# Arousal and Regulation:

# Ecological Momentary Assessment in Adolescents with a Range of Exposure to ACEs

By

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*As a very small tribute*
*to their unconditional love and unwavering encouragement of me pursuing my dreams,*
*this work is dedicated to my incredible parents,*
*Gail and Dennis Henry.*

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| | |
|---|---|
| ACE | adverse childhood experience |
| ANS | autonomic nervous system |
| CBCL | Child Behavior Checklist |
| CTQ-SF | Childhood Trauma Questionnaire – Short Form |
| ECG | electrocardiogram |
| EMA | ecological momentary assessment |
| ER | emotion regulation |
| DC | disengagement coping |
| HPA | hypothalamic-pituitary-adrenocortical |
| HR | heart rate |
| HR-R | heart rate - reactivity |
| HRV | heart rate variability |
| PCC | primary control coping |
| PDS | Pubertal Development Scale |
| RA | research assistant |
| RSQ-FD | Responses to Stress Questionnaire – Family Discussion |
| SCC | secondary control coping |
| VMR | video-mediated recall |
| YSR | Youth Self Report |

# CHAPTER I

# INTRODUCTION

## Importance of Studying ACEs

Exposure to adverse childhood experiences (ACEs) is a transdiagnostic risk factor that can alter developmental trajectories in mental and physical health (Albott et al., 2018). The seminal paper from the original ACEs study found a dose-response effect between ACEs and psychopathology (Felitti et al., 1998), and since that time, exposure to ACEs has been linked to almost every psychiatric disorder and syndrome (Baldwin et al., 2021; Shonkoff et al., 2012). Given the profound impact of ACEs on development, enhancing our understanding of the pathways by which ACEs contribute to psychopathology is an important research focus. Elucidating underlying mechanisms of dysfunction can help identify targets for intervention, detect subgroups of responders to inform intervention selection, and reduce the impact of ACEs on development (Insel, 2010). Stress reactivity and coping/emotion regulation (ER) have been shown to mediate and moderate the association between stress and psychopathology and thus are two promising targets for research and intervention (McLaughlin & Lambert, 2017; Miu et al., 2022).

## Stress Reactivity and Coping/ER in Typically Developing Adolescents

ACEs are thought to become embedded in psychobiology to shape future responses to stress (Del Giudice et al., 2011; McEwen, 1998), and disruption in the development of the automatic stress response system may partially explain the strong association between ACEs and psychopathology (Heim & Nemeroff, 2001; Tarullo & Gunnar, 2006). One critical pathway by which automatic self-regulation and reactivity occurs in response to stress is through the autonomic nervous system (ANS; Scarpa, 2015). ANS stress reactivity can be measured as heart

rate variability (HRV), or the variation in the period between consecutive heartbeats over time (Berntson et al., 1997). HRV is thought to reflect the ability of the ANS and cardiovascular system to adapt to changing circumstances by detecting and responding to stimuli (Kim et al., 2018). At rest, higher HRV, or a greater variation in the time interval between heartbeats, reflects the ability to quickly adapt to challenge (Fabes & Eisenberg, 1997), and lower HRV, or a smaller variation in the time interval between heartbeats, suggests restricted ability to respond to challenges and predicts increased risk for morbidity, mortality, and psychopathology (Beauchaine & Thayer, 2015; Thayer & Lane, 2007). However, research has been mixed with regard to changes in HRV *in response to stress*; research shows both heightened and blunted reactivity are related to internalizing and externalizing problems (e.g., Phillips et al., 2013). Emerging evidence suggests that moderate HRV reactivity may be the most adaptive response (Hamilton & Alloy, 2016).

Stress reactivity and coping/ER are intertwined (Thayer & Lane, 2000), in that coping/ER represents individuals' ability to influence their stress reactivity from moment to moment (Gross, 1998). The closely related constructs of coping and ER (see Compas et al., 2014) involve conscious, controlled, and purposeful efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to a stressful event (Compas et al., 2017). Understanding coping/ER as a mediator and moderator of the relation between ACEs and psychopathology is particularly important because coping/ER skills are malleable; interventions incorporating coping/ER skills training have shown effects in preventing psychopathology (Compas et al., 2011). One model of coping/ER organizes strategies around the controllability of the stressor (e.g., Compas et al., 2001; Weisz et al., 1994). Three types of coping responses are outlined in this control-based model: primary control coping (PCC) involves acting directly on a

stressor or related emotions (e.g., problem solving, emotion expression); secondary control coping (SCC) involves adapting to a stressor (e.g., acceptance, cognitive reappraisal); and disengagement coping (DC) describes efforts to avoid, deny, or wish away a stressor (Connor-Smith et al., 2000). Greater use of PCC and SCC have been associated with lower levels of internalizing and externalizing symptoms, and greater use of DC has been associated with higher levels of symptoms (Compas et al., 2017). This model has been validated in adolescents from diverse cultural backgrounds with exposures to a variety of stressors (e.g., Benson et al., 2011; see Compas et al., 2017 for a review).

**Stress Reactivity and Coping/ER in Adolescents with ACEs**

Animal models provide a basis for understanding the impact of early adversity on developing physiological responses to stress (O'Donnell & Meaney, 2020; Plotsky & Meaney, 1993). In rodent and non-human primate models, offspring separated from their mothers show heightened stress reactivity in adolescence and adulthood, along with elevated behavioral manifestations of psychological symptoms (Huot et al., 2001; Schneider et al., 2004). Sensitization of developing corticolimbic pathways to be hypervigilant to stress may be adaptive in the short-term (i.e., the individual becomes mobilized to respond to stress) but physiologically taxing over time (Heim et al., 2000). When response systems are overactivated during extended periods of adversity, adaptive reactions to stress may be extinguished; blunted stress reactivity represents a conditioned avoidance of threatening stimuli associated with minimal physiological activation in response to stress (e.g., De Bellis et al., 1994). Accordingly, research examining stress reactivity in humans exposed to early adversity is more varied (e.g., Lovallo et al., 2012). Some studies suggest that adolescents with ACEs exhibit heightened reactivity to stress (e.g.,

Fries et al., 2008; Oosterman et al., 2010), and other studies show a blunted response (e.g., Fisher et al., 2012; Gunnar et al., 2009).

Exposure to ACEs themselves, as well as various co-occurring contextual factors, may affect the development of coping/ER in childhood and adolescence. For example, while promoting survival in dangerous environments, bodily systems that become vigilant to threat may impede functioning and learning in more typical social contexts (Frankenhuis & Del Giudice, 2012). Further, deficits in parents' socialization of emotions may be more common in maltreating families (e.g., less validation of emotions and emotion coaching; Shipman et al., 2007). Correspondingly, meta-analyses suggest that exposure to ACEs is associated with greater emotional dysregulation and impaired emotion regulation skills, with effects small to medium in magnitude (Gruhn & Compas, 2020; Lavi et al., 2019). However, the meaning of this finding is ambiguous and may have somewhat limited clinical utility, considering that few studies have identified specific strategies that youth with ACEs use to regulate their emotions in response to stress. Notable exceptions include Dvir et al. (2014) and Min et al. (2017), both finding exposure to adversity to be associated with less use of SCC. Further, in a neuroimaging study, maltreated adolescents showed greater recruitment of the prefrontal cortex during cognitive reappraisal than controls, suggesting that cognitive reappraisal may be more effortful for maltreated youth (McLaughlin et al., 2015).

In studies examining coping/ER and stress reactivity in typically developing youth, SCC and PCC have been associated with more adaptive reactivity to stress, and DC has been associated with less adaptive reactivity to stress (Compas et al., 2017). In youth with ACEs, however, few studies examine stress reactivity and coping/ER *together*. Of 47 studies included in a meta-analysis of stress reactivity and coping/ER in maltreated children, seven studies measured

both constructs and two studies reported their association (Gruhn, 2018). Both Cook et al. (2012) and Min et al. (2017) revealed heightened and blunted patterns of stress reactivity to be associated with less SCC and PCC and more DC in youth with ACEs.

**Methodological Challenges in Studying Stress Reactivity and Coping/ER in Adolescents with ACEs**

In the body of research examining stress reactivity and coping/ER in youth with ACEs, progress has been stymied by several methodological issues. First, the umbrella term "emotion dysregulation" is commonly used to describe coping/ER in youth with ACEs (Gruhn & Compas, 2020). Emotion dysregulation, however, encompasses a broad array of strategies and trait-like aspects of cognition and behavior that are often confounded with symptoms of psychopathology (Compas et al., 2017). For example, Kim and Cicchetti (2010) found poor emotion regulation to mediate the pathway between early maltreatment and internalizing and externalizing symptoms in childhood, with emotion regulation broadly defined as socially appropriate emotional displays, empathy, and emotional self-awareness. In a study by Heleniak et al. (2016), cognitive coping did not mediate the association between exposure to ACEs and internalizing and externalizing psychopathology across adolescence. Similar to Kim and Cicchetti (2010), Heleniak et al. (2016) used a coping measure that conflated automatic stress responses (e.g., rumination) and diverse coping strategies (e.g., distraction, problem solving). With the ultimate goal of identifying targets for preventing psychopathology in youth with ACEs, specificity in operationalizing key constructs is crucial. In the current study, I examine coping/ER at the factor level (i.e., PCC, SCC, and DC) rather than the domain level (i.e., regulation vs. dysregulation) to enhance understanding of the specificity of associations with ACEs, stress reactivity, and psychopathology.

Second, the most common strategy for measuring the effects of ACEs on the stress response system is through cortisol, the end-product of the hypothalamic-pituitary-adrenocortical (HPA) axis (Bernard et al., 2017; Koss & Gunnar, 2018). Consistent with the brief review of stress reactivity above, hypercortisolism and hypocortisolism have both been found to be characteristic of youth with ACEs. However, stress responses via the HPA axis take minutes to hours to unfold, and studies measuring cortisol are plagued by methodological inconsistencies (e.g., sampling lag time post stress induction, time of day of data collection), making comparisons of findings across studies very challenging (Dickerson & Kemeny, 2004). In the current study, changes in HRV as an index of ANS reactivity to stress are used, mitigating methodological complications and enhancing comparability with previous and future research.

Third, coping/ER and stress reactivity have been measured in ways that limit generalizability of findings. Coping/ER has generally been measured with questionnaires (e.g., Kim & Cicchetti, 2010; Messman-Moore et al., 2010). Although they have a number of strengths, questionnaires are limited by retrospective recall, and some questionnaires contain items that overlap considerably with psychological symptoms (e.g., DERS; Gratz & Roemer, 2004). An alternative to questionnaire methods is single-session laboratory experiments, in which participants are instructed to use specified coping/ER skills to regulate negative emotions in response to stimuli (e.g., faces; Bettis, et al., 2019). Although few laboratory paradigms have been used to measure coping/ER in children with ACEs, in one example, Maughan and Cicchetti (2002) found profiles of dysregulation to mediate the association between ACEs and psychological symptoms. Conversely, with regard to measuring stress reactivity, laboratory-based stress-induction paradigms are the most common approach (e.g., Trier Social Stress Test; McLaughlin et al., 2014). Although laboratory methods represent a critical departure from

questionnaire methods, their ecological validity is still limited (Kamarack & Lovallo, 2003; Turner et al., 1994). Data obtained from laboratory experiments reflect responses to carefully constructed (and often impersonal) stressors in a controlled environment. Taken together, questionnaire and laboratory methodologies provide an important but incomplete understanding of the stress response system as it functions in daily life (Wilhelm & Grossman, 2010). In the current study, I use ecological momentary assessment (EMA) as a method for measuring responses to stress with heightened ecological validity.

**Correspondence Among Laboratory Paradigms, Questionnaires, and Ecological Momentary Assessment (EMA) in Measuring Responses to Stress**

EMA encompasses a range of research methods that allow for the repeated, moment-to-moment collection of data on participants' experiences and behaviors in their natural environments (Stone & Shiffman, 1994). The body of research on the correspondence between questionnaires and EMA methods varies widely, with questionnaires providing over-estimates (e.g., Broderick et al. 2006), under-estimates (e.g., Litt et al., 2000), and close estimates (e.g., Shrier et al., 2005) of different aspects of behavior relative to EMA (see Van den Brink et al., 2001 for a review). Further, the degree of correspondence between questionnaires and EMA can be moderated by individual and contextual factors (Shiffman et al., 2008). The correspondence between laboratory paradigms and EMA has been examined in a smaller body of research (Wilhelm & Grossman, 2010). However, recent studies suggest that correspondence between EMA and laboratory methods is promising (Schlute et al., 2018), especially when adequate methodological and data analytic procedures are used (e.g., controlling for activity and posture when measuring ambulatory stress reactivity; using multilevel modeling to account for nested data; Kamarack & Lovallo, 2003). Nevertheless, additional research is necessary to understand

the correspondence among laboratory paradigms, questionnaires, and EMA in research measuring responses to stress, especially in populations where heterogeneous findings are common (i.e., adolescents with exposure to ACEs).

**Additional Considerations in ACEs Research**

Many mental disorders emerge during adolescence (Kessler et al., 2007; Merikangas et al., 2010), making this period key for studying the processes that mediate and moderate risk for psychopathology. Correspondingly, adolescence is developmentally dynamic, especially with regard to pubertal maturation. Youth display increased reactivity to stress during puberty (Spear, 2009), and this association may differ for boys and girls (Carlo et al., 2012). Exposure to ACEs has been associated with both advanced and delayed pubertal stage (Magnus et al., 2018; Sumner et al., 2019), and so pubertal timing is especially important to measure in research involving this subpopulation of adolescents. Sex differences also emerge in coping/ER (McRae et al., 2008), stress reactivity (Koskinen et al., 2009; Ordaz & Luna, 2012), and psychopathology (Zahn-Waxler et al., 2008), and race and ethnicity can influence stress responses (DeSantis et al., 2007; Wang et al., 2005) and the incidence of psychopathology (Nguyen et al., 2007). As such, age, pubertal timing, sex, and race/ethnicity should all be accounted for when studying responses to stress in youth with ACEs.

**The Current Study**

The current study provides a robust measurement of two identified mediators and moderators of the ACEs-psychopathology association (i.e., stress reactivity and coping/ER) using two established methods (i.e., a laboratory paradigm and questionnaires) and a novel method (i.e., EMA). Further, the study pursues secondary and tertiary aims of identifying associations among ACEs, stress reactivity and coping/ER, and symptoms of psychopathology.

