Harsh Parenting and Trajectories of Adjustment across Late Adolescence: Autonomic Nervous System Activity as a Moderator of Risk

by

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Abstract

Adjustment problems including internalizing symptoms (e.g., anxiety, depression) and externalizing behaviors (e.g., rule-breaking, aggression, hostility) increase across earlier adolescence but little is known about their trajectories across mid-to-late adolescence (ages 15 to 18). Socioemotional problems and maladjustment during this developmental period often forecast worsening mental health as youth transition to adulthood and it is imperative to identify individual and family factors that may exacerbate risk for poor outcomes. Harsh parenting, characterized by physically and verbally aggressive behavior, is associated with a wide range of poor adjustment outcomes such as depression, delinquency, and substance use. Longitudinal associations have been reported in adolescence; however, little is known if harsh parenting influences increases in internalizing symptoms or externalizing behaviors. Individual differences in autonomic nervous system (ANS) reactivity are processes that tend to moderate adjustment outcomes in harsh parenting contexts. For example, dysregulation via blunted or inappropriate ANS responding (e.g., lower levels of ANS indices in response to a stressor) may confer vulnerability and enhance risk for maladaptive outcomes. Conversely, greater vagal withdrawal (e.g., decline in respiratory sinus arrhythmia [RSA] from baseline to task) and greater SNS reactivity (i.e., shortening of pre-ejection period [PEP], greater skin conductance reactivity [SCL]) have been associated with better adjustment in the context of harsh parenting. Many studies that have examined harsh parenting and ANS indices have been conducted with children and it is unclear if such resilience occurs in later adolescence. Sex-related effects have also been found to moderate these associations. For example, boys have often been implicated to be at

greatest risk for increasing externalizing problem trajectories in the context of greater harsh parenting when they have poor ANS regulation (e.g., low SCL reactivity [SCLr]).

The present study examines harsh parenting as a predictor of trajectories of adjustment across mid-to-late adolescence, as well as how ANS reactivity may moderate such associations. Using a multi-method approach, we assessed (1) youth and parent-reported harsh parenting as predictors of individual trajectories of internalizing symptoms (anxiety, depression) and externalizing behaviors (rule-breaking, aggression) over three waves (ages 16, 17, 18); (2) the moderating role of ANS reactivity on the aforementioned relations via RSAr, SCLr, and PEPr; and (3) examine the conjoint influence of ANS reactivity and adolescent sex as moderators of risk. Data for the present study were collected at ages 16, 17, and 18. Youth (N = 242; 53% girls, 66.8% White, 33.2% Black/African American) reported on their experiences of physical and verbal aggression from their parents at age 16, as well as completed physiological assessments. Each parent also reported on their own use of harsh parenting and reports were composited for a single score. Additionally, youth also reported on their internalizing symptoms and externalizing problems at each age. Results from the study indicate that youth-reported harsh parenting was a significant predictor of elevated adjustment problems at age 18 but did not predict change over time. Moderation effects of ANS indices and adolescent sex revealed that girls who reported greater harsh parenting and had longer PEPr were at greatest risk for elevated and increasing anxiety and depression symptoms across ages 16 to 18. Girls with low ANS reactivity also had the highest levels of aggressive behavior at age 18. Contrary to expectations, no moderation effects predicted trajectories of externalizing behavior. No direct or moderation effects for parent-reported harsh parenting on adjustment outcomes were found. Overall, results add to our understanding of relations between harsh parenting and maladjustment, particularly when

examining how ANS regulation may contribute to this risk, while highlighting which groups may be especially vulnerable to poor mental health during this developmental period.

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Table of Contents Abstract 2
Acknowledgement
List of Tables
List of Figures
List of Abbreviations
Chapter 1: Introduction
Chapter 2: Literature Review
Adjustment in Adolescence19
Harsh Parenting in Adolescence
Autonomic Nervous System
Moderation by Gender/Sex
Proposed Study47
Chapter 3: Methods
Participants51
Procedures
Measures
Plan of Analysis
Chapter 4: Results
Preliminary Analysis60
Unconditional Growth Models of Adjustment62
Harsh Parenting as Predictor of Adjustment (Aim 1)64
Physiological Reactivity as Moderator (Aim 2)66

Physiological Reactivity and Sex as Conjoint Moderators (Aim 3)	
Chapter 5: Discussion	71
Harsh Parenting and Adjustment	72
Individual Differences in Physiological Reactivity	75
Limitations and Future Considerations	
References	91
Appendix A: Tables and Figures	
Appendix B: Additional Analyses	171
Appendix C: Measures	

List of Tables

Table 1 Descriptive Statistics of Continuous Study Variables
Table 2 Sex and Race Differences in Harsh Parenting, Physiological, and Adjustment among
Adolescents
Table 3 Correlations among Harsh Parenting, Physiological Reactivity and Adjustment for
Adolescents
Table 4 Unconditional Models for Growth in Adjustment in Late Adolescence 142
Table 5 Effects of Youth-Reported Harsh Parenting on Growth in Internalizing Symptoms and
Externalizing Behaviors in Adolescence ($N = 242$)
Table 6 Effects of Parent-Reported Harsh Parenting on Growth in Internalizing Symptoms and
Externalizing Behaviors in Adolescence ($N = 242$)
Table 7 Interaction Effects of Youth-Reported Harsh Parenting and RSAr on Growth in
Internalizing Symptoms and Externalizing Behaviors in Adolescence ($N = 242$)
Table 8 Interaction Effects of Youth-Reported Harsh Parenting and SCLr on Growth in
Internalizing Symptoms and Externalizing Behaviors in Adolescence ($N = 242$)
Table 9 Interaction Effects of Youth-Reported Harsh Parenting and PEPr on Growth in
Internalizing Symptoms and Externalizing Behaviors in Adolescence ($N = 242$)
Table 10 Interaction Effects of Youth-Reported Harsh Parenting, RSAr, and Adolescent Sex on
Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence ($N = 242$)
Table 11 Interaction Effects of Youth-Reported Harsh Parenting, SCLr, and Adolescent Sex on
Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence ($N = 242$)

Table 12 Interaction Effects of Youth-Reported Harsh Parenting, PEPr, and Adolescent Sex on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242) Table 1a Fit Indices for Models examining Youth-Reported Harsh Parenting, RSAr, and Table 2a Fit Indices for Models examining Youth-Reported Harsh Parenting, SCLr, and Table 3a Fit Indices for Models examining Youth-Reported Harsh Parenting, PEPr, and Table 1b Interaction Effects of Parent-Reported Harsh Parenting and RSAr on Growth in Table 2b Interaction Effects of Parent-Reported Harsh Parenting and SCLr on Growth in Table 3b Interaction Effects of Parent-Reported Harsh Parenting and PEPr on Growth in Table 4b Interaction Effects of Parent-Reported Harsh Parenting, RSAr, and Adolescent Sex on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242) Table 5b Interaction Effects of Parent-Reported Harsh Parenting, SCLr, and Adolescent Sex on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

Table 6b Interaction Effects of Parent-Reported Harsh Parenting, PEPr, and Adolescent Sex on
Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence ($N = 242$)
Table 7b Fit Indices for Models examining Parent-Reported Harsh Parenting, RSAr, and
Adolescent Sex
Table 8b Fit Indices for Models examining Parent-Reported Harsh Parenting, SCLr, and
Adolescent Sex
Table 9b Fit Indices for Models examining Parent-Reported Harsh Parenting, PEPr, and
Adolescent Sex

List of Figures

Figure 1 Relations between Youth-Reported Harsh Parenting and Aggressive Behavior
Moderated by Pre-Ejection Period Reactivity151
Figure 2 Relations between Youth-Reported Harsh Parenting and Depression Symptoms
Moderated by Respiratory Sinus Arrhythmia Reactivity and Adolescent Sex
Figure 3 Relations between Youth-Reported Harsh Parenting and Aggressive Behavior
Moderated by Respiratory Sinus Arrhythmia Reactivity and Adolescent Sex
Figure 4 Relations between Youth-Reported Harsh Parenting and Aggressive Behavior
Moderated by Skin Conductance Level Reactivity and Adolescent Sex
Figure 5 Relations between Youth-Reported Harsh Parenting and Depression Symptoms
Moderated by Pre-Ejection Period Reactivity and Adolescent Sex
Figure 6 Relations between Youth-Reported Harsh Parenting and Anxiety Symptoms Moderated
by Pre-Ejection Period Reactivity and Adolescent Sex
Figure 7 Relations between Youth-Reported Harsh Parenting and Rule-breaking Behavior
Moderated by Pre-Ejection Period Reactivity and Adolescent Sex
Figure 8 Relations between Youth-Reported Harsh Parenting and Aggressive Behavior
Moderated by Pre-Ejection Period Reactivity and Adolescent Sex
Figure 1a Conceptual model
Figure 2a Interaction of Youth-Reported Harsh Parenting and Pre-Ejection Period Reactivity at
16 on Aggressive Behavior at Age 18162
Figure 3a Interaction of Youth-Reported Harsh Parenting, Respiratory Sinus Arrythmia
Reactivity, and Adolescent Sex at 16 on Depression Symptoms at Age 18

Figure 4a Interaction of Youth-Reported Harsh Parenting, Respiratory Sinus Arrythmia
Reactivity, and Adolescent Sex at 16 on Aggressive Behavior at Age 18
Figure 5a Interaction of Youth-Reported Harsh Parenting, Skin Conductance Reactivity, and
Adolescent Sex at 16 on Aggressive Behavior at Age 18165
Figure 6a Interaction of Youth-Reported Harsh Parenting, Pre-Ejection Period Reactivity, and
Adolescent Sex at 16 on Rule Breaking Behavior at Age 18166
Figure 7a Interaction of Youth-Reported Harsh Parenting, Pre-Ejection Period Reactivity, and
Adolescent Sex at 16 on Aggressive Behavior at Age 18167
Figure 1b Relations between Parent-Reported Harsh Parenting and Depression Symptoms
Moderated by Skin Conductance Level Reactivity
Figure 2b Relations between Parent Reported Harsh Parenting and Rule-breaking Behavior
Moderated by Skin Conductance Level Reactivity
Figure 3b Interaction of Parent-Reported Harsh Parenting and Skin Conductance Level
Reactivity at age 16 on Depression at Age 18
Figure 4b Interaction of Parent-Reported Harsh Parenting and Skin Conductance Level
Reactivity at age 16 on Rule-breaking Behavior at Age 18

List of Abbreviations

INR = Income-to-needs ratio ANS = Autonomic nervous system functioning PNS = Parasympathetic nervous system SNS = Sympathetic nervous system RSA = Respiratory sinus arrhythmia RSAb = Baseline respiratory sinus arrhythmia RSAr = Respiratory sinus arrhythmia reactivity SCL = Skin conductance level SCLb = Baseline Skin conductance level SCLr = Skin conductance level reactivity PEP = Pre-ejection period PEPb = Baseline pre-ejection period PEPr = Pre-ejection period reactivity BSC = Biological sensitivity to context

SES = Socioeconomic status

Chapter 1: Introduction

Experiences of harsh parenting are associated with a wide range of poor mental health outcomes (Pinquart, 2017; Repetti, Taylor, & Seeman, 2002), most prominently for internalizing (e.g., depression, anxiety) and externalizing problems (rule-breaking, delinquency) (Burnette et al., 2012; Lansford et al., 2011; Weymouth et al., 2016). Investigations of such relations have primarily focused on harsh parenting during childhood (Bubier, Drabick, & Breiner, 2009; El-Sheikh, Keller, & Erath, 2007), pre-adolescence (Burnette et al., 2012; Fletcher et al., 2017), and early adolescence (Diamond, Fagundes, & Cribbett, 2012). However less is known about the impact of harsh parenting on adjustment in later adolescence. The proposed study will examine links between harsh parenting and trajectories of internalizing symptoms and externalizing behaviors from mid-to-late adolescence. Additionally, the study will examine how individual differences in autonomic nervous system activity may confer protective effects or exacerbate risk for youth experiencing harsh parenting. Furthermore, adolescent sex will be examined as a moderator to ascertain if certain youth are more vulnerable or resilient in examined associations between harsh parenting and adjustment.

Poor adjustment, including anxiety, depression, and externalizing problems, afflicts 1 in 5 adolescents from ages 12 to 18 (CDC, 2020). The increased mental health risk during mid-to-late adolescence is a notable public health problem and has been identified as a contributor to widening health and economic disparities seen in adulthood (NIH, 2017). However, not much is known about the conjoint influence of harsh parenting and physiological regulation on trajectories of adjustment symptoms during the period before emerging adulthood.

Adolescence is a critical developmental period during which youth begin to make autonomous decisions concerning their future goals and interpersonal relationships (Erikson,

1968; Patton et al., 2018). Although the influence of parents tends to recede during adolescence, parents continue to play important roles that have long lasting ramifications (Allen et al., 1994; Steinberg, 1990; Weymouth et al., 2016). Harsh parenting may undermine adolescents' sense of self (Silk et al., 2003) and emotion regulation (Davies, Winter, & Cicchetti, 2006; Saritas, Grusec, & Gençoz, 2013), and thereby contribute to maladjustment across adolescence (Di Giunta et al., 2020; Repetti et al., 2002). The proposed study will examine harsh parenting, defined as physical and verbal aggression towards the adolescent, as a predictor of developmental trajectories of internalizing (depression, anxiety) and externalizing problems (rule-breaking, aggression) from mid-to-late adolescence, as well as adjustment symptoms at age 18.

Developmental research has implicated indices of autonomic nervous system (ANS) activity as moderators of relations between harsh family environments and youth outcomes (El-Sheikh & Erath, 2011). Diathesis-stress models of development (Sameroff, 1983) and psychopathology (Burnette & Cicchetti, 2012; Cicchetti & Natsuaki, 2014; Sroufe 2007) indicate that individual and environmental vulnerability factors interact to confer "dual risk" for maladaptive outcomes. Individual differences in physiological regulation may interact with the family environment to predict adjustment problems. For example, optimal ANS activity may provide protection against negative outcomes associated with adverse family environments. Conversely, youth with less optimal ANS functioning may be vulnerable to negative outcomes in the context of family risk. However, few studies have conducted thorough assessments of the moderating role of ANS functioning in links between harsh parenting and developmental trajectories of adjustment in youth. A large proportion of research in early to mid-adolescence has examined only one branch of the ANS (Benito-Gomez, Fletcher, & Buehler, 2019; Erath, El-

Sheikh & Cummings, 2009; Fletcher et al., 2017). Similarly, many of these studies of ANS moderation have primarily focused on externalizing behavior problems rather than internalizing symptoms (Bubier et al., 2009; Erath, et al., 2011; Huffman, Oshri, & Caughy, 2020; Sijtsema et al., 2013).

The ANS is comprised of two main branches: the parasympathetic nervous system (PNS) regulates processes of rest and digestion, and the sympathetic nervous system (SNS) controls fight, flight, or freeze responses to stressors in the environment (Porges, 2007). According to the Polyvagal Theory (Porges, 2007), respiratory sinus arrhythmia (RSA) represents PNS control on the heart through the vagus nerve. The vagus nerve exerts an inhibitory influence, or a "brake," that allows the body to maintain calm by reducing physiological arousal through slower heart rate in normal, non-threatening situations. In response to stress, decreases in RSA (vagal withdrawal) generally reflect flexible and adaptive reactions that are associated with positive outcomes for children, such as lower levels of internalizing symptoms and externalizing behaviors (Beauchaine, 2001; Calkins, Graziano, & Keane, 2007; Graziano & Derefinko, 2013). Associations in early to mid-adolescence have been less clear; both increased RSA (vagal augmentation) and decreased RSA (vagal withdrawal) have been linked to internalizing symptoms (Benito-Gomez et al., 2019; Fletcher et al., 2017) as well as externalizing behavior (Huffman et al., 2020; Sijtsema et al., 2013) in the context of harsh or stressful family environments.

The SNS is the branch of the ANS primarily involved in allocating and directing metabolic resources for responses to stress (Boucsein, 1992; Porges, 2007). SNS activity is often indexed by electrodermal responses (skin conductance) or cardiac responses (pre-ejection period). Skin conductance (SCL) is thought to reflect anxiety and inhibition, whereas pre-

ejection period (PEP) has been theorized to indicate approach motivation and impulsivity (Beauchaine et al., 2007; Beauchaine et al., 2001). Responses to stress indexed by these SNS markers may exacerbate or attenuate the effects of harsh parenting on youth well-being (Brenner & Beauchaine, 2011). Empirical research examining interactions between harsh parenting and SNS has mostly been conducted in younger developmental age groups (i.e., children, preadolescents), and associations have been reported for both greater SCL reactivity (Diamond et al., 2012) and lower SCL reactivity (Kochanska et al., 2015) in relation to externalizing behavior.

Studies examining ANS functioning as a moderator of links between of harsh parenting and adjustment have focused on externalizing problems. Huffman and colleagues (2020) utilized RSA reactivity (RSAr) and PEP reactivity (PEPr), whereas Erath and colleagues (2009, 2011) have examined SCL reactivity (SCLr) as a moderator of those relations in diverse community samples of pre-adolescents. These studies found that greater RSAr (withdrawal) and blunted lower SCLr in response to lab stressors exacerbated risk for externalizing problems (Erath et al., 2009), delinquency, and aggressive behaviors (Huffman et al., 2020) in the context of harsh parenting. Longitudinal associations also revealed that lower SCLr exacerbated the risk for stable elevated externalizing problems (Erath et al., 2011). The role of PEPr is not well-understood in the context of harsh family environments; no known studies have examined PEPr as a moderator of associations between harsh parenting and adjustment in adolescence. Thus, a more comprehensive understanding of ANS functioning in the context of harsh parenting is needed. The second aim of the proposed study is to examine parasympathetic (RSAr) and sympathetic (SCLr, PEPr) activity as moderators of relations between harsh parenting and trajectories of adjustment problems across adolescence.

In addition to examining ANS as a moderator, some of the aforementioned studies sought to ascertain if associations between harsh parenting, ANS functioning, and adjustment differed by youth sex/gender. No differences were noted for RSAr or PEPr by Huffman et al. (2020); however, boys with lower SCLr were particularly vulnerable to greater externalizing behavior problems in the context of harsh parenting (Erath et al., 2009). In addition to the scant evidence regarding sex-related effects in these associations, many studies have reported that rates of adjustment problems differ across boys and girls during adolescence (Collishaw 2015; Thapar et al., 2012). This highlights the importance of examining sex as an additional moderator of the associations between harsh parenting and adjustment among adolescents.

In summary, the proposed study will examine harsh parenting as a predictor of multiple dimensions of internalizing symptoms (i.e., depression, anxiety) and externalizing behavior (i.e., rule-breaking, aggression) trajectories across ages 16, 17, and 18 (Aim 1). Additionally, the proposed study will examine RSA, SCL, and PEP reactivity to a lab challenge (star-tracer) as moderators of associations between harsh parenting and multiple indicators of internalizing and externalizing outcomes (Aim 2). Finally, the study will explore sex as an additional moderator of these relations to help elucidate which youth may be at greatest risk for adjustment problems (Aim 3).

Chapter 2: Literature Review

Adjustment in Adolescence

Adjustment problems including internalizing symptoms (e.g., anxiety, depression) and externalizing behaviors (e.g., rule-breaking, aggression, and hostility) increase across early to mid-adolescence (Collishaw, 2015), however less is known about changes occurring during mid-to-late adolescence (age 15 to 18), especially in the context of family risk. Recent statistics in the United States indicate that across adolescence, 11% of youth report clinical levels of anxiety and 6% report depression (CDC, 2020). The rates of depression and anxiety symptoms are even higher among mid-to-late adolescents of whom 17% report depressive symptoms and 32% indicate anxiety symptoms in the past year (NIMH, 2017). Furthermore, the highest rates of depression among adults occur during the ages immediately following adolescence (18 – 25 years), indicating a need to examine adjustment from mid to late adolescence (NIMH, 2017; Galambos, Barker, & Krahn, 2006).

Despite overlap in depression and anxiety symptoms, differences are noted in their prevalence and symptom presentation in adolescents (Krueger, 1999; Merikangas et al., 2010). Negative affect is a common trait shared by depression and anxiety; however, each dimension of internalizing is distinctively characterized (Clark & Watson, 1991). Depression symptoms are particularly identified through anhedonia (lack of pleasure) whereas overarousal and preoccupation are more distinctive to anxiety. An examination of change in these core dimensions of depression and anxiety revealed different trajectories of these symptoms that are often missed in composite internalizing scores (Conway et al., 2017). Although both depression and anxiety have long-reaching consequences on individual health and well-being, specific aspects of their symptomology may confer unique risk for detrimental outcomes. Furthermore,

stressors experienced during adolescence may uniquely influence the trajectories of these internalizing problems. Further research is needed to better understand their distinct trajectories in late adolescence and emerging adulthood (Merikangas et al., 2010).

In addition to internalizing symptoms, externalizing behavior problems are also elevated across this developmental period (Tremblay, 2010). Two distinct dimensions of externalizing behavior have been identified, *aggressive behavior* and *rule-breaking/delinquent behaviors* (Achenbach & Ruffle, 2000; Prinzie, Onghena, & Hellinckx, 2006; Reef et al., 2010), each having a particular etiology and trajectory across development (Bongers et al., 2004; Evans et al., 2019; Stanger, Achenbach, & Verhulst, 1997). Specifically, risk-taking and rule-breaking behaviors have been noted to peak during mid-to-late adolescence (Steinberg, 2010). Some forms of aggression (i.e., indirect) have been documented to increase during early adolescence (Connor, 2004), although trajectories into later adolescence have been less clear (Archer & Coyne, 2005; Orpinas, McNicholas, & Nahapetyan, 2015; Vaillancourt & Farrell, 2021). Frequently, the literature does not differentiate between various manifestations of externalizing behaviors, and examination of both rule-breaking and aggressive behavior may help explicate the risk associated with these developmental outcomes in the context of family risk.

