement strategies. Means and standard deviations were derived and reported for all measures, and bivariate correlations were conducted as a first step in examining relations among variables.

Hypothesis Testing. Bivariate correlations and linear regression analyses were used to address Aim 1 and Aim 2. Correlations were conducted to first examine the bivariate relations between ELA (measured as a continuous score via the STRAIN) and each index of physiology (i.e., SCL-B, SCL-R, RSA-B, RSA-R). Hypothesis 1a would be supported by significant positive correlations in associations between ELA and SCL-B, SCL-R, indicating higher sympathetic activity at resting states and higher change in sympathetic nervous system activity from resting to stress tasks, lower RSA-B, indicating lower parasympathetic activity when at rest, and higher RSA-R, indicating an increase in RSA from baseline to stressor (i.e., blunted RSA withdrawal). Correlation analyses also include relations between ELA and coping. Significant negative correlations are hypothesized to emerge between ELA and primary control coping, secondary control coping and reappraisal, indicating that higher severity in adverse experiences is linked to lower self-reported use of these strategies, and positive correlations are hypothesized to emerge between ELA, disengagement coping, and expressive suppression, indicating that higher severity in adverse experiences is linked to higher self-reported use of disengagement coping and expressive suppression.

Aim 2 (Hypothesis 3) was addressed via bivariate correlations and linear regression analyses. First, bivariate correlations between RSA and SCL baseline and reactivity scores were examined in relation to coping and ER strategies. Results for primary control, secondary control, and cognitive reappraisal are expected to include significant negative correlations in relation to SCL-B, SCL-R, and RSA-R and positive correlations in relation to RSA-B, indicating lower

sympathetic activity when resting, lower sympathetic reactivity, RSA withdrawal, and higher basal RSA (i.e., high resting vagal tone indicating more variability) observed in adolescents who utilize these coping/ER strategies. Significant positive correlations are expected to emerge between disengagement coping and expressive suppression with SCL-B, SCL-R, and RSA-R, indicating higher sympathetic activity when resting, higher sympathetic reactivity, and blunted RSA withdrawal in response to stress tasks, and significant negative correlations are expected for basal RSA (i.e., low resting vagal tone).

Hypothesis tests for the primary aim of the study (Aim 3), to examine main and interaction effects of ELA and coping/ER on ANS, were tested via moderated regression analyses using the PROCESS custom dialog box for SPSS (Hayes, 2013). Variables were mean-centered, independent variables and interaction effects were entered simultaneously, and the PROCESS macro was employed, which automatically calculates the unique variability accounted for by XM in Y, following recommendations by Hayes (2013), Dawson (2014), and Ntoumanis and Appleton (2016). Unstandardized regression coefficients (*B*), standard errors of unstandardized regression coefficients (*SE*), *t*-values, and associated *p*-values were obtained for each independent variable and the interaction. The unstandardized regression coefficient quantifies how the effect of disempowering climates on the outcome variable changes as ELA changes by one unit, and whether the interaction is significant (i.e., $p < 0.05$).

Significant interactions were graphically plotted and probed via simple slopes analyses and the Johnson-Neyman (J-N) technique (Bauer, Curran, & Thurstone, 2005) in PROCESS. The J-N technique describes the variability about the estimate produced by the regression analysis via confidence bands around the simple slope. This technique highlights specific points of the moderator (ELA) where the effect of the X (coping/ER) on Y (ANS) transitions from being

statistically significant to non-significant by finding the value of M for which the ratio of the conditional effect to its standard error is equal to the critical t-score (Barnhofer, Duggan, & Griffith, 2011). By employing the J-N technique, we are able to provide specific empowering climate values at which the negative effects of ELA on the targeted ANS outcomes are buffered or exacerbated by coping/ER.

To address Aim 3, bivariate correlations and linear regression analyses were used to better understand the association between ELA history, physiological reactivity, coping/ER, and internalizing and externalizing problems in youth. Regression equations included ELA severity, physiology, and coping/ER as independent variables with internalizing and externalizing problems serving as dependent variables in separate equations. Given the possibility of multicollinearity between variables, all variables were centered in regression analyses via PROCESS. All predictors were entered simultaneously.

Last, Aim 4 was examined through bivariate correlation analyses that included ELA, coping, ER, SCL and RSA baseline and reactivity scores, and parent- and child- reported internalizing and externalizing problems. CBCL and YSR scores were highly correlated for indices of internalizing ($r = .52, p < .001$) and externalizing ($r = .72, p < .001$) problems. To reduce the number of analyses and address single-informant bias, composite scores were created for internalizing and externalizing problems by converting scores from adolescent and caregiver reports on the CBCL and YSR to z -scores and calculating the mean for each coping factor. A probability level of 0.05 adopted a priori as the cutoff for determining statistical significance in all analyses. Results at a probability level of 0.10 are also reported as approaching significance in order to provide a comprehensive overview of findings.

CHAPTER III

RESULTS

Preliminary Analyses

Means and standard deviations for main study variables are presented in Table 2. Data were screened for quality and distribution. Because SCL and RSA values were substantially skewed, these data were log transformed for all analyses following standard protocol (El-Sheikh et al., 2009; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Tabachnick & Fidell, 2012), though Table 2 displays raw means for interpretability. Reactivity scores for ANS can be interpreted as follows: Positive values of SCL-R indicate an increase of sympathetic nervous system activity from baseline to stress task (i.e., high SNS response), and negative values of SCL-R indicate a decrease of sympathetic nervous system activity from baseline/resting to stress. Negative RSA-R scores indicates that RSA decreased (vagal withdrawal, or relatively less application of the vagal brake) in response to the stimulus. Positive RSA reactivity score indicates that RSA increased (vagal augmentation, or relatively more application of the vagal brake) in response to the stress task. Additionally, associations between demographic characteristics (e.g., age, gender) and key study variables were assessed, and no significant relations emerged (p -values $> .10$). Thus, no demographic variables were controlled for in regression analyses.

As a test of the degree to which the laboratory parent-child discussion task was stressful for the participants, SCL and RSA were compared at baseline and during the star tracing and conflict discussion task via paired sample t -tests. Lab challenges were designed to elicit SCL

reactivity, and a paired sample t test revealed that SCL increased from baseline to stress task for star tracing $t(54) = 6.83, p < .001$ and the conflict discussion task $t(52) = 5.73, p < .001$. RSA significantly decreased ($t(48) = -2.56, p < .02$) from baseline to star tracing but did not significantly change from baseline to conflict task ($t(49) = .27, p = .78$). Correlations among baseline and reactivity scores were nonsignificant for SCL (SCL-B and SCL-R Star Tracing: $r = -.01, p > .10$; SCL-B and SCL-R Conflict Task: $r = .05, p > .10$), but significantly negatively correlated for RSA (RSA-B and RSA-R Star Tracing: $r = -.36, p < .01$; RSA-B and RSA-R Conflict Task: $r = -.55, p < .01$).