*Aim 1.* Test a novel method (i.e., EMA) to measure stress reactivity and coping/ER in adolescents with a range of exposure to ACEs and examine its convergence with a laboratory paradigm and a well-validated questionnaire as indicators of stress reactivity and coping/ER. Hypothesis 1 (H1). Three measures of stress reactivity and three measures of coping/ER will converge in response to current stress in the adolescent's life.

*Aim 2.* Examine the associations among exposure to ACEs, stress reactivity, and coping/ER in response to stress using data collected from an EMA paradigm, a laboratory paradigm, and questionnaires. Hypothesis 2 (H2). Greater exposure to ACEs will be associated with heightened and blunted reactivity in response to stress. Hypothesis 3 (H3). Greater exposure to ACEs will be associated with greater use of DC and less use of PCC and SCC. Hypothesis 4 (H4). Heightened and blunted reactivity to stress will be associated with greater use of DC and less use of PCC and SCC.

*Aim 3*. Provide a preliminary test of the associations of exposure to ACEs, stress reactivity, and coping/ER with symptoms of internalizing and externalizing psychopathology. Hypothesis 5 (H5). Greater exposure to ACEs will be associated with more internalizing and externalizing psychopathology. Hypothesis 6 (H6). Greater use of DC will be associated with more internalizing and externalizing psychopathology. Greater use of PCC and SCC will be associated with less internalizing and externalizing psychopathology. Hypothesis 7 (H7). Heightened and blunted reactivity to stress will be associated with more internalizing and externalizing psychopathology.

# CHAPTER II

# METHODS

## Participants

Adolescents and their caregivers were initially recruited to participate in a study involving a laboratory-based task to understand stress and emotions in families (5R21HD098454; PI: Compas). Recruitment sites in Nashville, Tennessee were selected to allow for a sample of adolescents with histories of exposure to a wide range of ACEs and adolescents without histories of ACEs, including Vanderbilt Center of Excellence for Children in State Custody, Vanderbilt Child and Adolescent Psychiatry Outpatient Clinic, Mental Health Co-operative of Middle Tennessee, Adoption Support and Preservation Program, and Vanderbilt University listservs. Caregivers who expressed interest in the study through one of the recruitment methods completed a phone screen with study research assistants (RAs) prior to enrollment. Caregiver–adolescent dyads meeting any of the following criteria were excluded from participation: the adolescent was outside the study age range (10 to 15 years old), the caregiver reported a diagnosis of schizophrenia in themselves or a diagnosis of autism spectrum disorder in their adolescent, the caregiver was not the legal guardian of the adolescent, or the dyad did not live together at least 50% of the time for the past 6 months. In families with multiple eligible adolescents, the older adolescent was invited to participate in the study.

All adolescents-caregiver dyads who participated in the laboratory-based study were eligible to participate in a home-based study examining stress and emotions in families in daily life (5UL1TR002243-03; PI: Henry). Laboratory-based study participants were contacted by phone and/or email by RAs and provided with information about the home-based study, and

families who expressed interest were enrolled.[1] Taken together, 53 caregiver-adolescent dyads completed both the laboratory-based study and the home-based study examining stress and emotions in families in daily life.

On average, adolescents were 11.98 years old (*SD* = 1.74), and 54.7% identified as female. Sixty-eight percent of adolescents were White, 15.1% Black or African American, 9.4% Asian, and 7.6% identified as more than one race or another race not provided. Caregivers were approximately 42.21 years old (*SD* = 6.29), primarily identified as female (88.7%), were married or living with a partner (73.6%), and ranged in education (45.3% completed a graduate degree, 7.5% some graduate education, 28.3% college graduate, 13.2% some college, 3.8% high school graduate). See Table 1 for additional sociodemographic characteristics of the sample.

**Procedures**

See Table 2 for the complete schedule of events for both the laboratory-based study and the home-based study.

***Laboratory study***

The laboratory study was approved by the Vanderbilt Institutional Review Board (IRB #181531). At home prior to the laboratory study, adolescents and their caregivers each completed a series of questionnaires via a secure online portal (REDCap; Harris et al., 2009). Relevant to the current research, adolescents completed the Youth Self Report (YSR; Achenbach & Rescorla, 2001), caregivers completed the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), and both participants completed a demographic questionnaire.

[1] Of note, due COVID-19-related delays in completing the laboratory-based study, a subset of participants (*N* = 2) were initially enrolled in the home-based study and later completed the laboratory-based study. This subset of participants was recruited from Vanderbilt Kennedy Center Study Finder and ResearchMatch/Vanderbilt Listserv. A similar phone screen as the laboratory-based study was used, and identical exclusion criteria determined eligibility. After completing the home-based study, participants were contacted by phone and/or email by RAs and provided with information about the laboratory-based study. All families completed the laboratory-based study.

The adolescent-caregiver dyad subsequently completed 4- to 5-hr laboratory visit in a private laboratory space at Vanderbilt University. Relevant to the current research, adolescents and their caregivers each completed the Childhood Trauma Questionnaire – Short Form (CTQ-SF; Bernstein et al., 2003) and an adapted version of the Issues Checklist (Robin & Foster, 1989). The Issues Checklist lists 44 topics (e.g., coming home on time, helping out around the house) and asks respondents to indicate which topics they discussed with their partner in the last 4 weeks. Respondents rated how they felt during discussed topics on a Likert-type scale from 1 (*calm*) to 5 (*angry*).

Next, dyads were seated in chairs facing each other and separated by a divider. They completed a series of resting and speaking baseline and acclimation/recovery periods (3 min each). The divider was removed, and the adolescent-caregiver dyad engaged in a 10-min discussion on the topic from the Issues Checklist (Robin & Foster, 1989) that was rated highly by both the caregiver and adolescent. RAs selected the topic rated highest by the adolescent if caregiver and adolescent rankings did not align. The dyad was asked to (a) describe the issue, (b) explain how they feel about it, (c) discuss why it has become a source of conflict, and (d) attempt to resolve the issue. Prior to the start of the conflict-topic discussion, RAs assisted the caregiver and adolescent in placing seven electrodes on the body (torso, hand) for the collection of physiological data. Physiological data were collected from both the caregiver and the adolescent, but adolescent physiological data were exclusively used in the current research. Similar conflict-topic discussion tasks have consistently produced ANS activation (Beijersbergen et al., 2008).

Finally, caregivers and adolescents then completed a video-mediated recall (VMR) task independently in separate rooms. On desktop computers, participants watched (with audio) the middle 4 min of the 10-min videotape in 30-s clips of the conflict-topic discussion (eight clips in

total). After each 30 s clip, participants were prompted to describe aloud what they were thinking or doing during that part of the interaction, resulting in eight separate reports of coping/ER during the 10-min interaction. Again, VMR data were collected from both the caregiver and the adolescent, but adolescent VMR data were exclusively used in the current research. Similar VMR procedures have been used successfully in past research (e.g., Ohr et al., 2010).

Adolescents received $50 and caregivers received $100 for the entire laboratory visit.

***Home-based study***

The home-based study was approved by the Vanderbilt University Institutional Review Board (IRB #191930). The full home-based study protocol is described below and depicted in Figure 1. For the purposes of testing the H1-H7, relevant data include stress reactivity and coping/ER data collected during the conflict-topic discussion task and two questionnaires (Pubertal Development Scale [PDS; Petersen et al., 1988] and Responses to Stress Questionnaire – Family Discussion [RSQ - FD; Connor-Smith et al., 2000]).

**Initial Laboratory Visit.** Caregivers and adolescents were invited to a private laboratory space at Vanderbilt University for an initial one-hr visit, during which dyads were introduced to the four-day home-based study protocol. This introduction included demonstrations on how to apply the EcgMove4, use the smartphone, and answer the surveys. In addition, the adolescent completed the Pubertal Development Scale (PDS; Petersen et al., 1988), and the adolescent and caregiver completed the Child and Parent Versions of the Responses to Stress Questionnaire – Family Discussion (RSQ - FD; Connor-Smith et al., 2000), respectively.

Variance in stressor type (e.g., peer, school, family) may interfere with analyses exploring convergence in stress reactivity and coping/ER across methodologies (Aim 1 H1). As such, dyads were asked to schedule a time to complete a family discussion (the same conflict-

topic discussion task completed in the laboratory study) and a family activity during the EMA protocol. In this way, responses to stress (i.e., stress reactivity and coping/ER) could be measured relative to family stressors, in particular, across methods (laboratory, questionnaire, and EMA). Accordingly, during the initial laboratory visit, dyads scheduled a time to engage in a 10-min conflict-topic discussion task and family activity. If still relevant for the family, dyads were asked to discuss the same topic selected from the Issues Checklist (Robin & Foster, 1989) that was discussed during the laboratory study. If the conflict-topic was no longer relevant (e.g., resolved), then the family was asked to select a new topic that the family had discussed in the past four weeks and generated anger in both partners. Dyads were also asked to plan a family activity that solicited dyadic engagement (e.g., more engagement than watching television) while mitigating physical activity (i.e., no more active than a walk to reduce interference from physical activity for the EcgMove4). Examples of family activities included a picnic, window shopping, and a game night. Adolescents were asked to complete surveys on the smartphone provided following the family discussion and family activity. RAs provided dyads with handouts containing written instructions of study procedures and contact information for an RA available for phone consultation during the home protocol.

**Home Protocol.** The four-day home protocol was completed on either Thursday, Friday, Saturday, and Sunday; or Saturday, Sunday, Monday, and Tuesday. Assessing adolescents on weekdays and the weekend increased the likelihood of adolescents reporting a variety of stressor types. Adolescents were asked to wear the EcgMove4 around their chest (underneath the clothes) to measure physiological responses to stress. On weekdays, adolescents were asked to wear the EcgMove4 continuously from the time they arrived home from school (or during the afternoon

for participants who are not attending school in-person due to the COVID-19 pandemic[2]) through the time they went to bed, given anticipated barriers (e.g., academic interference, prohibition of cellphone use by school administration) to adolescents reporting stressors during the school day. On weekends, adolescents were asked to wear the EcgMove4 continuously from the time they woke up through the time they went to bed. Participants also completed surveys on the smartphones provided throughout the four-day home protocol to track stressors, coping/ER, and emotions. Participants received prompts to complete several different types of surveys throughout the four-day protocol. (1) EMA surveys. EMA surveys assessed exposure to stressors throughout the day, as well as coping/ER and negative and positive emotions in response to those stressors. On two weekdays, participants received two EMA surveys randomized between 4:00 pm and 7:30 pm. On Saturday and Sunday, participants received two EMA surveys randomized between 9:00 am and 12:30 pm, two EMA surveys randomized between 12:30 pm and 4:00 pm, and two EMA surveys randomized between 4:00 pm and 7:30 pm. (2) Follow-up surveys. Follow-up surveys assessed coping/ER and emotions in response to a specific stressful event. If participants endorsed having experienced a stressor in an EMA survey, they received follow-up surveys 15 min and 30 min after their initial EMA survey (i.e., these surveys were only distributed if participants endorsed a stressor on an EMA survey). (3) Daily surveys. Daily surveys asked participants to describe the worst stressor they experienced over the course of the day and their coping/ER and emotions in response to that stressor. On each of the four days, participants received the daily survey at 8:00 pm. Despite research suggesting EMA to be

[2] Due to COVID-19, participants were engaged in diverse school routines over the course of the study (e.g., in-school learning, synchronous at-home learning, asynchronous at-home learning). Students completing at-home learning sometimes had inconsistent or not clearly defined school vs. after-school hours. While returning home from school might trigger in-school learning students to apply the EcgMove4, at-home learning students might have difficulty remembering to apply the EcgMove4 on weekdays. As such, at-home learning participants were given the option to wear the EcgMove4 from their wake time until their bedtime on weekdays.

feasible with adolescent samples (76% survey completion rate across studies, similar to rates observed with adult samples; Heron et al., 2017), there is a risk of incomplete data. Daily surveys represent a daily diary approach to EMA (Larson & Csikszentmihalyi, 2014) to be used in analyses if in-vivo reporting was low (Shiffman et al., 2008). Further, daily surveys allowed for significant school-day stressors to be captured during the week. (4) Family Discussion and Family Activity surveys. The Family Discussion and Family Activity surveys measured stressors, coping/ER, and emotions experienced during the 10-min conflict-topic discussion task and the family activity. Participants completed these surveys after finishing their scheduled conflict-topic discussion and family activity.

**Final Laboratory Visit.** After the final day of data collection for the four-day home protocol, the dyad returned to the laboratory for a final one-hr visit. During the final laboratory visit, adolescents completed a semi-structured interview, and dyads were debriefed, returned the devices (i.e., EcgMove4 and smartphone), and received payment. Caregivers received $35 for completing the study. Adolescents received $50 for completing the study. Adolescents who missed three or fewer of the smartphone surveys over four days received a bonus payment of $15 (for a total of $65), and adolescents who missed one or fewer of the smartphone surveys over four days received a bonus payment of $25 (for a total of $75).[3]

[3] Risks to confidentiality were minimized in the current study through data management protocols. Smartphones and the EcgMove 4 were used repeatedly for data collection across participants. Data on the smartphone were encrypted (256 Bit), protecting participant privacy in the event that the device was lost or stolen. Of note, no device was lost or stolen during the study. Information transfer from MovisensXS to the Movisens web console was encrypted with SSl, and the data were decrypted when they reached the web console. Data stored on the Movisens secure web console were de-identified, and Movisens servers were hosted in a secure, ISO 27001 certified environment. After each adolescent completed the four-day EMA paradigm, data from their EcgMove 4 was downloaded onto the secure server. To extract physiological data, the EcgMove 4 was placed into a cradle connected via USB to a designated PC. Without the accompanying cradle, data cannot be extracted from the device. When not in use, all materials (i.e., smartphones, EcgMove 4 devices, EcgMove 4 cradle) were stored in a locked cabinet. Of note, the current study has undergone review from two separate review boards: the Vanderbilt IRB (#191930) and the Vanderbilt Institute for Clinical and Translational Research (VR53797).