Increases in internalizing symptoms across adolescence are associated with greater risk for mental health disorders during the transition to adulthood (Johnson et al., 2018) and poor social relationships in adulthood (Schwartz-Mette & Rose, 2016). Similarly, externalizing behavior problems have been identified as risk factors for the development of later psychopathology (Dishion & Patterson, 2016; Mullin & Hinshaw, 2007; Yap, Allen, & Sheeber, 2007), as well as difficulties with educational and occupational attainment (Carter, 2019; Tanner, Davis, & O'Grady, 1999). In addition, aggressive behaviors during adolescence have been

associated with later engagement in violent or criminal behavior (Oshri et al., 2018; Patterson, 2002; Reef et al., 2010). Thus, adjustment problems in adolescence forecast mental health problems in adulthood.

The family environment influences the development of psychopathology in youth (e.g., Cole et al., 2002; Repetti et al., 2002). For example, families characterized by interparental conflict (Buehler & Gerard, 2002; Davies et al., 2016), parent-adolescent conflict (Branje et al., 2010; Weymouth et al., 2016), and poor cohesion (White, Shelton & Elgar, 2014) are key contributors to growing internalizing and externalizing problems across early to late adolescence. Harsh parenting also contributes to adjustment problems during this critical period (Bender et al., 2007). However, it is less clear how harsh parenting influences trajectories of specific internalizing and externalizing problems in mid-late adolescence.

Harsh Parenting in Adolescence

Prevalence and Terminology

Although adolescence is typically characterized as a period when parental influences on youth wane as peer influences increase, parents continue to play a necessary role in positive socio-emotional development during adolescence (Patterson, DeBaryshe, & Ramsey, 2017; Sarıtaş et al., 2013). Parent-adolescent interactions often involve more conflict and negative affect during early to middle adolescence (ages 10 - 15) compared to childhood, though improvements have been noted in mid-to-late adolescence (16 - 19 years; Brouillard et al., 2019; Shanahan, McHale, Crouter, et al., 2007). In these later years of adolescence, prior to the transition to adulthood, youth report valuing their relationships with their parents (Tsai, Telzer, & Fuligini, 2013). At the same time, they continue to report negative interactions and conflict with their parents (De Goede, Branje, & Meeus, 2009; Shanahan, McHale, Osgood, et al, 2007;

Steinberg & Morris, 2001; Van Doorn, Branje & Meeus, 2011). For example, 90% of parents in a nationally representative sample endorsed the use of verbal and physical disciplinary tactics with children and adolescents (Straus & Field, 2003). Compared to childhood, parents endorse greater use of verbal and psychological conflict tactics (50%) and a reduction in physical aggression with their adolescents.

The literature examining harsh parenting in adolescence has been somewhat disjointed due to the use of multiple measures and terminology. Harsh parenting is often defined as parental physical aggression (e.g., slapping, spanking) and verbal and psychological aggression (e.g., shouting, insulting) directed at the child and intended to be coercive in response to the child's behavior (Erath et al., 2009; Shaw et al., 2003). Across studies, terminology has varied when describing these types of behaviors, at times referred to as "parent-child hostility," "parent-child conflict," or simply an examination of a specific dimension of coercive parenting such as verbal aggression or physical discipline (e.g., corporal punishment, spanking). A recent meta-analysis examining harsh parenting used the term "hostility" and operationalized hostility as "overt behaviors and expression that includes arguing, angry comments, contempt, yelling, name calling, or physical aggression;" this term is akin to the aforementioned definition of harsh parenting (Weymouth et al., 2016). Despite differences in terminology, the review of literature below will refer to these aggressive or hostile parenting behaviors collectively as "harsh parenting" unless otherwise specified.

Harsh parenting in childhood and adolescence has been assessed using a variety of methods, including observation of dyadic interactions between parent and child, but has most often been assessed using parental and/or youth report. A widely used and validated measure is the parent-child version of the Conflict Tactics Scale (Straus et al., 1998), which assesses the

frequency of physical and verbal/psychological aggression. Often, researchers use parent selfreports of their harsh parenting; however, self-reports may lead to underreporting due to concerns with social desirability. Adolescent reports provide important information about aggression directed at them, including accounts of harsh parenting from non-residential parents.

Harsh Parenting and Adjustment: Theoretical and Conceptual Approaches

Mid to late adolescence is a particularly sensitive period where youth are frequently engaged in interactions and environments that shape trajectories of achievement, health, and well-being (Patton et al., 2018). A salient developmental task for youth is fostering identity and a sense of self while establishing their needs for independence and autonomy (Erikson, 1968; Deci & Ryan, 2000). Harsh parenting behaviors may undermine these developmental tasks (Erikson, 1968; Steinberg, 1990) and interfere with self-regulation (Collins & Steinberg, 2007)

Self-determination theory posits that environments and relationships can shape fulfillment of basic psychological needs, including perceived autonomy, competence, as well as relatedness (Deci & Ryan, 2000; Erikson, 1968). Parenting that is coercive and punitive may counteract progress toward meeting these developmental needs and limit opportunities to develop autonomy and connectedness with others (Soenens et al., 2008). Furthermore, disruption of these needs during late adolescence would be detrimental to emotional well-being and adjustment in young adulthood (Vansteenkiste & Ryan, 2013).

Harsh parenting may also convey negative self-evaluative messages and trigger rumination (Bruce et al., 2006; Deci & Ryan, 2000). Indeed, physical aggression assessed via self-report on the Conflict Tactics Scale has been associated with internalizing and externalizing behaviors (Bender et al., 2007; Tang et al., 2018). Based on observational assessments of 16year-olds and their parents, Bender and colleagues (2007) reported that youth who reported

experiencing greater physical aggression were less likely to express autonomy during interactions. Similarly, in large scale study with Chinese 9th graders (*N*= 890), Tang and colleagues (2018) found indirect associations between youth reported harsh parenting and depression symptoms via negative cognitions about oneself. Similarly, parent verbal aggression is also disruptive of autonomy through intrusion on adolescents' thoughts and emotions (Soenens & Vansteenkiste, 2010). Acts of verbal or psychological aggression contribute to internalizing problems via negative views of the self (Miller-Perrin, Perrin, & Kocur, 2009), as well as a more self-critical cognitive style (Sachs-Ericsson et al., 2006).

In addition to undermining autonomy development, harsh parenting may affect youths' internal working models of self and relationships (Bowlby, 1973). Harsh and negative treatment by caregivers is associated with poor internal conceptualization of the self, including diminished self-worth and low self-esteem (Allen et al., 1994; Kuhlberg, Pena, & Zayas, 2010; Silk et al., 2003). Further, the use of harsh discipline and aggression may contribute to hostile relationship schemas (Bowlby 1973; Dodge & Pettit, 2003), particularly regarding the parent-adolescent relationship. Such experiences may contribute to suspicion and distrust of others, leading to the use of aggressive behaviors in parent-youth interactions and engagement in various rule-breaking behaviors and delinquency (Dodge, Bates, & Pettit, 1990). In a longitudinal study examining pre-adolescent African American youth, verbal aggression was associated with greater negative views on relationships, which in turn were associated with more delinquent behavior two years later (Evans, Simons, & Simons, 2012), indicating the long-term effect that harsh parenting may have on problematic externalizing behaviors via negative attributions.

Furthermore, youth may replicate similar aggressive behaviors in peer and romantic relations. Social learning theory postulates that experiences of harsh parenting will reinforce the

use of aggressive tactics (Bandura, 1977). Indeed, experiences of aggressive verbal and physical discipline from parents are associated with exacerbation of anger and hostility towards others (Chang et al., 2003; Patterson, 2002), as well as replication of such behaviors in other interpersonal contexts (Gámez -Guadix et al., 2010; Jouriles et al., 2012; Sarıtaş et al., 2013). Harsh parent-child interactions may demonstrate to youth that aggressive and controlling tactics are an effective way to change the behaviors of others (Simons, Lin, & Gordon, 1998). Early work with children has found that negative and coercive parent-child interactions are associated with children seeking physically aggressive responses to solutions where there is conflict, perhaps to control their environment (Graziano, 1994).

Harsh and aggressive parenting has also been linked to emotion regulation difficulties in youth (Morris et al., 2017; Morris et al., 2007; Saritas et al., 2013). Coercive and psychologically aggressive parenting was associated with poor adjustment for adolescents via their difficulties in regulating anger (Cui et al., 2014). Such parental hostility uniquely predicts youths' emotion dysregulation and adjustment difficulties, even after accounting for positive parenting behaviors (e.g., warmth; Hoeve et al., 2009; Valiente et al., 2004). Furthermore, difficulties in emotion regulation have been linked to increases in rule-breaking and conduct problems (Walton & Flouri, 2010), aggression (Cui et al., 2014), as well as greater internalizing problems (Soenens et al., 2008).

Harsh Parenting and Adolescent Adjustment

Extensive empirical research has established strong links between harsh parenting and adjustment outcomes, particularly in childhood and early adolescence. Through cross-sectional and longitudinal designs, harsh parenting is associated with internalizing and externalizing problems (Erath et al., 2009). These adjustment problems, particularly anxiety and depression

symptoms, have also been reported in early adolescence (Thapar et al., 2012), but less is known about the influence of harsh family environments in the later years of adolescence.

Internalizing Symptoms. A review of studies found that harsh parenting is linked to low self-confidence, anxiety symptoms, and negative emotionality during childhood and adolescence (Barber & Harmon, 2002). A recent meta-analysis across 52 studies that utilized a variety of terminology and methods of assessment also reported associations between parents' harsh and controlling behavior and adolescents' depression and anxiety symptoms (Weymouth et al., 2016). A large-scale study from Tang and colleagues (2018) found that adolescent-reported experiences of harsh parenting (verbal and physical) predicted greater depression symptoms among Chinese 9th graders. Further, the use of physical aggression (e.g., hitting, slapping, pushing/grabbing) was associated with adjustment problems among 16-year-olds (Bender et al., 2007). Bender et al. (2007) noted that greater use of harsh physical aggression by both mothers and fathers predicted greater depression symptoms above and beyond other parenting factors (i.e., attachment, affection).

In comparison to cross-sectional studies, fewer longitudinal investigations have ascertained links between harsh parenting and adolescent internalizing symptoms. A metaanalysis examining parenting behavior reported that harsh control was associated with increases in internalizing symptoms in children and adolescents over time (Pinquart, 2017). In addition, a cross-cultural study by Di Giunta and colleagues (2020) examined 1,298 adolescents and their parents in 9 countries. They found that youth had greater internalizing problems at age 15 when they had parents who used harsh parenting practices and disciplinary measures the previous year. Similarly, a longitudinal and cross-lagged study examining 13-year-old youth also indicated that parent-reported verbal aggression (both mothers and fathers) was associated with greater

depression symptoms in youth one year later (Wang & Kenny, 2014). These limited longitudinal studies have primarily examined harsh parenting as a predictor of outcomes across a single year. Less is known about how harsh parenting may influence trajectories of internalizing symptoms over a longer period. Additionally, many studies have established links with depression symptoms; however, associations between harsh parenting and adolescent anxiety are less clear.

Externalizing Behaviors. Numerous studies have linked harsh parenting and externalizing behavior problems cross-sectionally and longitudinally. Such associations have been noted in specific developmental periods (e.g., adolescence), as well as across development, most notably from childhood to adolescence (Deardorff et al., 2013; Lansford et al., 2011). Cross-sectional studies in adolescence have reported that harsh verbal aggression perpetrated by both mothers and fathers against 16-year-olds was associated with a variety of externalizing behavior problems (Bender et al., 2007). Similar links were reported in another study utilizing multiple informants in a large sample of 857 Chinese early to mid-adolescents (12 – 16 years). Specifically, Wang (2019) found that parent-reported harsh parenting (cross parent reports of physical and verbal aggression) was associated with greater peer-endorsed aggression, particularly for harsh parenting from fathers even after accounting for parental warmth.

Longitudinal associations between experiences of harsh parenting in childhood and later adjustment have been reported for a variety of externalizing behaviors in pre-adolescents (ages 10 to 12; Erath et al., 2011; Hinnant, Erath, & El-Sheikh, 2015; Kingsbury et al., 2020; Pettit et al., 2001). These longitudinal links are also evident in later years of adolescence. A cross-cultural study reported that harsh parenting at age 14 was associated with severe externalizing problems one year later, even when controlling for earlier externalizing behaviors (Di Giunta et al., 2020). Among Mexican-origin girls, harsh parenting during middle school years (7th grade) was

associated with increased externalizing behavior in 10th grade when controlling for earlier externalizing behaviors (Deardorff et al., 2013). In a large study examining 976 families, Wang and Kenny (2014) found that both maternal and paternal reports of harsh verbal aggression when youth were age 13 was associated with increased conduct behavior problems at age 14 using a longitudinal cross panel study design. Similarly, a study of African American youth found that experiences of harsh parenting during pre-adolescence (ages 11 to 13) were associated with greater anger during late adolescence (age 16 to 18) and worse physical health (Brody et al., 2013).

Others have examined longitudinal process models to examine links between harsh parenting in pre-adolescence and externalizing problems. For example, harsh parenting during childhood (age 10) was associated with externalizing behavior problems in pre-adolescence (age 12), which later predicted anti-social and rule-breaking behaviors in mid-adolescence at age 15 (Burnette et al., 2012). These studies highlight the long-term consequences of negative and harsh parenting on adjustment, as well as help elucidate the processes and mechanisms that are involved. However, thus far, no known studies have examined how harsh parenting in adolescence may contribute to growth or trajectories of externalizing behavior across later periods of adolescence.

Despite evidence linking harsh parenting and negative socioemotional outcomes, not all youth who experience such harsh parenting or negative family environments develop internalizing or externalizing problems (Yap et al., 2014). Developmental perspectives suggest that individual differences in biological and regulatory processes also influence socioemotional functioning (El-Sheikh, Keiley, & Hinnant, 2010; Lopez-Duran, Kovacs, & George, 2009; Steinberg & Avenevoli, 2000). Dual-risk or diathesis-stress models indicate that youth with

biological vulnerability, such as poor physiological regulation, are more likely to experience negative outcomes in harsh or stressful environments (Sameroff, 1983; Sroufe, 2007). More specifically, conceptualizations and empirical evidence suggest that ANS activity can moderate relations between family conflict and adjustment in children and adolescents (El-Sheikh & Erath, 2011). Several studies have found markers of the ANS to moderate associations between harsh parenting and adjustment outcomes (Erath et al., 2011; Fletcher et al., 2017; Huffman et al., 2020). A discussion of these ANS markers and their role as potential moderators in links between harsh parenting and internalizing and externalizing behaviors follows.

Autonomic Nervous System

The autonomic nervous system (ANS) is a regulatory system that controls the body's internal organs and metabolic resources for homeostatic functioning, as well as the mobilization of resources during stress responses. As indicted previously, the ANS is comprised of two main branches – parasympathetic nervous system (PNS) and sympathetic nervous system (SNS) – that operate in conjunction to allow for cardiovascular, endocrine, and electrodermal systems to respond to stress. Both divisions of the ANS are significant contributors to emotion regulation and stress response systems (Gentzler et al., 2009; Graziano & Derefinko, 2013) and related to a broad range of symptoms of psychopathology (Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007; Porges, 2007).

Social-ecological experiences, particularly those with stressful stimuli, are known to produce dynamic ANS responses through innervation of cardiac and electrodermal responses in order to self-regulate (Bell & Deater-Deckard, 2007). According to the Polyvagal theory (Porges, 2007), the ANS responds to stressful stimuli in the environment in a hierarchical manner. First, the ANS relies primarily upon the more recently evolved myelinated vagus nerve of the PNS for

an energy efficient and rapid mobilization of resources. If the response is insufficient the more primitive SNS is further activated, requiring greater metabolic resources (Porges, 2007). Though these processes typically operate in a hierarchical manner when reacting to stressors in the environment, various response patterns are possible in order to provide flexible and adaptive approaches in a dynamic stress environment (Porges, 2007). These processes are detailed below.

Parasympathetic nervous system. Polyvagal theory posits that the ventral vagus nerve exerts an inhibitory influence on the heart, or operates as a "brake," allowing the body to maintain calm through a slow heart rate in non-threatening situations. This basal functioning is referred to as vagal tone, and a higher level of vagal tone is commonly conceptualized as capacity for emotion regulation (Beauchaine, 2015; Porges, 2007). Respiratory sinus arrhythmia (RSA) is a non-invasive marker of vagal tone that assesses variations in heart rate across spontaneous breathing cycles.

When threatening or stressful stimuli are perceived in the environment, the vagal "brake" is withdrawn, allowing for increased heart rate and mobilization of metabolic resources (e.g., increased blood flow, oxygen) to facilitate an active cognitive and emotional response. Withdrawal of vagal tone is marked by decreases in RSA from baseline in response to a stressor (greater RSAr). Increases in vagal tone during a stressor, or vagal augmentation, are characterized by a slowing of heart rate (lower RSAr). Vagal augmentation may reflect disengagement and compromise effective coping with environmental demands and stressors (Moore & Calkins, 2004). Thus, basal RSA levels are indicative of regulatory and attentional capacity and maintenance of regulation, whereas RSA reactivity is indicative of regulatory activity and ability to actively cope and engage with environmental demands (Porges, 2007).

Higher levels of baseline RSA, or vagal tone, as well as greater vagal withdrawal in response to stressors are generally associated with lower levels of internalizing symptoms and externalizing behavior (Beauchaine, 2001; Graziano & Derefinko, 2013), better emotion regulation (Appelhans & Luecken, 2006), and improved cognitive performance (Forman-Alberti & Hinnant, 2016). However, excessive vagal withdrawal may indicate dysregulated PNS activity (Zisner & Beauchaine, 2016). Indeed, links between RSAr and internalizing problems have been found, yet results have varied across clinical (Pang & Beauchaine, 2013) and community samples (El-Sheikh, Keiley, Erath, & Dyer, 2013). Lower vagal withdrawal is typically associated with higher internalizing problems in community samples (Graziano & Derefinko, 2013). Likewise, blunted vagal withdrawal to stressors has been consistently linked to greater externalizing problems in community samples (Beauchaine, 2001; Calkins et al, 2007; Graziano & Derefinko, 2013). For example, lower levels of RSA withdrawal in response to challenging math-based tasks among adolescents was associated with higher externalizing behavior (Busso, McLaughlin, & Sheridan, 2017).

Sympathetic Nervous System. The SNS serves as a complementary branch to the PNS by enabling fight, flight, or freeze reactions, which may be as characterized as "approach and avoidance" responses. Activation of the SNS involves a complex cascade of energy-intensive responses that mobilize one's approach or avoidance to stressors or challenges (Beauchaine, 2001). In reaction to stress, the SNS produces physiological responses such as increased cardiac output and changes in oxygen and blood flow to key organs and muscles (Sapolsky, Romero, & Munck, 2000). Individual differences in SNS activation have been found; over-arousal and under-arousal in response to stressors have been implicated in maladaptive outcomes (Lazarus, Speisman, & Mordkoff, 1963).

Skin conductance level (SCL) refers to electrodermal activity produced by sweat glands that are innervated by cholingeric fibers connected to the SNS and independent of PNS input (Boucsein, 1992; Fowles, 1988). SCL reactivity (SCLr) is an indicator of the Behavioral Inhibition System which is a neurophysiological motivation system activated during situations of threat and negative consequences (Beauchaine, 2001; Gray, 1987). Blunted SCL reactivity (decreases or small increases in response to stressor from baseline) may be indicative of fearlessness and poor inhibitory control, whereas elevated SCL reactivity may be reflective of anxiety and passive avoidance in threatening or risky environments (Boucsein, 1992).

Fearlessness and disinhibition are associated with reduced sensitivity to punishment, which may contribute to individual differences in externalizing behaviors including aggression and conduct problems (Beauchaine et al., 2007; Raine, 2002). Lower SCLr is an index of underarousal that is often linked to externalizing behaviors during childhood and adolescence (Beauchaine et al., 2001; Gregson, Tu, & Erath, 2014; Hinnant et al., 2016; Murray-Close, 2013). Similarly, blunted SNS reactivity via lower SCLr is related to sensation seeking, rulebreaking and delinquency among youth (Ortiz & Raine, 2004; Sijtsema et al., 2010). These associations exist across the developmental spectrum persisting into adolescence and adulthood where lower SCLr is linked to antisocial behaviors, conduct problems, and psychopathy (Fung et al.,2005; Gatzke-Kopp et al., 2002; Lorber, 2004; van Bokhoven et al., 2005). Relations between blunted SNS activity and internalizing problems are less well understood.

In contrast, high SCLr has been conceptualized as a marker of sensitivity to environmental or stressful challenges and has been found to be associated with fearfulness (Fowles, Kochanska, & Murray, 2000) and inhibition (Matthys, van Goozen, & van Engeland, 2004). Thus, higher SCLr responses have been found among youth with internalizing problems

(Beauchaine, 2001) and anxious arousal (Weems et al., 2005), as well as anger and aggression (Hubbard et al., 2002; Hubbard et al., 2004). Greater SNS activation via higher SCLr may reflect emotional arousal linked to fear and anxiety (Beauchaine, 2001).

Pre-ejection period (PEP) is a marker of beta-adrenergic SNS influences on the heart (Cacioppo, Uchino, & Berntson, 1994; Sherwood et al., 1986), defined as the time (in milliseconds) between the onset of the heart beat and ejection of blood out of the left ventricle into the aorta (Berntson et al., 2004. Sherwood et al, 1990). This established cardiac measure is indicative of the sympathetic influence on the myocardium and is indicated by the shortening or lengthening of the pre-ejection period (Imrich et al., 2008). Shorter PEPb and greater PEPr (shortening) during challenges are indicative of greater sympathetic activity, whereas longer PEPb and lower PEPr (lengthening) represent a blunted sympathetic response. PEPr is an indicator of the Behavioral Approach System, a neurophysiological system that reflects approach motivation and impulsivity to seek rewards (Beauchaine et al., 2007; Brenner, Beauchaine, & Sylvers, 2005).