Aim 1: ELA and Stress Responses

Bivariate correlation analyses were conducted in order to assess associations between ELA, coping, ER, and ANS activity in resting and stress tasks. Consistent with hypothesis 1A, ELA was significantly negatively correlated with RSA-B ($r = -.31, p < .05$), indicating that higher severity of cumulative stressful experiences is related to decreased vagal tone when adolescents are in a resting state or baseline. In correlations examining relations between ELA and reactivity scores (i.e., SCL and RSA change from baseline to star tracing, SCL and RSA change from baseline to conflict task), two of 8 bivariate correlations emerged as significant. First, ELA was significantly positively correlated with SCL-R in the star tracing task in the hypothesized direction ($r = .29, p < .05$), indicating higher SNS reactivity to the nonsocial stress task for individuals with higher ELA.

Hypothesis 1B was partially confirmed. ELA was significantly negatively correlated with secondary control coping as hypothesized, such that higher ELA severity is related to lower use of secondary control coping ($r = -.47, p < .001$). Supplementary analyses examining relations

between ELA and specific coping/ER strategy were conducted. Of the strategies that comprise secondary control coping, acceptance ($r = -.50, p < .001$) and positive thinking ($r = -.33, p < .05$) were significantly negatively correlated with ELA, and the correlation between cognitive restructuring and ELA approached significance ($r = -.27, p < .07$), indicating that higher ELA severity is linked to lower use of these strategies (see Appendix Table 1). Contrary to hypotheses, ELA was not significantly related to reappraisal, expressive suppression, primary control or disengagement on survey measures.

Aim 2: Coping, Emotion Regulation, and Autonomic Nervous System Activity

Associations between autonomic nervous system activity and child self-reported efforts to cope with stress and regulate emotions were examined to address study Aim 2 via bivariate correlations, regression analyses, and simple slope analyses to probe significant interaction effects. Primary control coping was significantly associated with RSA-R-Star Tracing ($r = -.31, p < .05$), indicating that more primary control coping is linked to greater RSA withdrawal in the nonsocial stress task, consistent with Hypothesis 2a. No significant associations emerged between secondary control coping, disengagement, reappraisal, or expressive suppression with ANS indices.

Supplementary analyses were conducted to assess whether item parcels that comprise each RSQ coping factor (i.e., primary control, secondary control, and disengagement coping) or uniquely drive associations between coping/ER and ANS activity (see Appendix Table 1). The correlation between acceptance and RSA-B approached significance ($r = .24, p = .06$), indicating that higher use of acceptance may be associated with increased vagal tone at baseline in the hypothesized direction. Emotion modulation was significantly negatively correlated with RSA-R

during the star tracing task ($r = -.41, p < .01$), highlighting an association between this strategy and greater vagal withdrawal.

Aim 3: ELA, Coping, Emotion Regulation, and ANS Activity

Moderated regression analyses were conducted to analyze ELA, coping/ER, and the interaction between ELA and coping/ER in order to better understand possible differential associations of coping efforts with autonomic nervous system activity for adolescents based on severity of childhood adversity experiences. First, models predicting basal SCL and RSA are presented in Table 4. Of the ten calculated models predicting resting ANS activity, only Model 3 (disengagement coping) approached significance for both SNS and PNS indices, SCL model: $F(3, 52) = 2.38, p = .08$; RSA model: $F(3, 52) = 2.42, p = .07$. A significant interaction effect of ELA and disengagement coping emerged predicting SCL-B ($t = -2.66, p = .01$). The Johnson-Neyman approach was used to probe the interaction (Hayes & Matthews, 2009). This approach identified values of ELA where the conditional effect of coping/ER on ANS transitioned from non-significant to significant ($\alpha = .05$). J-N significance regions were -33.32 (7.55%) and 16.90 (26.42%). Individuals with higher ELA demonstrated significant negative associations between disengagement coping and SCL-B, highlighting higher resting-state SCL ($t = -2.27, p = .03$), in line with study hypotheses (see Figure 1).

Model 3 (Disengagement) predicting RSA-B yielded a significant main effect of ELA and interaction effect for ELA and disengagement coping in predicting basal RSA. First, ELA was negatively linked to RSA-B, such that lower vagal tone at baseline is associated with higher ELA ($t = -2.06, p = .05$), consistent with hypotheses. The interaction effect for disengagement coping by ELA approached significance at $\alpha = .06$ ($t = 1.91, p = .06$). The J-N significance

region was identified as 7.51, such that individuals who experienced ELA above this threshold (32% of the sample) demonstrated higher RSA-B when using disengagement coping ($t = 2.31, p = .03$) (see Figure 2). This finding, contrary to study hypotheses, suggests that disengagement coping is linked to higher basal vagal tone for individuals with high ELA. No significant findings emerged between disengagement coping and RSA-B for individuals with low ELA. In SCL-B Model 5, the interaction between reappraisal and ELA neared significance ($t = 1.84, p = .07$), such that greater use of reappraisal on the RSQ was associated with lower basal skin conductance for individuals with low ELA only ($t = -1.70, p = .09$). However, this model was not significant, $F(3,49) = 1.44, p > .10, R^2 = .04$ and results should be interpreted with caution.

Next, models predicting autonomic nervous system reactivity to the nonsocial stress task (i.e., difference in mean SCL and RSA during star tracing task and baseline) were calculated. Results are presented in Table 5. No significant main or interaction effects emerged in models predicting ANS reactivity to the nonsocial (star tracing) stress task with the exception of RSA-R: Star Tracing, Model 1. In this model, one significant main effect emerged such that primary control coping was significantly negatively related to RSA-R ($t = -2.30, p = .03$). This finding suggests that higher levels of primary control coping are related to greater RSA withdrawal, consistent with hypotheses.

Last, in models predicting autonomic nervous system reactivity to the social (conflict discussion) stress task, Model 4 (Reappraisal) approached significance for SCL-R, $F(3, 53), p = .07, R^2 = .34$. In this model, main effects for ELA ($t = 2.01, p = .05$), reappraisal ($-1.83, p = .07$), and an interaction effect between ELA and reappraisal ($t = -2.48, p = .02$) emerged as significant or approaching significance in predicting SNS reactivity. The interaction was further probed, and J-N significance yielded 6.44 as the value of ELA at which the reappraisal-SCLR association

becomes significant, which includes 34.69% of the sample. Results suggest that reappraisal is related to higher SCL-R at low levels of ELA and significantly lower levels of SCL-R for individuals with high levels of ELA (see Figure 3). Of note, the interaction between disengagement coping and ELA in SCL-R: Conflict Task Model 3 approached significance ($t = 1.71, p = .09$); however, the overall model was not significant ($F(3, 47) = 1.12, p = .35$) and simple slope and J-N tests did not yield further significant findings.

Aim 4: Internalizing and Externalizing Problems

Although not the primary aim of the study, association between ELA, stress responses, and internalizing and externalizing problems were examined via correlations in order to better understand possible links between these risk pathways to psychosocial problems in this population. Results are presented in Table 3. ELA was positively correlated with internalizing ($r = .42, p < .091$) and externalizing ($r = .38, p < .01$) symptoms, indicating that higher cumulative severity of adverse experiences is linked to more psychosocial problems in youth.