**Three-Month Follow-up.** Three months after the final study visit, caregivers and adolescents were asked to complete the CBCL (Achenbach & Rescorla, 2001) and YSR (Achenbach & Rescorla, 2001), respectively, via a secure, web-based system (REDCap; Harris et al., 2009). Caregivers and adolescents each received a $10 for completing the surveys.

## Measures

### *Demographics*

Demographic information, including age, gender, and race/ethnicity, was collected from adolescents and caregivers in a brief questionnaire.

### *Pubertal Timing*

Pubertal timing was assessed using the Pubertal Development Scale (PDS; Petersen et al., 1988). The PDS is an adolescent self-report measure of five indices of pubertal growth. Questions surround physical growth, body hair, and skin changes. Some questions are gender-specific, such that males are asked about voice changes and growth of facial hair, and females are asked about breast development and onset of menstruation. Items are measured using a 4-point Likert scale (1 = *no changes yet*, 4 = *seems completed*). This scale has been used successfully in studies of adolescents with ACEs (Mendle et al., 2014). Overall scores on the PDS are averages of the five gender-specific items ranging from 1 to 4, with higher scores indicating more advanced pubertal development. In the current study, the PDS showed good internal consistency reliability for females ($\alpha = .88$) and excellent internal consistency reliability for males ($\alpha = .90$) (Nunnally & Bernstein, 1994).

### *Adverse Childhood Experiences (ACEs)*

Adolescent ACEs were assessed using the Childhood Trauma Questionnaire – Short Form (CTQ-SF; Bernstein et al., 2003) as both parent report on child and child self-report. The

CTQ-SF is a 28-item scale that assesses exposure to maltreatment. Each item is measured on a 5-point scale from *never true* (1) to *very often true* (5). The CTQ-SF produces scores on five clinical scales: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Individual clinical scale scores range from 5 to 25 and total maltreatment scores range from 25 to 125, with higher scores indicating greater severity of maltreatment. The CTQ is among the most commonly used measures of child maltreatment, and has excellent internal consistency, test-retest reliability, and convergent and discriminant validity with interview measures and clinician reports (Bernstein et al., 1994). All but one subscale in the current study (child report of physical abuse $\alpha = .69$) demonstrated acceptable to excellent internal consistency reliability (Nunnally & Bernstein, 1994), including emotional abuse (parent report on child $\alpha = .90$; child report $\alpha = .79$), physical abuse (parent report on child $\alpha = .90$; child report $\alpha = .69$), sexual abuse (parent report on child $\alpha = .98$; child report $\alpha = .96$), emotional neglect (parent report on child $\alpha = .79$; child report $\alpha = .84$), and physical neglect (parent report on child $\alpha = .87$; child report $\alpha = .87$).

***Stress Reactivity***

Adolescent stress reactivity was assessed using three methods: questionnaire, laboratory task, and EMA task.

**Questionnaire.** Adolescents and their caregivers reported on youth involuntary stress responses using the Responses to Stress Questionnaire – Family Discussion Version (RSQ-FD; Connor-Smith et al., 2000). The 57-item RSQ-FD assesses the ways adolescents cope with and react to stress specifically in the context of the selected conflict topic. Concurrent validity of the RSQ has been established through correlations with heart rate reactivity during a laboratory task (Connor-Smith et al., 2000). Involuntary engagement (e.g., emotional arousal, stress reactivity) is

an involuntary response to stress (which reflects stress reactivity) and has been confirmed in factor analytic studies (e.g., Connor-Smith et al., 2000). In the current study, missing items were prorated through substitution with the mean of their factor score. Of note, only .5% of item level data were missing. Adolescent report of involuntary engagement demonstrated good internal consistency reliability ($\alpha = .88$) and caregiver report demonstrated excellent internal consistency reliability ($\alpha = .93$) (Nunnally & Bernstein, 1994).

**Laboratory Task.** Electrodes were placed on the adolescent (central clavicle, right clavicle, xiphoid process, and the right and left sides of the torso below the rib cage) to collect physiological data during the 10-min conflict-topic discussion task, as described in the laboratory study procedures above. Equipment to facilitate physiological data collection included the MW1000A acquisition system, BioNex 8-slot chassis (MW50371108), BioNex Impedance Cardiograph and GSC (M371100-00), and disposable pediatric snap electrocardiogram (ECG) electrodes from MindWare, Technologies Ltd (Gahanna, OH).

**Ecological Momentary Assessment task.** Adolescents wore an ambulatory ECG (EcgMove4; Movisens GmbH, Karlsruhe, Germany) to collect continuous physiological data over the course of four days, as described in the home-based study procedures above. Physiological data collected during the 10-min conflict-topic discussion task were used for the current research. The EcgMove4 has been used previously in psychophysiological research (e.g., Humbel et al., 2018). The EcgMove4 (62.3 mm x 38.6 mm x 11.5 mm; 26 g) is equipped with a 3.7 V Lithium-Polymer-Battery that can withstand 300 charging cycles and has a maximum recording capacity of two consecutive weeks. The EcgMove4 is fixed to the body using a chest belt.

***Coping/Emotion Regulation (ER)***

Adolescent coping/ER was assessed using three methods: questionnaire, laboratory task, and EMA task.

**Questionnaire.** Adolescents reported on their own coping behaviors using Responses to Stress Questionnaire – Family Discussion Version (RSQ - FD; Connor-Smith et al., 2000). As described above, the RSQ-FD is a 57-item measure that assesses the ways in which adolescents cope with and react to stress specifically in the context of the selected conflict topic. Three factors of coping have been confirmed in factor analytic studies (e.g., Compas et al., 2006a): primary control coping (PCC), secondary control coping (SCC), and disengagement coping (DC). The PCC scale includes items assessing problem solving, emotional modulation, and emotional expression; the SCC scale includes items assessing acceptance, cognitive reappraisal, positive thinking, and distraction; and the DC scale includes items assessing avoidance, denial, and wishful thinking. In the current study, all scales demonstrated acceptable or good internal consistency reliability, including PCC (parent report on child $\alpha = .80$; child report $\alpha = .85$), SCC (parent report on child $\alpha = .78$; child report $\alpha = .75$), and DC (parent report on child $\alpha = .76$; child report $\alpha = .84$) (Nunnally & Bernstein, 1994).

**Laboratory task.** As described in the laboratory study procedures above, video-mediated recall (VMR; Welsh & Dickson, 2005) was used to elicit adolescent coping/ER responses during the 10-min conflict-topic discussion task. RAs subsequently transcribed the audio recording from each participant's VMR task, and the transcripts of each of the eight separate reports of coping/ER were consensus coded using a novel coding scheme (Watson et al., 2020). Each of the eight reports were coded as 0 (coping/ER response not observed) or 1 (coping/ER response observed) for PCC, SCC, and DC, yielding total scores ranging from 0 (no sections coded) to 8 (all sections coded) for each of the three coping/ER response across the VMR task. Reliability,

as measured by percent agreement, was calculated for each coping/ER response: PCC (*M* = 87.99, *SD* = 14.57), SCC (*M* = 90.69, *SD* = 12.46), and DC (*M* = 95.59, *SD* = 7.84).

**Ecological Momentary Assessment task.** The EMA coping/ER data were collected using MovisensXS software (Movisens GmbH, Karlsruhe, Germany). MovisensXS is a smartphone application-based experience sampling software with a web platform for survey design and data processing. The MovisensXS smartphone application is functional for research participants with or without an internet connection. Android smartphone devices (Motorola Moto G, Motorola Mobility) that met specifications recommended by Movisens GmbH to maximize the functionality of MovisensXS were provided to adolescents. MovisensXS has been used previously in psychological research with high-risk participants (e.g., suicide risk; Kleiman et al., 2017).

As described in the home-based study procedures above, surveys on the smartphones throughout the four-day home protocol were used to elicit adolescent in-vivo coping/ER. After completing the conflict-topic discussion task, participants completed a survey on their smartphone which included the prompt, "Please rate how much you tried to do each of the following while you were trying to deal with your stressor." Participants were then presented with 11 items that have been previously identified (see Connor-Smith et al., 2000) as having significant factor loadings onto PCC ("I took steps to try to solve the problem," "I tried to let my feelings out," "I tried not to show my emotions," and "I tried to keep my feelings under control"), SCC ("I tried to think of a way to make it less stressful," "I told myself that I just need to accept things as they are," "I tried to think about things in a more positive way," and "I tried to think about something else that made me feel good or less stressed"), and DC ("I wished the problem would go away and that everything would work itself out," "I tried to not talk about it,"

and “I told myself that it wasn’t really a problem or that it wasn’t real”). Adolescents rated each item on a 4-point Likert-type scale from 1 (*not at all*) to 4 (*a lot*). Total scores for PCC and SCC had a possible range of 4 to 16, and the total score for DC had a possible range of 3 to 12. Internal consistency reliability ranged from very low to good: PCC (four items; $\alpha = .43$), SCC (four items; $\alpha = .80$), and DC (three items; $\alpha = .49$).

### *Internalizing and Externalizing Symptoms of Psychopathology*

The Youth Self Report and Child Behavior Checklist (YSR, CBCL; Achenbach & Rescorla, 2001) were completed by the adolescent and their caregiver, respectively, to assess adolescent internalizing and externalizing problems over the past six months. Behaviors are rated on a 3-point scale (0 = *not true*, 2 = *very true or often true*). The reliability and validity of the CBCL and YSR are well established (Achenbach & Rescorla, 2001).

## Data Analytic Approach

### Preliminary Analyses

### *Preprocessing Physiological Data*

Preprocessing of the physiological data was completed for the laboratory study (MindWare HRV Analysis Software 3.0.17; MW60-1100, MindWare Technologies Ltd, Gahanna, OH) and the EMA study (DataAnalyzer software; movisens GmbH, Karlsruhe, Germany). Laboratory and EMA data were corrected for artifacts using MindWare HRV Analysis Software 3.0.17 and DataAnalyzer software, respectively. Due to technical malfunction with the ambulatory ECG during the EMA study, full or partial HRV data could only be calculated for 15 participants (28% of the sample). Both HRV and heart rate (HR) are regulated by the ANS (Wehrwein et al., 2016), HR is significantly associated with HRV in ambulatory research (Tsuji et al., 1996), and previous work has demonstrated associations between exposure

to ACEs and blunted HR-R (HR reactivity) (Bourassa et al., 2021; Lovallo et al., 2012; Voellmin et al., 2015). As such, HR (beats per minute [bpm]) was calculated for both the EMA and laboratory data. For both the laboratory and EMA tasks, HR-R was computed by subtracting average HR collected during the speaking baseline laboratory task from HR collected during the conflict-topic discussion task (laboratory or EMA, as appropriate), such that higher (i.e., more positive) HR-R scores indicate greater reactivity. HR-R was averaged across the 10-min EMA and laboratory conflict-topic discussion tasks for correlational analyses and multiple linear regression analyses. In multilevel model analyses, mean HR-R was calculated for ten 60-s intervals, resulting in 530 observations (epochs) for analysis.

***Data Reduction***

To address single-informant bias in measuring child and adolescent emotional and behavioral problems (Achenbach et al., 1987), both parent reports and adolescent reports were collected for questionnaire measures of ACES (i.e., CTQ-SF, parent report on child version and CTQ-SF, child self-report version), stress reactivity (i.e., RSQ-FD, parent report on child version and RSQ-FD, child self-report version), coping/ER (i.e., RSQ-FD, parent report on child version and RSQ-FD, child self-report version), and psychological symptoms (i.e., CBCL and YSR). To reduce the number of analyses for hypothesis testing, composite scores were created for each construct of interest pending sufficiently high cross-informant correlations (Piacentini et al., 1992). This method has been successfully implemented in previous work (e.g., Reising et al., 2018). Given a high cross-informant correlation for the overall maltreatment scale of the CTQ-SF ($r = .83$, $p < .001$), a composite score was created by calculating $z$ scores and averaging across parent and child reports. The CTQ-SF composite score was used when testing all relevant hypotheses. For stress reactivity and coping/ER on the RSQ-FD, cross-informant correlations

were all significant but were not considered sufficiently high for involuntary engagement ($r$ = .37, $p$ = .007), PCC ($r$ = .39, $p$ = .005), SCC ($r$ = .51, $p$ <.001), or DC ($r$ = .44, $p$ <.001). Adolescent reports of their own involuntary engagement and coping/ER on the RSQ-FD were used for all analyses to promote consistency with coping/ER as measured in the laboratory (child self-report via video-mediated recall) and in daily life (child self-report via EMA). For psychological symptoms, cross-informant correlations were not considered sufficiently high for internalizing symptoms ($r$ = .64, $p$ <.001) or externalizing symptoms ($r$ = .66, $p$ <.001). Accordingly, parent reports of child symptoms on the CBCL were used for all analyses.

**Hypothesis Testing**

SPSS Version 26 was used for all analyses. Descriptive statistics were generated to summarize sociodemographic information (e.g., adolescent age, gender, race/ethnicity) and study variables (i.e., stress reactivity, coping/ER, ACEs, pubertal development, internalizing and externalizing symptoms) across methods (i.e., EMA, laboratory, questionnaires) for the full sample.

***Aim 1***

H1 was tested using a multitrait-multimethod correlation matrix (Campbell & Fisk, 1959). Results were evaluated based on the guidelines described by Campbell & Fisk (1959), in that convergent validity was supported by (1) validity values significantly different from zero and sufficiently large, (2) validity values higher than corresponding heterotrait-heteromethod values, (3) validity values higher than corresponding heterotrait-monomethod values, and (4) the same pattern of trait interrelationship is evident for the monomethod and heteromethod blocks. Cohen's (1988) guidelines were used to interpret the magnitude of the effect size for correlations, with $r$ = 0.10 representing a small effect, $r$ = 0.30 representing a medium effect, and

$r$ = 0.50 representing a large effect. Fisher's $z$ transformation was used for comparing correlations (Howell, 1997).

***Aims 2 and 3***

Zero-order correlations were computed to understand simple associations among the study variables, inform the selection of covariate variables to retain for H2-H7, and provide preliminary tests of H2-H7. Pearson correlations were conducted with pairs of continuous variables and point-biserial correlations were conducted with continuous-dichotomous pairs. Cohen's (1988) guidelines were used to interpret the magnitude of effect size.