Blunted SNS activity via longer PEPb and lower PEP reactivity (lengthening) during reward challenges has been associated with conduct problems (Beauchaine et al., 2007; Crowell et al., 2006) and delinquent behaviors (Beauchaine et al., 2007) across developmental age groups (e.g., elementary school, adolescents), mostly commonly among youth with clinical levels of externalizing behavior problems (Beauchaine et al., 2007; Crowell et al., 2006; Shannon et al., 2007). Similar findings have been detected in a community sample of youth in which blunted SNS activation via lengthening of PEPr was linked to externalizing behavior problems (Boyce et al., 2001). However, greater SNS reactivity (shortening PEPr) has also been linked to aggressive and conduct problems among preschoolers (Beauchaine et al., 2013), as well as escalation of

alcohol and substance use among middle-schoolers (Brenner & Beauchaine, 2011). Little research has examined the role of PEP in relation to internalizing problems. Individuals who express low affect, motivation, and anhedonia associated with depression may be insensitive to rewards (Whitton, Treadway, & Pizzagalli, 2015) and appetitive stimuli (Fowles, 1988), and thus may exhibit lengthening of PEP in response to challenge. However, studies examining depression have found inconsistent results in youth (Ahles, Mezulis, & Crowell, 2017; Boyce et al., 2001; Brinkmann & Franzen, 2015).

Inconsistencies among relations between high and low SNS reactivity and externalizing problems may be due to the specific dimensions of externalizing behaviors that are linked with over- and under-arousal (Beauchaine, 2001; Brenner at al., 2005). For example, greater reactivity via shortening of PEPr (overarousal) may be associated with reactive dimensions of externalizing problems whereas blunted SNS via lengthening of PEPr (under arousal) may be linked to more proactive aspects of externalizing behavior that provide stimulation. The type of task used to elicit reactivity may also contribute to the lack of clarity across findings. Tasks with social and relational components such as Trier Social Stress Task or conflict tasks, may evoke different levels of reactivity compared to frustrating or challenging cognitive tasks such as memory (e.g., number matching) or arithmetic tasks. Additionally, much of the extensive work to understand SNS activity has focused on youth with diagnosed psychopathology or clinical levels of adjustment problems (Beauchaine, 2015; Beauchaine, et al., 2001; Boyce et al., 2001; Brenner & Beauchaine, 2011; Shannon et al., 2007), as well as primarily involve younger developmental age groups, such as children and pre-adolescents (Ahles et al., 2017; Boyce et al., 2001; Zisner & Beauchaine, 2016).

The prevailing and dominant theory concerning the role of physiological regulation in stressful environments is heavily influenced by transactional/dual-risk models (Sameroff, 1983) and diathesis-stress models of psychopathology (Burnette & Cicchetti, 2012; Cicchetti & Natsuaki, 2014; Zuckerman, 1999). Such models propose that some individuals are more likely to experience adverse effects from contextual or environmental stressors due to a "vulnerability," such as dysregulation in stress responding. The compounding negative effects of environmental risk and individual vulnerability would confer heightened, or dual risk, for poor outcomes, whereas those without the individual vulnerability or those with the individual vulnerability within a more supportive environment would have better outcomes.

Despite significant evidence for this theory in relation to ANS functioning (for review, see Calkins, Propper, & Mills-Koonce, 2013; El-Sheikh & Erath, 2011), others have proposed alternate models such as differential susceptibility (Belsky & Pluess, 2009) and biological sensitivity to context (BSC; Boyce & Ellis, 2005; Ellis & Boyce, 2008). Both models contend that neurobiological factors moderate relations between environment and developmental outcomes (Ellis et al., 2011). Research focusing in ANS functioning and family environments has relied upon BSC theory. This theory postulates that physiological stress responses are *plastic* and individuals may become more sensitive to changes in stress or support in their environment, such that highly reactive youth would have the worst outcomes in adverse conditions or the best outcomes in a positive and supportive environment. Some of the studies presented in the following section support the BSC perspective concerning the moderating role of ANS in the family environment (i.e., Bubier et al., 2009; Huffman et al., 2020; Obradović, Bush & Boyce, 2011). Although the current study will not fully compare these theoretical perspectives, the results will provide needed evidence related to the theories during late adolescence.

The Moderating Role of Autonomic Nervous System Activity

Family environments characterized by harsh parenting and hostility represent stressful contexts that undermine and disrupt emotion regulation processes (Morris et al., 2007; Saritas et al., 2013) and are associated with psychopathology (Weymouth et al., 2016). Physiological reactivity, to a greater degree than baseline activity, reflects dynamic stress responsivity, emotion arousal, and self-regulation that may occur in the context of harsh parenting (Beauchaine & Thayer, 2015; Sturge-Apple et al., 2012). Thus, the use of physiological reactivity markers can assist in the identification of processes that promote resilience in stressful contexts (Obradović, 2012). Frequent and unmitigated activation of regulatory systems in response to stress can produce dysregulation via blunted or volatile response patterns and contribute to wear and tear on multiple systems (i.e., allostatic load; McEwen, 1998, 2000). Modulation of physiological stress responses in stressful environments is necessary for adaptive and optimal outcomes, whereas dysregulation may cascade into adverse outcomes such as psychological distress and poor physical health (Gunnar & Vasquez, 2006; Repetti et al., 2002; Seeman et al., 2010). Indeed, a growing body of research has identified individual differences in ANS reactivity as protective and vulnerability factors for youth in relations between adverse family environment and well-being (El-Sheikh & Erath, 2011; Obradović et al, 2011).

Harsh Parenting and PNS.

Links between harsh parenting, PNS functioning, and internalizing problems are scant and, at times, inconsistent. Though greater vagal withdrawal has been linked with lower internalizing symptoms, studies examining harsh parenting have not always found moderating effects of vagal reactivity. Among children, RSAr did not moderate relations between harsh parenting (physical or verbal aggression) and internalizing symptoms (Whitson & El-Sheikh,

2003). Among pre-adolescents, studies are limited; however, vagal withdrawal (greater RSAr) has been found to protect against internalizing problems. One study noted that those with RSA augmentation to a social stress task reported the highest levels of internalizing behaviors when they experienced greater family stress, including harsh parenting, interparental conflict, and cumulative family risk (Benito-Gomez et al., 2019). Similarly, another study reported that greater RSA was protective against depressive symptoms for boys, but not girls, in the context of harsh physical discipline (Fletcher et al., 2017). The same study, however, also noted disparate findings where greater RSA withdrawal was a risk factor for depression symptoms in the context of greater maternal psychological control for girls, and that greater withdrawal was a protective factor for boys.

Existing research on related constructs of interparental or marital conflict have noted the protective effects of RSA withdrawal in relation to internalizing symptoms. Greater RSA withdrawal has been frequently linked with fewer depression and anxiety symptoms among children (El-Sheikh, Harger, & Whitson, 2001; El-Sheikh & Whitson, 2006; Katz & Gottman, 1997; Whitson & El-Sheikh, 2003) and young adolescents (Khurshid, Peng & Wang, 2019) who experienced greater interparental discord and conflict. Evidence suggests that greater RSA withdrawal during parent-child interaction is protective for youth against depression (Tu, Li, & Cohen, 2019), and similar associations with internalizing outcomes have been noted in the context of marital conflict (El-Sheikh & Whitson, 2006). Similarly, adolescents with blunted RSAr and greater childhood adversity had greater depression scores (McLaughlin, Alves, & Sheridan, 2014). No known studies have ascertained such associations between harsh parenting, RSAr, and internalizing symptoms in mid-to-late adolescence. The limited research has focused primarily on depression symptoms or aggregated internalizing symptoms, further highlighting

the limited understanding of RSAr as a moderator in relations between harsh parenting and anxiety.

A small number of studies have investigated PNS activity as a moderator of associations between harsh parenting and externalizing behavior during adolescence, and even fewer have utilized longitudinal designs. The direction of effects has been somewhat mixed, potentially due to differences in study populations (community or clinical) as well as the developmental period. A recent study of 101 racially diverse preadolescents (9- to 12-year-olds) investigated RSA reactivity as a moderator of relations between observed harsh parenting and mother reported externalizing outcomes (delinquency and aggressive behaviors) across one year (Huffman et al., 2020). Adolescents with higher levels of RSA withdrawal to a social stress task had worse externalizing behavior when they experienced greater levels of harsh parenting.

Studies examining related family stress contexts have found blunted vagal reactivity to be risk factor for externalizing problems in adolescents. A recent study examining trajectories of externalizing behavior in early adolescence (ages 13 to 15) found RSA augmentation to be a risk factor for elevated and increasing externalizing problems for youth who experienced greater marital conflict at age 13 (Peng, Wang, & Zhang, 2021). Additionally, among 16-year-old youth with poor family cohesion, those with blunted cardiac reactivity were at greater risk for aggressive behavior, particularly for boys (Sijtsema et al., 2013). Similarly, RSAr moderated associations between risky family environment (e.g., single parent status, parent mental health) and externalizing behavior problems, such that girls with less RSA withdrawal were at most risk (Diamond et al., 2012).

Collectively, studies examining various forms of family aggression, RSA, and adjustment are consistent with the overarching literature indicating that blunted cardiac responses (lower

RSA withdrawal, or augmentation) are associated with worse internalizing and externalizing behaviors among adolescents in adverse family contexts. Furthermore, the findings indicate the need to clarify the moderating role of PNS functioning in the links specifically between harsh parenting and multiple adjustment outcomes in adolescence as well as to examine sex-related effects.

Harsh Parenting and SNS. Skin conductance reactivity has been implicated as an important moderator in the link between harsh parenting and adjustment, particularly for externalizing behavior problems. Sympathetic reactivity indexed by SCLr may be indicative of how youth experience harsh parenting and may exacerbate or attenuate the effects of such parenting on adjustment. For youth with blunted sympathetic arousal, harsh disciplinary tactics and coercive behaviors may not evoke sufficient physiological reactivity to signal risk in the environment and therefore may be ineffective in socializing youth to associate their problem behavior with negative behavioral consequences or negative emotional consequences such as feelings of shame or guilt (Hoffman, 1994; Raine, 2002). Additionally, for youth with blunted SNS arousal, harsh parental behavior may trigger uninhibited defiant or aggressive responses during interactions (Dadds & Salmon, 2003; Patterson et al., 1992) and reinforce social learning of aggressive and coercive behavior. Conversely, physiologically over-aroused youth may find harsh parental discipline and aggression to be overwhelming and punishing, thus leading to negative cognitions of rejection and failure associated with internalizing symptoms (Erath et al., 2009; Hoffman, 1994; Kochanska, 1997).

The role of sympathetic reactivity in associations between harsh parenting and internalizing symptoms is understudied. However, research examining marital conflict suggests that higher SCLr is associated with parent-reported internalizing symptoms as well as increases

in symptoms among children over time in the context of high marital conflict (El-Sheikh, 2005; El-Sheikh et al., 2007). Based on findings from these studies, as well as conceptualizations of SCLr as a marker of anxiety, higher levels of SCL reactivity may be a vulnerability factor associated with greater internalizing symptoms in the context of harsh parenting.

Some studies have found SCLr to moderate associations between harsh parental control and externalizing behaviors such that children with low SCLr in these contexts show greater externalizing problems (Erath et al., 2009; Kochanska et al., 2015). Using a multi-informant cross-sectional design in a sample of 251 children, Erath and colleagues (2009) examined SCLr as a moderator of relations between parent- and child-reported harsh parenting and parentreported externalizing behaviors. Youth with low SCLr in response to a challenge task (star tracing task) had the highest levels of externalizing behavior in the context of greater harsh parenting (both parent and child reported). Similar findings emerged with SCLr to a stress task (listening to a stressful argument): parent-reported harsh parenting was positively associated with greater externalizing behavior problem at lower levels of SCLr, particularly for boys. Similar to the cross-sectional findings, externalizing behaviors were elevated and stable across childhood (ages 8 - 10) for boys with blunted SCLr in response to a stress task and high parent-reported harsh parenting (Erath et al., 2011). A similar pattern of high and stable externalizing across time was reported for girls with low SCLr and greater harsh parenting, whereas girls with higher SCLr exhibited declines in externalizing behavior. These longitudinal associations across childhood and into pre-adolescence have also been noted in research by Kochanska and colleagues, who also found blunted SCL reactivity to be a susceptibility factor for worsening externalizing problems in the context of coercive and power assertive parenting (Kochanska, Brock, & Boldt, 2017; Kochanska et al., 2015). This pattern of blunted SCLr and poor externalizing outcomes

has also been noted in a sample of emerging adults (18 – 23 years old): Parental psychological control was positively associated with the use of proactive (goal-oriented actions motivated by expectations in reward) relational aggression among those with low SCLr (Wagner & Abaied, 2016).

Fewer associations between harsh parenting and externalizing problems at higher levels of SCLr have been reported with adolescents. Family risk (e.g., single parent status, parent mental health) and higher SCLr was associated with greater externalizing behavior for a sample of 14-year-old boys (Diamond et al., 2012). Among emerging adults, positive associations between parental psychological control and reactive aggression (e.g., retaliatory response to perceived threat such as excluding a friend who forgot to invite you) were found for youth with greater sympathetic reactivity (higher SCLr in response to challenge; Wagner & Abaied, 2016). Though perhaps counterintuitive, these findings may reflect overarousal and reactive dimensions of externalizing behavior.

Taken altogether, despite some exceptions within the broader family stress literature, the research findings highlight the exacerbation of risk for externalizing behavior problems among youth with blunted SCLr (lower SCLr) and experiences of punitive disciplinary practices and harsh parenting (Kochanska et al., 2017). The present study builds on this literature and extends it by examining SCLr as a moderator of links between harsh parenting and adjustment symptoms over time during adolescence.

Little is known about the role of cardiosympathetic influences (PEPb or PEPr) on adjustment outcomes in adolescence in the context of harsh parenting. Youth with blunted PEP may not experience adequate stimulation and incentives in their environment and may seek sensation and stimulation in risky peer and social settings. A blunted PEPr profile is often

implicated with lower mesolimbic dopaminergic responses and associated with inability to derive pleasure, leading to seeking greater levels of stimulation (Beauchaine et al., 2007). Poor sympathetic reactivity may increase sensation seeking in the form of externalizing behaviors (e.g., risk taking), particularly when coupled with aversive family environments.

Few studies have examined PEP reactivity as a moderator between family stress and adjustment outcomes. Two studies have noted that shorter PEPb as moderator between harsh parenting and externalizing behavior problems among children (Huffman et al., 2020; Bubier et al., 2009). Greater harsh parenting was associated with increased delinquency for pre-adolescents with shortened PEPb (Huffman et al., 2020), as well as with externalizing behavior problems for urban ethnic minority children with short PEPb when they reported greater experiences of harsh parenting (Bubier et al., 2009). The aforementioned studies did not find that PEPr moderated relations between harsh parenting and externalizing problems.

Blunted PEPr among youth has been associated with a variety of rule-breaking and delinquency behaviors in the context of permissive parenting (Hinnant et al., 2016). In contrast, Obradović and colleagues (2011) reported that greater PEPr (shortening) was a risk factor for greater internalizing symptoms for children in high marital conflict homes, but no associations were found for externalizing behavior. Existing evidence supports further investigation to better understand how PEP may protect or exacerbate risk for adjustment problems in the context of harsh parenting.

Limited research has been done to examine the moderating role of physiological activity on relations between harsh parenting and adjustment outcomes during mid-to-late adolescence. Taken together, this small set of studies examining harsh parenting, in conjunction with findings in other family stress contexts, suggest that greater vagal withdrawal and greater SNS reactivity

(i.e.., shortening PEP, greater SCL) are associated with better adjustment during childhood and early adolescence. These associations are consistent with those reported in the broader literature examining youth well-being and ANS functioning within family stress contexts (Calkins, Propper, & Mills-Koonce, 2013; El-Sheikh & Erath, 2011), despite evidence for alternate ANS patterns that may confer risk for adjustment problems (Obradović et al., 2010; Obradović et al., 2011; Tabachnick et al., 2020). Further investigation is necessary to expand our understanding of ANS functioning in the link between harsh parenting and youth well-being; the proposed study aims to better understand these processes during late adolescence.

Moderation by Gender/Sex

Across the literatures examining harsh parenting and aversive family environments, adjustment outcomes, and ANS functioning, there have been notable sex-related effects. Mixed evidence has emerged to indicate that negative family environments may impact girls more than boys (Davies & Windle, 1997), whereas others have found that boys are more influenced by family conflict and harsh parenting (Davies & Linsday, 2001). Boys and girls may be exposed to different dimensions of harsh parenting. For example, girls were more likely to experience elevated and stable harsh verbal aggression from mothers (Donovan & Brassard, 2011), whereas boys were more likely to be physically punished during childhood and preadolescence (Grogan-Kaylor & Otis, 2007). Gender socialization theories indicate that girls are likely to be given messages about the importance and value of social relationships, such as parent-child relationships, more so than boys (Gilligan, 1982). Additionally, developmental trends also indicate greater relationship orientation and emotional needs among adolescent girls (Cyranowski et al., 2000; Rudolph, 2002). Difficulties with parents during adolescence may serve as a greater vulnerability for girls, thus greater harsh parenting and strained parent-child

relationships would potentially be more socio-emotionally detrimental to girls than boys. These disparate experiences highlight the need to better ascertain who may be at greater risk for maladjustment in stressful family environments.

An abundance of research implicates adolescent sex as a critical moderator in understanding the links between harsh parenting and adjustment outcomes. Sex differences in the development of depressive symptoms have been noted, such that adolescent girls are more vulnerable to developing depression in the context of greater conflict or poor parent-child relationship (Hankin et al., 2015; Slavin & Rainer 1990; Windle 1992). Social learning and gender socialization may encourage girls to emote and behave in indirect and less overtly aggressive or hostile ways (Letendre, 2007). Boys, on the other hand, may be socialized to believe that physical and aggressive behavior and emotional outbursts (e.g., anger) are more acceptable (Card et al., 2008). This type of socialization may contribute to greater levels of internalized symptomology among adolescent girls, and externalizing behaviors among boys, especially when youth live in harsh and aggressive family environments. Elevated and increasing levels of depressive symptoms for girls persist across adolescence in relation to harsh parenting behaviors (Lansford et al, 2006; Rudolph et al., 2000) and parent-child conflict (Chen & Harris, 2019), whereas depressive symptoms may decrease for boys. In addition, girls who experienced high and stable levels of maternal verbal aggression were rated by peers to be more physically and relationally aggressive compared to other girls. No such associations for aggression were noted for boys (Donovan & Brassard, 2011). For adolescent boys, harsh parenting is most often associated with delinquency or rule-breaking behaviors (Shaw & Scott, 1991). Longitudinal research has shown that harsh parenting during early childhood uniquely predicted externalizing problems for boys, but not girls, at age 17 (Leve, Kim, & Pears, 2005). Furthermore, pre-

adolescent boys experiencing harsh parenting had higher physical and social aggression during early adolescence (Kingsbury et al., 2020). The timing of harsh parenting experiences (e.g., preadolescence vs. later adolescence) and assessment of externalizing behaviors (e.g., self vs. parent report) may contribute to the variability in findings.

Moderation by Sex/ Gender in ANS Functioning

It is important to consider sex differences along with ANS functioning to specify risk for maladjustment in the context of harsh parenting. Research across multiple stress response systems have found that sex differences may help explain individual variation in youth outcomes related to ANS activity (Del Giudice, Ellis, & Shirtcliff, 2011; Koss & Gunnar, 2018). However, identification of who is most vulnerable for maladjustment and poor health has been inconsistent across studies and is likely due to measurement of stressors, outcomes, and developmental timing (Koss & Gunnar, 2018).

Empirical evidence of sex differences has emerged among several studies examining interactions between harsh parenting practices and ANS markers. Among preadolescents, the protective effects of greater vagal withdrawal on depressive symptoms and aggression were found only for boys with greater maternal psychological control and harsh discipline (Fletcher et al., 2017; Sijtsema et al., 2013). Vagal withdrawal did not confer the same protection for girls but rather was associated with higher depression symptoms when they experienced greater maternal psychological control. Notable sex differences for the role of RSA withdrawal in aversive family environments have found that girls with less vagal withdrawal are more vulnerable to negative affect and externalizing problems (Diamond et al., 2012). Other studies did not find any sex-related effects (Huffman et al., 2020; Mezulis et al., 2015). Thus, further

clarification about how RSA and sex function as moderators is needed, particularly among older adolescents.

Studies examining SNS correlates and externalizing behaviors within the family aggression context have noted risk predominantly for boys with lower SCLr, as well as limited evidence for girls with higher SCLr. Cross sectional and longitudinal findings have identified boys with lower SCLr to be at greatest risk for externalizing problems in the context of greater harsh parenting (Erath et al., 2009; Erath et al., 2011) and permissive parenting via deviant peer affiliation (Hinnant et al., 2019). Girls with greater SCL reactivity were more vulnerable to elevated negative affect and externalizing problems than boys in the context of risky family environments characterized by single parent households or parental mental health problems (Diamond et al, 2012). In related family stress contexts, differences have been found in longitudinal relations between marital conflict and adjustment. Stronger associations were found for girls with greater SCLr, who had increased internalizing symptoms and externalizing behavior in the context of marital conflict, compared to girls with lower SCLr (El-Sheikh et al., 2007). Higher SCLr was not a vulnerability or protective factor for boys in the context of marital conflict (El-Sheikh et al., 2007). This study will further investigate the role of SCLr in associations between harsh parenting and adjustment outcomes in late adolescence.

For cardiosympathetic reactivity (PEPr), no sex differences have been indicated in relations between harsh family environments and adjustment outcomes. However inconsistent differences in PEP have been noted. Some studies find evidence that boys have longer PEPb (Hinnant, Elmore-Stanton, & El-Sheikh, 2010; Matthews et al., 2002), whereas others find no differences between boys and girls (Alkon et al., 2003). Scarce findings indicate the need to

further investigate how PEP and sex differences impact adjustment in the context of harsh parenting.