In analyses of coping/ER strategies, primary control coping and secondary control coping were significantly negatively correlated with internalizing (PC: $r = -.38, p < .01$; SC: $r = -.56, p < .001$) and externalizing problems (PC: $r = -.29, p < .05$; SC: $r = -.34, p < .05$) problems in the hypothesized directions. Disengagement coping was significantly positively correlated with internalizing ($r = .47, p < .001$) problems, and the association between disengagement coping and externalizing problems approached significance ($r = .24, p = .08$). No relations emerged between self-reported cognitive reappraisal or expressive suppression on the ERQ with internalizing or externalizing problems.

Last, in bivariate correlation analyses of ANS activity and internalizing and externalizing

symptoms, SCL-R-Star Tracing was significantly positively correlated with externalizing problems ($r = .28, p < .05$). This finding suggests that high sympathetic reactivity to the nonsocial (star tracing) stress task is related to increased self- and parent- reported externalizing problems in youth, consistent with Hypothesis 4. The correlation between SCL-R-Conflict task and externalizing problems approached significance ($r = .24, p = .10$) in the expected direction, indicating a possible link between higher increases in sympathetic activity to interpersonal stress. The correlation between RSA-R in the Conflict Task and externalizing problems also approached significance, indicating a possible negative relation between these constructs ($r = -.26, p = .06$). Notably, finding is contrary to study hypothesis, and indicates that greater RSA withdrawal may be linked to higher externalizing problems.

CHAPTER IV

DISCUSSION

Disruptions in the development of stress response systems have been posited to be a central mechanism underlying the association between ELA and psychopathology (Gunnar & Quevedo, 2007; McLaughlin et al., 2014), yet patterns of physiologic response and specific coping and regulation strategies observed among adolescents exposed to adverse experiences have varied widely across studies (Fries et al., 2008; MacMillan et al., 2009). Additionally, very few investigations have incorporated indices of both SNS and PNS activity, nor have studies examined relations between intentional coping and emotion regulation efforts with automatic physiologic responses. As a result, considerable gaps exist between prevailing theories and existing evidence of how ELA influences stress responses in youth.

The current study applied a well-validated theoretical model that differentiates between primary, secondary, and disengagement coping (Compas et al., 2001; Connor-Smith et al., 2000; Rudolph, Dennig, & Weisz, 1995) that has not previously been utilized in the study of ELA in conjunction with real-time physiological measurements in order to better understand the interplay of these processes and relations to early life experiences. Aims of the current study include examining associations between: (1) ELA and stress responses, including both automatic, biological responses and intentional, psychological responses (2) coping/emotion regulation and sympathetic and parasympathetic nervous system activity, (3) coping/ER with ANS indices, moderated by ELA and (4) ELA, stress responses, and internalizing and externalizing problems in youth. Results provide important implications for the impact of

childhood adversity on stress responses and functioning, and provide a foundation for future research.

Preliminary analyses demonstrated a significant change in SNS and PNS measures from baseline to star tracing and a significant change in SNS, but not PNS, from baseline to conflict task. These tasks were designed to elicit sympathetic nervous system activity (El-Sheikh, 2005); thus the results confirm that the tasks were effective in yielding sympathetic nervous system response. The lack of change in RSA between baseline and conflict task raises a possibility that, given the significant increase in sympathetic nervous system activity, the parasympathetic nervous system may not be working in tandem with this response to either withdrawal or release the ‘vagal break.’ Reciprocal SNS and PNS patterns (i.e., high SNS/low PNS; low SNS/high PNS) are conceptualized as adaptive automated responses, as they reflect more normative and directional responses to stress at the physiological level promoting homeostasis. Preliminary evidence suggests that examining patterns of SCL *and* RSA may provide unique implications for either buffering against or compound negative effects of ELA (El-Sheikh et al., 2009; Gordis et al., 2010), highlighting this as an important direction for future research. Additionally, the lack of correlation between SCL at baseline and reactivity to tasks is surprising, given that reactivity scores are calculated in relation to baseline. However, past research has found minimal associations between rest and reactivity levels of ANS indices (e.g., Calkins & Keane, 2004; El-Sheikh, 2005, El-Sheikh, 2007), indicating that each parameter may provide unique information about physiological activity.

To address Aim 1, associations between ELA and stress responses, including cognitive-behavioral (coping/ER) and physiological (SCL, RSA) responses were examined via bivariate correlation analyses in order to assess how the cumulative severity of previous stressful life

events impact later stress responses. ELA exposure was hypothesized to be linked to higher sympathetic (SCL-B) and lower parasympathetic activity (RSA-B) in resting conditions, and a threat pattern of autonomic nervous system reactivity characterized by heightened skin conductance (high SCL-R) and blunted respiratory sinus arrhythmia/vagal withdrawal (high RSA-R) in response to stress tasks. Only two of the six hypothesized effects emerged in analyses, thereby partially supporting this hypothesis. First, ELA was significantly related to basal RSA, indicating that higher severity of cumulative stressful experiences is related to decreased vagal tone when adolescents are in a resting state or baseline in this sample. This finding is consistent with study hypothesis and past work linking lower resting RSA following chronic stress (Friedman & Thayer, 1998; Lipschutz, Gray, Weems, & Scheeringa, 2017). ELA was not significantly related to SCL-B, indicating possible specific links between cumulative stress experiences and the parasympathetic, rather than sympathetic, nervous system at when resting. Second, SCL-R-Star Tracing was significantly related to ELA, indicating that higher severity of cumulative stressful experiences is related to higher SNS reactivity. However, ELA was not significantly related to SCL reactivity in the conflict task. Further research is needed to understand why ELA was related to SCL-R in the star tracing but not conflict interaction task.

One possible explanation for the differential relation between ELA and the two stress tasks in the present study arises from various correlates that may be specific to social vs. nonsocial stressors. For example, (Chen et al., 2002) found that greater ANS reactivity during a social stressor were related to higher adolescent reports of interpersonal strivings for others' acceptance and affiliation, whereas greater ANS reactivity during a nonsocial stressor were associated with higher ratings of competitiveness, self-defensiveness, and expressiveness. Thus, it is possible that ELA differentially impacts factors such as competitiveness, self-defensiveness,

and expressiveness that heighten sympathetic reactivity to nonsocial stress. No significant correlations between ELA and RSA reactivity in the star tracing or stress task, suggesting that associations between ELA and autonomic nervous system activity in this sample are more prominent for SNS than PNS indices.

Following analyses of ELA and ANS activity, ELA was examined in relation to intentional efforts to cope with stress or regulate emotions. Greater severity of stressful life events was hypothesized to be related to lower levels of primary control coping, secondary control coping, and reappraisal, and higher use of disengagement and expressive suppression. Partial support was found for this hypothesis. A significant association emerged for ELA and secondary control coping emerged indicating that ELA was significantly related to lower use of secondary control coping. Supplementary analyses of ELA and item parcels that comprise secondary control coping suggest that this finding may be primarily driven by lower levels of self-reported acceptance, positive thinking, and cognitive restructuring. These findings are consistent with previous work (e.g., Bal, Crombez, Van Oost, & Debourdeaudhuij, 2003; Min et al., 2017; Tremblay, Hébert, & Piché, 1999), and build upon findings by highlighting acceptance, positive thinking, and cognitive restructuring as specific strategies that may be impacted by ELA. This may be because continued exposure to stressful experiences interfere with children's ability to acquire or employ these strategies, or altered thoughts about the usefulness of these strategies. For example, an individual who has continually faced traumatic experiences at higher levels of severity may acquire a belief that using a strategy like positive thinking is ineffective, as it does not prevent exposure to additional problems.