H2 – H7 were tested separately in each of the three methods (i.e., EMA, laboratory, and questionnaire). A two-level structure was expected for models predicting HR (H2 and H4) in both the EMA and laboratory data, such that level 1 = epoch (60-s interval) and level 2 = person; intraclass correlation coefficients for the null models were estimated to establish the need for multilevel modeling (Pornprasertmanit et al., 2014). Given that the dependent variables of interest in the remaining equations (i.e., physiology [involuntary engagement] in the questionnaire data for H2 and H4; coping/ER data for EMA, laboratory, and questionnaire data in H3; internalizing and externalizing symptoms in H5, H6, and H7) are person-level variables, nesting was irrelevant and multiple linear regressions were used for hypothesis testing. All final equations are provided in the Appendix.

# CHAPTER III

# RESULTS

## Descriptive Statistics

See Table 3 for descriptive statistics for study variables. Taken together, adolescents reported their overall pubertal development as being "definitely underway" ($M$ = 2.80, $SD$ = .81). On average, parents and children reported exposure to ACEs in the "none or low" category across all subscales (Bernstein et al., 2003). Still, there was variation in the sample: 32% of caregivers and 30% of adolescents endorsed some emotional abuse (i.e., greater than *never true* (1)); 6% of caregivers and 17% of adolescents endorsed some physical abuse; 11% of caregivers and 6% of adolescents endorsed some sexual abuse; 11% of caregivers and 21% of adolescents endorsed some emotional neglect; and 11% of caregivers and 9% of adolescents endorsed some physical neglect. On the RSQ-FD, adolescents' reports of their relative (proportional) use of PCC ($M$ = .19, $SD$ = .04), SCC ($M$ = .25, $SD$ = .05), DC ($M$ = .15, $SD$ = .03), and involuntary engagement ($M$ = .24, $SD$ = .04) were similar to levels from other samples of children with uncontrollable stressors (e.g., parental depression, Henry et al., 2018; cancer, Murphy et al., 2017; sickle cell disease, Prussien et al., 2017). During the 10-min laboratory-based conflict-topic discussion task, adolescents were coded as having used PCC in approximately 3 of 8 VMR segments ($SD$ = 2.38), SCC in 2 of 8 VMR segments ($SD$ = 1.56), and DC in 1 of 8 VMR segments ($SD$ = 1.31). During the 10-min EMA-based conflict-topic discussion task, adolescents reported using PCC ($M$ = 2.36, $SD$ = .60) and SCC ($M$ = 2.43, $SD$ = .87) between "a little" (2) and "some" (3), and adolescents reported using DC ($M$ = 2.03, $SD$ = .75) "a little" (2). On average, adolescent heart rate (bpm) was 83.78 ($SD$ = 11.67) during the laboratory task talking baseline task, 86.03 ($SD$ = 10.82) during the 10-min laboratory conflict-topic discussion task,

and 114.79 (*SD* = 27.94) during the 10-min EMA conflict-topic discussion task. Average change in heart rate from the laboratory talking baseline to the laboratory conflict-topic discussion task (i.e., laboratory HR-R) was 3.98 (*SD* = 12.45). Average change in heart rate from the laboratory talking baseline to the EMA conflict-topic discussion task (i.e., EMA HR-R) was 35.51 (*SD* = 33.77). Parent report on child internalizing symptoms (*M* = 56.96, *SD* = 12.14) produced *T*-scores about one half *SD* above the normative mean. Parent report on child externalizing symptoms (*M* = 50.98, *SD* = 10.31) produced *T*-scores at the normative mean.

**Aim 1: Multitrait-Multimethod Correlation Matrix**

In H1, three measures of stress reactivity and three measures of coping/ER were expected to converge in response to current stress in the adolescent's life. The multitrait-multimethod correlation matrix is presented in Table 4.

***Stress Reactivity***

***Reliability.*** The conflict-topic discussion task was completed one time in each of the EMA and laboratory methods, precluding our temporal assessment of reliability (test-retest reliability) for stress reactivity (via HR-R). The questionnaire measure of stress reactivity (RSQ-FD adolescent report of involuntary engagement) demonstrated good internal consistency reliability (α = .88).

***Validity.*** With regard to the trait of stress reactivity, the validity value for EMA and laboratory methods (*r* = .15, *p* = .35) was nonsignificant. The validity values for EMA and questionnaire methods (*r* = -.05, *p* = .77) and laboratory and questionnaire methods (*r* = -.12, *p* = .41) were nonsignificant. These data do not support H1.

***Coping/ER***

***Reliability.*** The EMA method for coping/ER demonstrated internal consistency reliability ranging from good to very low: PCC (four items; α = .43), SCC (four items; α = .80), and DC (three items; α = .49). The questionnaire method for coping/ER (RSQ-FD, child self-report) demonstrated acceptable or good internal consistency reliability for all scales, including PCC (α = .85), SCC (α = .75), and DC (α = .84). The laboratory method for coping/ER demonstrated acceptable percent agreement: PCC (*M* = 87.99, *SD* = 14.57), SCC (*M* = 90.69, *SD* = 12.46), and DC (*M* = 95.59, *SD* = 7.84). Kappa statistics for determining inter-rater reliability for the laboratory method were not available.

***Validity.*** With regard to the trait of coping/ER, the PCC validity value for EMA and laboratory methods ($r = .29$, $p = .05$) was significant with medium effect size. The PCC validity value was larger (significant and approaching significance, respectively) than corresponding heterotrait-heteromethod values for EMA PCC and laboratory stress reactivity ($r = -.12$, $p = .50$; $z = 1.82$, $p = .03$) and EMA stress reactivity and laboratory PCC ($r = -.01$, $p = .98$; $z = 1.29$, $p = .09$). The PCC validity value was not significantly larger than corresponding heterotrait-monomethod values for EMA PCC and EMA stress reactivity ($r = .19$, $p = .30$; $z = .48$, $p = .32$) or laboratory PCC and laboratory stress reactivity ($r = .36$, $p = .03$; $z = -.37$, $p = .36$).

The SCC ($r = .49$, $p < .001$) and DC ($r = .43$, $p = .002$) validity values for EMA and questionnaire methods were also significant with medium-to-large effect sizes. For SCC, the validity value was larger than corresponding heterotrait-heteromethod values for EMA SCC and questionnaire stress reactivity ($r = .01$, $p = .93$; $z = 2.50$, $p = .006$) and EMA stress reactivity and questionnaire SCC ($r = .01$, $p = .97$; $z = 2.33$, $p = .01$). For DC, the validity value was larger than (approached significance) the corresponding heterotrait-heteromethod value for EMA stress

reactivity and questionnaire DC ($r$ = .11, $p$ = .56, $z$ = 1.48, $p$ = .07) but not EMA DC and questionnaire stress reactivity ($r$ = .22, $p$ = .13; $z$ = 1.12, $p$ = .13).

For SCC, the validity value was larger (approached significance and significant, respectively) than corresponding heterotrait-monomethod values for EMA SCC and EMA stress reactivity ($r$ = .17, $p$ = .34; $z$ = 1.48, $p$ = .07) and questionnaire SCC and questionnaire stress reactivity ($r$ = .15, $p$ = .29; $z$ = 1.87, $p$ = .03). The DC validity value was larger than the corresponding heterotrait-monomethod value for EMA DC and EMA stress reactivity ($r$ = .02, $p$ = .93; $z$ = 1.91, $p$ = .03) but not questionnaire DC and questionnaire stress reactivity ($r$ = .74, $p$ < .001; $z$ = -2.39, $p$ = .01). Accordingly, in partial support for H1, there was some preliminary evidence for the convergence between EMA and laboratory methods for PCC, and there was some preliminary evidence for the convergence between EMA and questionnaire methods for SCC and DC.

**Aims 2 and 3: Multiple Linear Regression and Multilevel Modeling Analyses**

***Bivariate Correlations between Demographics and Key Variables***

Bivariate correlations between demographics and key variables are presented in Table 5. Older age was associated with more advanced pubertal development ($r$ = .67, $p$ < .001), increased use of PCC measured in the laboratory ($r$ = .26, $p$ = .06), lower average stress reactivity measured in daily life ($r$ = -.30, $p$ = .07), and higher stress reactivity measured by questionnaire ($r$ = .27, $p$ = .05). Female gender was associated with more advanced pubertal development ($r$ = .40, $p$ = .004), greater use of DC measured by questionnaire ($r$ = .42, $p$ = .002), and increased involuntary engagement ($r$ = .34, $p$ = .01). White race was associated with less use of DC measured by EMA ($r$ = -.31, $p$ = .03). More advanced pubertal development was associated with less use of SCC measured in daily life ($r$ = -.27, $p$ = .06), greater use of DC as

measured by questionnaire ($r$ = .44, $p$ = .001), and greater involuntary engagement ($r$ = .45, $p$ < .001).

Given the large effect size of the correlation between older age and more advanced pubertal development, we conducted exploratory partial correlations replicating the above associations while controlling for age. The association between pubertal development and SCC measured in daily life was nonsignificant ($r$ = -.12, $p$ = .46). Associations between pubertal development and greater use of DC as measured by questionnaire ($r$ = .45, $p$ = .003) and greater involuntary engagement ($r$ = .47, $p$ = .002) remained significant. There were no significant associations among demographics and parent-reported internalizing or externalizing symptoms.

***Associations Among Exposure to ACEs, Stress Reactivity, and Coping/ER***

***Hypothesis 2.*** As a preliminary test of H2, bivariate correlations (Table 5) did not support an association between exposure to ACEs and stress reactivity measured in daily life ($r$ = -.18, $p$ = .29), in the laboratory ($r$ = -.04, $p$ = .78), or by questionnaire ($r$ = -.01, $p$ = .95).

Given significant associations with stress reactivity in correlational analyses, age, gender, and pubertal development were included as covariates in all equations. Multilevel models were used to test the hypothesized associations using the EMA and laboratory methods. The ICCs derived from the null univariate models predicting EMA and laboratory stress reactivity (HR-R) indicated that 75% of the observed variation in EMA heart rate and 81% of the observed variation in laboratory heart rate was due to differences between individuals (Table 6). These values suggest that multilevel modeling is an appropriate analytic method for these data. In the final models (Table 7), ACEs did not predict stress reactivity in daily life ($\gamma_{04}$= -3.04, $p$ = .69) or in the laboratory model ($\gamma_{04}$= .72, $p$ = .44).

Multiple linear regression analyses (Table 8) were used to test the hypothesized associations using the questionnaire method. Consistent with the results from zero-order correlation analyses, ACEs (β = -.05, $p$=.74) were not significantly associated with involuntary engagement as measured by questionnaire (RSQ-FD), $F(4, 46)=3.73$, $p$=.01.

***Hypothesis 3.*** As a preliminary test of H3, bivariate correlations (Table 5) did not support an association between exposure to ACEs and coping/ER measured in daily life (PCC: $r$ = -.08, $p$ = .89; SCC: $r$ = -.16, $p$ = .33; DC: $r$ = .10, $p$ = .25), in the laboratory (PCC: $r$ = .04, $p$ = .88; SCC: $r$ = -.03, $p$ = .42; DC: $r$ = .22, $p$ = .22), or by questionnaire (PCC: $r$ = .19, $p$ = .13; SCC: $r$ = .004, $p$ = .76; DC: $r$ = .02, $p$ = .67).

Multiple linear regression analyses were used to examine associations using all three (EMA, laboratory, questionnaire) methods (Table 9). Given significant associations with coping/ER in bivariate correlation analyses, gender, race, and pubertal development were included as covariates in all equations. Analyses were consistent with the results from correlation analyses. Equations predicting PCC, SCC, and DC as measured by EMA and laboratory task were nonsignificant. Equations predicting PCC and SCC as measured by questionnaire were nonsignificant. The equation for DC as measured by questionnaire was significant, $F(4,46)=4.86$, $p$=.002, but exposure to ACEs (β = .01, $p$=.91) was a nonsignificant predictor.

***Hypothesis 4.*** As a preliminary test of H4, bivariate correlations (Table 5) did not support an association between stress reactivity and coping/ER measured in daily life (PCC: $r$ = .08, $p$ = .63; SCC: $r$ = .23, $p$ = .16; DC: $r$ = -.02, $p$ = .89) or in the laboratory (PCC: $r$ = .06, $p$ = .68; SCC: $r$ = .19, $p$ = .22; DC: $r$ = -.15, $p$ = .32), The questionnaire measure demonstrated that greater child self-reported involuntary engagement was associated with greater use of PCC ($r$

= .28, $p$ = .006) and DC ($r$ = .74, $p$ < .001). There was no association between involuntary engagement and SCC ($r$ = .15, $p$ = .75).

Age, gender, and pubertal development were included as covariates in all equations considering significant associations with stress reactivity. Multilevel models were used to examine associations using the EMA and laboratory methods. As described above, the ICCs derived from the null univariate models predicting EMA stress reactivity (ICC: 75%) and laboratory stress reactivity (ICC: 81%) indicated that multilevel modeling is an appropriate analytic method for these data. In the final EMA model (Table 10), H4 was not supported, with nonsignificant effects for PCC ($\gamma_{04}$= 1.17, $p$ = .61), SCC ($\gamma_{05}$= .67, $p$ = .67), and DC ($\gamma_{06}$= -.51, $p$ = .81). Further, H4 was also unsupported in the final laboratory method model (Table 10), with nonsignificant effects for PCC ($\gamma_{04}$= .59, $p$ = .17), SCC ($\gamma_{05}$= -.25, $p$ = .69), and DC ($\gamma_{06}$= -.62, $p$ = .40).

Multiple linear regression analyses were used to examine associations using the questionnaire method (Table 11). Providing partial support for H4, increased use of DC (β = .67, $p$<.001) was associated with increased reported involuntary engagement. PCC (β = .23, $p$=.04) also emerged as a predictor of involuntary engagement, albeit in the opposite direction hypothesized.