Proposed Study

Building upon the literature, the first aim is to examine the role of harsh parenting during adolescence as a predictor of developmental trajectories of internalizing symptoms (depression, anxiety) and externalizing behaviors (rule-breaking, aggression). Trajectories of specific externalizing symptoms, specifically rule-breaking and aggressive behavior are known to differ across adolescence (Evans et al., 2019; Steinberg, 2010), but it is unclear how they vary in later adolescence despite known consequences for youth outcomes in adulthood. Similarly, depression and anxiety are known to increase across adolescence; however, much research has utilized aggregated internalizing symptoms, without ascertaining potentially unique trajectories of anxiety and depressive symptoms. Latent growth curve modeling will be used to examine these trajectories across ages 16, 17, and 18. Consistent with research examining associations between harsh parenting and adjustment (Erath et al., 2011; Hinnant et al., 2015; Repetti et al., 2002), we expect that greater levels of harsh parenting will be associated with increasing internalizing and externalizing problems across late adolescence (e.g., an increasing slope from ages 16 to 18) and that higher levels of adjustment problems would occur at age 18 (intercept effects).

The second aim will examine parasympathetic (indexed by RSAr) and sympathetic (indexed by SCLr and PEPr) reactivity to a moderately frustrating task at age 16 as a moderator of relations between harsh parenting at age 16 and adjustment trajectories across ages 16 and 18, as well as intercept effects at age 18. The star-tracer task is a challenging task that elicits cognitive responses such as attention and inhibition. The task may also elicit performance-related emotion responses including frustration or anxiety about failure or excitement about success.

Compared to studies that have used socially evaluative or reward scenarios, the star-tracer may operate in a similar fashion that would be consistent with approach and avoidance conceptualizations for ANS reactivity.

Consistent with predominantly cross-sectional studies done with younger children (Benito-Gomez et al., 2019; Fletcher et al., 2017; Graziano & Derefinko, 2013), we hypothesize that RSAr will moderate relations between harsh parenting and adjustment such that greater vagal withdrawal (decline in RSA from baseline to task) will serve as a protective factor against internalizing and externalizing problems. Conversely, we expect that augmentation (increase in RSA from baseline to task) will be a vulnerability factor and exacerbate the relationship between harsh parenting and adjustment problems. Difficulties in physiological regulation, particularly blunted vagal reactivity, may reflect disengagement and thus predict internalizing and externalizing problems (Beauchaine, 2001; Porges, 2007).

Additionally, in accordance with the previous longitudinal research examining SNS activity in the context of harsh parenting (Erath et al., 2011, Wagner & Abaied, 2016), we postulate that youth with blunted SCLr (low SCLr) will be vulnerable to elevated externalizing behaviors (e.g., intercept at 18 and increasing slope for rule-breaking problems and aggression). Empirical evidence for associations with internalizing problems in the context of harsh parenting have not been consistently observed. Some have reported higher SCLr to be associated with internalizing symptoms (Beauchaine, 2001; Weems et al., 2005) including those with marital conflict (El-Sheikh et al., 2007). However, others have noted associations between blunted SCLr and internalizing problems and negative affective in youth experiencing other stressful contexts such as peer victimization (Erath, Su, & Tu, 2018) and poor maternal mental health (Diamond et

al., 2012). Due to mixed evidence concerning the role of SCLr as moderator in links between family stress and internalizing symptoms, no directional hypothesis is offered.

No concrete hypothesis is offered for the moderating role of PEP as it relates to internalizing symptoms due to the limited empirical findings. Short baseline PEP and longer PEPr (lengthening of PEP; less SNS activity) has been implicated as risk factors for increased delinquent behavior and externalizing problems (Hinnant et al., 2016), and it is expected that such findings will emerge when examining PEPr as a moderator in relations between harsh parenting and externalizing outcomes. Additionally, theoretical considerations for role of PEP in sensation seeking also suggest that under-arousal of reward motivation systems would be associated with decreased PEPr and may confer risk for externalizing behaviors, particularly rule-breaking and proactive aggression. The conjoint influence of harsh parenting and lengthening of PEP (i.e., lower or blunted reactivity) may further undermine youth adjustment. It is hypothesized that youth who experience greater harsh parenting in conjunction with lengthening of PEPr would be at greater risk for elevated rule-breaking and aggressive behavior. No strong proposition is offered concerning the direction of effects for internalizing outcomes; however, it would be reasonable to expect that greater PEP reactivity is associated with internalizing problems, consistent with findings in the context of marital conflict (Obradović et al., 2011).

Finally, an exploratory study aim will address the moderating role of adolescent sex in associations between harsh parenting, physiological regulation, and adjustment outcomes. This aim will be explored through assessments of three-way interactions among harsh parenting, ANS reactivity, and adolescent sex. Due to the inconsistent and limited findings across the literature

concerning the role of sex as a moderator, no specific hypotheses about differential effects of ANS markers are proffered.

Chapter 3: Methods

Participants

Adolescents and their families participated in the latter three waves of a larger 6-wave longitudinal study across middle childhood and adolescence examining health, bioregulation, and adjustment in the context of family functioning (Family Stress and Youth Development). The six waves of data were collected across a period of 10 years. Youths were approximately 8 years old at the first wave (W1) and 18 years old at the last wave (W6); 2005- 2015. Initial recruitment of children and families occurred in 2005 through the distribution of flyers at three elementary schools in small towns and adjacent less populated rural communities predominantly in Alabama; a few participants resided in Georgia. Children were eligible for participation if they had cohabiting parents residing together for at least two years and did not have a diagnosis of an attention disorder, developmental delay, or chronic illness. A majority of participants (79%) returned to participate after a 4-year gap between W3 and W4, and an additional cohort of 53 adolescents and families was recruited at W4 using the same recruitment methods. No differences were noted among demographics or primary study variables between participants recruited during W1 and W4.

At age 16 (W4), the sample included 252 adolescents, 10 of whom were excluded from the analytic sample due to unavailable physiological data. No significant differences on variables of interest were found between the full sample at age 16 (N= 252) and the analytic sample (N= 242). The analytic sample for the proposed analyses utilizes participants from W4 to W6 where there was a 1-year time lag between successive waves (2012- 2015). Specifically, the analytic sample was composed of 242 adolescents (53% girls, 66.8% White, 33.2% Black/African American [AA]) who visited the laboratory and provided assessments at least at one of the three study waves. Adolescents were approximately 16 ($M_{age} = 15.79$ years, SD = .79), 17 ($M_{age} = 16.78$ years, SD = .77), and 18 ($M_{age} = 17.71$ years, SD = .74) years during the three waves, respectively. For enhanced clarity throughout, participants' mean ages will be referenced rather than study waves. At age 16, adolescents primarily resided in two-parent households and a small number lived in a single parent household (n = 27); some parents divorced or separated after initial recruitment. Family income-to-needs ratio (annual family income divided by the poverty threshold considering the family size; U.S. Department of Commerce, 2012) was used as an index of socio-economic status (SES). Approximately 14% of families lived below the poverty line (ratio < 1), 29% lived near the poverty line (ratio >1 and ≤2), 22% were classified as lower middle class (ratio >2 and ≤3), 26% were middle class (ratio >3 and ≤4), and 9% were upper middle class (ratio >4). Retention across study waves was good, with 94% of the sample from age 16 participating at age 17, and 85% of the sample at age 16 participating at age 18.

Procedure

The study procedures were approved by the University's institutional review board and parents and adolescents provided written consent and assent at each wave; procedures pertinent to the current investigation are discussed. At each wave, parents and adolescents visited our oncampus laboratory where each participant answered questionnaires in a separate room using Qualtrics (Qualtrics; Provo, UT). In addition, at age 16, adolescents' ANS activity (RSA, PEP, SCL) was assessed. Cardiovascular (RSA, PEP) and electrodermal (SCL) data were sampled at 1000 Hz using a Mindware BioNex 8-slot chassis and MW1000A acquisition system (Gahana, OH, USA). Data were filtered and smoothed using acquisition software BioLab 3.2. After the completion of a 3-minute acclimation period, adolescents' ANS activity was assessed during a 3minute resting condition while sitting quietly by themselves in a room (baseline assessment).

This assessment was followed by a frustrating stress task where the participants were asked to trace a star using only the reflection in the mirror as guide (LaFayette Instrument Company, Lafayette, IN). The task has been demonstrably used to evoke ANS reactivity among adolescents, including RSA-r (El-Sheikh, 2004; Jennings et al., 2002), SCL-r (Allen & Matthews, 1997; Erath et al., 2011), and PEPr (Hinnant et al., 2016).

Measures

Harsh Parenting

Youth report. At age 16, adolescents completed the well-established and validated Parent-Child Conflict Tactics Scale (CTSPC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998) to report on aggression directed at them by their parents during the past year. Youth reported on conflict with mothers and fathers separately. Items assessed the frequency of acts of verbal/psychological (5 items, "Shouted, yelled, or screamed at you"; "Called you dumb or lazy or some other name like that") and physical aggression (9 items; "Slapped you on the face or head or ears"; "Shook you") in the past year using a 7-point scale (0 = this never happened to 6 =more than 20 times in the past year). Adolescents' reports on verbal/ psychological (r = .54, p <.001) and physical (r = .63, p < .001) aggression across mothers and fathers were correlated and were averaged to derive total scores for each scale. High internal consistency was found for the combined mother and father scores on verbal/psychological aggression ($\alpha = .86$) and physical aggression ($\alpha = .85$). In addition, the verbal and physical scores were highly associated (r = .56, p < .001), and a mean composite score of harsh parenting was created across both scales with high internal consistency ($\alpha = .89$). A majority of youth reported about their experiences of harsh parenting for both parents (93%); the composite score across verbal and physical aggression was used for youth who reported only for one parent.

Parent report. Both mothers and fathers also completed the Parent-Child Conflict Tactics Scale (Straus et al, 1998) to report on their acts of aggression towards their adolescents in the past year. Mothers and fathers' reports on verbal/ psychological (r = .33, p < .001) and physical (r = .40, p < .001) aggression were correlated and averaged for a total score for each scale. High internal consistency was found for the combined mother and father scores on verbal/psychological aggression ($\alpha = .80$) and physical aggression ($\alpha = .85$). Verbal and physical aggression scores were moderately associated (r = .46, p < .001), and a mean composite score of harsh parenting was created across both scales and had high internal consistency ($\alpha = .85$). Similar to adolescent report, a majority of harsh parenting scores were derived using reports from two parents (70%) and composite score across verbal and physical was used when only one parent responded.

Autonomic Nervous System Activity

Respiratory sinus arrhythmia (RSA). Electrocardigram (ECG) data were collected using disposable snap electrodes and respiration was derived from thoracic impedance (Z0; Ernst, Litvack, Lozano, Cacioppo, & Berntson, 1999) using well validated procedures (Hinnant et al., 2015). RSA data was quantified using spectral analysis with Mindware HRV software (Version 3.1.5) where data were scored in 1-minute intervals. RSA was derived as the natural log of the variance in the heart period within the respiratory frequency range (.15 - .40 Hz), a validated means to ascertain parasympathetic influence on cardiac activity (Berntson et al., 1997).

Baseline RSA (RSAb; 3 min) was obtained following an adaptation period to the lab. RSA reactivity (RSAr; 3 min) was calculated as a difference score between the star-tracing task and baseline RSA, such that lower (negative) scores represent suppression or withdrawal (decreases in RSA) and higher scores (positive) are indicative of augmentation (increases in RSA).

Pre-ejection period (PEP). Pre-ejection period is defined as the interval (in milliseconds) between the electrical innervation of the ventricular myocardium (from the Q wave on the ECG) and the isovolumetric contraction of the left ventricle and opening of the aortic valve. Cardiac impedance (Z_0) data was collected using validated measures with a 4-spot electrode configuration placed on the thoracic cavity (Berntson & Cacioppo, 2004). Voltage electrodes were placed at the apex and base of the thorax and dual electrodes were placed on the participant's back 1.5 inches above and below the voltage electrodes. Respiration was derived from the Z_0 signal and PEP was quantified using Mindware Impedance (IMP) software (version 3.1.6).

Baseline PEP (PEPb) was obtained during a resting period where a lower number denotes higher SNS activity. PEP reactivity (PEPr) was calculated as a difference score between task (star-tracing) and baseline values, using the same method as noted above for RSA (task minus baseline). Negative values indicate shorter or quickening of PEP (higher SNS reactivity) during the stress task, and positive values indicate a lengthening of PEP. Negative values for reactivity are indicative of greater SNS activity during the stressor.

Skin conductance level (SCL). Skin conductance level was assessed using two silver/silver -chloride (Ag-AgCl) electrodes (1" × 1" foam, 0 % chloride gel) that were placed on the palm of adolescent's non-dominant hand. SCL levels were analyzed in 1-minute interval (units = microsiemens or μ S) with a gain of 10mV and low filter pass of 10 Hz using Mindware EDA software (version 3.0.21). Higher values of baseline SCL (SCLb) indicate greater SNS activity. SCL reactivity (SCLr) was calculated as a difference score using the same method as

noted above for RSA and PEP. Higher values (positive) denote greater SNS activity during the stressor task.

Adjustment Symptoms

Depression. Adolescents reported on their depressive symptoms using the well validated and reliable Children's Depression Inventory (CDI; Kovacs, 1992). Items (27 items; one item concerning suicidal ideation was removed) assessed the severity of symptoms using three statements (e.g., "I am sad all the time"; "I am sad many times"; "I am sad once in while"). Adolescents selected the statement that best described their feelings across the last 2 weeks and were scored on a 3-point scale ($0 = absence \ of \ symptom$ to $2 = definite \ symptom$). Scores across all items were summed to create an overall depressive symptoms score. Internal consistency for the overall score was high for all waves of the study (α s = .88 – .89). Elevated and clinically significant scores (scores ≥ 20) were reported by 6.9%, 9.2%, and 8.3% of youth at ages 16, 17, and 18, respectively.

Anxiety. Adolescents reported on their symptoms of anxiety using the validated and reliable Revised Children's Manifest Anxiety Scale – 2 (RCMAS2; Reynolds & Richmond, 2008). The measure utilizes 40 items to assess worries, social concerns, and physiological symptoms (e.g., "I fear other kids will laugh at me"; "I get nervous around people"; "Often I feel sick in my stomach") and youth reported "yes" or "no" to each statement. Scores were summed to create a total anxiety score and internal consistency was high for all study waves ($\alpha s = .92 - .93$). High anxiety symptoms (2 SDs > mean score) were reported by a small percentage of youth; 5.2%, 7.2%, and 6.3% at ages 16, 17, and 18, respectively.

Externalizing behaviors. Youth reported on their externalizing behaviors in the past 6 months using two subscales on the Youth Self-Report (YSR; Achenbach & Rescorla, 2001). Due

to increasing differentiation of externalizing behaviors across adolescence, the two subscales of Rule-Breaking behavior and Aggressive behavior were examined. Rule-Breaking (15 items, e.g., "I cut classes or skip school"; "I drink alcohol without my parents' approval") and Aggressive behavior (17 items; "I argue a lot"; "I threaten to hurt people") were assessed using a 3-point scale (0 = not true to 2 = very often or often true). Responses were summed for total scores for each scale and internal consistency was moderate for rule-breaking ($\alpha s = .71 - .83$) and aggressive behavior ($\alpha s = .81 - .88$) across waves. Youth reporting borderline or clinical levels (T score ≥ 60) across all study waves were 14.6%, 16.7%, and 18.5% for rule breaking behavior at ages 16, 17, and 18, respectively. Borderline and clinical levels (T score ≥ 60) of aggressive behavior were reported for 23%, 14.8% and 14.2% of youth at ages 16, 17, and 18, respectively.

Controls

Other variables that may be associated with the primary model variables were considered as potential covariates in analyses including youth age, race/ethnicity (0 = White; 1 =Black/AA), sex (0 = girls, 1 = boys), and SES (family income-to-needs ratio).

Plan of Analysis

Preliminary analyses examined means, standard deviations, and correlations among primary study variables. Variables were assessed for potential outliers and skew using skew statistics and visual inspection of histograms. Outlier variables that exceed 3 *SD* were winsorized and recoded to the highest or lowest value (Cousineau & Chartier, 2010) and multivariate outliers were assessed using Mahalanobis distance (Kline, 2015). An examination of missing data was assessed using Little's Missing Completely at Random (MCAR) test and handled using full information maximum likelihood (FIML; Acock, 2005). The use of such an approach to handle missing data has been shown to produce the least-biased estimates and the fewest Type 1

errors through estimation of model parameters using all available data (Enders & Bandalos, 2001; Raykov, 2005).

A series of latent growth curve models (LGCM) were fit in Mplus 8.1 (Muthen & Muthen, 2017) to examine trajectories of adjustment outcomes using established guidelines for growth modeling (Singer & Willet, 2003). First, unconditional growth models were fit to examine between-individual variance in the intercepts and slopes for each adjustment outcome (anxiety, depression, rule-breaking, aggressive behavior) across three time points (ages 16, 17, and 18). Intercepts for adjustment outcomes were set at age 18 and time coding for the slopes was -2, 1, and 0 which represented ages 16, 17, and 18, respectively. Residuals of the repeated measures for adolescent adjustment were constrained to be equal across time to ensure measurement invariance.

For adjustment outcomes with significant variability in the intercepts and slopes, conditional models examined harsh parenting and physiological markers as predictors of adjustment. Interaction terms between harsh parenting and each physiological marker were created as products of mean-centered predictors to reduce multicollinearity. Models were fit separately for each report of hash parenting (e.g., youth report) and physiological variable interaction (e.g., Harsh Parenting x RSAr) and each outcome (e.g., depressive symptoms). In all models assessing physiological markers, the baseline level of the physiological marker was retained as a control. The law of initial values stipulates that baseline levels of functioning limit the capacity for reactivity (Hinnant et al, 2018; Wilder, 1967). Conditional moderation models examined these interactions as predictors of adjustment at the intercepts (age 18) and slopes for parameters that had significant variance. Additionally, final models explored three-way

interactions between harsh parenting, ANS reactivity (RSA, SCL, PEP), and adolescent sex (Appendix A: Figure 1a).

Results from significant growth models are presented using the well-established method of prototypical plots (Singer & Willet, 2003). The effect of predictors on adjustment trajectories is best illustrated by "identifying a prototypical individual/child distinguished by particular predictor values" (Singer & Willett, 2003, p. 60). Significant values indicative of "high" (+ 1 SD) and "low" (-1 SD) for each predictor were used in the final fitted models, along with estimated values for adjustment outcomes over time to represent prototypical youth (e.g., those in the population with high/low levels of harsh parenting). Simple slopes testing will be used to determine if slopes are significantly different from zero. Furthermore, differences in the intercept between prototypical youth were examined by constraining intercepts and conducting delta-chi square tests. All conditional growth models included time invariant covariates assessed at age 16 (age, sex, race, family SES). Exogenous variables were allowed to covary. Models were considered an acceptable fit if they satisfied at least two of the three following criteria: $\chi^2/df < 3$, comparative fit index (CFI) = .95 or greater, and nonsignificant root mean square error of analysis (RMSEA) ≤ 0.05 (Hu & Bentler, 1999; Schreiber et al., 2006).

Chapter 4: Results

Preliminary Analysis

Primary study variables (predictor and moderating variables) were examined for mulitvariate outliers using Mahalanobis distance. Three values for youth-reported harsh parenting, two values for parent-reported harsh parenting, and three values for physiological reactivity were winsorized and recoded to the highest or lowest values. Adjustment variables were also examined for outliers and values greater than 3 *SD* were winsorized. For depression symptoms, 3, 2, and 4 values were recoded at ages 16, 17, and 18, respectively. For anxiety symptoms, one value was recoded at age 17. For rule-breaking behavior, 3, 5, and 4 values were recoded at ages 16, 17, and 18, respectively. Finally, for aggressive behaviors, 3 values were recoded at age 16, 2 values at age 17, and 1 value at age 18. An examination of missingness of study variables revealed that values were missing at random based on Little's Missing Completely at Random test γ^2 (619) = 780.417, p > .001.

Descriptive statistics for all variables are reported in Table 1. An examination of group differences between boys and girls using independent samples *t*-tests revelaed significant differences, primarily in adjutsment outcomes (Table 2). Compared to boys, girls had greater depression symptoms at age 16 and anxiety symptoms at ages 16, 17, and 18. Likewise, compared to girls, boys had greater rule-breaking behavior at ages 16, 17, and 18, as well as greater parent-reported harsh parenting at age 16. Black adolescents, compared to White adolescents, reported more harsh parenting. No race differences were observed in physiological reactivity or adjustment variables. Additionally, there was a significant difference between youth and parent-reported harsh parenting (t(208)= 2.36, p = 0.018), where youth reported more harsh parenting (M = 4.36, SD = 4.80) than their parents (M = 3.50, SD = 3.51).

Bivariate correlations between covariates and primary study variabes (not depicted in table) reveal that age was positively associated with adolescent sex (r = .22, p < .01), as well as rule breaking behavior at age 16 (r = .18, p < .01), age 17 (r = .18, p < .001), and age 18 (r = .20, p < .01), indicating that older adolescents were boys, and had higher rule-breaking behavior at each timepoint. Family INR at age 16 was negatively associated with anxiety (r = -.17, p < .05) and depression symptoms at age 18 (r = -.26, p < .001), as well as with rule-breaking ($r_{age 17} = .18, p < .01; r_{age18} = .15, p < .05$) and aggressive behaviors (rs = -.21 - .22, p < .01) at ages 17 and 18. Race was positively correlated with baseline RSAb (r = .14, p < .05), and negatively associated with SCLb (r = -.38, p < .001) and INR (r = -.25, p < .001) indicating that Black youth had higher RSAb, lower SCLb, and lower INR at age 16. Adolescent sex was positively associated with parent-reported harsh parenting (r = .14, p < .05), PEPb (r = .16, p < .01), and rule breaking behavior at each age (rs = .16 - .24, ps < .05 - .001) indicating that boys had higher parent-reported harsh parenting, higher baseline PEP, and rule-breaking behavior. Negative associations were found between adoelscent sex and anxiety symptoms at each age (rs = -.19 --.64, ps < .01 - .001), as well as depression at age 16 (r = -.17, p < .01), indicating that girls reported greater internalizing symptoms.