Contrary to the hypothesis, primary control coping and disengagement strategies, including expressive suppression, was not significantly related to ELA severity. The lack of

association between ELA and disengagement strategies is particularly surprising, given that avoidance and suppression strategies are among the only consistently cited strategies to be linked to ELA (Gruhn & Compas, 2020). Importantly, the lack of significant associations between ELA and these strategies in the current study may be a function of the sample, which was not recruited based on one specific type of ELA, in conjunction with the assessment of ELA employed. The STRAIN, which was used to index ELA in the current study, incorporates a wide variety of stressors across multiple domains (e.g., housing, education, financial, death). It is possible that the connection between ELA with primary control coping or disengagement coping may be driven by specific types of stressful experiences (e.g., deprivation/neglect, Milojevich, Norwalk, & Sheridan, 2019). Future studies should work to disentangle broad and specific effects of ELA type on cognitive-behavioral responses to stress.

The second aim of the current study was to assess relations between automatic, physiological stress responses and intentional, cognitive-behavioral responses to stress in order to better understand the interplay between these processes. At the bivariate level, primary control coping was negatively related to RSA-R in the star tracing task, and this association remained significant in the linear regression analysis. The direction of this effect indicates that greater self-reported use of strategies directed at changing either the stressor (i.e., problem-solving) or one's own emotions (i.e., emotion modulation, emotional expression) are related to greater vagal withdrawal during the nonsocial stress task. Past work suggests that vagal withdrawal (disengaging the vagal brake or RSA decrease from resting to stressor) in response to stress may be an adaptive response that permits an individual's biological system to respond to challenge (i.e., increased heart rate and arousal; Graziano & Derefinko, 2013). The star-tracing was designed as an inherently controllable, yet challenging, nonsocial stress task, as it requires

participants to carefully trace within the outline of a star looking only through a mirror. Given the nature of this task, the association between RSA-R and primary control coping supports the conceptualization of primary control coping as particularly beneficial for stress that can be changed/controlled (Compas et al., 2017), and extends this work by demonstrating a direct link to parasympathetic reactivity in response to a controllable-stress task. Interestingly, subsequent exploratory analyses of item-parcels within the primary control coping factor suggest that this finding may be driven by emotional modulation. Working to directly alter one's own emotions during a nonsocial controllable stress task may therefore be particularly beneficial in regulating parasympathetic nervous system reactivity and increasing vagal withdrawal.

No other significant associations between coping/ER and ANS indices emerged at the bivariate level, however several interaction effects emerged in linear regression analyses in the examination of ELA, coping/ER, and ANS activity (Aim 3). At baseline, the interaction between ELA and disengagement was associated with basal SCL and RSA, suggesting that, despite the lack of direct association to early adversity, disengagement coping may have strong implications for physiology at rest. Specifically, results indicate that disengagement coping is linked to lower SCL and higher RSA only for individuals with high levels of ELA, suggesting that strategies of avoidance, wishful thinking, and denial may predict lower sympathetic activity and higher parasympathetic activity at baseline. This pattern of resting-state ANS activity has been conceptualized in the literature as adaptive, indicating decreased hyper-vigilance in the absence of threat and a greater capacity for self-regulation (Porges, 2007). Resting vagal tone in particular has been generally viewed as the individual's ability to maintain homeostasis and autonomic flexibility to generate adequate responses to environmental demands, with high RSA-B (high vagal tone) representing an increased capacity to respond appropriately to environmental

demands and low RSA-B (low vagal tone) representing a restricted ability to respond to stress. Thus, it is possible that disengagement coping is adaptive in preparing the parasympathetic nervous system to respond to environmental threat for adolescents with greater experiences of ELA. It is important to acknowledge that the interaction effect only approached significance in the model predicting RSA-B (alpha level = .06), however. Additionally, firm conclusions on the “adaptive” vs. “maladaptive” nature of basal SCL and RSA in this sample cannot be drawn, given the variability of past findings (Loman & Gunnar, 2010). One important future direction of this work includes 12- month longitudinal follow-ups of internalizing and externalizing symptoms, which will provide further insight into how this interaction relates to current functioning.

Only one significant interaction effect (ER-Reappraisal x ELA predicting SCL-R: Conflict Task) emerged in analyses examining associations between ELA, coping/ER, and reactivity to the star tracing and conflict discussion stress tasks. For individuals with high levels of ELA severity, greater self-reported reappraisal was significantly related to lower reactivity to the conflict task, but this effect was not significant for individuals with low ELA. Items on the measure of reappraisal used in this study focus on cognitive-based techniques with the end-goal of down-regulating one’s own reactivity to stress (e.g., “When I’m faced with a stressful situation, I make myself think about it in a way that helps me stay calm”). Thus, this result suggests that changing the way that one is thinking about a stressor may be particularly effective in down-regulating sympathetic activity when dealing with conflict in the caregiver-child relationship following ELA.

Taken together, results highlight disengagement coping and cognitive reappraisal as two types of coping and emotion regulation that may have a differing impact on physiology

depending on the cumulative severity of stressful experiences that an adolescent has been exposed to. The lack of significant interactions between ELA and primary control coping, secondary control coping, and expressive suppression predicting ANS may indicate that these strategies work similarly for individuals regardless of childhood adversity. However, these associations should be further investigated, as the lack of significance could be attributable factors such as the relatively low sample size of $N = 56$ for conducting moderation analyses (see *Limitations*).

In the final aim of the study, associations between ELA, stress responses, and internalizing and externalizing problems were examined in order to better understand ELA and stress response patterns in relation to current psychosocial functioning. Consistent with past work, greater severity of ELA was linked to increased caregiver and self-reports of adolescent internalizing and externalizing symptoms. Links between psychosocial problems and coping/ERQ partially supported hypothesis 4, such that primary and secondary control coping were linked to lower internalizing and externalizing problems and disengagement coping was linked to higher levels of internalizing and externalizing problems in the current sample. However, reappraisal and expressive suppression on the ERQ did not correlate with symptoms in this sample.

In analyses of PNS and SNS, higher sympathetic reactivity in response to the star tracing task was significantly related to greater externalizing problems, consistent with past empirical work and study hypotheses. Importantly, the cross-sectional design limits conclusions on directionality. Therefore, it is unknown whether sympathetic reactivity to nonsocial stress is a function of symptoms, or whether this reactivity pattern instigates externalizing behaviors. The correlation between SCL reactivity to the conflict task approached significance, in line with research highlighting links between anxiety and reactivity to interpersonal stress (Yoon &

Joormann, 2012). The association of high SCL-R in the star-tracing (nonsocial) task to externalizing problems and high SCL-R in the conflict (social) task to internalizing problems warrants further study to assess whether the nature of stress (i.e., interpersonal vs. individual) uniquely predicts relations between SCL-R and psychopathology symptoms.