***Associations of Exposure to ACEs, Stress Reactivity, and Coping/ER with Symptoms of Internalizing and Externalizing Psychopathology***

Bivariate correlations and multiple linear regression analyses (Table 12) were used to examine the associations of exposure to ACEs, stress reactivity, and coping/ER with internalizing and externalizing psychopathology. Six regression models were calculated, with two models (internalizing and externalizing) including stress reactivity and coping/ER as

measured by EMA, two as measured by the laboratory task, and two as measured by the questionnaire. Given nonsignificant associations with internalizing and externalizing symptoms in bivariate correlation analyses, we excluded covariates (age, gender, race, pubertal development) to simplify the model and thus maximize power to observe true effects. Four models were significant (EMA externalizing: $F(5, 31)=3.78$, $p=.01$; laboratory internalizing: $F(5,38)=2.66$, $p=.04$; laboratory externalizing: $F(5,38)=3.91$, $p=.006$; questionnaire internalizing $F(5,46)=3.84$, $p=.01$), one model approached significance (EMA internalizing: $F(5,31)=2.22$, $p=.08$), and one model was nonsignificant (questionnaire externalizing: $F(5,46)=1.52$, $p=.20$).

***Hypothesis 5.*** Bivariate correlations provided preliminary partial support for H5. Exposure to ACEs was associated with more externalizing symptoms ($r = .27$, $p = .055$). No significant associations emerged between exposure to ACEs and internalizing symptoms ($r = .20$, $p = .15$).

Regression analyses also provided partial support of H5. Exposure to ACEs was associated with more psychological symptoms in three of the five significant models (EMA internalizing: $\beta = .43$; EMA externalizing: $\beta = .52$; questionnaire internalizing approached significance: $\beta = .23$).

***Hypothesis 6.*** Bivariate correlations provided preliminary partial support for H6. No associations emerged between coping/ER (PCC, SCC, DC) in daily life and internalizing or externalizing symptoms. In the laboratory, greater use of DC was associated with more internalizing symptoms ($r = .39$, $p < .01$) and externalizing symptoms ($r = .54$, $p < .001$). The associations between greater use of SCC and fewer internalizing symptoms ($r = -.26$, $p = .07$) and externalizing symptoms ($r = -.25$, $p = .08$) approached significance. On the questionnaire, greater use of DC was associated with more internalizing symptoms ($r = .40$, $p < .01$).

Regression analyses also provided partial support of H6. Greater use of DC was associated with more psychological symptoms in three of the five significant models (laboratory internalizing: β = .43; laboratory externalizing: β = .51; questionnaire internalizing: β = .49). Of note, greater use of PCC was associated with more internalizing symptoms (β = .32) in the laboratory model, which is the opposite direction of the hypothesized effect.

***Hypothesis 7.*** Neither bivariate correlations nor regression analyses provided support for H7. In bivariate correlations, stress reactivity measured in daily life (internalizing: $r$ = -.11, $p$ = .53; externalizing: $r$ = .02, $p$ = .90), in the laboratory (internalizing: $r$ = -.01, $p$ = .97; externalizing: $r$ = -.05, $p$ = .73), and on the questionnaire (externalizing: $r$ = .22, $p$ = .12) was not significantly associated with internalizing or externalizing symptoms. The association between greater questionnaire stress reactivity (involuntary engagement) and more internalizing symptoms approached significance ($r$ = .24, $p$ = .08). There were no significant effects of stress reactivity on psychological symptoms in any of the five significant regression models.

# CHAPTER IV

# DISCUSSION

Exposure to ACEs is a prevalent and powerful predictor of psychopathology in children and adolescents (Baldwin et al., 2021). Researchers are tasked with identifying modifiable pathways from ACEs to psychopathology to ultimately prevent or mitigate the impact of ACEs on youth. Stress reactivity and coping/ER are two promising targets for intervention (Miu et al., 2022; Compas et al., 2011). However, these constructs have infrequently been examined together in a single study, and methodological limitations in existing studies have obscured our understanding of stress reactivity and coping/ER in the daily lives of youth exposed to ACEs. The current research (1) provides a robust measurement of stress reactivity and coping/ER using a novel method of youth lived experience (i.e., EMA), along with two established methods (i.e., a laboratory task and questionnaire), and identifies associations among (2) ACEs, stress reactivity and coping/ER, and (3) symptoms of psychopathology, across methods.

## *Aim 1*

***Feasibility.*** A primary goal of the current research was to test EMA as a novel method for measuring stress reactivity and coping/ER in adolescents with a range of exposure to ACEs. Ninety-five adolescents with a range of exposure to ACEs were initially recruited to participate in the 4-to 5-hr laboratory study. Fifty-three percent of the laboratory study sample ($n$ = 51) subsequently agreed to participate in the four-day home-based EMA study. (Two additional participants were initially recruited into the home-based study and later completed the laboratory study.) There were no differences in total maltreatment scores (CTQ-SF) between adolescents who completed the home-based study and adolescents who did not consent to participate (parent report: $t(94) = 1.12$, $p = .27$; child report: $t(93) = .26$, $p = .80$). Of note, data collection for the

home-based study occurred between November 2020 and April 2022, and the COVID-19 pandemic impacted participation in terms of adolescent and caregiver capability, comfort, and interest. Of the full sample of adolescents who participated in the home-based study, both EMA stress reactivity and coping/ER data were available for 81% of the sample ($n$ = 43), with missingness due to unusable physiological data ($n$ = 7), caregiver-adolescent dyads completing their discussion on a topic that differed from the specified topic ($n$ = 2), and lack of completion of the conflict-topic discussion task ($n$ = 1). Taken together, recruitment and compliance data highlight that an intensive four-day home-based study (including measures of *both* physiology using an ambulatory ECG and coping/ER using surveys on a smartphone) with adolescents exposed to a range of ACEs is feasible (yet challenging).

***Hypothesis 1.*** Data provide partial support for the convergence of an EMA method, a laboratory method, and a questionnaire method in measuring coping/ER. In particular, there was evidence for convergence of EMA and questionnaire methods in measuring SCC and DC during a family conflict-topic stressor. Given that EMA items representing PCC, SCC, and DC were selected directly from corresponding RSQ scales, it is surprising that evidence of convergent validity emerged for SCC and DC but not PCC. Of note, internal consistency reliability was low for both PCC (four items; $\alpha$ = .43) and DC (three items; $\alpha$ = .49) in the EMA method. It could be that the four items selected to represent EMA PCC ("I took steps to try to solve the problem," "I tried to let my feelings out," "I tried not to show my emotions," and "I tried to keep my feelings under control") were not the best representations of the RSQ PCC scale. Researchers might consider testing other items from the RSQ to represent EMA PCC in future research. Still, convergence of EMA and laboratory methods for PCC was supported in the current data. Accordingly, the selected EMA PCC items are capturing, in part, content similar to what was

captured by the novel VMR coding scheme (Watson et al., 2020). Participant influence from task instructions is another potential factor in the lack of convergence of PCC across all methods. The laboratory and EMA conflict-topic tasks instruct dyads to (a) describe the issue, (b) explain how they feel about it, (c) discuss why it has become a source of conflict, and (d) attempt to resolve the issue, which may solicit greater use of PCC (e.g., problem solving) from dyads. In the laboratory task, adolescents' use of PCC ($M$ = 2.85, $SD$ = 2.38) was almost double their use of SCC ($M$ = 1.62, $SD$ = 1.56). Results represent preliminary success in the selection and implementation of SCC and DCC EMA items that correspond to an established measure of coping/ER (RSQ-FD), while highlighting the obstacles that EMA researchers face in balancing the creation of psychometrically sound measures with consideration for participant burden.

An EMA method, a laboratory method, and a questionnaire method of a family conflict-topic stressor did not converge as indicators of stress reactivity. At least a couple of issues may have interfered with the ability to detect a true effect of convergence in stress reactivity across methods. Even with the substitution of HR for HRV, the sample size was small for all stress reactivity pairs in correlation analyses ($N$ = 27 for EMA and laboratory methods; $N$ = 33 for EMA and questionnaire methods; $N$ = 37 for EMA and questionnaire methods). In addition, activity and posture were controlled in the algorithm calculating HRV but not HR. While adolescents were seated for the entire 10-min conflict-topic discussion task in the laboratory, activity class ranged in valid measurements for the home-based study, including seated or standing (76%), lying down (15%), walking (5%), cycling (2%), and engaging in other activity classes (2%). On average, HR collected during the laboratory conflict-topic discussion task ($M$ = 86.03, $SD$ = 10.82) was significantly lower than HR collected during the EMA conflict-topic discussion task ($M$ = 114.79, $SD$ = 27.94), $t(37) = -6.68$, $p<.001$. It is also possible that a true

effect of nonconvergence across EMA and laboratory methods emerged, reflecting greater reactivity in daily life than in the controlled conditions of the laboratory. In a group of young adult men, Pollak (1991) found average HR during daily ambulatory wake-time (controlling for activity level) to be comparable to average HR during a lab-based stressor task. In addition to lack of convergence between EMA and laboratory methods, lack of convergence was also observed between the questionnaire method and EMA and laboratory methods. While EMA and laboratory methods both used HR to measure physiological reactivity, the child self-report questionnaire of involuntary engagement during the conflict-topic discussion task was maximally dissimilar from the other two methods. Still, prior research using the RSQ found a medium correlation between involuntary engagement on the RSQ and HR reactivity measured during a series of laboratory-based stressor tasks (Connor-Smith et al., 2000). In the current study, internal consistency reliability for the involuntary engagement scale of the RSQ-FD was good. However, test-retest reliability for HR in the laboratory and EMA methods was not available. Future research on convergence across methods for measuring stress reactivity would benefit from the use of physiological indices that can account for movement in ambulatory assessments, as well as collecting data on test-retest reliability for laboratory and EMA methods.

***Aim 2***

The second aim of the current research was to examine the associations among exposure to ACEs, stress reactivity, and coping/ER in response to stress using data collected from an EMA paradigm, a laboratory paradigm, and questionnaires.

***Hypothesis 2.*** The hypothesis that greater exposure to ACEs would be associated with increased reactivity to stress was not supported by correlational analyses or regression analyses in any of the three methods (laboratory, EMA, and questionnaire). Previous studies examining

stress reactivity in individuals with histories of exposure to ACEs has produced mixed results (Lovallo et al., 2012), with some work demonstrating heightened reactivity (e.g., Fries et al., 2008; Oosterman et al., 2010), other work showing blunted reactivity (e.g., Bourassa et al., 2021; Fisher et al., 2012; Gunnar et al., 2009; Voellmin et al., 2015), and recent meta-analytic research finding no effects (Sigrist et al., 2021). The current research was designed to use HRV as an index of ANS in operationalizing stress reactivity, mitigating methodological complications associated with research on the stress response system through cortisol (Bernard et al., 2017; Koss & Gunnar, 2018). However, technical malfunction with the ambulatory ECG rendered the EMA-based HRV data insufficient for testing the current hypotheses, and HR data were used instead for both EMA and laboratory methods. Notwithstanding great potential for elucidating stress reactivity in daily life, ambulatory physiological measurement is vulnerable to motion artifacts, improper device application (especially in studies with children and adolescents), and environmental factors (e.g., temperature, moisture) that can compromise data quality (Smets et al., 2018). Additional research is needed across methods to clarify patterns of physiological responding in youth exposed to ACEs.

***Hypothesis 3.*** The hypothesis that greater exposure to ACEs would be associated with greater use of DC and less use of PCC and SCC was not supported in any of the three methods (EMA, laboratory, or questionnaire). These data stand in contrast to previous research characterizing youth with exposure to ACEs as "dysregulated" (Gruhn & Compas, 2020; Lavi et al., 2019), as well as the few studies targeting coping/ER strategies and finding exposure to ACEs to be associated with less use of SCC. The current study focused on understanding responses to family-based stressors. Children first acquire the skills needed to regulate their emotions through interactions with their parents (Tan et al., 2020), and throughout childhood,

parents and children develop predictable and stable patterns of behavior following repeated interactions (Granic et al., 2003). Adolescents with ACEs may become well-adapted to managing family-based stressors, while newer contexts (e.g., relationships, friendships, school) may pose fresh challenges (Bonanno & Burton, 2013). Future research should explore coping/ER in a variety of other stress-generating contexts in the lives of adolescents with histories of ACEs.

***Hypothesis 4.*** Although no associations emerged between stress reactivity and coping/ER in daily life or in the laboratory, consistent with previous research on the RSQ (Connor-Smith et al., 2000), increased endorsement of involuntary engagement was associated with reports of greater use of DC. In contrast to previous RSQ research (Connor-Smith et al., 2000), increased endorsement of involuntary engagement was also associated with reports of greater use of PCC. Participants completed the RSQ-FD about their stress responses relating to their conflict topic following their participation in the laboratory study and prior to completing the EMA conflict-topic discussion task. Having already completed the conflict-topic discussion task in the laboratory (which, as mentioned above, potentially primes adolescents to use more PCC), youth may have been oriented toward using more action-oriented strategies to manage that specific stressor, along with their corresponding stress reactivity.

***Aim 3***

The third aim of the current research was to provide a preliminary test of the associations of exposure to ACEs, stress reactivity, and coping/ER with symptoms of internalizing and externalizing psychopathology.

***Hypothesis 5.*** Consistent with a large body of research on the association between exposure to ACEs and psychopathology (e.g., Baldwin et al., 2021; Shonkoff et al., 2012), the hypothesis that greater exposure to ACEs would be associated with more internalizing and

externalizing psychopathology was largely supported in the current research. Given that, on average, parents and children reported exposure to ACEs in the "none or low" category across all subscales, these data suggest that even low levels of ACEs exposures may impact youth psychological functioning. Of note, a total maltreatment score was used to operationalize exposure to ACEs in the current research. The cumulative risk approach (Evans et al., 2013) has demonstrated a dose-response relation between exposure to ACEs and psychological and physical problems (Anda et al., 2005; Felitti et al., 1998) but assumes all ACEs are equal and additive in conferring risk and omits potentially important information (e.g., frequency, duration, severity). Still, selection of an approach for operationalizing ACEs is dependent on research goals (Henry et al., 2022). Approaches that can provide a more nuanced understanding of the associations between exposure to ACEs and stress reactivity, coping/ER, and psychopathology will be important in future studies with larger sample sizes to detect significant effects (e.g., Dimensional Model of Adversity and Psychopathology; Sheridan & McLaughlin, 2014).