Correlations among primarily study variables are reported in Table 3. Each adjustment variable was positively associated with it's own subsquent scores across time. For example, depression symptoms at age 16 were correlated with symptoms at ages 17 and 18. Additionally, adjustment variables at all time points were consistently, positively associated with each other. Youth-reported harsh parenting was positively associated with all adjustment variables at age 16, 17, and 18. Furthermore, youth and parent reports of harsh parenting were weakly but positively correlated with one another. Baseline physiological activity variables were negatively correlated with their respective reactivity scores. For example, greater RSAb was associated with lower RSAr reactivity (greater withdrawal). Parent-reported harsh parenting was negatively associated with SCLb, but no other correlations involving such parenting and baseline or reactivity were found. Youth-reported harsh parenting was not correlated with any physiological baseline or reactivity indices. Furthermore, physiological reactivity (e.g., RSAr, SCLr) was not significantly correlated with any adjustment outcomes.

Unconditional Growth Models of Adjustment.

To examine change in adjustment symptoms across late adolescence, unconditional latent growth models were fit using adolescents' self-reported scores from ages 16, 17, and 18 for each adjustment symptom (Table 4).

Depression Symptoms.

Growth models for depression symptoms fit the data well: χ^2 (1) = 0.54, p = .82; χ^2/df = 0.54; RMSEA = .00, p = .86; CFI = 1.00. At age 18 (intercept), the mean value was 8.07 (*SE* = 0.48) and had significant variability (σ^2 = 43.94, *SE* = 6.37, p < .001), indicating that there are individual differences in depression symptoms at that age. The slope was not significant, suggesting that depression symptoms were stable across time at the group level. However, marginal variance in the slope was detected (σ^2 = 15.30, *SE* = 2.25, p < .10), suggesting that there may be individual differences in depression trajectories across from ages 16 to 18, despite no average change. The intercept and slope covariances (s^2_{is} = 7.97, p < .05) indicated that those with greater change in depression across time had higher levels of depression at age 18.

Anxiety Symptoms.

The unconditional growth model for anxiety symptoms fit the data well: χ^2 (1) = 4.29, p = .03; χ^2/df = 4.29; RMSEA = .12, p = .10; CFI = 0.99 and the intercept at age 18 was significant

(M = 9.41, SE = 0.61) and had significant variability ($\sigma^2 = 71.64, SE = 9.95, p < .001$). The slope of anxiety was not significant and thus indicative of stability in such symptoms across age 16 to 18 at the mean level. However, significant variance in the slope was detected ($\sigma^2 = 10.20, SE =$ 3.43, p < .01), suggestive of potential individual differences. The covariance between the intercept and slope ($s^2_{is} = 13.98, p < .01$) indicated that those with greater change in anxiety across time had higher levels of symptoms at age 18.

Rule-Breaking Behavior.

The model fit rule-breaking data well: χ^2 (1) = 1.21, p = .27; $\chi^2/df = 1.21$; RMSEA = .03, p = .40; CFI = 0.99. The intercept at age 18 was significant (M = 4.06, SE = 0.20) and had significant variability ($\sigma^2 = 7.63$, SE = 1.23, p < .001). The slope for rule-breaking was not significant, suggesting stability in rule-breaking behaviors across time. Marginal variance in the slope was detected ($\sigma^2 = 0.87$, SE = 0.48, p < .10), suggestive of potential individual differences. A marginal covariance between the intercept and slope covariance indicated that youth with increasing rule-breaking behavior had higher rule-breaking behavior at age 18 ($s^2_{is} = 1.06$, p < .10).

Aggressive Behavior

Growth models for aggressive behavior fit the data less than ideally and some fit statistics were acceptable: $\chi^2(1) = 11.07$, p = .00; $\chi^2/df = 11.07$; RMSEA = .21, p = .86; CFI = 0.97. A nogrowth model where the slope variable was constrained to zero was fit to examine if such a model would better fit the data: $\chi^2(4) = 27.20$, p = .00; $\chi^2/df = 6.80$; RMSEA = .16, p = .001; CFI = 0.92. Chi-square testing revealed that the model with the slope not constrained was the better fitting model ($\Delta \chi^2(3) = 25.99$) and was thus utilized for all analyses examining aggressive behavior. The intercept of aggressive behavior at age 18 was significant (M = 5.13, SE = 0.31) and had significant variability ($\sigma^2 = 14.73$, SE = 2.23, p < .001). The slope for aggressive behavior was significant (B = -0.54, SE = 0.15 p < .001) indicating that aggressive behavior decreased across mid-to-late adolescence. However, there was no variance in the slope ($\sigma^2 = 0.86$, SE =1.03, p = .41) indicating lack of interindividual differences in the pattern of change in aggressive behavior.

Harsh Parenting as a Predictor of Adjustment (Aim 1).

Models examining youth-reported harsh parenting as a predictor of adjustment symptoms fit the data adequately (model fit statistics provided below). Across all models examining youthreported harsh parenting, adolescent sex and family income-to-needs predicted the intercepts for adjustment symptoms (Table 5). Specifically, a negative association emerged such that boys and higher SES youth had lower levels of all adjustment outcomes at age 18. Furthermore, greater SES was also associated with declines in depression and anxiety symptoms. Race was a significant predictor of the intercept of aggression and a marginal predictor of rule-breaking, such that Black youth had lower levels of externalizing behaviors at age 18.

In models with parent-reported harsh parenting, a similar pattern of effects emerged for the covariates (Table 6). Greater SES predicted the intercepts for all adjustment outcomes as well as declines in internalizing symptoms (depression, anxiety). Sex was also associated with anxiety symptoms, rule-breaking, and aggressive behavior at age 18. The direction of effects for anxiety and aggressive behavior were the same as those reported previously for models with youth reported harsh parenting where boys had lower anxiety and depression. However, boys had higher levels of rule-breaking behavior than girls. Additionally, though models fit the data well, parent-reported harsh parenting did not predict any intercepts or slopes of adjustment.

Furthermore, only a single significant two-way interaction between harsh parenting and SCLr predicted the intercept of rule-breaking behavior, as well as one marginal interaction predicted depressive symptoms. There were no significant three-way interactions involving any of the ANS indices and adolescent sex.

Due to the null findings and lack of interaction effects, models with parent-reported harsh parenting will not be explicated in detail throughout the results section. An explication of findings, along with tables, model fit statistics, and figures for relevant results are provided in Appendix B.

Depression symptoms. The model with youth-reported harsh parenting fit the data well: $\chi^2(5) = 7.042, p = .22; \chi^2/df = 1.40; \text{RMSEA} = .04, p = .51; \text{CFI} = 0.99$. Harsh parenting at age 16 had a significant positive association with depression symptoms at age 18, however there was no significant association with the slope (Table 5).

Anxiety symptoms. Youth-reported harsh parenting had a significant positive association with the intercept of anxiety symptoms (Table 5; χ^2 (5) = 10.269, p = .06; $\chi^2/df = 2.05$; RMSEA = .07, p = .27; CFI = 0.98). Greater harsh parenting at age 16 predicted greater anxiety symptoms at age 18.

Rule-breaking behavior. Youth-reported harsh parenting at age 16 predicted greater rulebreaking behavior at age 18 (Table 5; $\chi^2(5) = 2.746$, p = .73; $\chi^2/df = 0.54$; RMSEA = .00, p = .90; CFI = 1.00). There was also a negative association of marginal significance for the slope indicating that greater harsh parenting predicted declines in rule-breaking behavior over ages 16 to 18.

Aggressive behavior. Youth-reported harsh parenting was a significant predictor of the intercept of aggressive behavior (Table 5; χ^2 (10) = 27.018, p = .003; $\chi^2/df = 2.70$; RMSEA = .08,

p = .07; CFI = 0.95. Greater harsh parenting at age 16 predicted greater aggressive behavior at age 18.

Physiological reactivity as a Moderator (Aim 2)

RSAr as a moderator. All models fit the data adequately and RSAb was included as a covariate (Appendix A: Table 1a). Among models with youth reported harsh parenting, neither RSAr nor RSAb predicted the intercepts of any adjustment outcomes at age 18 (Table 7). Similarly, baseline and reactivity did not predict trajectories for depression, anxiety, or rule-breaking behavior across late adolescence. Furthermore, no two-way interactions between harsh parenting and RSAr emerged.

SCLr as a moderator. All models fit the data adequately and SCLb was included as a covariate (Appendix A: Table 2a). In models with youth reported harsh parenting, no direct effects of SCLb were found, yet SCLr was a significant predictor of anxiety symptoms and aggressive behavior intercepts (Table 8). Greater SCLr at age 16 was associated with lower anxiety and lower aggressive behavior at age 18. No moderation effects of SCLr were detected in relations between harsh parenting and adjustment intercepts and slopes.

PEPr as a moderator. All models fit the data adequately (Appendix A: Tables 3a) and PEPb was included as a covariate in all models. PEPb did not predict adjustment intercepts or slopes in models with youth reported harsh parenting (Table 9). PEPr was marginally associated with the slope of anxiety symptoms such as that adolescents with lengthening of PEP in response to stressors (longer PEP during stress task compared to baseline) predicted increases in anxiety symptoms across ages 16 and 18. Additionally, longer PEPr also predicted a marginally elevated aggressive behavior at age 18. This was qualified by a significant two-way interaction between PEPr and harsh parenting. As shown in Figure 1, youth with greater harsh parenting and lengthening of PEPr (orange solid line) had the highest levels of aggressive behavior (M = 8.28). These youth had a 0.62 SD significant difference in aggressive behavior scores at age 18 in comparison to youth with shortening PEPr (orange dashed line; $\Delta \chi^2$ [1] = 17.65, p < 0.001). Similarly, a 1.07 SD significant difference in aggressive behavior at age 18 ($\Delta \chi^2$ [1] = 79.59, p <0.001) was found among youth with lengthening of PEP and those with less (blue solid line, M =3.60) and greater harsh parenting. No significant differences in aggressive behavior were found for youth with less harsh parenting, regardless of PEPr. The interaction effect on the intercept of aggression at age 18 is also depicted in Appendix A Figure 2a.

Physiological Reactivity and Sex as Conjoint Moderators (Aim 3)

Sex and RSAr as moderators. All models fit the data adequately (Appendix A: Table 1a). A marginal three-way interaction for the intercept of depression (Table 10) indicated that girls with greater harsh parenting and less RSA withdrawal had the highest levels of depression at age 18 (Figure 2; dark orange solid line, M = 13.97). There is a significant .37 *SD* difference in depression symptoms ($\Delta \chi^2$ [1] = 3.98, p < 0.05) between these girls and their counterparts with more RSA withdrawal (dark orange dashed line), and a 1.03 *SD* difference with boys with high harsh parenting and less RSA withdrawal (orange dashed line; $\Delta \chi^2$ [1] = 40.89, p < 0.001). Both youth with less harsh parenting regardless of RSA reactivity (blue lines), and boys with greater harsh parenting and less RSA withdrawal (blue dashed line) had lower levels depression at age 18. The interaction effects at age 18 are also depicted in Appendix A: Figure 3a.

A single significant interaction between youth reported harsh parenting, RSAr, and adolescent sex emerged to predict aggressive behavior at age 18 (Table 10). The slope is significant and indicates that on average, levels of aggressive behaviors were decreasing across ages 16 to 18. Girls with greater harsh parenting and less RSA withdrawal had the highest aggressive behaviors at age 18 (Figure 3; dark orange solid line, M = 10.09). A similar pattern of effects as depression symptoms emerged such that there was a significant 0.58 *SD* difference in aggressive behaviors between these girls and their counterparts with more RSAr withdrawal (dark orange dashed line; $\Delta \chi^2$ [1] = 14.12, p < 0.001). Additionally, there was a 1.42 *SD* difference in aggressive behavior between girls and boys with greater harsh parenting and less RSA withdrawal (orange solid line; $\Delta \chi^2$ [1] = 60.01, p < 0.001). Appendix A Figure 4a depicts the interaction effects on aggression at age 18. No significant three-way interactions predicted the slopes of any adjustment outcomes.

Sex and SCLr as moderators. All models fit the data adequately (Appendix A: Tables 2a). A significant three-way interaction for the intercept of aggression (Table 11) indicated that girls with greater harsh parenting and lower SCLr had the highest levels of aggression at age 18 (Figure 4; dark orange solid line, M = 15.44). Compared to their counterparts with high SCLr, there was a 0.86 *SD* difference in aggressive behaviors at age 18 (dark orange dashed line; $\Delta \chi^2$ [1] = 16.51, p < 0.001). A similar significant difference of 1.06 *SD* existed between them and boys with greater harsh parenting with lower SCLr (orange dashed line; $\Delta \chi^2$ [1] = 32.34, p < 0.001). Additionally, the specific interaction effects on aggression at age 18 are depicted in Appendix Figure 7.

Sex and PEPr as moderators. Data fit the models well (Appendix A: Table 3a). A significant three-way interaction for the intercept, as well as a marginally significant interaction predicting the slope of depression emerged (Table 12). In the prototypical plot represented in Figure 5, individual differences in the trajectory of depression symptoms are depicted. Girls with greater harsh parenting and lengthening of PEP (lower PEPr; dark orange solid line) had the highest and marginally increasing levels of depression symptoms across mid-to-late adolescence.

Specifically, there was a significant 1.25 *SD* difference in depression symptoms for these girls compared to their counterparts with greater PEPr (shortening of PEP) at age 18 (dark orange dashed line; $\Delta \chi^2$ [1] = 16.05, *p* < 0.001). There was also a 1.01 *SD* difference in depressive symptoms between girls and boys with greater harsh parenting and lengthening of PEP (orange solid line; $\Delta \chi^2$ [1] = 31.87, *p* < 0.001). Overall, boys had relatively lower levels of depression symptoms compared to girls.

A significant three-way interaction also predicted the intercept and slope of anxiety symptoms. Similar to models for depression, girls with greater harsh parenting and lengthening of PEP (dark orange solid line) had the highest and increasing levels of anxiety symptoms across ages 16 to 18 (Figure 6). Additionally, girls with greater harsh parenting and shortening PEPr (dark orange dashed line) had higher levels of anxiety and decreasing symptoms across mid-tolate adolescence. There was a significant 1.03 SD difference in anxiety symptoms at age 18 among girls with lengthening and shortening of PEP and greater harsh parenting ($\Delta \chi^2$ [1] = 14.35, p < 0.001). A marginal slope also emerged for girls with less harsh parenting and lengthening of PEP (dark blue solid line), and there was a 1.40 SD difference in anxiety between these girls and their counterparts with greater harsh parenting ($\Delta \chi^2$ [1] = 45.05, p < 0.001). No significant slopes emerged for boys, and they had relatively lower levels of anxiety symptoms compared to girls. Among boys, those with greater harsh parenting and lengthening of PEP had the highest anxiety symptoms at age 18 (blue solid line, M = 9.40). There was also a 1.13 SD difference in anxiety between boys and girls with greater harsh parenting and lengthening of PEP $(\Delta \chi^2 [1] = 38.56, p < 0.001).$

A marginal interaction between harsh parenting, PEPr, and adolescent sex predicted the intercept of rule-breaking behavior. Girls with greater harsh parenting and lengthening of PEP

had the highest levels of rule breaking behavior at age 18 (Figure 7; dark orange solid line, M = 6.76). A 1.05 SD difference was found among their counterparts with shortening PEP (dark orange dashed line; $\Delta \chi^2 [1] = 13.93$, p < 0.001). Generally, boys had higher levels of rule-breaking behavior, particularly those with greater harsh parenting and lengthening of PEP (orange solid line, M = 6.09), however no significant differences were detected between them and their counterparts with shortened PEP at age 18 (dashed orange line; $\Delta \chi^2 [1] = 2.33$, ns). Appendix A Figure 8a depicts the three-way interaction on the intercept of rule-breaking behavior at age 18.

Finally, a significant three-way interaction predicted aggressive behavior at age 18. Girls with greater harsh parenting and lengthening of PEPr had the highest levels of aggressive behavior (Figure 8; dark orange solid line, M = 11.15). There was a 1.21 *SD* difference in aggression between these girls and those with shortening PEP (dark orange dashed line; $\Delta \chi^2$ [1] = 22.62, p < 0.001). A similar difference of 0.89 *SD* emerged between boys (orange solid line) and girls who had greater harsh parenting and lengthening of PEPr ($\Delta \chi^2$ [1] = 30.22, p < 0.001). For boys with greater harsh parenting, a 0.65 *SD* difference emerged for those with longer and shorter PEPr (dashed orange line; $\Delta \chi^2$ [1] = 10.97, p < 0.001). An additional plot depicting this three-way interaction on the intercept of aggression at age 18 can be seen in Appendix A: Figure 7a.

Chapter 5: Discussion

Socio-emotional adjustment and mental health in mid-to-late adolescence are influenced by environmental context, which can be critical to shaping the trajectories of internalizing and externalizing symptoms (Cicchetti, 2008; Patton et al., 2018). Adverse family environments characterized by harsh parenting contribute to the development of internalizing and externalizing symptoms, however it is unclear which factors may contribute to increased vulnerability or protection in such environments (Cole et al., 2002; Repetti et al., 2002). Individual differences in physiological regulatory ability via ANS reactivity may help identify youth who are more protected or less resilient to adjustment problems in childhood (Erath et al., 2009; 2011; Huffman et al., 2020). The current study examined harsh parenting as a predictor of trajectories and intercepts of depression and anxiety symptoms, as well as rule-breaking and aggressive behaviors, during mid-to-late adolescence. To address research questions, we utilized three waves of data spanning ages 16 to 18 with one-year intervals. The moderating role of ANS reactivity in such relations was also examined using indices of RSA, SCL, and PEP reactivity. The conjoint influences of ANS reactivity and adolescent sex were also examined as moderators of relations between harsh parenting and internalizing symptoms and externalizing behaviors.

Harsh parenting at age 16 was a significant predictor of internalizing symptoms (anxiety, depression) and externalizing behaviors (rule-breaking, aggression) at age 18. However, it did not predict changes in adjustment outcomes. Furthermore, although no unique two-way interactions emerged, three-way interactions were found between some ANS indices and adolescent sex in the prediction of adjustment outcomes. Generally, the three-way interactions revealed that girls with greater harsh parenting and lower levels of ANS reactivity were at greatest risk for increases in the slope adjustment problems across adolescence, as well elevated

problems at age 18. Specifically, girls with higher harsh parenting and lower SNS reactivity indicated by the lengthening of PEP (greater PEP during stressor than baseline) had increases in anxiety symptoms across ages 16 to 18. Additionally, the same girls also showed marginally increasing depression symptom trajectories and greater rule-breaking behavior at age 18. Findings did not indicate similar risk for boys with low reactivity for any adjustment outcomes.

Low ANS reactivity was also as a risk factor for aggressive behavior in girls. Across all ANS markers (RSA, SCL, PEP), girls with low reactivity who reported greater levels of harsh parenting had the highest levels of aggression at age 18. These intercept effects for aggression are indicative of the potential risk of blunted ANS responding in stressful family contexts and extend understanding of who is at most risk for maladjustment (El-Sheikh & Erath, 2011). The findings highlight a constellation of risk factors across individual and family environment that may contribute to elevated risk for adjustment problems during this developmental period.

Harsh Parenting and Adjustment

Harsh parenting, specifically based on youth report, was a significant predictor of adjustment outcomes. Intercept effects indicated that experiences of greater harsh parenting at age 16 were associated with higher depression and anxiety symptoms, as well as rule-breaking and aggressive behaviors at age 18. Consistent with the overarching literature and study hypothesis, harsh parenting was associated with adolescent internalizing and externalizing outcomes (Di Giunta et al., 2020; Kingsbury et al., 2020; Weymouth et al., 2016). Few studies have examined the role of harsh parenting on adjustment during later years of adolescence (after age 16), and those have been primarily cross-sectional (Bender et al., 2007) or have focused on harsh parenting in childhood as a predictor of adolescent well-being (Clayborne et al., 2021). The current findings add to the limited longitudinal studies during this period. Taken together,

these studies show that family environment and parenting are relevant risk factors for negative outcomes during late adolescence (Di Giunta et al., 2020; Wang, 2019).

Contrary to expectations, no main effects of harsh parenting were observed for the slopes of any adjustment outcome. Harsh parenting at age 16, based on both youth and parent reports, did not predict increases in internalizing symptoms or externalizing behaviors from ages 16 to 18. Some studies that have investigated the role of harsh parenting practices on trajectories of internalizing symptoms and externalizing behavior have also reported similar null effects (Davis, Votruba-Drzal, & Silk, 2015; Leve et al., 2005) but primarily from childhood to adolescence. The lack of slope effects is somewhat contrary to those reported in studies from childhood where harsh parenting predicted changes in internalizing and externalizing behaviors. Growth model and person-centered approaches have reported increases in adjustment problems primarily in later childhood and into early adolescence. For example, Erath and colleagues (2011) reported that harsh parenting at age 8 predicted trajectories of externalizing problems in late childhood (ages 8-10). Additionally, high maternal verbal aggression was associated with increases in depression symptoms during middle school years (6th – 8th grade; Donovan & Brassard, 2011).

Although it is plausible that no causal association exists, effects of adolescents' perspectives on their reports of parenting or effects of adolescents' behaviors on parenting may underlie the correlation between harsh parenting and adjustment. Youth may attribute harshness to parents as a function of their internalizing symptoms and externalizing behavior. Studies examining reciprocal effects between parenting and adjustment have found child-driven effects of internalizing and externalizing on parental behaviors of warmth and control across cultures (Rothenberg et al., 2019). Similarly, greater internalizing symptoms, as well as greater

engagement in delinquency and aggression during adolescence is associated with greater parental control (Janssens et al., 2017).