Notably, the relation between RSA reactivity in response to the conflict task approached significance, indicating that higher RSA withdrawal may be related to more externalizing problems. This finding is contrary to the study hypotheses, and suggests that greater vagal withdrawal from baseline to an interpersonal stress task may be linked to higher levels of problem behaviors directed outward. Findings of RSA-R and functioning have been mixed in adolescent populations, with studies citing low RSA-R as both a protective factor against developing internalizing or externalizing symptoms (El-Sheikh, Harger, & Whitson, 2001; El-Sheikh & Whitson, 2006; Katz & Gottman, 1997), as well as a risk factor (Boyce et al., 2001; Calkins, Graziano, et al., 2007). Future studies should continue to investigate this association, employing longitudinal designs and assessing possible moderators (e.g., demographic characteristics, type of ELA), in order to clarify links between ANS activity and internalizing and externalizing problems in youth with and without ELA.

Limitations and Future Directions

The current study compliments and extends past research by integrating measures of ELA, coping, emotion regulation, SCL, and RSA to investigate the impact of adverse experiences on the association between biological and psychological dimensions of the stress response system. However, results should be interpreted within the limitations of the study design. First, the sample size of $N = 56$ resulted in relatively low statistical power to detect

effects, particularly interaction effects. Analyses yielding a significance value of $p < .10$ were reported in order to provide a more comprehensive picture of the link between study variables, but results at this threshold ($.05 < p < .10$) should be interpreted with caution (Aiken & West, 1991). This study is ongoing, and 20 families are currently on the waitlist to complete the study protocol who were unable to engage in research due to the COVID-19 pandemic. Thus, an important future direction of this research includes enrolling additional participants, which will allow for increased power to detect effects.

A second important limitation of the current study is the cross-sectional study design, which limits conclusions on the direction of effects. Given the lack of consistent ANS patterns in relation to ELA, coping/ER, future studies should employ longitudinal designs to better understand the causal paths between adverse experiences and physiology. Third, coping and emotion regulation were assessed through child self-reports on survey measures. Questionnaires are the most widely used method to assess coping and emotion regulation in childhood and adolescence and demonstrate consistent links to psychopathology in youth (Compas et al., 2017). However, reliance on questionnaire measures is subject to a number of limitations. While these measures have consistently shown associations with symptoms of psychopathology in youth, reliance on questionnaire measures is subject to a number of limitations. First, utilizing child-reports of coping/ER introduces potential shared method variance, such that associations between coping/ER and internalizing and externalizing problems may become inflated as a result of method bias (Podsakoff, MacKenzie, & Podsakoff, 2012). Additionally, these measures assess adolescents' typical use of coping and emotion regulation strategies rather than the strategies that adolescents were using during the stress tasks. Although individuals are generally thought to have coping/ER styles representative of their typical responses to (Compas et al., 2017),

assessing the specific strategies used during stress tasks would highlight real-time links between intentional and automatic/physiological stress responses

Conclusions

The current study represents the first investigation of how ELA differentially impacts the association between specific coping and emotion regulation strategies with sympathetic and parasympathetic activity in adolescents. Findings indicate that ELA severity is related to lower use of secondary control coping, particularly acceptance and positive thinking, higher sympathetic reactivity to nonsocial stress, lower vagal tone at baseline and decreased vagal withdrawal from baseline to an interpersonal stress task. Additionally, interaction effects highlight disengagement coping as a strategy that may have unique implications for resting-state ANS for individuals with more severe ELA history, while reappraisal may have specific effects on sympathetic nervous system reactivity to social stress for individuals with high ELA. Findings underscore the importance of considering the ANS in concert with intentional, cognitive behavioral efforts to facilitate responses to stress. Individual differences in ANS functioning were not solely attributable to ELA, but instead may be better conceptualized as resulting from dynamic processes that develop over time and are influenced by interactions among multiple levels. Disentangling the independent and interrelated engagement of biological and psychosocial stress response patterns in adolescents with ELA remains an important target for future research, and could enhance knowledge about the etiology of stress response patterns and inform prevention and early intervention efforts in this high-risk population.

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TABLES

Table 1. Schedule of Measurement Strategies

Construct	Format	Instrument (Respondent)
Demographics	Survey	Demographics Survey (P)
Early Life Adversity		
Stressful Life Experiences	Survey	STRAIN (C)
Automatic Stress Response		
Sympathetic Nervous System: SCL	Physiological Assessment	MW100A Acquisition System (O)
Parasympathetic Nervous System: RSA	Physiological Assessment	MW100A Acquisition System (O)
Controlled Stress Response		
Coping	Survey	RSQ (C)
Emotion Regulation	Survey	ERQ (C)
Psychopathology Symptoms		
Internalizing Problems	Survey	CBCL (P)
		YSR (C)
Externalizing Problems	Survey	CBCL (P)
		YSR (C)

Note. P = Parent report on child; STRAIN = Stress and Adversity Inventory; C = Child; SCL = Skin Conductance Level; MW = MindWare; O = Objective/Observational; RSA = Respiratory Sinus Arrhythmia; RSQ = Responses to Stress Questionnaire; ERQ = Emotion Regulation Questionnaire; CBCL = Child Behavior Checklist; YSR = Youth Self Report.

Table 2. Descriptive Statistics ($N = 56$)

Variable	Mean	SD	Minimum	Maximum
Child Characteristics				
Child Age	12.30	1.67	10.00	15.00
ELA Severity	41.58	25.53	1.00	102.00
Emotion Regulation				
ERQ Reappraisal	4.40	1.22	1.50	7.00
ERQ Suppression	3.69	1.16	1.00	5.75
Coping				
RSQ Primary Control Coping	0.17	0.04	0.08	0.25
RSQ Secondary Control Coping	0.24	0.06	0.14	0.44
RSQ Disengagement Coping	0.16	0.03	0.10	0.24
Physiology: SNS				
SCL-B	11.31	7.41	0.56	35.72
SCL-R Star Tracing	2.14	2.48	-2.11	10.07
SCL-R Conflict	3.27	3.95	-2.53	18.34
Physiology: PNS				
RSA-B	6.52	1.10	3.71	8.83
RSA-R Star Tracing	-0.39	0.83	-3.09	1.01
RSA-R Conflict	0.04	0.94	-2.27	2.30
Psychosocial Problems				
CBCL Internalizing Problems (T)	67.10	10.85	33.00	83.00
CBCL Externalizing Problems (T)	51.46	10.79	34.00	74.00
YSR Internalizing Problems (T)	56.10	9.92	39.00	81.00
YSR Externalizing Problems (T)	52.12	9.14	29.00	74.00

Note. ERQ = Emotion Regulation Questionnaire; RSQ = Responses to Stress Questionnaire; SNS = Sympathetic Nervous System; SCL-B = Baseline Skin Conductance Level; SCL-R = Skin Conductance Level Reactivity from Baseline; PNS = Parasympathetic Nervous System; RSA-B = Basal Respiratory Sinus Arrhythmia; RSA-R = Respiratory Sinus Arrhythmia Reactivity from Baseline; CBCL = Child Behavior Checklist; YSR = Youth Self Report.