***Hypothesis 6.*** Greater use of DC was hypothesized to be associated with more internalizing and externalizing psychopathology, while greater use of PCC and SCC would be associated with less internalizing and externalizing psychopathology. Consistent with prior research linking greater use of DC to poorer adjustment (Compas et al., 2012), the hypothesized association between DC and psychological symptoms was fairly well supported when DC was measured in the laboratory and by questionnaire. However, an association between DC and psychological symptoms was not detected when DC was measured using EMA. No validated scale for assessing in-vivo coping/ER exists, and most commonly, single-item measures are used to assess a range of coping/ER strategies (e.g., Brans et al., 2013). Although the current research represents a considerable improvement from single items, DC was measured using three items

with relatively low internal consistency reliability (α = .49). The current research provides a step toward comprehensive assessment of coping/ER in daily life, but additional work is needed in this area.

Greater use of PCC measured in the laboratory was unexpectedly associated with more internalizing symptoms, and although the relation between greater use of SCC and fewer psychological symptoms approached significance in correlational analyses, no other associations between PCC or SCC and symptoms emerged in regression analyses. Research suggests that the adaptiveness of different coping/ER strategies depends on context (Aldao et al., 2015; Troy et al., 2013). In the current research, the context for adolescent coping was a family-based stressor rated highly by caregiver and child. The use of problem solving (one aspect of PCC) may be maladaptive when individuals have little control over a given situation (e.g., cannot change or modify the stressor). The family setting during early adolescence is characterized by caregiver-youth relationships that are largely still vertical (Branje, 2018). Accordingly, youth may have less control during conflict-topic discussions with caregivers, and their use of PCC may be less effective and adaptive. Understanding the sensitivity of coping/ER to context is a continued area of importance for future work which may be particularly well-suited for EMA methods.

***Hypothesis 7.*** The hypothesis that reactivity to stress would be associated with more internalizing and externalizing psychopathology was not supported by correlational analyses or regression analyses in any of the three methods. HRV is recognized as a transdiagnostic biomarker of psychopathology (Appelhans & Luecken, 2006; Beauchaine & Thayer, 2015). As mentioned above, although HRV was the intended measure of stress reactivity for the laboratory and EMA tasks, technical malfunctions rendered these data unusable, and HR was implemented in the present analyses. However, previous research has demonstrated that other measures of

stress reactivity (e.g., respiratory sinus arrhythmia, pre-ejection period) are more effective at distinguishing youth with internalizing and externalizing symptoms from controls (e.g., Boyce et al., 2018), and these measures should be pursued for the measurement of stress reactivity in future research.

### *Strengths*

The current research is characterized by methodological strengths that advance the state of the research on stress reactivity and coping/ER in youth with a range of exposure to ACEs. First, stress reactivity and coping/ER were examined together (Gruhn, 2018) to provide insight into relations between two potential mediators and moderators of the association between exposure to ACEs and psychopathology, and thus two potential targets for intervention. Second, three maximally dissimilar methods (EMA, laboratory, questionnaire) were used to measure stress reactivity and coping/ER under highly similar contexts (i.e., EMA: 10-min adolescent-caregiver discussion task on a conflict topic in daily life; laboratory: 10-min adolescent-caregiver discussion task on the same conflict topic in the laboratory; questionnaire: adolescent recollection of typical stress responses [stress reactivity, coping/ER] to the same conflict topic). Third, while questionnaires and laboratory tasks have been the most common methods for examining responses to stress, they are limited by retrospective recall and ecological validity, respectively (Wilhelm & Grossman, 2010). EMA was implemented in the current research design to elucidate adolescent stress reactivity and coping/ER in their natural environments (Stone & Shiffman, 1994). Fourth, coping/ER is typically operationalized as "emotion dysregulation," or a broad array of strategies and trait-like aspects of cognition and behavior that are often confounded with psychological symptoms, in research on youth exposed to ACEs (Compas et al., 2017). In reality, different coping/ER strategies likely have unique associations

with ACEs, stress reactivity, and psychopathology (Heleniak et al., 2016). In contrast, the current research operationalized coping/ER as three specific responses (PCC, SCC, DC) from a control-based model of coping/ER (e.g., Compas et al., 2001; Weisz et al., 1994) that have demonstrated associations with psychological symptoms (Compas et al., 2017) and shown malleability to intervention (Compas et al., 2011). Finally, in examining associations among ACEs, stress reactivity, coping/ER, and psychopathology, the current research accounts for potential associations with age (Kessler et al., 2007; Merikangas et al., 2010), pubertal development (Carlo et al., 2012; Magnus et al., 2018; Spear, 2009), gender (Koskinen et al., 2009; McRae et al., 2008; Ordaz & Luna, 2012; Zahn-Waxler et al., 2008), and race and ethnicity (DeSantis et al., 2007; Wang et al., 2005; Nguyen et al., 2007).

***Limitations***

In addition to hypothesis-specific recommendations discussed above, several overall limitations should be considered when evaluating the results of the current research and designing future studies. First, hypothesis testing was limited by a small sample size. The subsample of participants with usable data for analyses including stress reactivity data (HR-R) in the EMA and laboratory methods was particularly small, reducing the likelihood of detecting a true effect. Researchers would benefit from minimizing factors that may compromise ambulatory physiological data, such as emphasizing the importance of proper ECG placement to children and families while orienting participants to study materials. Second, the current analyses were cross-sectional and therefore the direction of the associations among the constructs of interest could not be determined. Prospective designs examining stress reactivity and coping/ER as mediators of the association between exposure to ACEs and psychopathology in daily life are needed. Third, although exposure to ACEs varied across participants, average caregiver and

adolescent reports (CTQ-SF) were in the "none or low" category. Accordingly, the results of the current research are generalizable to a similar population of youth. Although six of the eight recruitment sites for the laboratory study were selected to maximize the likelihood of recruiting adolescents with histories of ACEs, 65% of the laboratory study sample was recruited from Vanderbilt University listervs. Greater researcher engagement with agencies, organizations, and individuals serving families of youth with ACEs, perhaps through a community-based participatory approach, might result in more balanced recruitment in future research (Collins et al., 2018).

### *Future Directions*

The aforementioned limitations of the current research provide clear directions for future research toward better *understanding* stress reactivity and coping/ER as pathways from exposure to ACEs to psychopathology. Importantly, EMA methods, including adaptations of the approaches used in the current research, provide opportunities for *modifying* stress reactivity and coping/ER in youth exposed to ACEs. Translational research toward delivering technology-based interventions in daily life (e.g., mHealth, ecological momentary interventions, digital health interventions; Russell & Gajos, 2020) rely on EMA methodology. The integration of adolescent reports of stress exposure, coping/ER, and emotions during their daily lives (e.g., EMA-based smartphone surveys) and passive sensing of physiological states (e.g., wearable devices like ambulatory ECG) allow for deep digital phenotyping, which can provide the basis for "just-in-time" adaptive interventions delivering skills and support at the moment and in the context at which they are most needed (Nahum-Shani et al., 2018).

### *Conclusion*

Stress reactivity and coping/ER have been identified as intervention targets with the potential to interrupt developmental cascades from ACEs exposure to psychopathology. In addressing methodological weaknesses in the field that have stymied related research progress, the current study provides evidence for the feasibility of conducting EMA research in a sample of youth with ACEs, preliminary support for convergence across methods for measuring stress reactivity and coping/ER, and preliminary support for associations among ACEs, stress reactivity and coping/ER, and psychological symptoms. EMA is no doubt a promising research method for shedding light on the daily lives of adolescents while providing opportunities for accelerating the translation of research to intervention. Still, additional research is necessary to understand the correspondence of EMA with other commonly used methods, especially in populations where mixed findings are common.

Appendix

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**Table 1**

*Sociodemographic Characteristics of the Sample (N = 53)*

| Focal | Characteristic | *M/%* | *SD* |
|---|---|---|---|
| Caregiver | Age | 42.21 | 6.29 |
| | Gender (% female) | 88.7 | |
| | Education (%) | | |
| | Graduate degree | 45.3 | |
| | Some graduate education | 7.5 | |
| | College graduate | 28.3 | |
| | Some college or technical school | 13.2 | |
| | High school graduate | 3.8 | |
| | Marital Status (%) | | |
| | Married/living with partner | 73.6 | |
| | Never married | 11.3 | |
| | Divorced or annulled | 13.2 | |
| | Widowed | 1.9 | |
| Adolescent | Age | 11.98 | 1.74 |
| | Gender (% female) | 54.7 | |
| | Race (%) | | |
| | White | 67.9 | |
| | Black/African American | 15.1 | |
| | Asian | 9.4 | |
| | More than one race/other race | 7.6 | |
| | Ethnicity (% Hispanic or Latino/a) | 9.4 | |

*Note.* All demographic data reported at the time of the laboratory study participation.

**Table 2**

*Schedule of Events*

| Measure | Lab Study | Initial Visit | EMA Protocol Day 1 | EMA Protocol Day 2 | EMA Protocol Day 3 | EMA Protocol Day 4 | Final Visit | 3-Month Follow-up[g] |
|---|---|---|---|---|---|---|---|---|
| Informed consent/assent[a] | X | X | | | | | | X |
| Ambulatory assessments | | | | | | | | |
| EMA survey | | | X[b] | X[b] | X[b] | X[b] | | |
| Follow-up survey | | | X[c] | X[c] | X[c] | X[c] | | |
| Daily survey | | | X[d] | X[d] | X[d] | X[d] | | |
| Family discussion survey | | | | X[e] | | | | |
| Family activity survey | | | | X[e] | | | | |
| Heart rate (via ECG) | | | X[f] | X[f] | X[f] | X[f] | | |
| Study tasks | | | | | | | | |
| Family (conflict-topic) discussion task | X | | | X | | | | |
| Family activity | | | | X | | | | |
| Video Mediated Recall (VMR) | X | | | | | | | |
| Questionnaires/interviews | | | | | | | | |
| Child Trauma Questionnaire – Short Form (CTQ - SF) | X | | | | | | | |
| Child Behavior Checklist (CBCL) | X | | | | | | | X |
| Youth Self Report (YSR) | X | | | | | | | X |
| Pubertal Development Scale (PDS) | | X | | | | | | |
| Responses to Stress Questionnaire (RSQ), Family Discussion – Child Version | | X | | | | | | |
| Responses to Stress Questionnaire (RSQ), Family Discussion – Caregiver Version | | X | | | | | | |
| Daily Inventory of Stressful Events (DISE) Interview | | | | | | | X | |

*Note.*

[a] The caregiver completed informed consent for their participation in each part of the study, and they provided consent for the participation of their minor child. The child completed an assent form for their participation in each part of the study.

[b] Ecological momentary assessment (EMA) surveys were distributed on the following schedule: On two weekdays, participants received two surveys randomized between 4:00 pm and 7:30 pm. On Saturday and Sunday, participants received two surveys randomized between 9:00 am and 12:30 pm, two surveys randomized between 12:30 pm and 4:00 pm, and two surveys randomized between 4:00 pm and 7:30 pm.

[c] If a stressor was endorsed on an EMA survey, participants received Follow-up Surveys 15- and 30-min after their EMA survey to track coping/ER and positive and negative emotions.

[d] The Daily Surveys are self-report measures of stress, coping/ER, and positive and negative emotions experienced over the course of the entire day. On each of the four days, participants received Daily Surveys at 8:00 pm.

[e] The Family Discussion and Family Activity Surveys are self-report measures of stress, coping/ER, and positive and negative emotions experienced during the 10-min family (conflict-topic) discussion task and the family activity. Participants recieved these surveys after completing a scheduled family discussion and family activity in the context of the EMA Study. The family discussion and family activity took place on any of the four days of the family's choosing. The day and time of the family discussion and activity were planned with the RA at the initial visit.

[f] Adolescents were asked to wear the ambulatory electrocardiogram (ECG; EcgMove4; Movisens) to collect data on physiological reactivity to stress. Physiological data were tracked continuously for four days, starting when adolescents applied the ambulatory ECG and ending when they removed the ambulatory ECG. On two weekend days (Saturday and Sunday), these data were collected continuously from the time participants woke up through the time they went to bed. On two weekdays, these data were collected continuously from the time participants came home from school through the time they went to bed.

[g] The three-month follow-up consisted of at-home online surveys. Informed consent and assent forms and surveys were sent to the caregiver and child through separate links to participant emails. Surveys were completed in REDCap (Harris et al., 2009).

**Table 3**

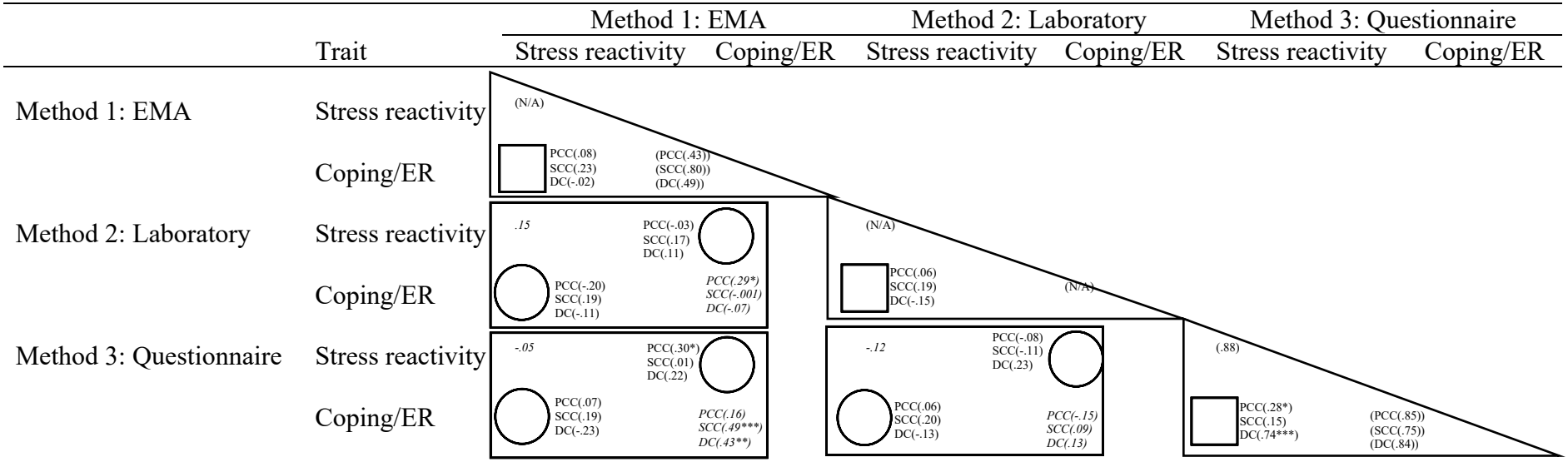
*Descriptive Statistics for Study Variables*