To date, no known studies have specifically examined the effects of harsh parenting on internalizing symptoms and externalizing behavior during mid-to-late adolescence. The lack of main effects on trajectories of adjustment may further support the notion that as youth progress further into later adolescence, parental influences wane (Patterson et al, 2017; Sarıtaş et al., 2013) whereas peer relations and bioregulatory and genetic influences may play an increasing role (Dick, Adkins, & I-Chun Kuo, 2016). Data from twin studies that has examined alcohol use among adolescents has shown a greater influence of genetics on drinking behavior, whereas at the same time family and other environmental influences decrease (Hopfer et al., 2003; Rose et al., 2001). Similarly, studies that have disentangled the impact of genetic and parent-adolescent conflict and aggression on depression among adolescents have found that genetic influences are a stronger contributor than environmental ones (Neiderhiser et al., 1999; Rice et al., 2013; Samek et al., 2018). The stability in adjustment symptoms throughout mid-to-late adolescence in the current investigation may also suggest the influence of other environmental variables and overarching influence of genes during this developmental period. The increasing salience of biological factors during late adolescence may be critical new areas of inquiry to consider when examining changes in adjustment and parenting behaviors (Morris et al., 2021).

In addition to null slope findings, associations between harsh parenting and adjustment only emerged for youth-reported harsh parenting and not parent report. A significant difference was detected between youth and parent report of harsh parenting; parents reported fewer instances of engaging in harsh behaviors compared to what their child reported. Parent reports on physical and verbal aggression towards their child may be susceptible to bias due to concerns

with social desirability. The findings for youth-reported harsh parenting also highlight the need to focus on how youth experience aggression in their relationship with their parents. Youth perceptions may be more salient because they directly influence their cognitions, notions of autonomy, and self-regulation (Bender et al., 2007; Cui et al., 2014; Miller-Perrin et al., 2009) and contribute to worsening mental health. Further, discrepancies between parents and adolescents concerning their relationship where youth report worse relationships have been linked to greater externalizing problems, particularly aggressive behavior (Dimler et al., 2016). **Individual Differences in Physiological Reactivity**

Physiological reactivity and adolescent sex conjointly were significant moderators of associations between harsh parenting and adjustment; however, findings were somewhat unexpected but consistent. In keeping with dual-risk perspectives, girls with low physiological reactivity, namely for PEP, appear to be at greatest risk for increases in internalizing problems across mid-to-late adolescence when they experienced greater harsh parenting. These effects were most pronounced for changes in anxiety symptoms for girls, and marginal trajectories for depression symptoms also provided further support for this pattern. No such slope effects emerged for rule-breaking behavior, nor were there any significant moderation effects on any adjustment outcomes for boys. Across mid-to-late adolescence, aggressive behavior generally decreased, however interactions between harsh parenting, ANS reactivity, and adolescent sex identified girls with low ANS reactivity to be at greatest risk for greater aggression at age 18. Physiological under-arousal has been identified as a significant moderator of relations between family aggression and socioemotional outcomes (El-Sheikh & Erath, 2013; Erath et al., 2011; Huffman et al., 2020), however, two-way interactions between harsh parenting and ANS reactivity indices did not predict internalizing or externalizing outcomes.

The current investigation found increases in anxiety symptoms during mid-to-late adolescence for all youth, as well as marginal increases in depression symptoms. Previous studies that have examined relations between harsh parenting and internalizing symptoms in conjunction with physiological indices have predominantly reported findings for depression symptoms (Fletcher et al., 2017), or a composite of internalizing symptoms (Benito-Gomez et al., 2019), but no known studies have specifically examined anxiety symptoms. Despite elevated levels of anxiety reported during the adolescent years, there is less clarity about trajectories of such symptoms, particularly in the context of harsh family environments. Additionally, evidence concerning trajectories of anxiety has been mixed where some have noted decreases (McLaughlin & King, 2015; van Oort et al., 2009), and others have noted increases across adolescence and early adulthood (Leadbeater, Thompson, & Gruppuso, 2012). The examination of anxiety and depression symptoms separately in the current study clarified trajectories, as well as identified who was at risk for specific outcomes in the context of harsh parenting and ANS reactivity.

Pre-ejection period reactivity is an important SNS marker that is understudied as related to internalizing symptoms, particularly anxiety symptoms (Beauchaine & Thayer, 2015). PEP activity is typically conceptualized as a marker of reward and appetitive motivation (Beauchaine et al., 2007; Brenner et al., 2005). Fluctuations in PEP are indicative of mesolimbic dopamine activity in response to challenging and/or rewarding situations, and symptoms of anxiety and depression are typically associated with alterations in neural circuitry associated with threat responses and reward processing (Dillon et al., 2013); lengthening of PEP during challenge tasks, indicative of reduced ANS activity may be reflective of dysfunction in such systems.

biopsychological model of challenge and threat (Blascovich, 2009) where dampened or reduced activity during challenge tasks may be reflective of adverse and maladaptive environmental contexts and motivational dysfunction (Hase et al.,2020; McLaughlin, Sheridan, et al., 2014). Blunted cardiosympathetic responding by youth may reflect a "shutting down" approach to cope with stressful family environments. Among youth with a spectrum of anxiety disorders, greater anxiety scores marked by apprehension and negative affect are associated with decreased or blunted physiological reactivity - suggestive of disruptions in the fear and defense neural circuitry (Lang & McTeague, 2009).

Among other studies that have examined other cardiovascular ANS markers, such as heart rate and RSA reactivity, blunted or dampened responses have been associated with poor mental health and difficulties in appropriately responding to stressors in the environment (Beauchaine, 2015; Lipschutz et al., 2017). Youth exposed to high levels of parent-child conflict may develop passive coping mechanisms in response to their adverse environment (Hare et al., 2015), and this may also be reflected in psychophysiological responses. The exertion of control by parents may contribute to withdrawal as a coping strategy (Booth-LaForce & Oxford, 2008; Barber & Harmon, 2002), which may alter physiological response patterns due to chronic exposure to parental aggression. Passive coping strategies, disengagement and withdrawal have been reported to serve as mediating and indirect mechanisms between parental psychological control during adolescence and heart rate and RSA reactivity among young adults (Loeb et al., 2021). Furthermore, similar blunted cardiovascular patterns have been reported among those who have experienced peer victimization and child maltreatment who report greater internalizing problems (Lambe, Craig, & Hollenstein, 2019; McLaughlin et al., 2014; Newman, 2014; Young-Southward et al., 2020).

A number of characteristics have been identified as significant contributors of risk for elevated or increasing depression symptoms during adolescence, including difficulties in the parent-child relationship, stressful life events, female sex/gender, conduct problems, substance use, and lower family SES (Shore et al., 2017). Many of these characteristics were also implicated in the current investigation as risk factors for depression. Supportive of the pattern of risk uncovered for anxiety symptoms, girls with greater harsh parenting and lengthening PEPr were also marginally at risk for increasing and elevated depression symptoms across age 16 to 18. This is in line with previously reported links between blunted PEPr and depression where lengthening of PEP in response to a challenge task was associated with greater depressive symptoms (Brinkman & Franzen, 2015) and anhedonia (Ahles et al., 2017). Furthermore, these findings contribute to ongoing conceptualizations concerning links between cardiovascular reactivity and depressive symptomatology. Low SNS reactivity via cardiovascular responses (e.g., blood pressure) have been linked to reduced motivation, low affect, and insensitivity to rewards (Brinkman et al., 2009; Whitton et al., 2015).

Only interactions with adolescent sex illuminated who was at risk for increases in anxiety and depression symptoms over time. A number of biological and environmental factors have been proposed as underlying contributors to help understand how gender/sex may be related to risk for psychopathology (Zahn-Waxler, Shirtcliff, & Marceau, 2008). Internalizing symptomatology, particularly depressive symptoms, has been identified as having adolescentonset. Genetic and biological differences between boys and girls during adolescence have been posited as potential etiological explanations for why there may be amplification of risk of internalizing problems for girls. Alterations in hormone response patterns (e.g., estradiol, cortisol) during puberty and early maturation of brain regions related to mood, emotions, and

emotion regulation in girls may influence stress responsivity in aversive environments. Furthermore, evidence from early childhood and into adolescence, indicates that girls are likely to be more socially sensitive and motivated towards affiliation (Zahn-Waxler et al., 2006), as well as attuned to the family environment and interpersonal relations (Davies & Lindsay, 2004). It may be likely that such characteristics, coupled with physiological disengagement (i.e., lengthening of PEP), may heighten increasing internalizing during this period for girls that experience higher harsh parenting. It may be that for boys, the slower pace of brain maturation, coupled with decreased focus on emotional cues and the family environment, may provide protection from internalizing problems.

Under-arousal in harsh and adverse environments may confer risk for maladjustment, however it appears that family emotion socialization processes may influence who is at greatest risk and how these problems may manifest (e.g., conduct problems, anxiety symptoms; Beauchaine et al., 2007; Beauchaine, Klein, Crowell, Debidge, & Gatzke-Kopp, 2009). Emotion socialization practices withing the family may be influenced by the gender of the child. Girls may be socialized to suppress impulsive behavior and thus their stress may manifest in a manner that is less overt, such as worries and pre-occupation (Letendre, 2007). It is possible that this may result in more elevated and pronounced anxiety symptoms and depressive behaviors rather than externalizing behavior such as rule-breaking and conduct problems. Additionally, the combination of disengagement or low engagement in a highly stressful environment of harsh parenting may produce a profile of risk that is similar to those observed among youth with conduct problems and anti-social behaviors; such that low ANS reactivity for those with adverse family environment may manifest as internalizing problems for girls. Elevated anxiety symptoms in girls and comorbidities with other disruptive and externalizing behaviors have been reported

(Bubier & Drabick, 2009; Chung et al., 2019), but it is unclear how ANS functioning may operate in such relations; of note is that at each age, anxiety was not strongly associated with externalizing behaviors for girls and such associations were similar for boys. The lack of clarity in studies is often further complicated by the use of composite measures of internalizing in which dimensions of guilt, worries, low affect, and self-worth are all assessed together. Disaggregation of such symptoms may help clarify how specific aspects of ANS activity may be related to adjustment and psychopathology.

The observed pattern of risk for internalizing symptoms did not emerge for boys with low PEP reactivity. Girls in the current study reported greater internalizing symptoms, compared to boys, and such associations have been well-documented (Galambos, Leadbeater, & Barker, 2004; Meadows, Brown, & Elder, 2006; Twenge & Nolen-Hoeksema, 2002) and girls may be more vulnerable than boys to elevated internalizing symptoms in adverse family environments and harsh parenting (Chen & Harris, 2019; Eberhart, Shih, Hammen, & Brennan, 2006; Hankin et al., 2015). Such explanations for the links between harsh parenting, low PEP, and increasing internalizing problems for girls are purely speculative. Though reported findings in this study are novel and provide new areas of inquiry, replication of such phenomenon is needed. No specific hypothesis was proffered concerning how PEPr would moderate associations between harsh parenting and internalizing outcomes and such propositions are tentative. Findings from this study suggest the importance of considering multiple ANS parameters as moderators of relations between harsh parenting and anxiety and depression symptoms to help identify which adolescents may be most at risk.

Unexpectedly and somewhat divergent from the broader literature, no significant interactions with any ANS indices predicted trajectories of rule-breaking behavior. A single

marginal association emerged for high harsh parenting and lengthening of PEP for elevated rulebreaking behavior at age 18 for girls. This intercept effect highlights co-occurring risk factors adverse environment and dampened physiological regulation – that may help identify anti-social behavior (ASB) and externalizing problems among girls. Interactions between experiences of adversity and PEP have been associated with ASB (e.g., broken or vandalized objects, hit someone) among 16-years olds (Sijtsema et al., 2015). Sex differences were noted where greater ASB for boys was associated with blunted PEPr whereas among girls, associations emerged for changes in PEP from baseline to recovery. However, among pre-adolescent youth, Huffman and colleagues (2020) did not find PEPr and youth sex to be moderators of relations between harsh parenting and delinquent behavior. The unclear and disparate findings from previous research and those presented in this paper highlight the need to further explore the role of PEPr in rulebreaking and delinquent behaviors.

The limited influence of harsh parenting, ANS reactivity, and adolescent sex on externalizing behavior trajectories is contrary to what has been previously reported in crosssectional and longitudinal studies that have been conducted with pre-adolescent youth, mostly with SCLr as the ANS index. In a pair of studies, Erath and colleagues found that pre-adolescent youth with lower SCLr exhibited higher externalizing behavior (Erath et al., 2009), as well as increases in externalizing over time (Erath et al., 2011). Similarly, lower SCLr has been identified as a risk factor for poor externalizing behaviors in the context of assertive and coercive parenting from childhood and into pre-adolescence (Kochanska et al., 2017; Kochanska et al., 2015), as well as permissive parenting (Hinnant, et al., 2019; Hinnant et al., 2016).

Assessments of youth sex as a moderator of effects have often identified boys with lower SCLr to be at most risk for a variety of externalizing behavior problems in adolescence when

they experience adverse family environments. Erath and colleagues (2011) found boys to be at greatest risk for higher and increasing externalizing behavior problems when they experienced greater harsh parenting. A study examining permissive parenting at age 16 also reported that boys with lower SCLr had increasing engagement with deviant peers at age 17, and such affiliation with deviant peers served as an indirect mechanism for greater externalizing problems at age 18 (Hinnant et al., 2019). Links among adolescent girls and higher levels of externalizing behavior problems have been noted when they are exposed to risky family environments, however with greater sympathetic reactivity (higher SCLr) rather than blunted responses (Diamond et al., 2012). These findings did not replicate in the current sample of older adolescents, perhaps indicative of potential developmental differences. Sensation seeking behaviors are often at their highest during later adolescence and into early adulthood and thus PEP, rather than SCL, may be a more salient risk factor. Such relations have been noted in clinical and non-community samples where low PEP reactivity is a risk factor for conduct problems and anti-social behaviors (Brenner & Beauchaine, 2011; Beauchaine et al., 2009).

Examination of aggressive behavior disaggregated from other externalizing behaviors revealed significant associations in interaction models across ANS indices. The current study did not find significant variability in change in aggressive behavior across mid-to-late adolescence, however, on average, aggressive behavior declined, and this is consistent with to previous reports (Orpinas et al., 2015; Vaillancourt & Farrell, 2021). Dampened or low ANS reactivity among girls with greater harsh parenting was associated with elevated risk for higher levels of aggressive behavior at age 18. This constellation of co-occurring risk factors (i.e., harsh parenting, low ANS reactivity, girls) for elevated levels of aggressive behavior was evident in models examining RSAr, SCLr, and PEPr.

Low ANS reactivity has been consistently implicated as a risk factor for aggressive behavior and poor externalizing outcomes among clinical and community youth (Beauchaine et al., 2008; Graziano & Derefinko, 2013). Fearlessness and sensation seeking theories suggest that physiologically under-aroused youth engage in rule-breaking, delinquent, or aggressive behavior as a means to experience optimal physiological states (Raine, 2002). Physiologically underaroused youth may exacerbate negative interactions with their parents due to insensitivity to punishment and thus interactions between harsh parenting and ANS reactivity may implicate physiological under-arousal as an indicator of risk for aggression and conduct problems (Beauchaine et al., 2007; Raine, 2002). The findings for SNS reactivity via SCLr corroborate associations that have been previously reported where lower SCLr has been linked with proactive aggression among children and adolescents (Hubbard et al., 2010), as well as emerging adults in the context of parental psychological control (Wagner & Abaied, 2016). Beauchaine and colleagues (2008) have reported that in clinical samples of youth with conduct problems, boys and girls have exhibited different patterns of physiological responding in relation to aggressive behaviors. Boys with lower SCLr appear to be at greater risk for aggressive behavior whereas girls with higher SCLr are at risk.

A small number of studies have reported conjoint influences of sex and cardiovascular ANS indices on aggressive behavior. A recent cross-sectional study with diverse pre-adolescent youth found that those with RSAr withdrawal and greater harsh parenting had elevated levels of aggression, however no sex differences were detected (Huffman et al., 2020). In related adverse family environments, boys with blunted cardiac reactivity (heart rate variability), but not girls, had greater aggressive behavior when they reported less family cohesion (Sijtsema et al., 2013). No evidence has been reported for lengthening of PEP to be a risk factor for aggression in the

limited studies that have investigated such relations (Beauchaine et al., 2008; Huffman et al., 2020).

Generally, indicators of aggressive behavior tend to be aggregated in externalizing behavior composites and thus have been examined infrequently in the context of family risk and ANS functioning. In studies that have examined aggression, greater attention is often given to the type of aggression (e.g., indirect, overt) or aggression in specific relational contexts (e.g., romantic relationships, peer victimization; Muñoz-Rivas et al., 2007). It is unclear why girls (compared to boys) with low ANS reactivity would be at particular risk for aggressive behavior. Developmental models indicate that externalizing behavior problems tend to be early-onset disorders that occur in childhood, however, this may be most pertinent to boys and not girls (Moffit & Caspi, 2001). In adolescence, girls may be more likely to have co-occurring symptomology of internalizing problems, such as anxiety, that may relate to increased use of behavioral strategies of relational and indirect forms of aggression (Zahn-Waxler et al., 2006).Biological and social vulnerabilities prominent in adolescence may also heighten the use of aggressive behavior among girls compared to boys. Low ANS reactivity via hypothalamic pituitary adrenal systems (e.g., cortisol) has been implicated as a risk factor for impulsive and aggressive behavior among adolescent girls (Susman, 2006; Susman et al., 2010), especially among those who have experienced greater stress (Roberts & Lopez-Duran, 2019). Furthermore, situations of conflict and discord within family systems have been reported to play a greater role in conduct behavior problems for adolescent girls compared to boys (Windle, 1992). Such constellation of risk factors may place girls in double jeopardy for maladjustment and interactions between biological vulnerabilities of dampened ANS reactivity and family

environment may be key to identify a subset of girls that are at risk for poor outcomes (Zahn-Waxler et al., 2008).

Additionally, a closer examination of the measure used to assess aggressive behavior is needed. The aggressive behavior sub-scale from the Youth Self Report is multi-faceted and taps into multiple domains of aggressive behavior. The measure includes items that reflect overt and relational forms of aggression, as well as items that reflect proactive and reactive functions of aggression. Girls are more likely to engage in relational and indirect forms of aggression (Archer & Côté, 2005; Côté, 2007) and thus may be reporting more elevated levels despite decreases over time (Cleverley et al., 2012). Studies that distinguish the forms and functions of aggression may help clarify gender differences in the predictors of aggression. Future work should continue to disentangle ANS functioning, gender, and harsh parenting influences on aggressive behaviors.

Contrary to expectations, girls had elevated self-reported rule-breaking and aggressive behaviors at age 18 compared to boys. Previous studies have relied upon teacher or parent report on externalizing behaviors and these informants may be more attuned to aggressive and rulebreaking behavior that is overt and deliberate. It may be teachers and parents believe that boys will engage in rule-breaking and delinquent behaviors based on empirical evidence (Liu & Miller, 2020) and socialization (Berg-Nielsen et al., 2012). Delinquent, aggressive and rulebreaking behavior by girls may be missed due to this type of biased attention and scrutinization of boys' behavior (Ferguson, 2000), and because girls may be more secretive and more likely to engage in covert forms of externalizing behavior due to socialization from parents and teachers (Keenan & Shaw, 1997). Such methodological distinctions between the current study and previous research may explain the somewhat novel but disparate findings concerning

externalizing behavior. Such propositions are speculative and further replication of such associations is needed.

No interactions among RSAr or SCLr, harsh parenting, and adolescent sex predicted anxiety or depression. Despite limited findings in pre-adolescence, interactions with RSAr were not associated with any internalizing outcomes. Other studies have found RSAr to moderate associations and protect against depression in the context of harsh parenting (Benito-Gomez et al., 2019; Fletcher et al., 2017), however these studies examined multiple dimensions of harsh parenting (e.g., physical discipline, psychological control), along with and other family stressors. Differences in the assessment of harsh parenting among these studies and the current investigation may contribute to lack of moderation effects of RSAr in relations between harsh parenting and internalizing symptoms. Additionally, RSAr and SCLr were not significant predictors of internalizing symptoms though previous evidence has linked low RSAr (Greaves-Lord et al., 2010) and greater SCLr to anxiety symptoms (Beauchaine, 2001; Weems et al., 2005). The lack of findings with SCLr in the current investigation may be related to rates of missing data for the variable.

The stress or challenge task that was utilized may not be best suited to assess stress reactivity, particularly within the behavioral inhibition/activation framework. A number of studies have reported ANS reactivity to different tasks such as more social evaluatively or emotionally stressful tasks such as the Trier Social Stress Task (Benito-Gomez et al., 2019; Fletcher et al., 2019), listening to a simulated argument (El-Sheikh, Harger, & Whitson, 2001; Obradović et al., 2011), and memory tasks. Emotionally valanced tasks may evoke more reactivity and be more closely aligned with stressful experiences of harsh parenting. However, studies have found significant moderating effects between harsh parenting and adjustment using

both reactivity to the star-tracing task and other evaluative tasks (Erath et al., 2009; 2011). Though the challenge task used to elicit ANS reactivity was sufficient to uncover associations with adjustment, future research should consider tailoring tasks more closely for ecologically valid assessments. Such methods would allow for a better understanding of how youth may be responding to harsh parenting "in the moment".

Low ANS functioning across physiological indices were hypothesized to be a risk factor for all youth, however the findings do not suggest a predictive or causal influence on changes in adjustment. The overarching literature has reported associations among low ANS reactivity indices and conduct and externalizing behavior problems (Beauchaine et al., 2007; Boyce et al., 2001, Crowell et al., 2006), as well as anxiety and depression (Greaves-Lord et al., 2010). Furthermore, the lack of interactive associations between harsh parenting and ANS reactivity on adjustment outcomes for boys may indicate that harsh parenting may be more salient for girls than boys during this developmental period. It will be important to explore how other dimensions of parenting may be particularly influential for boys. Permissive parenting and lack of parental monitoring have been associated with greater delinquency and substance use (Hinnant et al., 2019; Hinnant et al., 2016), as well as greater internalizing problems (Cai & Tu, 2020) among adolescent boys with blunted ANS functioning. Though the current results add further complexity to this unique development period, additional research will be needed for a more comprehensive understanding of interactions between family environment, physiological functioning, and mental health outcomes.