Table 3. Correlation Matrix of ELA, Stress Responses and Internalizing and Externalizing Problems ($N = 55$)

	ELA	PC	SC	DIS	ERR	ES	SCLB	RSA B	SCL RST	RSA RST	SCL R CT	RSA R CT	Int	Ext
ELA	---													
PC	-.12	---												
SC	-.49**	.30*	---											
DIS	.14	-.57**	-.43**	---										
ERR	.09	.18	.41**	-.23	---									
ES	.08	-.35*	.03	.32*	.30*	---								
SCL-B	-.07	-.11	-.11	.04	-.12	.16	---							
RSA-B	-.31*	.19	.15	.05	.07	-.04	-.04	---						
SCLR-ST	.29*	-.05	-.18	-.08	-.15	.01	-.01	-.01	---					
RSAR-ST	.05	-.31*	-.07	.16	.02	.06	.01	-.36**	.22	---				
SCLR-CT	-.14	.05	.02	-.08	-.18	.02	.05	.41**	.38**	-.17	---			
RSAR-CT	-.15	.07	.02	.02	-.01	-.05	.21	-.44**	-.01	.51**	-.12	---		
Int	.42**	-.38**	-.56**	.34*	-.21	.21	-.16	.05	.19	.04	.24†	-.18	---	
Ext	.38**	-.29*	-.34*	.24†	-.05	.10	-.16	-.09	.28*	-.04	.03	-.26†	.32*	---

Note. † $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$. ELA = Early Life Adversity; PC = Primary Control Strategies; SC = Secondary Control Strategies; DIS = Disengagement Strategies; ERR = Emotion Regulation Reappraisal; ES = Expressive Suppression; SCL-B = Skin Conductance Level at Baseline; RSA-B = Respiratory Sinus Arrhythmia at Baseline; SCLR-ST = Skin Conductance Level Reactivity from Baseline to Star Tracing; RSAR-ST = Respiratory Sinus Arrhythmia Reactivity from Baseline to Star Tracing; SCLR-CT = Skin Conductance Level Reactivity from Baseline to Conflict Task; RSAR-CT = Respiratory Sinus Arrhythmia Reactivity from Baseline to Conflict Task; Int = Internalizing Problems; Ext = Externalizing Problems.

Table 4. Regression Models of Baseline ANS ($N = 54$)

Variable	<u>SCL-Baseline</u>					<u>RSA-Baseline</u>					
	<i>B</i>	<i>SE (B)</i>	<i>t</i>	<i>p</i>	<i>Model R²</i>	<i>B</i>	<i>SE (B)</i>	<i>t</i>	<i>p</i>	<i>Model R²</i>	
Model 1: Primary Control (PC)					.01						.13
ELA	.00	.00	-.65	.52		.00	.00	-1.51	.14		
PC	-.38	1.33	-.29	.78		.30	.30	.98	.33		
PC X ELA	.02	.06	.36	.72		.00	.01	-.27	.78		
Model 2: Secondary Control (SC)					.06						.11
ELA	.00	.00	-.87	.39		.00	.00	-.85	.40		
SC	-.99	1.00	-.99	.33		.02	.33	.06	.95		
SC X ELA	.00	.05	.00	.90		.00	.01	.38	.70		
Model 3: Disengagement (DC)					.14						.19
ELA	.00	.00	-1.07	.29		.00	.00	-2.06	.05*		
DC	-1.96	1.68	-1.16	.25		.58	.38	1.54	.13		
DC X ELA	-.18	.07	-2.66	.01*		.03	.02	1.91	.06 [†]		
Model 4: Emotion Regulation- Reappraisal (ERR)					.04						.10
ELA	.00	.00	-.96	.34		-.01	.01	-1.63	.11		
ERR	.01	.01	.92	.36		-.09	.15	-.63	.53		
ERR X ELA	.00	.00	1.84	.07 [†]		.45	.00	.01	.82		
Model 5: Emotional Suppression (ES)					.01						.13
ELA	.00	.00	-.35	.73		.00	.00	-1.82	.08 [†]		
ES	.00	.01	-.21	.83		.00	.00	1.16	.25		
ES X ELA	.00	.00	-.37	.71		.00	.00	-.31	.76		

Note. [†] $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$. SCL = Skin Conductance Level; RSA = Respiratory Sinus Arrhythmia; ELA = Early Life Adversity.

Table 5. Regression Models of Star Tracing Task ANS Reactivity ($N = 54$)

Variable	<u>SCL-R: Star Tracing</u>					<u>RSA-R: Star Tracing</u>					
	<i>B</i>	<i>SE (B)</i>	<i>t</i>	<i>p</i>	<i>Model R²</i>	<i>B</i>	<i>SE (B)</i>	<i>t</i>	<i>p</i>	<i>Model R²</i>	
Model 1: Primary Control (PC)					.05	Model 1: Primary Control (PC)					.10
ELA	.00	.00	1.26	.22		.00	.00	-.07	.94		
PC	-.18	1.14	-.16	.87		-1.11	.48	-2.30	.03*		
PC X ELA	-.03	.05	-.60	.54		-.01	.03	-.48	.63		
Model 2: Secondary Control (SC)					.09	Model 2: Secondary Control (SC)					.00
ELA	.02	.02	1.08	.29		.00	.00	.02	.99		
SC	-.13	.66	-.20	.84		-.10	.40	-.25	.80		
SC X ELA	-.15	.31	-.47	.64		.00	.03	.12	.90		
Model 3: Disengagement (DIS)					.10	Model 3: Disengagement (DIS)					.03
ELA	.01	.00	1.37	.17		.00	.01	.05	.96		
DIS	1.15	2.32	.49	.62		.81	.87	.93	.36		
DIS X ELA	.12	.12	1.07	.29		.01	.04	.35	.73		
Model 4: Emotion Regulation- Reappraisal (ERR)					.14	Model 4: Emotion Regulation- Reappraisal (ERR)					.04
ELA	.01	.01	1.38	.17		.00	.01	.28	.78		
ERR	-.01	.01	-1.16	.25		-.01	.12	-.08	.94		
ERR X ELA	-.01	.01	-1.20	.24		-.01	.01	-1.01	.32		
Model 5: Emotional Suppression (ES)					.06	Model 5: Emotional Suppression (ES)					.00
ELA	.01	.01	1.10	.28		.00	.01	.13	.89		
ES	-.01	.01	-.31	.76		.02	.10	.22	.82		
ES X ELA	-.01	.01	-.92	.36		.00	.00	.08	.93		

Note. † $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$. SCL-R = Skin Conductance Level Reactivity from Baseline; RSA-R = Respiratory Sinus Arrhythmia Reactivity from Baseline; ELA = Early Life Adversity.