| Construct | Measure | *M*/% | *SD* |
|---|---|---|---|
| Covariate | | | |
| | Pubertal Development Scale | 2.80 | .81 |
| ACEs | | | |
| | Child Trauma Questionnaire – Short Form (PR/CR) | | |
| | Emotional abuse | 8.51/7.94 | 4.13/3.20 |
| | Physical abuse | 6.00/6.34 | 2.79/2.54 |
| | Sexual abuse | 5.62/5.21 | 2.89/1.26 |
| | Emotional neglect | 6.81/7.85 | 2.36/3.29 |
| | Physical neglect | 6.03/6.17 | 2.35/2.56 |
| | Total maltreatment score | 32.98/33.52 | 11.95/10.38 |
| Coping/ER | | | |
| | Responses to Stress Questionnaire – FD (CR) | | |
| | Primary control coping | 22.25 | 5.98 |
| | Secondary control coping | 29.30 | 6.54 |
| | Disengagement coping | 17.42 | 5.63 |
| | Video-mediated recall | | |
| | Primary control coping | 2.85 | 2.38 |
| | Secondary control coping | 1.62 | 1.56 |
| | Disengagement coping | 0.63 | 1.31 |
| | EMA | | |
| | Primary control coping | 9.43 | 2.40 |
| | Secondary control coping | 9.73 | 3.48 |
| | Disengagement coping | 6.10 | 2.26 |
| Reactivity | | | |
| | Responses to Stress Questionnaire – FD (CR) | | |
| | Involuntary engagement | 28.50 | 9.42 |
| | Laboratory | | |
| | Heart rate (bpm) talking baseline | 83.78 | 11.67 |
| | Heart rate conflict-topic discussion task | 86.03 | 10.82 |
| | Heart rate R (Δ from laboratory baseline) | 3.98 | 12.45 |
| | EMA | | |
| | Heart rate conflict-topic discussion task | 114.79 | 27.94 |
| | Heart rate R (Δ from laboratory baseline) | 35.51 | 33.77 |
| Symptoms | | | |
| | Internalizing problems (CBCL *T*-score) | 56.96 | 12.14 |
| | Externalizing problems (CBCL *T*-score) | 50.98 | 10.31 |

*Note.* ACEs = adverse childhood experiences; FD = family discussion; PR = parent report; CR = child report; CBCL = Child Behavior Checklist; R = reactivity.
Raw (total) scores are provided for all measures of coping/ER and the involuntary engagement scale from the Responses to Stress Questionnaire – FD (CR). All other scores are presented as is typical for the given measure (and described in the Method section) or as listed in the table.

**Table 4**

*Multitrait-Multimethod Correlation Matrix*




*Note.* ER = emotion regulation, PCC = primary control coping, SCC = secondary control coping, DC = disengagement coping, N/A = not available in the current research.

Validity is represented by italics. Reliability is represented by parentheses (e.g., intraclass correlation coefficients). Heterotrait-monomethod is represented by square enclosures. Heterotrait-heteromethod is represented by circle enclosures. Heteromethod blocks are represented by rectangle enclosures. Monomethod blocks are represented by triangle enclosures.

Following guidance from Campbell and Fiske (1959), evidence of convergent validity is supported by (1) validity values significantly different from zero and sufficiently large, (2) validity values higher than corresponding heterotrait-heteromethod values, (3) validity values higher than corresponding heterotrait-monomethod values, and (4) the same pattern of trait interrelationship is evident for the monomethod and heteromethod blocks.

Figure adapted from Campbell and Fiske (1959).

*$p$<.05

**$p$<.01

***$p$<.001

**Table 5**

*Bivariate Correlations Among Sociodemographics and Key Study Variables*

| Measure | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Age | | | | | | | | | | | | | | | | |
| 2. Gender | -.03 | | | | | | | | | | | | | | | |
| 3. Race | .19 | .05 | | | | | | | | | | | | | | |
| 4. PDS | .67*** | .40** | .23 | | | | | | | | | | | | | |
| 5. ACEs | .11 | -.13 | .11 | .17 | | | | | | | | | | | | |
| 6. EMA PCC | .04 | -.01 | -.02 | -.03 | -.08 | | | | | | | | | | | |
| 7. EMA SCC | -.18 | -.16 | -.03 | -.27$^{+}$ | -.16 | .53*** | | | | | | | | | | |
| 8. EMA DC | -.08 | .06 | -.31* | .05 | .10 | .30* | .11 | | | | | | | | | |
| 9. Lab PCC | .26$^{+}$ | -.07 | -.04 | .09 | .04 | .29* | .08 | .17 | | | | | | | | |
| 10. Lab SCC | -.21 | .02 | .24 | .02 | -.03 | .02 | -.001 | .13 | .08 | | | | | | | |
| 11. Lab DC | .06 | .21 | -.07 | -.03 | .22 | .09 | -.06 | -.07 | -.27* | -.24 | | | | | | |
| 12. Ques PCC | -.09 | .03 | .23 | .19 | .19 | .16 | .25 | .12 | -.15 | .12 | -.06 | | | | | |
| 13. Ques SCC | .08 | .01 | .02 | .20 | .004 | .31* | .49*** | .20 | -.18 | .09 | -.04 | .42** | | | | |
| 14. Ques DC | .21 | .42** | -.02 | .44*** | .02 | .21 | -.08 | .43** | -.01 | -.09 | .13 | .15 | .18 | | | |
| 15. EMA SR | -.30$^{+}$ | .17 | -.17 | -.30 | -.18 | .08 | .23 | -.02 | -.20 | .19 | -.11 | .07 | .19 | -.23 | | |
| 16. Lab SR | -.13 | .03 | -.08 | -.001 | -.04 | -.03 | .17 | .11 | .06 | .19 | -.15 | .06 | .20 | -.13 | .50 | |
| 17. Ques SR | .27* | .34* | -.09 | .45*** | -.01 | .30* | .01 | .22 | -.08 | -.11 | .23 | .28* | .15 | .74*** | .05 | -.18 |

*Note.* PDS=pubertal development; ACEs=adverse childhood experiences; EMA=ecological momentary assessment; PCC=primary control coping; SCC=secondary control coping; DC=disengagement coping; Lab=laboratory; Ques=questionnaire; SR=stress reactivity. Male=0, female=1; white=0, nonwhite=1.
$^{+}p<.07$

**Table 6**

*Null Models Predicting Stress Reactivity Using EMA and Laboratory Methods*

| | EMA | | | Laboratory | | |
|---|---|---|---|---|---|---|
| Predictor | PE | *SE* | *p* | PE | *SE* | *p* |
| Fixed effects | | | | | | |
| Intercept ($\gamma_{00}$) | 33.38 | 4.72 | <.001 | 2.54 | .90 | .007 |
| Random effects | | | | | | |
| Intercept ($\tau_{00}$) | 801.28 | 199.96 | | 36.84 | 8.92 | |
| Residual($\sigma_e^2$) | 272.88 | 23.12 | | 8.92 | .63 | |
| ICC | | 75% | | | 81% | |

*Note.* PE = parameter estimate, SE = standard error, ICC = intraclass correlation coefficient.

**Table 7**

*Multilevel Models Predicting Stress Reactivity Using EMA and Laboratory Methods*

| | EMA | | | Laboratory | | |
|---|---|---|---|---|---|---|
| Predictor | PE | *SE* | *p* | PE | *SE* | *p* |
| Fixed effects | | | | | | |
| Intercept ($\gamma_{00}$) | 55.00 | 37.82 | .16 | 5.47 | 6.62 | .41 |
| Age ($\gamma_{01}$) | -.19 | 3.39 | .96 | .29 | .73 | .69 |
| Gender ($\gamma_{02}$) | 20.17 | 10.14 | .06 | .93 | 2.06 | .65 |
| PDS ($\gamma_{03}$) | -17.71 | 8.16 | .04 | -2.52 | 1.68 | .14 |
| ACEs ($\gamma_{04}$) | -3.04 | 7.57 | .69 | .72 | .92 | .44 |
| Random effects | | | | | | |
| Intercept ($\tau_{00}$) | 659.62 | 167.74 | | 35.26 | 7.64 | |
| Residual($\sigma_e^2$) | 212.19 | 18.49 | | 9.00 | .65 | |

*Note.* PE = parameter estimate, SE = standard error, ACEs = adverse childhood experiences, PDS = pubertal development.

**Table 8**

*Linear Regression Model Predicting Stress Reactivity Using the Questionnaire Method*

| Predictor | *b(SE)* | 95% CI | β | *t* | *p* |
|---|---|---|---|---|---|
| Age | .22(.97) | [-1.73, 2.18] | .04 | .23 | .82 |
| Gender | 4.08(2.85) | [-1.65, 9.81] | .21 | 1.43 | .16 |
| PDS | 4.04(2.33) | [-.66, 8.73] | .35 | 1.73 | .09 |
| ACEs | -.45(1.32) | [-3.09, 2.21] | -.05 | -.34 | .74 |
| | | *Adjusted* $R^2$=.18, $F(4,46)$=3.73** | | | |

*Note.* PDS=pubertal development; ACEs=adverse childhood experiences.

**$p$<.01.

**Table 9**

*Linear Regression Models Predicting Coping/Emotion Regulation Using EMA, Laboratory, and Questionnaire Methods*

| | | EMA | | | | Laboratory | | | | Questionnaire | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Model | Predictor | *b(SE)* | 95% CI | β | *t* | *b(SE)* | 95% CI | β | *t* | *b(SE)* | 95% CI | β | *t* |
| PCC | Gender | -.48(.82) | [-2.13, 1.17] | -.10 | -.58 | -1.15(.79) | [-2.74, .43] | -.24 | -1.47 | 2.28(1.81) | [-1.37, 5.93] | .19 | 1.26 |
| | Race | -.10(.82) | [-1.75, 1.55] | -.02 | -.13 | -.35(.76) | [-1.89, 1.18] | -.07 | -.46 | 2.30(1.77) | [-1.26, 5.86] | .19 | 1.30 |
| | PDS | .09(.53) | [-.99, 1.17] | .03 | .17 | .62(.49) | [-.37, 1.62] | .22 | 1.27 | .28(1.14) | [-2.02, 2.57] | .04 | .24 |
| | ACEs | -.27(.70) | [-1.69, 1.14] | -.06 | -.39 | -.01(.37) | [-.76, .74] | -.006 | -.04 | 1.01(.88) | [-.75, 2.78] | .17 | 1.16 |
| | | *Adjusted* $R^2$=-.08, *F*(4,41)=.13 | | | | *Adjusted* $R^2$=-.02, *F*(4,44)=.58 | | | | *Adjusted* $R^2$=.05, *F*(4,46)=1.58 | | | |
| SCC | Gender | .36(1.12) | [-1.91, 2.63] | .05 | .32 | -.36(.51) | [-1.39, .67] | -.11 | -.70 | .90(2.10) | [-3.32, 5.12] | .07 | .43 |
| | Race | -.18(1.12) | [-2.45, 2.09] | -.02 | -.15 | .87(.50) | [-.13, 1.87] | .26 | 1.76 | -.62(2.04) | [-4.73, 3.50] | -.05 | -.30 |
| | PDS | -1.06(.73) | [-2.54, .42] | -.25 | -1.44 | .04(.32) | [.60, .69] | .02 | .14 | 1.51(1.32) | [-1.14, 4.16] | .19 | 1.15 |
| | ACEs | -.84(.96) | [-2.79, 1.10] | -.14 | -.87 | -.09(.24) | [-.58, .40] | -.06 | -.38 | -.25(1.01) | [-2.29, 1.79] | -.04 | -.25 |
| | | *Adjusted* $R^2$=.01, *F*(4,41)=1.05 | | | | *Adjusted* $R^2$=-.01, *F*(4,44)=.89 | | | | *Adjusted* $R^2$=-.04, *F*(4,46)=.58 | | | |
| DC | Gender | .15(.74) | [-1.34, 1.64] | .03 | .20 | .72(.42) | [-.12, 1.56] | .27 | 1.72 | 3.71(1.57) | [.55, 6.88] | .33 | 2.36* |
| | Race | -1.57(.74) | [-3.05, -.08] | -.33 | -2.13* | -.20(.40) | [-1.01, .62] | -.07 | -.49 | -1.60(1.53) | [-4.68, 1.49] | -.13 | -1.04 |
| | PDS | .27(.48) | [-.70, 1.24] | .09 | .55 | -.30(.26) | [-.82, .23] | -.19 | -1.14 | 2.37(.99) | [.38, 4.35] | .34 | 2.40* |
| | ACEs | .17(.63) | [-1.11, 1.45] | .04 | .27 | .41(.20) | [.02, .81] | .31 | 2.10* | .08(.76) | [-1.45, 1.61] | .01 | .11 |
| | | *Adjusted* $R^2$=.02, *F*(4,41)=1.27 | | | | *Adjusted* $R^2$=.05, *F*(4,44)=1.58 | | | | *Adjusted* $R^2$=24, *F*(4,46)=4.86** | | | |

*Note.* PDS=pubertal development; ACEs=adverse childhood experiences; PCC=primary control coping; SCC=secondary control coping; DC=disengagement coping.

*$p$<.05.

**$p$<.01.

**Table 10**

*Multilevel Models Predicting Stress Reactivity Using EMA and Laboratory Methods*

| | EMA | | | Laboratory | | |
|---|---|---|---|---|---|---|
| Predictor | PE | *SE* | *p* | PE | *SE* | *p* |
| Fixed effects | | | | | | |
| Intercept ($\gamma_{00}$) | 49.30 | 41.06 | .24 | 5.94 | 6.75 | .38 |
| Age ($\gamma_{01}$) | -.90 | 3.50 | .80 | .04 | .77 | .95 |
| Gender ($\gamma_{02}$) | 18.44 | 9.61 | .06 | .76 | 2.02 | .71 |
| PDS ($\gamma_{03}$) | -16.51 | 8.20 | .05 | -1.92 | 1.62 | .24 |
| PCC ($\gamma_{04}$) | 1.17 | 2.27 | .61 | .59 | .42 | .17 |
| SCC ($\gamma_{05}$) | .67 | 1.56 | .67 | -.25 | .61 | .69 |
| DC ($\gamma_{06}$) | -.51 | 2.13 | .81 | -.62 | .73 | .40 |
| Random effects | | | | | | |
| Intercept ($\tau_{00}$) | 634.26 | 159.37 | | 32.53 | 7.15 | |
| Residual($\sigma_e^2$) | 207.68 | 17.80 | | 8.94 | .65 | |

*Note.* PDS=pubertal development; PCC=primary control coping; SCC=secondary control coping; DC=disengagement coping.