Limitations and Future Considerations

The present study is among the first to highlight the role of SNS activity and adolescent sex as important conjoint moderators of relations between harsh parenting and adjustment in

later adolescence, however the study is not without limitations. Participants in the study reside primarily in two-parent households in small towns and semi-rural areas and findings may not generalize to other types for family structures and those living in urban and dense environments. Family SES and youth race were significantly associated with adjustment outcomes across all models and may additionally serve as potential moderators of risk. Though they were considered as covariates, the current study is underpowered to assess four-way interactions or multi-group models. Furthermore, adolescents in this study were all youth who were attending high school and living at home and thus findings may not extend to youth who are working full-time or in other independent living situations. A more diverse representation of experiences in mid-to-late adolescence is needed to better understand how harsh parenting and ANS functioning may impact well-being.

The Conflict Tactics Scale is a widely used measure to assess experiences of harsh and aggressive behaviors, however, it may not capture more subtle forms of aggression, particularly those that may be more psychological. Additionally, the measure asks respondents to report on the previous year, which may cause underreporting of the frequency of harsh parenting. For example, it is possible that respondents are reporting the frequency of physical and verbal aggression that were salient or memorable, rather than capturing the total experiences. Nevertheless, significant associations have emerged to indicate that youths' experiences of harsh parenting have long-term effects on maladjustment (Deardorff et al., 2013; Di Giunta et al., 2020; Pinquart, 2017). Future studies could examine multiple dimensions of harsh parenting, including those related to psychological control to better assess how parenting impacts youth. Additionally, recent reviews on parenting in adolescence have called for greater attention to how positive and supportive aspects of parenting may serve as a protective factor in relations between

stressful family environments and internalizing and externalizing outcomes, particularly for youth who may be vulnerable to poor outcomes (Morris et al., 2021). Though it was beyond the scope of the current study, future investigations may benefit from examining positive dimensions of parenting (i.e., warmth, support) in addition to harsh parenting in investigations of youth wellbeing.

A few methodological issues should be considered when interpretating the results. Though this study examined both parent and youth reports of harsh parenting as predictors, mono-informant bias may be a concern as youth reported on both harsh parenting and their own adjustment symptoms. It may be important to consider additional models in future studies to parse out potentially more nuanced and complex associations between harsh parenting, ANS reactivity, and trajectories of adjustment outcomes. It is possible that centering the intercept at an earlier time point (i.e., age 16) may provide further clarification about the stability in adjustment outcomes. However, for analyses in the current study in which no slope effects were detected, it would be excepted that intercept effects at age 16 would be similar to intercept results at age 18, consistent with the observed non-significant slopes.

Despite evidence for dual/risk concerning the role of ANS functioning in relations between harsh parenting and adjustment, other theories are plausible. The inclusion of positive parenting dimensions would allow for a comparison of biological sensitivity to context and dual risk/diathesis stress perspectives in later adolescence and provide clarity concerning the role of ANS reactivity as risk or resilience factor. Furthermore, due to the null effects of harsh parenting on change in adjustment outcomes, future studies may need to examine bi-directional associations between adjustment and harsh parenting, as well as moderation by ANS reactivity in such links. The current findings illustrate that blunted physiological reactivity is a risk factor for

poor adjustment outcomes for older adolescents in the context of harsh parenting. Overall, lengthening of PEP (lower PEPr) served as a risk factor for increasing anxiety and depression symptoms across mid-to-late adolescence for girls who experience greater harsh parenting. In addition to worsening internalizing outcomes, girls with lower ANS reactivity across multiple indices (RSA, SCL, and PEP) had elevated levels of aggressive behavior at age 18. The use of a longitudinal and multi-method design allowed for a better understanding of how individual and family factors interact to predict socioemotional well-being, as well as highlight which groups may be especially vulnerable during this developmental period. Furthermore, this study sheds new light on the conjoint influence of PEPr and adolescent sex on relations between harsh parenting and adjustment, and future research is necessary to replicate or refute these findings.

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		Age 16			Age 17			Age 18	
	п	M	SD	п	M	SD	п	M	SD
Age (in months)	225	0.87	0.98	_	—	_	_	—	_
SES	232	2.41	1.30	_	_	_	—	—	_
Harsh Parenting (youth)	227	4.28	4.50	_	_	_	—	—	_
Harsh Parenting (parent)	217	3.42	3.51	_	_	_	_	—	_
RSAb	222	6.90	1.14	_	_	_	_	—	_
RSAr	222	-0.21	0.84	_	_	_	_	—	_
SCLb	152	9.16	4.98	_	_	_	_	—	_
SCLr	142	1.87	2.24	_	—	_	—	_	_
PEPb	194	119.78	10.53	_	—	_	_	_	_
PEPr	167	-4.01	7.28	_	_	_	_	_	_
Depression symptoms	231	7.64	6.37	218	7.83	6.88	205	8.21	7.20
Anxiety symptoms	231	10.17	8.46	219	9.14 ^a	8.68	205	9.93	9.16
Rule-breaking behavior	226	3.81	3.13	209	3.72	3.08	189	3.95 ^a	2.87
Aggressive behavior	226	6.53	5.20	209	5.24 ^a	4.32	186	5.33	4.34

Descriptive Statistics of Continuous Study Variables

Note. SES = socioeconomic status, operationalized as family income-to-needs ratio. RSAb = Respiratory Sinus Arrythmia baseline ^a = differs significantly from previous time point.

Sex and Race Differences in Harsh Parenting, Physiological, and Adjustment among Adolescents

	Whi	ite	В	lack		Gi	rls	В		
-	М	SD	М	SD	<i>t</i> -value	М	SD	М	SD	<i>t</i> -value
Harsh Parenting (youth)	3.87	4.33	5.10	4.77	-1.95*	3.84	3.79	4.76	5.16	-1.51
Harsh Parenting (parent)	3.42	3.21	3.43	2.99	-0.01	3.00	2.69	3.86	3.50	-1.99*
RSAr	-0.18	0.80	-0.28	0.91	0.80	-0.25	0.81	-0.17	0.88	-0.68
SCLr	1.79	2.32	2.06	2.00	-0.62	2.02	2.30	1.73	2.20	0.74
PEPr	-4.06	6.66	-3.90	8.67	-0.14	-3.39	7.06	-4.60	7.48	1.06
Depression symptoms (Age 16)	7.56	6.46	7.84	6.23	-0.32	8.67	6.33	6.51	6.24	2.61**
Depression symptoms (Age 17)	7.82	7.06	7.78	6.65	0.04	7.93	6.63	7.65	6.63	0.30
Depression symptoms (Age 18)	8.16	7.38	8.44	7.05	-0.25	8.77	6.87	7.63	7.68	1.11
Anxiety symptoms (Age 16)	10.05	8.36	10.42	8.73	-0.31	12.25	8.35	7.84	8.00	4.09***
Anxiety symptoms (Age 17)	9.56	9.15	8.28	7.74	1.02	11.11	8.78	6.78	8.05	3.74***
Anxiety symptoms (Age 18)	9.73	9.13	10.46	9.52	-0.52	11.53	8.69	8.06	9.56	2.70**
Rule-breaking behavior (Age 16)	3.90	3.17	3.62	3.06	0.63	3.32	2.81	4.32	3.37	-2.42*
Rule-breaking behavior (Age 17)	3.81	3.28	3.49	2.45	0.71	3.06	2.34	4.47	3.56	-3.28***
Rule-breaking behavior (Age 18)	4.11	2.96	3.65	2.72	1.02	3.35	2.46	4.74	3.19	-3.27***
Aggressive behavior (Age 16)	6.55	4.95	6.49	5.68	0.08	7.16	5.31	5.85	5.00	1.90
Aggressive behavior (Age 17)	5.25	4.34	5.12	4.18	0.21	5.52	4.09	4.83	4.49	1.15
Aggressive behavior (Age 18)	5.34	4.28	5.23	4.54	0.16	5.61	4.48	4.93	4.18	1.06

Note: RSAr = Respiratory Sinus Arrythmia reactivity; SCLr = Skin Conductance Level Reactivity; PEPr = Pre-Ejection Period reactivity.

* *p* < .05. ** *p* < .01. *** *p* < .001.

	1	2	ng Harsh 3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. HP (youth)																				Ī
2. HP (parent)	.27***																			ľ
3. RSAb	.07**	05																		ľ
4. RSAr	01	.05	29***																	ľ
5. SCLb	14	24**	01	17*																ſ
6. SCLr	.13	.08	.04	10	27***															ľ
7. PEPb	.08	.14	11	.17*	.01	07														I
8. PEPr	01	.00	.15*	.07	.00	18	24**													ľ
9. Dep. – 16y	.26***	01	03	03	10	11	05	.07												ľ
10. Dep. –17y	.22**	.05	02	.00	03	24*	07	.16	.63***											I
11. Dep. –18y	.32***	04	04	08	.03	08	04	.05	.61***	.72***										I
12. Anx. –16y	.24***	09	.02	02	08	00	10	.01	.77***	.50***	.50***									I
13. Anx. –17y	.19**	.03	13	.01	01	18 ∗	04	.01	.60***	.67***	.58***	.67***								
14. Anx. –18y	.22**	01	04	03	01	12	08	.09	.53***	.60***	.75***	.59***	.73***							
15. Rule -16y	.40***	.09	06	.07	10	04	.12	.02	.33***	.28***	.25***	.17**	.15*	.11						
16. Rule –17y	.30***	.03	04	02	.10	.05	.02	.03	.28***	.40***	.43***	.09	.17**	.22**	60***					
17. Rule –18y	.35***	.07	03	05	.03	11	.03	.10	.36***	.39***	.43***	.16*	.17*	.31***	.59***	.69***				
18. Agg. –16y	.43***	.08	.01	.05	10	01	06	.09	.55**	.42***	.41***	.45***	.36***	.39***	.68***	.44***	.49***			
19. Agg. –17y	.37***	.12	.01	05	13	07	11	.11	.46***	.51***	.46**	.31***	.39***	.41***	.42***	.65***	.51 **.	.71***		
20. Agg. –18y	.30**	.11	06	04	07	05	07	.11	.53***	.57***	.64***	.36***	.43***	.57***	.43***	.54***	.65***	.63***	.73***	

Table 3	
Correlations among Harsh Parenting, Physiological Reactivity and Adjustment for Adolescents	

Note: RSAb = Respiratory Sinus Arrythmia baseline; RSAr = Respiratory Sinus Arrythmia reactivity; SCLb = Skin Conductance Level baseline; SCLr = Skin Conductance Level Reactivity; PEPb = Pre-Ejection Period baseline; PEPr = Pre-Ejection Period reactivity.

* p < .05, ** p < .01, *** p < .001.

		Depre	ssion			ety]	reaking		Aggressive Behavior							
	Interce	pt	Slope		Intercept		Slope		Intercept		Slope		Interce	pt	Slope		
	Estimate SE		SE Estimate SE		Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	
Means (µ)	8.07***	0.48	0.16	0.21	9.41***	0.61	-0.25	0.28	4.06***	0.20	0.15	0.10	5.13***	0.31	-0.54***	0.15	
Variances (σ^2)	43.94***	6.37	4.13~	2.29	71.64***	9.55	10.20**	3.43	7.63***	1.23	0.87~	0.48	14.73***	2.32	0.86	1.03	
I/S correlation	7.97**				13.98**					1.0	6~		-0.22				

Unconditional Models for Growth in Adjustment in Late Adolescence

Note. ~ *p* < .10. * *p* < .05. *** *p* < .001.

Effects of Youth-Reported Harsh Parenting on Growth in Internalizing Symptoms and Externalizing Behaviors in	
Adolescence ($N = 242$)	

		Depres	ssion			Anxi	ety]	Rule Br	eaking		Aggressive Behavior				
-	Intercept Slope		e	Interce	Slop	Slope		Intercept		Slope		Intercept		e			
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	
SES	-1.40***	0.36	-0.58***	0.16	-1.23**	0.46	-0.63**	0.22	-0.43**	0.15	-0.08	0.80	-0.72***	0.20	-		
Race	-1.24	0.99	-0.47	0.46	-1.31	1.30	-0.24	0.63	-0.77~	0.42	-0.05	0.22	-1.14*	0.55	-		
Sex	-2.12*	0.91	0.01	0.42	-4.60***	1.16	0.20	0.56	1.02***	0.38	0.15	0.20	-1.56**	0.50	-		
Harsh Parenting (youth)	0.47***	0.11	0.05	0.05	0.44***	0.13	-0.04	0.07	0.19***	0.04	-0.04~	0.02	0.39***	0.06	-		
Means (µ)	10.87***	1.25	1.51**	0.57	13.11***	1.60	1.46~	0.78	4.08***	0.54	0.46†	0.28	6.29***	0.73	-0.55***	0.15	
Variances (σ^2)	37.70***	5.79	4.96*	2.11	59.79***	8.51	8.90**	3.22	6.02***	1.06	0.88*	0.44	11.09***	1.39	0.46	0.52	
I/S correlation	7.62**		12.37**				1.10*					-					
R^2	18.2%	5	10.8%	⁄0	14.99	%	7.0	%	19.6%		4.3%		27.7%		-		

Effects of Parent-Reported Harsh Parenting on Growth in Internalizing Symptoms and Externalizing Behaviors in	
Adolescence ($N = 242$)	

		Depres	ssion			Anxi	ety			Rule Br	eaking		Aggressive Behavior				
-	Intercept Slope		е	Interce	pt	Slop	Slope		Intercept		Slope		Intercept		е		
_	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	
SES	-1.43***	0.37	-0.57***	0.16	-1.28**	0.47	-0.63**	0.22	-0.49**	0.16	-0.07	0.80	-0.56***	0.22	-		
Race	-0.66	1.03	-0.41	0.46	-0.79	1.32	-0.34	0.63	-0.55	0.43	-0.09	0.22	-0.77	0.60	-		
Sex	-1.52	0.94	0.12	0.42	-4.21***	1.19	0.06	0.56	1.19**	0.40	0.13	0.20	-1.30*	0.55	-		
Harsh Parenting (parent)	-0.06	0.16	-0.04	0.07	0.07	0.20	0.10	0.09	0.02	0.07	-0.01	0.03	0.15~	0.09	-		
Means (µ)	12.68***	1.33	1.77**	0.58	14.50***	1.66	1.00	0.78	4.80***	0.56	0.35	0.28	7.53***	0.78	-0.54***	0.15	
Variances (σ^2)	41.48***	6.13	4.49*	2.21	63.11***	8.86	8.75**	3.32	6.62***	1.14	0.81~	0.47	13.38***	1.59	-1.09*	0.56	
I/S correlation	7.60**		11.71**				0.87					-					
R^2	8.5%		10.6%	⁄o	9.6%	<u>⁄o</u>	7.7	%	10.7%	1	1.6%		11.1%		-		

Interaction Effects of Youth-Reported Harsh Parenting and RSAr on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depres	ssion			Anx	iety		-	Rule Br	eaking		Ag	gressive	e Behavior	
	Interce	pt	Slop	е	Interce	pt	Slope	2	Interc	ept	Slo	ре	Interce	ept	Slope	е
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estim	SE	Estimate	SE	Estimate	SE
			0.50111				0.50111	0.1.6	0.1011		ate		0 60444			
SES	-1.42***	0.36	-0.59***	0.16	-1.42***	0.36	-0.59***	0.16	-0.42**	0.15	-0.07	0.08	-0.69***	0.20	-	-
Race	-1.07	1.00	-0.46	0.46	-1.07	0.99	-0.46	0.46	-0.67	0.42	-0.03	0.22	-0.98~	0.55	-	-
Sex	-2.22**	0.90	0.02	0.42	-2.22**	0.90	0.02	0.42	0.97**	0.38	0.15	0.20	-1.66***	0.50	-	-
Harsh Parenting (youth)	0.49***	0.10	0.06	0.05	0.49***	0.13	0.06	0.05	0.20***	0.04	-0.04~	0.02	0.40***	0.06	-	-
RSAb	-0.60	0.43	-0.11	0.20	-0.60	0.43	-0.11	0.20	-0.20	0.18	-0.02	0.09	-0.37	0.23	-	-
RSAr	-0.69	0.59	-0.27	0.28	-0.69	0.59	-0.27	0.28	-0.22	0.24	-0.17	0.12	-0.17	0.31	-	-
HP x RSAr	0.02	0.13	-0.04	0.07	0.02	0.13	-0.04	0.07	0.07	0.05	0.03	0.03	0.09	0.06	-	-
Means (µ)	8.09***	0.44	0.19	0.20	8.09***	0.44	0.19	0.20	4.08***	0.19	0.16†	0.10	5.10***	0.27	-0.56***	0.15
Variances (σ^2)	37.52***	5.78	5.10*	2.13	37.52***	5.78	5.10*	2.13	5.84***	1.05	0.83~	0.44	10.79***	1.36	0.56	0.52
I/S correlation		7.81	**			7.8	[**			1.0	2~			-		
R^2	20%		12%)	19.8	%	129	⁄₀	21.3%)	7.6%	6	29.5%	⁄ 0	-	

Interaction Effects of Youth-Reported Harsh Parenting and SCLr on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depre	ssion			Any	kiety			Rule E	Breaking		Ag	gressive	Behavior	
	Interce	pt	Slop	е	Interce	pt	Slope	2	Interce	ept	Slop	е	Interce	ept	Slope	е
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.33***	0.36	-0.63***	0.16	-1.10*	0.47	-0.68**	0.23	-0.42**	0.15	-0.07	0.08	-0.64**	0.21	-	-
Race	-0.18	1.10	0.13	0.50	-0.72	1.43	0.11	0.69	-0.67	0.42	-0.03	0.22	-1.26*	0.61	-	-
Sex	-2.31**	0.89	-0.03	0.41	-4.98***	1.16	0.08	0.56	0.97**	0.38	0.15	0.20	-1.67***	0.50	-	-
Harsh Parenting (youth)	0.47***	0.11	0.04	0.05	0.48***	0.14	-0.01	0.07	0.20***	0.04	-0.04~	0.02	0.40***	0.06	-	-
SCLb	0.19	0.13	0.16**	0.06	0.07	0.16	0.10	0.08	-0.20	0.18	-0.02	0.09	-0.07	0.07	-	-
SCLr	-0.41	0.30	0.13	0.14	-0.82*	0.35	-0.19	0.17	-0.22	0.24	-0.17	0.12	-0.33*	0.15	-	-
HP x SCLr	0.09	0.06	0.03	0.03	0.08	0.07	-0.001	0.03	0.07	0.05	0.03	0.03	0.01	0.03	-	-
Means (µ)	8.02***	0.44	0.19	0.20	9.34***	0.58	-0.20	0.28	4.08***	0.19	0.16†	0.10	5.07***	0.28	-0.55***	0.15
Variances (σ^2)	33.75***	5.77	3.49†	2.12	54.22***	8.38	7.70*	3.21	5.84***	1.05	0.83~	0.44	10.62***	1.37	0.47	0.52
I/S correlation	6.13*				10.4	10**			1.	02~			-			
R^2	25.2%)	25.8%	6	22.0)%	13.2	2%	22.5%	6	9.2%	<i>,</i> 0	31.1%	, D	-	

Interaction Effects of Youth-Reported Harsh Parenting and PEPr on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depres	ssion			Anx	iety			Rule E	Breaking		Ag	gressive	e Behavior	
	Interce	pt	Slop	е	Interce	pt	Slope	2	Interco	ept	Slop	е	Interce	ept	Slope	2
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.39***	0.36	-0.60***	0.16	-1.23**	0.46	-0.67**	0.22	-0.42**	0.15	-0.06	0.08	-0.69***	0.20	-	-
Race	-1.23	0.99	-0.49	0.46	-1.27	1.30	-0.30	0.63	-0.76~	0.42	-0.03	0.21	-1.12*	0.54	-	-
Sex	-1.98*	0.94	-0.06	0.44	-4.61***	1.20	0.04	0.58	1.10**	0.39	0.24	0.20	-1.52**	0.51	-	-
Harsh Parenting (youth)	0.47***	0.10	0.05	0.05	0.44***	0.13	-0.05	0.07	0.19***	0.04	-0.04~	0.02	0.40***	0.06	-	-
PEPb	0.00	0.05	0.01	0.02	0.02	0.07	0.04	0.03	-0.00	0.02	-0.02	0.01	-0.02	0.03	-	-
PEPr	0.10	0.08	0.01	0.04	0.12	0.10	0.09~	0.05	0.05	0.03	0.01	0.02	0.07~	0.04	-	-
HP x PEPr	0.003	0.02	0.01	0.01	0.01	0.02	0.02	0.01	0.01	0.01	0.001	0.00	0.03**	0.01	-	-
Means (µ)	8.07***	0.44	0.19	0.20	9.43***	0.58	-0.22	0.28	4.09***	0.19	0.16~	0.10	5.10***	0.28	-0.55***	0.15
Variances (σ^2)	36.41***	5.75	4.43*	2.14	59.17***	8.50	8.45**	3.20	5.70***	1.06	0.78~	0.45	10.32***	1.34	0.46	0.51
I/S correlation	7.11**				11.8	34**			0.	94~			-			
R^2	19.7% 12.6%			/ ₀	16.0)%	12.5	5%	22.3%	0	9.5%	0	32.8%	, D	-	