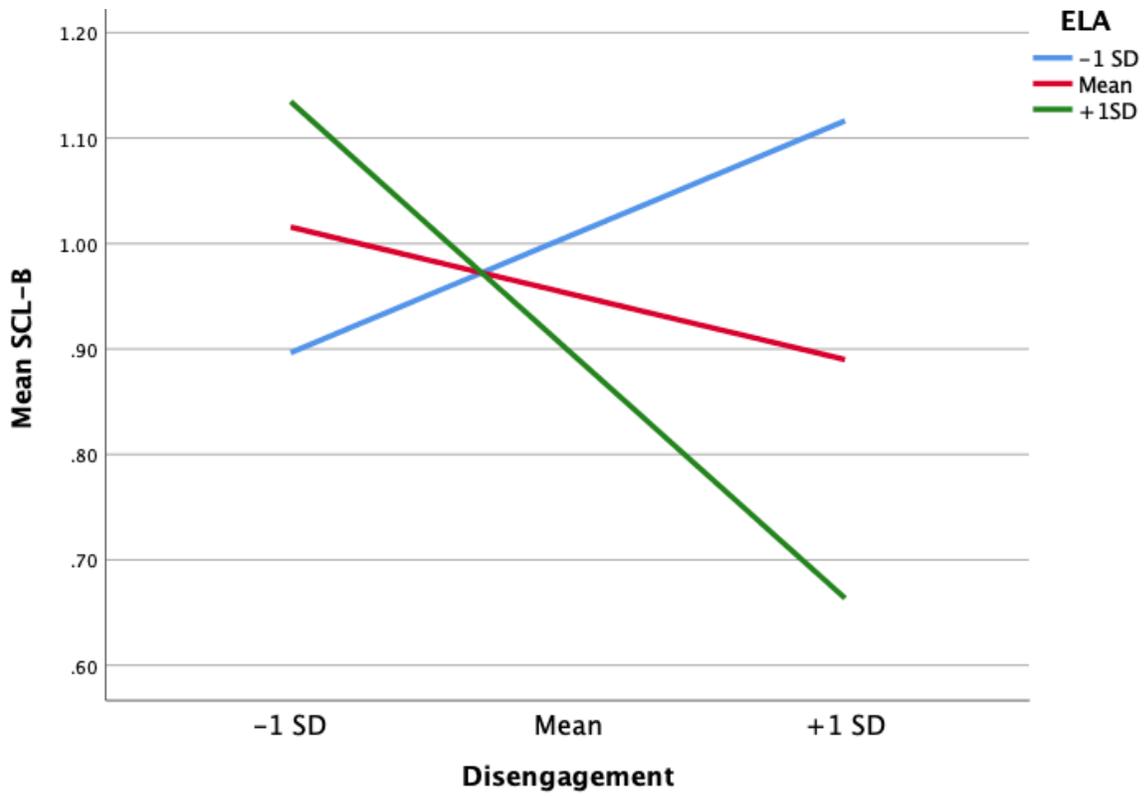
Table 6. Regression Models of Conflict Task ANS Reactivity ($N = 53$)

<u>SCL-R: Conflict Task</u>						<u>RSA-R: Conflict Task</u>				
Variable	<i>B</i>	<i>SE (B)</i>	<i>t</i>	<i>p</i>	<i>Model R²</i>	<i>B</i>	<i>SE (B)</i>	<i>t</i>	<i>p</i>	<i>Model R²</i>
Model 1: Primary Control (PC)						Model 1: Primary Control (PC)				
					.07					.03
ELA	.00	.00	1.30	.20		.00	.00	-.48	.63	
PC	.02	1.21	.02	.98		-.61	.47	-1.30	.20	
PC X ELA	-.07	.07	-.98	.33		-.01	.03	-.47	.64	
Model 2: Secondary Control (SC)						Model 2: Secondary Control (SC)				
					.03					.02
ELA	.00	.00	1.16	.25		.00	.00	-.63	.53	
SC	.52	1.00	.53	.60		-.17	.37	-.47	.64	
SC X ELA	-.03	.06	-.56	.58		.00	.02	.15	.88	
Model 3: Disengagement (DIS)						Model 3: Disengagement (DIS)				
					.16					.02
ELA	.00	.00	1.46	.15		.00	.00	-.45	.66	
DIS	1.72	2.28	.75	.46		.23	.66	.35	.73	
DIS X ELA	.20	.11	1.71	.09 [†]		.00	.03	-.19	.85	
Model 4: Emotion Regulation- Reappraisal (ERR)						Model 4: Emotion Regulation- Reappraisal (ERR)				
					.34					.09
ELA	.01	.00	2.01	.05*		.00	.00	-.63	.53	
ERR	-.02	.01	-1.83	.07 [†]		-.05	.15	-.29	.77	
ERR X ELA	-.01	.00	-2.48	.02*		-.01	.01	-1.43	.16	
Model 5: Emotional Suppression (ES)						Model 5: Emotional Suppression (ES)				
					.06					.03
ELA	-.01	.02	-.36	.72		.00	.00	-.42	.68	
ES	.02	.26	.09	.93		.00	.00	-.28	.78	
ES X ELA	.00	.01	-.11	.92		.00	.00	.50	.62	

Note. [†] $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$. SCL-R = Skin Conductance Level Reactivity from Baseline; RSA-R = Respiratory Sinus Arrhythmia Reactivity from Baseline; ELA = Early Life Adversity.