**Table 11**

*Linear Regression Model Predicting Stress Reactivity Using the Questionnaire Method*

| Predictor | *b(SE)* | 95% CI | β | *t* | *p* |
|---|---|---|---|---|---|
| Age | .80(.75) | [-.71, 2.31] | .15 | 1.07 | .29 |
| Gender | .02(2.11) | [-4.22, 4.27] | .001 | .01 | .99 |
| PDS | .35(1.79) | [-3.26, 3.97] | .03 | .20 | .85 |
| PCC | .37(.18) | [.02, .73] | .23 | 2.12 | .04 |
| SCC | -.10(.15) | [-.41, .20] | -.07 | -.69 | .50 |
| DC | 1.11(.19) | [.74, 1.49] | .67 | 5.98 | <.001 |
| *Adjusted* $R^2$=.55, $F(6,45)$=11.47*** | | | | | |

*Note.* PDS=pubertal development; PCC=primary control coping; SCC=secondary control coping; DC=disengagement coping.

**Table 12**

*Linear Regression Models Predicting Internalizing and Externalizing Symptoms Using EMA, Laboratory, and Questionnaire Methods*

| | | EMA | | | | Laboratory | | | | Questionnaire | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Model | Predictor | *b(SE)* | 95% CI | β | *t* | *b(SE)* | 95% CI | β | *t* | *b(SE)* | 95% CI | β | *t* |
| Intern | ACEs | 7.55(2.75) | [1.94, 13.16] | .43 | 2.74** | .85(1.41) | [-2.00, 3.70] | .09 | .60 | 2.40(1.31) | [-.25, 5.04] | .23 | 1.83$^{+}$ |
| | PCC | .47(.80) | [-1.16, 2.11] | .11 | .59 | 1.35(.63) | [.08, 2.61] | .32 | 2.16* | -.34(.24) | [-.82, .15] | -.20 | -1.39 |
| | SCC | -.88(.56) | [2.02, .25] | -.29 | -1.60 | -.97(.96) | [-2.91, .98] | -.15 | -1.01 | -.24(.21) | [-.66, .18] | -.16 | -1.16 |
| | DC | .15(.76) | [-1.40, 1.70] | .03 | .20 | 3.10(1.13) | [.81, 5.40] | .43 | 2.74** | .85(.33) | [.19, 1.50] | .49 | 2.60** |
| | SR | .01(.05) | [-.09, .12] | .04 | .27 | .04(.11) | [-.19, .26] | .05 | .32 | -.04(.20) | [-.44, .36] | -.04 | -.19 |
| | | *Adjusted* $R^2$=.15, $F$(5,31)=2.22$^{+}$ | | | | *Adjusted* $R^2$=.16, $F$(5,38)=2.66* | | | | *Adjusted* $R^2$=.22, $F$(5,46)=3.84** | | | |
| Extern | ACEs | 6.68(1.85) | [2.91, 10.46] | .52 | 3.61** | 1.28(1.03) | [-.81, 3.36] | .17 | 1.24 | 2.20(1.08) | [.03, 4.38] | .29 | 2.04* |
| | PCC | .97(.54) | [-.13, 2.08] | .30 | 1.80 | .27(.46) | [-.65, 1.20] | .08 | .60 | -.11(.20) | [-.51, .29] | -.09 | -.55 |
| | SCC | -.84(.37) | [-1.60, -.08] | -.38 | -2.26* | -.58(.70) | [-2.00, .85] | -.11 | -.82 | -.11(.17) | [-.45, .24] | -.10 | -.63 |
| | DC | .04(.51) | [1.00, 1.09] | .01 | .09 | 2.88(.83) | [1.20, 4.56] | .51 | 3.48*** | .05(.27) | [.49, .59] | .04 | .17 |
| | SR | .04(.03) | [-.03, .11] | .19 | 1.24 | .03(.08) | [-.13, .20] | .05 | .40 | .18(.16) | [-.15, .51] | .23 | 1.09 |
| | | *Adjusted* $R^2$=.28, $F$(5, 31)=3.78** | | | | *Adjusted* $R^2$=.25, $F$(5,38)=3.91** | | | | *Adjusted* $R^2$=.05, $F$(5,46)=1.52 | | | |

*Note.* Intern=internalizing symptoms; Extern=externalizing symptoms; ACEs=adverse childhood experiences; PCC=primary control coping; SCC=secondary control coping; DC=disengagement coping; SR=stress reactivity.

$^{+}$p<.08.

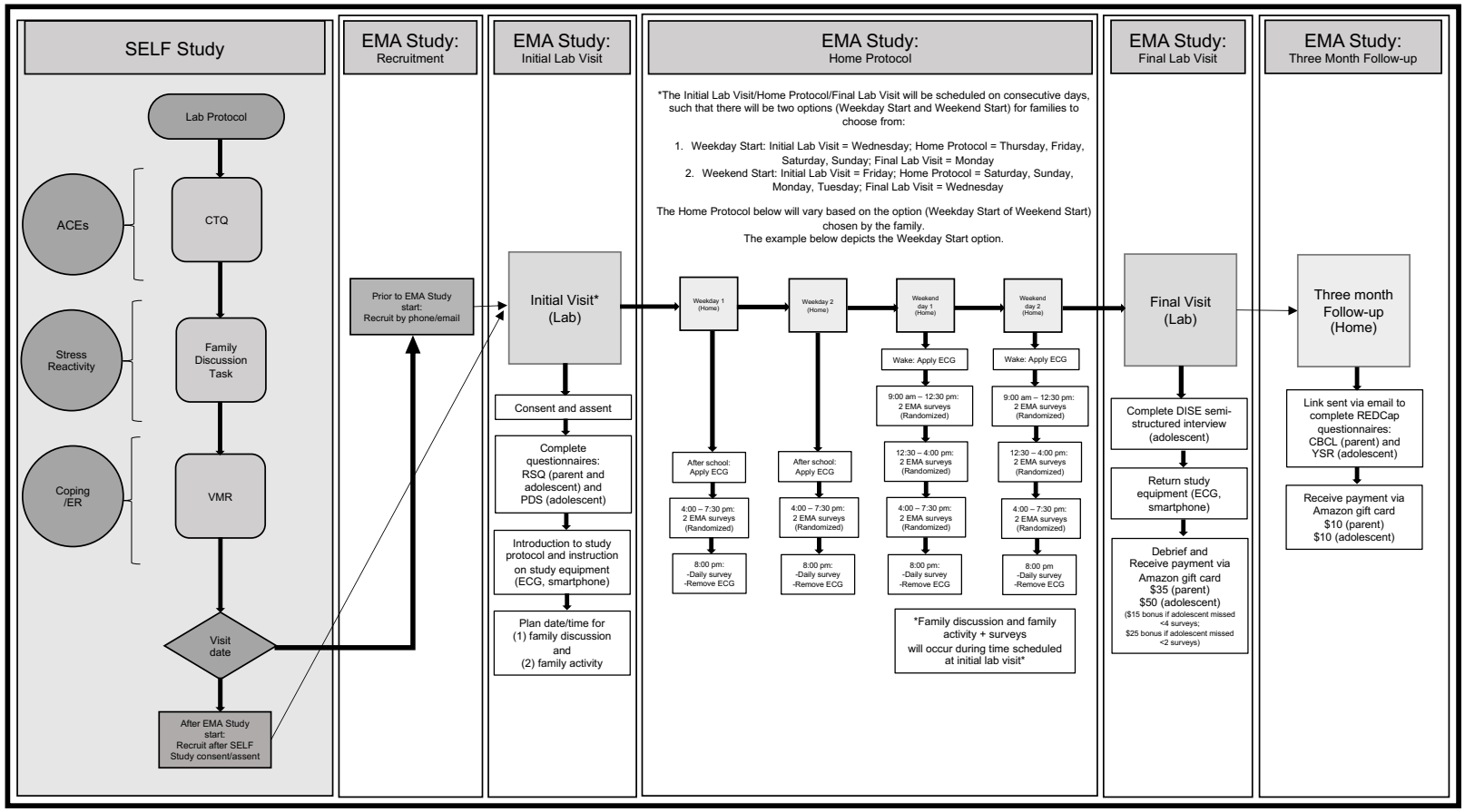
*p<.05.

**p<.01.

***p<.001.

**Figure 1**

*Schematic of Study Flow*




*Note.* Includes key datapoints collected from the laboratory study (IRB #181531) and the home-based study (IRB #191930). ACEs = adverse childhood experiences; ER = emotion regulation; CTQ = Childhood Trauma Questionnaire-Short Form; VMR = video mediated recall task; RSQ-C/P = Responses to Stress Questionnaire – Family Discussion, Child/Parent; PDS = Pubertal Development Scale; ECG = electrocardiogram; DISE = Daily Inventory of Stressful Events; YSR = Youth Self Report; CBCL = Child Behavior Checklist.

## Appendix[4]

**Hypothesis 2:** Greater exposure to ACEs will be associated with heightened and blunted reactivity in response to stress.

***Null Multilevel Model for EMA and Laboratory Data***

$$SR_{ij} = \beta_{0j} + e_{ij}$$
$$\beta_{0j} = \gamma_{00} + u_{0j}$$

$$e_{ij} \sim N(0, \sigma_e^2)$$
$$u_{0j} \sim N(0, \tau_{00})$$

$$\text{ICC}_{epoch} = \frac{\sigma_e^2}{\tau_{00} + \sigma_e^2}$$

$$\text{ICC}_{person} \frac{\tau_{00}}{\tau_{00} + \sigma_e^2}$$

[4]ACEs = adverse childhood experiences (measured via Child Trauma Questionnaire – Short Form); PCC = primary control coping, DC = disengagement coping, SCC = secondary control coping (measured via ecological momentary assessment surveys [EMA], video mediated recall [VMR; laboratory], and RSQ-FD [questionnaire]; SR = stress reactivity (measured via heart rate reactivity [EMA and laboratory] and involuntary engagement scale on RSQ-FD [questionnaire;); PDS = Pubertal Development Scale.

***Multilevel Model for EMA and Laboratory Data***

$$SR_{ij} = \beta_{0j} + e_{ij}$$

$$\beta_{0j} = \gamma_{00} + \gamma_{01}Age_j + \gamma_{02}Gender_j + \gamma_{03}PDS_j + \gamma_{04}ACEs_j + u_{0j}$$

$e_{ij} \sim N(0, \sigma_e^2)$
$u_{0j} \sim N(0, \tau_{00})$

***Multiple Linear Regression Model for Questionnaire Data***

$$SR_i = \beta_0 + \beta_1 Age_i + \beta_2 Gender_i + \beta_3 PDS_i + \beta_4 ACEs_i + e_i$$

**Hypothesis 3:** Greater exposure to ACEs will be associated with greater use of DC and less use of PCC and SCC.

***Multiple Linear Regression Models for EMA, Laboratory, and Questionnaire Data***

$$PCC_i = \beta_0 + \beta_1 Gender_i + \beta_2 Race_i + \beta_3 PDS_i + \beta_4 ACEs_i + e_i$$

$$SCC_i = \beta_0 + \beta_1 Gender_i + \beta_2 Race_i + \beta_3 PDS_i + \beta_4 ACEs_i + e_i$$

$$DC_i = \beta_0 + \beta_1 Gender_i + \beta_2 Race_i + \beta_3 PDS_i + \beta_4 ACEs_i + e_i$$

**Hypothesis 4:** Heightened and blunted reactivity to stress will be associated with greater use of DC and less use of PCC and SCC.

***Null Multilevel Model for EMA and Laboratory Data***

$$SR_{ij} = \beta_{0j} + e_{ij}$$
$$\beta_{0j} = \gamma_{00} + u_{0j}$$

$e_{ij} \sim N(0, \sigma_e^2)$
$u_{0j} \sim N(0, \tau_{00})$

$$\text{ICC}_{epoch} = \frac{\sigma_e^2}{\tau_{00} + \sigma_e^2}$$

$$\text{ICC}_{person} \frac{\tau_{00}}{\tau_{00} + \sigma_e^2}$$

***Multilevel Model for EMA and Laboratory Data***

$$SR_{ij} = \beta_{0j} + e_{ij}$$

$$\beta_{0j} = \gamma_{00} + \gamma_{01} Age_j + \gamma_{02} Gender_j + \gamma_{03} PDS_j + \gamma_{04} PCC_j + \gamma_{05} SCC_j + \gamma_{04} DC_j + u_{0j}$$

$e_{ij} \sim N(0, \sigma_e^2)$
$u_{0j} \sim N(0, \tau_{00})$

***Multiple Linear Regression Model for Questionnaire Data***

$$SR_i = \beta_0 + \beta_1 Age_i + \beta_2 Gender_i + \beta_3 PDS_i + \beta_4 DC_i + \beta_5 PCC_i + \beta_5 SCC_i + e_i$$

**Hypothesis 5:** Greater exposure to ACEs will be associated with more internalizing and externalizing psychopathology.
**Hypothesis 6*:*** Greater use of DC will be associated with more internalizing and externalizing psychopathology. Greater use of PCC and SCC will be associated with less internalizing and externalizing psychopathology.
**Hypothesis 7:** Heightened and blunted reactivity to stress will be associated with more internalizing and externalizing psychopathology.

***Multiple Linear Regression Model for EMA, Laboratory, and Questionnaire Data***

$$intern_i = \beta_0 + \beta_1 ACEs_i + \beta_2 PCC_i + \beta_3 SCC_i + \beta_4 DC_i + \beta_5 SR_i + e_i$$

$$extern_i = \beta_0 + \beta_1 ACEs_i + \beta_2 PCC_i + \beta_3 SCC_i + \beta_4 DC_i + \beta_5 SR_i + e_i$$