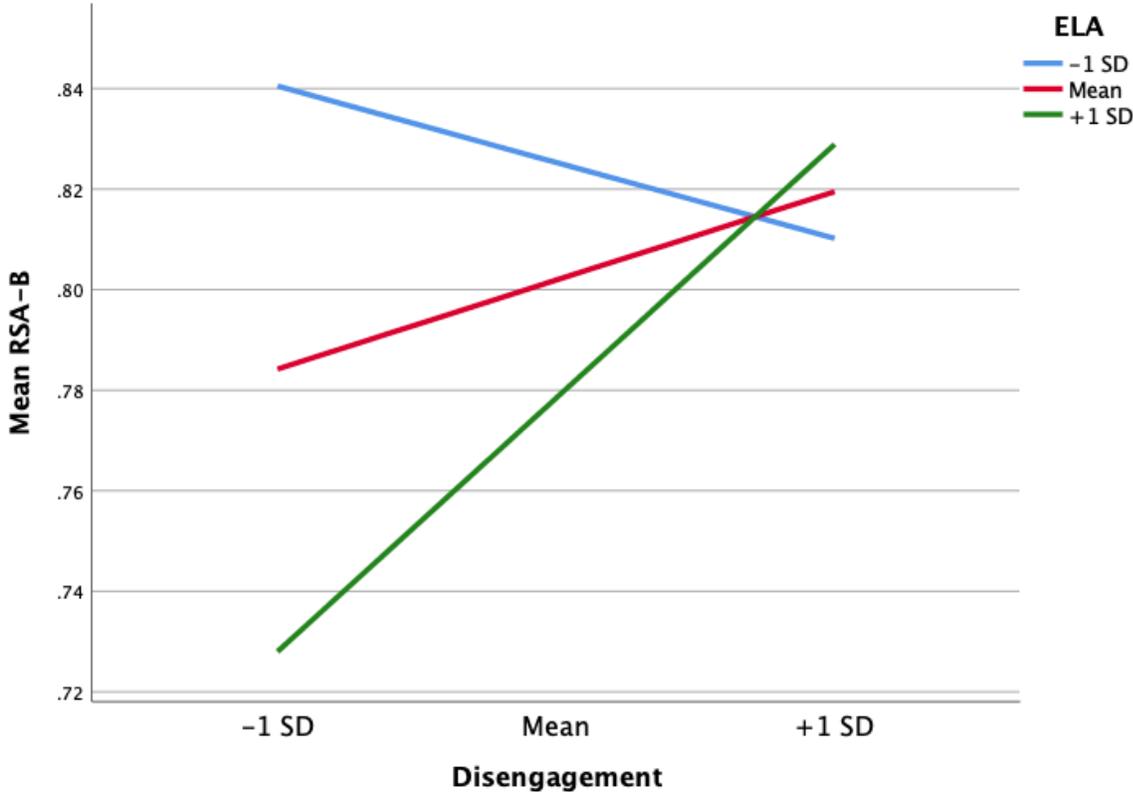
FIGURES

Figure 1. Disengagement and ELA Predicting Baseline SCL



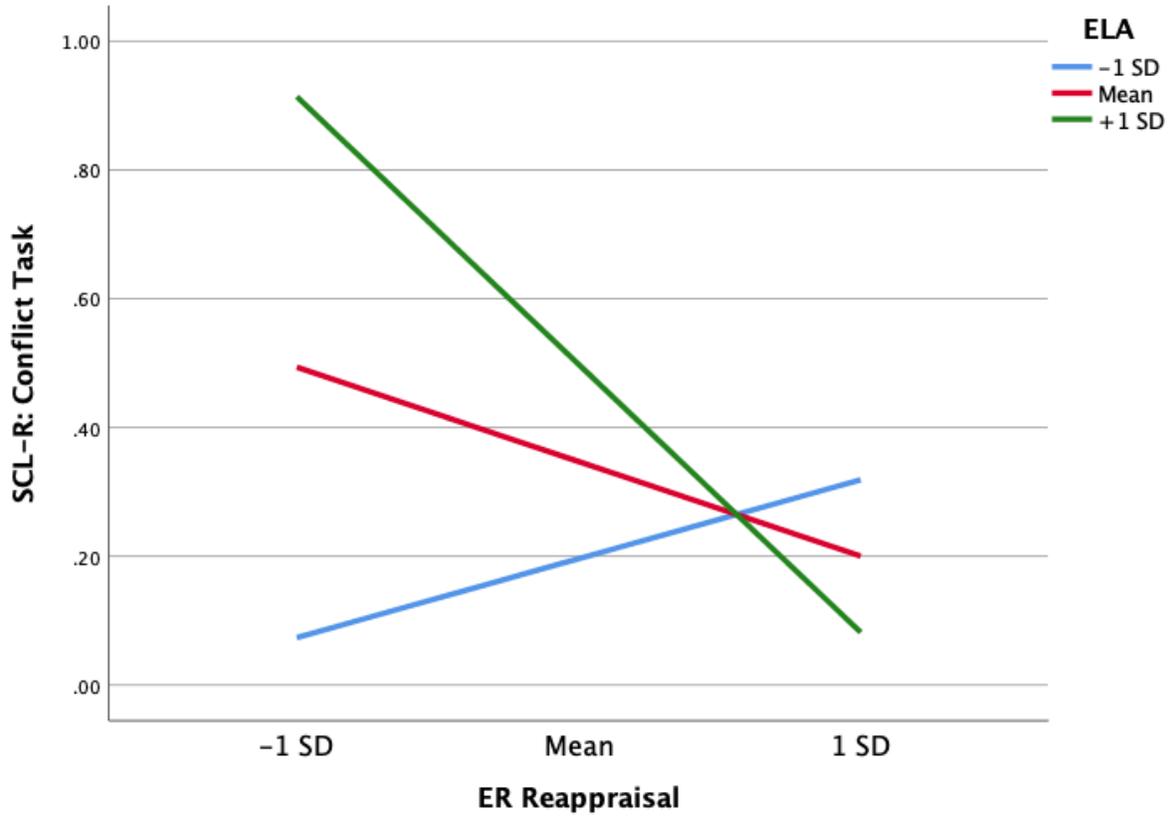
Note. SCL-B = Skin Conductance Level at Baseline; ELA = Early Life Adversity.

Figure 2. Disengagement and ELA Predicting Baseline RSA



Note. RSA-B = Respiratory Sinus Arrhythmia at Baseline; ELA = Early Life Adversity.

Figure 3. Reappraisal and ELA Predicting SCL-R (Conflict Task)



Note. SCL-R = Skin Conductance Level Reactivity from Baseline; ER Reappraisal = Reappraisal on the Emotion Regulation Questionnaire; ELA = Early Life Adversity.

APPENDIX

Appendix Table 1. Bivariate Correlation Matrix of Coping/ER Strategies, ELA, and ANS (N = 55)

	ELA	SCL-B	RSA-B	SCLR-ST	RSAR-ST	SCLR-CT	RSAR-CT
Primary	-.12	-.11	.19	-.05	-.31*	.16	.13
EE (RSQ)	.00	-.04	.09	.05	-.20	.19	.15
EM (RSQ)	-.19	-.07	.12	-.26	-.41**	-.03	-.01
PS (RSQ)	-.08	-.11	.11	.05	-.10	-.08	.21
Secondary	-.49**	-.08	.10	-.18	-.07	.02	-.05
Acpt (RSQ)	-.50**	-.04	.24 [†]	-.20	-.23	.02	-.02
Dist (RSQ)	-.20	-.12	.14	.01	-.18	.17	-.22
PT (RSQ)	-.33*	-.04	-.07	-.15	.04	-.01	.01
CR (RSQ)	-.27 [†]	-.04	-.05	-.14	.21	-.11	.03
Disengagement	.14	.04	.02	-.08	.16	-.08	-.04
Avd (RSQ)	.18	-.02	.03	-.04	.11	-.06	-.20
WT (RSQ)	.05	.06	.01	.03	.16	.01	-.09
Den (RSQ)	.07	.03	.02	-.20	.03	-.15	.26

Note. [†] $p < .10$. * $p < .05$. ** $p < .01$. ELA = Early Life Adversity; SCL-B = Skin Conductance Level at Baseline; RSA-B = Respiratory Sinus Arrhythmia at Baseline; SCLR-ST = Skin Conductance Level Reactivity from Baseline to Star Tracing; RSAR-ST = Respiratory Sinus Arrhythmia Reactivity from Baseline to Star Tracing; SCLR-CT = Skin Conductance Level Reactivity from Baseline to Conflict Task; RSAR-CT = Respiratory Sinus Arrhythmia Reactivity from Baseline to Conflict Task; EE = Emotional Expression; RSQ = Response to Stress Questionnaire; EM = Emotional Modulation; PS = Problem Solving; Acpt=Acceptance, Dist = Distraction; PT = Positive Thinking; CR = Cognitive Reappraisal; ERR = Emotion Regulation-Reappraisal; ERQ = Emotion Regulation Questionnaire; Avd = Avoidance; WT = Wishful Thinking; Den. = Denial; ES = Expressive Suppression.