Interaction Effects of Youth-Reported Harsh Parenting, RSAr, and Adolescent Sex on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depres	ssion	·1		Any	xiety	·	-	Rule B	reaking		Ag	gressive	e Behavior	
	Intercep	pt	Slope	e	Interce	pt .	Slop	<i>ie</i>	Interce	ept	Slope	e	Interce	ept	Slope	2
· · · · · ·	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.42***	0.35	-0.60***	0.16	-1.17**	0.45	-0.63**	0.22	-0.44**	0.15	-0.10	0.08	-0.68***	0.19	-	-
Race	-0.76	1.00	-0.32	0.48	-0.81	1.32	0.01	0.65	-0.58	0.41	-0.07	0.22	-0.92~	0.55	-	-
Sex	-2.17**	0.88	0.09	0.42	-4.91***	1.15	0.18	0.57	0.98**	0.36	0.15	0.19	-1.72***	0.48	-	-
Harsh Parenting (youth)	0.59***	0.11	0.06	0.05	0.57***	0.14	-0.04	0.07	0.24	0.04	-0.02	0.02	0.47***	0.06	-	-
RSAb	-0.60	0.42	-0.13	0.20	-0.90	0.55	-0.35	0.27	-0.20	0.17	-0.02	0.09	-0.33	0.23	-	-
RSAr	-0.83	0.58	-0.30	0.28	-0.35	0.75	-0.03	0.38	-0.24	0.23	-0.16	0.12	-0.21	0.31	-	-
HP x RSAr	0.00	0.13	-0.05	0.07	0.25	0.16	0.02	0.08	0.05	0.05	0.03	0.03	0.09	0.06	-	-
HP x Sex	-0.29	0.22	0.02	0.11	-0.37	0.28	0.01	0.14	-0.18*	0.09	-0.11**	0.04	-0.27*	0.12		
RSAr x Sex	-2.76*	1.14	-0.80	0.55	-2.10	1.48	-0.84	0.74	-1.54***	0.46	-0.38	0.24	-1.33*	0.60		
HP x RSAr x Sex	-0.48~	0.26	-0.04	0.14	-0.27	0.32	0.02	0.17	-0.06	0.10	0.07	0.05	-0.31**	0.13		
Means (μ)	8.29***	0.43	0.21	0.20	9.56***	0.58	-0.24	0.28	4.18***	0.18	0.20*	0.09	5.25***	0.27	-0.56***	0.15
Variances (σ^2)	36.13***	5.64	5.68**	2.09	54.75***	8.20	8.03**	3.24	5.06***	0.99	0.67	0.43	9.76***	1.26	0.60	0.52
I/S correlation		8.16)**			10.8	84**			0.7	76			-		
R^2	24.8%	<u>)</u>	12.4%	%	20.4	1%	10.	.4%	30.2%	0	21.3%	ó	35.9%	ó	-	

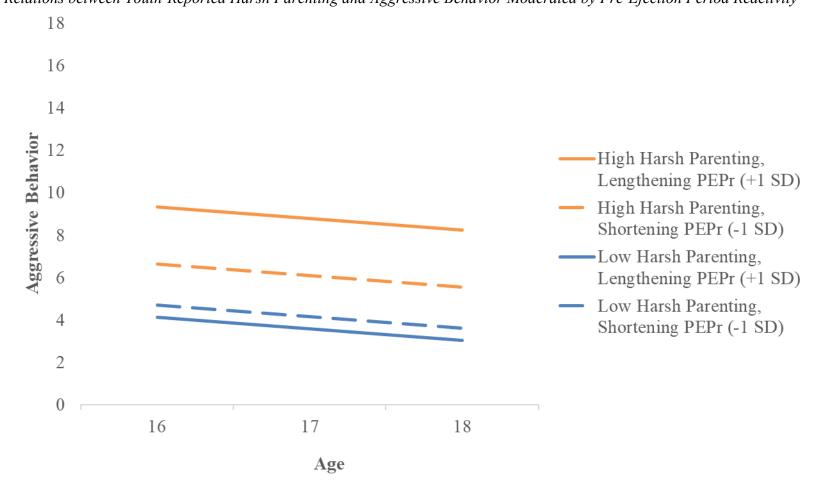
Interaction Effects of Youth-Reported Harsh Parenting, SCLr, and Adolescent Sex on Growth in Internalizing Symptoms	
and Externalizing Behaviors in Adolescence ($N = 242$)	

		Depres	ssion			Any	xiety		-	Rule B	reaking		Ag	gressive	e Behavior	
	Intercep	pt	Slope	e	Interce	pt	Slope	е	Interce	ept	Slope	е	Interce	ept	Slope	?
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.34***	0.36	-0.64***	0.16	-1.23**	0.47	-0.72***	0.23	-0.45**	0.15	-0.11	0.08	-0.68***	0.21	-	-
Race	-0.48	1.10	-0.02	0.50	-1.02	1.44	0.04	0.69	-0.90*	0.46	-0.02	0.23	-1.43*	0.61	-	-
Sex	-2.46**	0.90	-0.01	0.41	-5.39***	1.18	-0.03	0.57	0.83*	0.38	0.04	0.20	-1.91***	0.51	-	-
Harsh Parenting (youth)	0.50***	0.11	0.04	0.06	0.52***	0.14	-0.01	0.07	0.23***	0.05	-0.02	0.02	0.44***	0.06	-	-
SCLb	0.20	0.13	0.17**	0.06	0.07	0.16	0.09	0.08	0.00	0.05	0.04	0.03	-0.07	0.07	-	-
SCLr	-0.23	0.31	0.26~	0.14	-0.86*	0.35	-0.20	0.17	-0.20~	0.11	-0.03	0.06	-0.38**	0.15	-	-
HP x SCLr	0.15*	0.08	0.07*	0.04	0.04	0.08	-0.02	0.04	0.00	0.03	0.00	0.01	-0.02	0.04	-	-
HP x Sex	-0.29	0.23	0.01	0.11	-0.36	0.29	-0.02	0.14	-0.19*	0.09	-0.14**	0.05	-0.26*	0.12		
SCLr x Sex	-0.86*	0.58	-0.69**	0.26	-0.68	0.68	-0.39	0.32	-0.17	0.22	-0.04	0.11	0.14	0.29		
HP x SCLr x Sex	-0.05	0.15	-0.07	0.07	0.24	0.16	0.08	0.07	0.05	0.05	0.04†	0.03	-0.13*	0.07		
Means (μ)	7.95***	0.45	0.09	0.21	9.35***	0.59	-0.24	0.29	4.11***	0.19	0.21*	0.09	5.16***	0.27	-0.56***	0.15
Variances (σ^2)	33.89***	5.81	3.15	2.18	52.73***	8.33	7.75*	3.21	5.68***	1.03	0.77~	0.43	10.04***	1.33	0.51	0.52
I/S correlation	6.20*		9*			10.1	10**			0.9	15~			-		
R^2	28.1%	3	38.2%	%	24.8	5%	15.7	7%	25.2%	, D	19.1%	Ó	34.6%	ó	-	

		Depres	ssion			Anx	iety			Rule Bı	reaking		Ag	gressive	e Behavior	
	Interce	pt	Slop	е	Interce	pt	Slope	2	Interce	ept	Slop	е	Interce	ept	Slope	2
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.41***	0.36	-0.61***	0.16	-1.25**	0.46	-0.67**	0.22	-0.41**	0.15	-0.06	0.08	-0.69***	0.20	-	-
Race	-1.42	1.00	-0.48	0.47	-1.58	1.31	-0.39	0.63	-0.94*	0.42	-0.12	0.22	-1.29*	0.54	-	-
Sex	-2.18*	0.92	-0.10	0.44	-4.95***	1.19	-0.15	0.58	1.05**	0.39	0.24	0.20	-1.64***	0.50	-	-
Harsh Parenting (youth)	0.51***	0.11	0.05	0.06	0.50***	0.14	-0.04	0.07	0.23***	0.05	-0.02	0.02	0.43***	0.06	-	-
PEPb	0.02	0.05	0.02	0.02	0.03	0.06	0.05	0.03	0.00	0.02	-0.01	0.01	-0.01	0.03	-	-
PEPr	0.15~	0.08	0.03	0.04	0.17	0.10	0.11*	0.05	0.07*	0.03	0.02	0.02	0.10*	0.04	-	-
HP x PEPr	0.04~	0.02	0.02	0.01	0.05	0.03	0.04	0.01	0.02~	0.01	0.00	0.01	0.04**	0.01	-	-
HP x Sex	-0.28	0.22	-0.01	0.11	-0.35	0.27	-0.04	0.14	-0.20*	0.09	-0.13**	0.05	-0.24*	0.12		
PEPr x Sex	0.01	0.16	0.01	0.07	-0.04	0.20	-0.01	0.10	0.02	0.07	0.03	0.04	0.01	0.09		
HP x PEPr x Sex	-0.13**	0.05	-0.04~	0.02	0.12*	0.05	-0.07**	0.03	-0.03~	0.02	-0.00	0.01	-0.04*	0.02		
Means (μ)	8.25***	0.43	0.23	0.20	9.66***	0.58	-0.13	0.28	4.23***	0.19	0.23*	0.10	5.25***	0.28	-0.54***	0.15
Variances (σ^2)	33.36***	5.60	4.39*	2.11	55.78***	8.37	7.81**	3.19	5.54***	1.04	0.82~	0.43	9.62***	1.29	0.67	0.52
I/S correlation		6.30	5*			10.2	3**			0.9	4~			-		
R^2	26.6%	, D	18.2	V ₀	20.4	%	21.8	8%	27.1%)	18.3%	⁄ 0	38.6%	, D	-	

Interaction Effects of Youth-Reported Harsh Parenting, PEPr, and Adolescent Sex on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

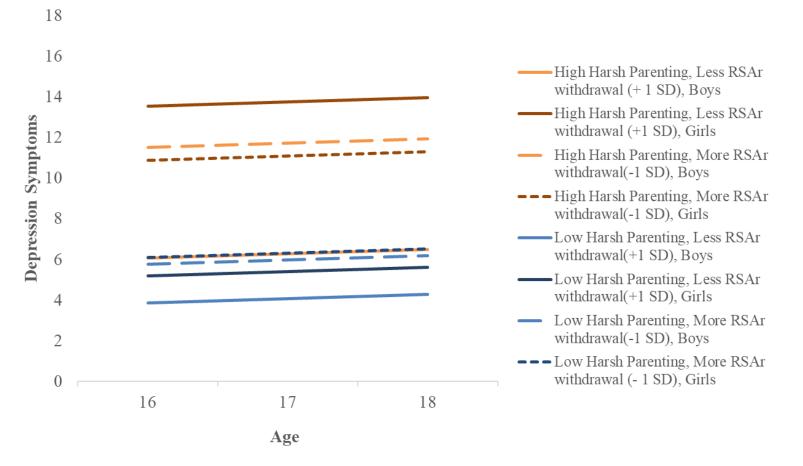




Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP in milliseconds (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Model accounts for the effects of sex, race, family SES, and baseline PEP.



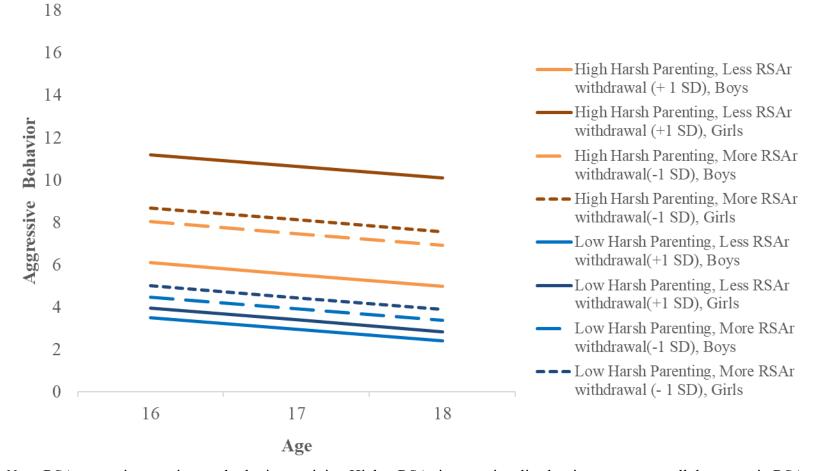
Relations between Youth-Reported Harsh Parenting and Depression Symptoms Moderated by Respiratory Sinus Arrhythmia Reactivity and Adolescent Sex



Note. RSAr = respiratory sinus arrhythmia reactivity. Higher RSAr is operationalized as increases or small decreases in RSA or less withdrawal (+1 SD, 0.63) and lower RSAr is operationalized as decreases in RSA or greater withdrawal (-1 SD, -1.05). Model accounts for the effects of sex, race, family SES, baseline RSA, and lower-order two-way interactions. *Marginal Interaction*.

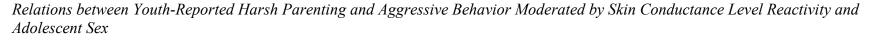
Figure 3.

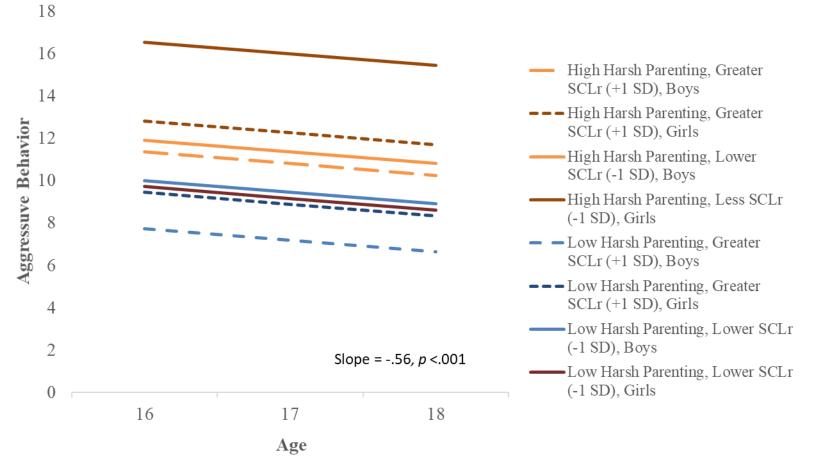
Relations between Youth-Reported Harsh Parenting and Aggressive Behavior Moderated by Respiratory Sinus Arrhythmia Reactivity and Adolescent Sex



Note. RSAr = respiratory sinus arrhythmia reactivity. Higher RSAr is operationalized as increases or small decreases in RSA or less withdrawal (+1 SD, 0.63) and lower RSAr is operationalized as decreases in RSA or greater withdrawal (-1 SD, -1.05). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline RSA, and lower-order two-way interactions.

Figure 4.

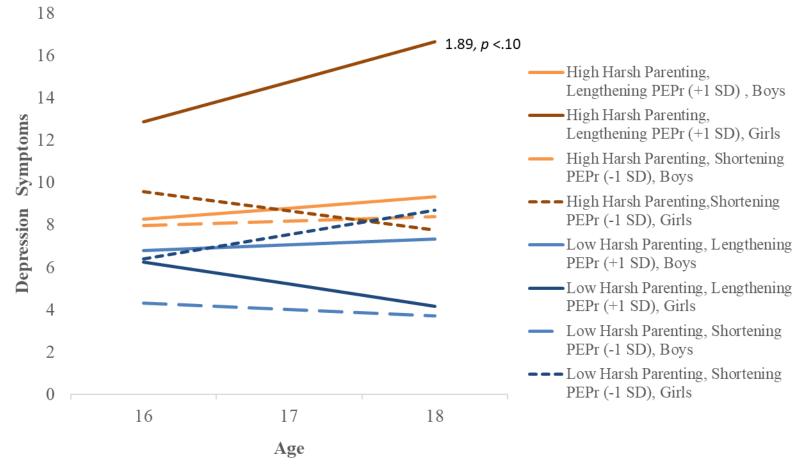




Note. SCLr = skin conductance level reactivity. Higher SCLr is operationalized as increases in SCL (+1 SD, 4.11) and lower SCLr is operationalized as small increases or decreases (-1 SD, -0.37). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline SCL, and lower-order two-way interactions.

Figure 5.

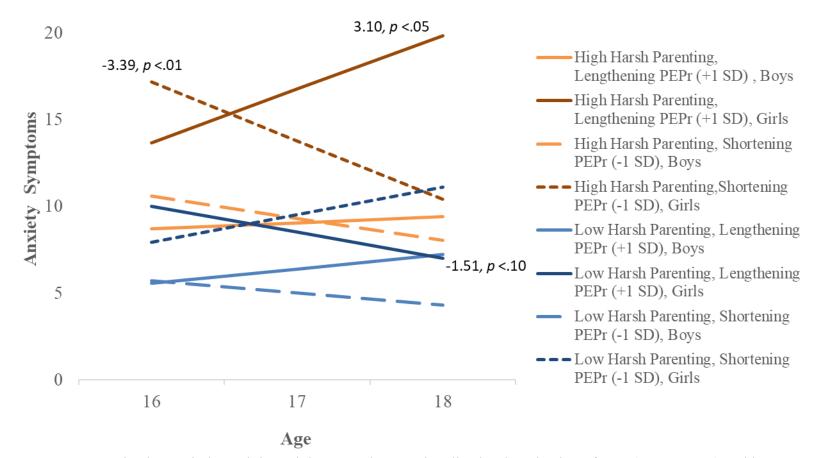
Relations between Youth-Reported Harsh Parenting and Depression Symptoms Moderated by Pre-Ejection Period Reactivity and Adolescent Sex



Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline PEP, and lower-order two-way interactions. *Marginal Interaction on Slope*.

Figure 6.

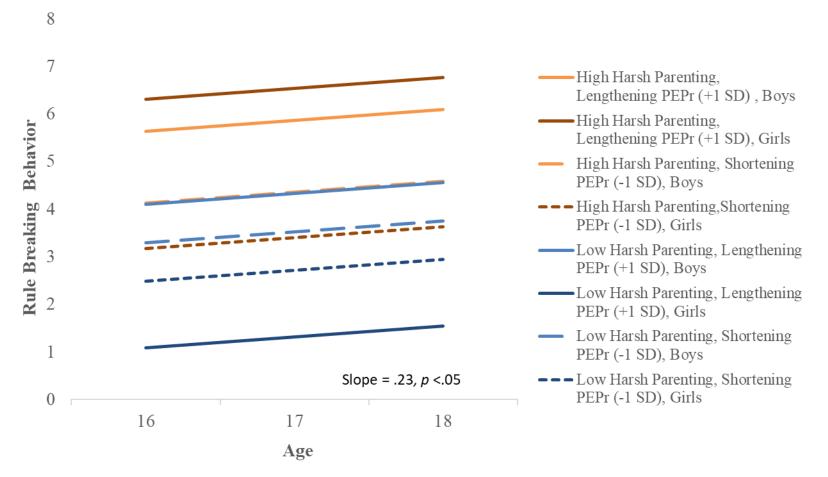
Relations between Youth-Reported Harsh Parenting and Anxiety Symptoms Moderated by Pre-Ejection Period Reactivity and Adolescent Sex



Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline PEP, and lower-order two-way interactions.

Figure 7.

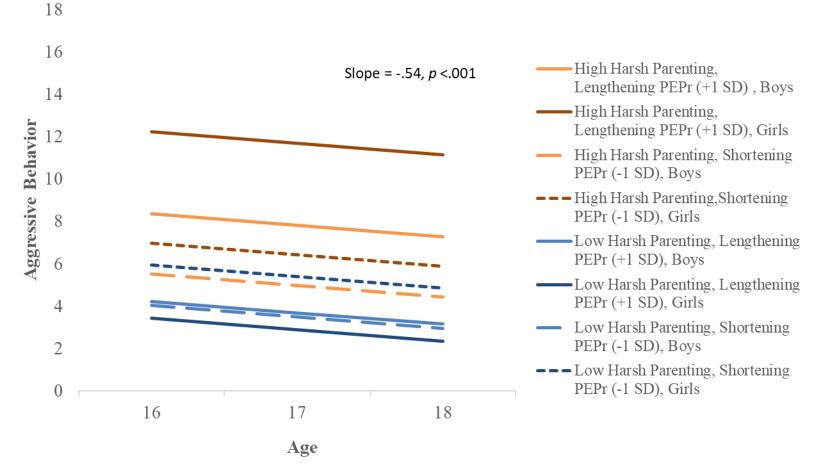
Relations between Youth-Reported Harsh Parenting and Rule-breaking Behavior Moderated by Pre-Ejection Period Reactivity and Adolescent Sex



Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline PEP, and lower-order two-way interactions. *Marginal Interaction*.

Figure 8.

Relations between Youth-Reported Harsh Parenting and Aggressive Behavior Moderated by Pre-Ejection Period Reactivity and Adolescent Sex



Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline PEP, and lower-order two-way interactions.

Appendices

Appendix A Figures and Tables

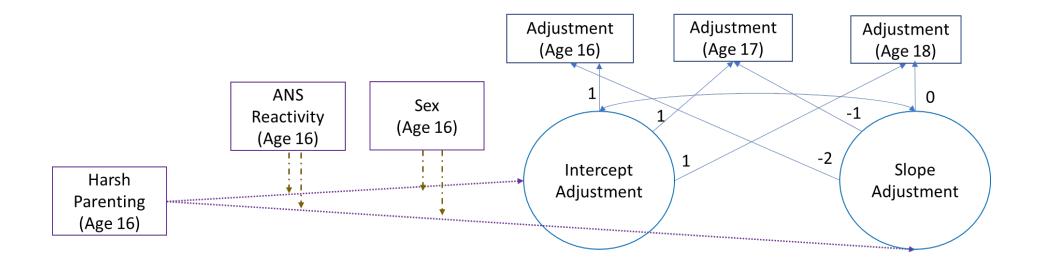
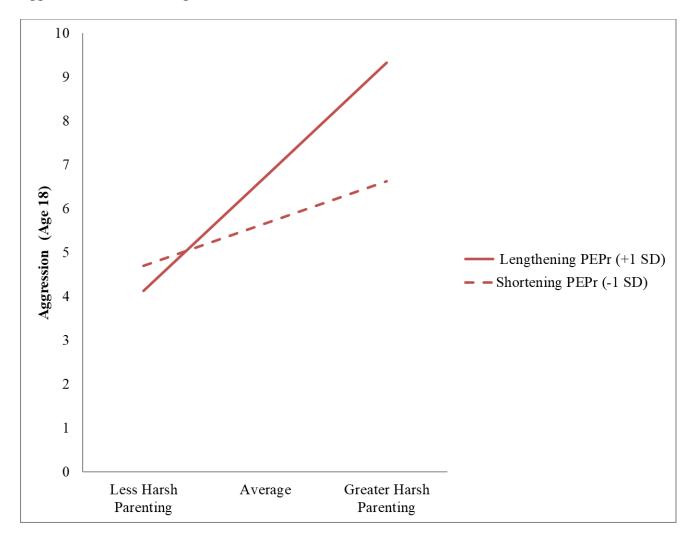


Figure 1a. Conditional model for effects of ANS reactivity and adolescent sex on relations between harsh parenting and growth in adolescent adjustment from ages 16 to 18.

Figure 2a.

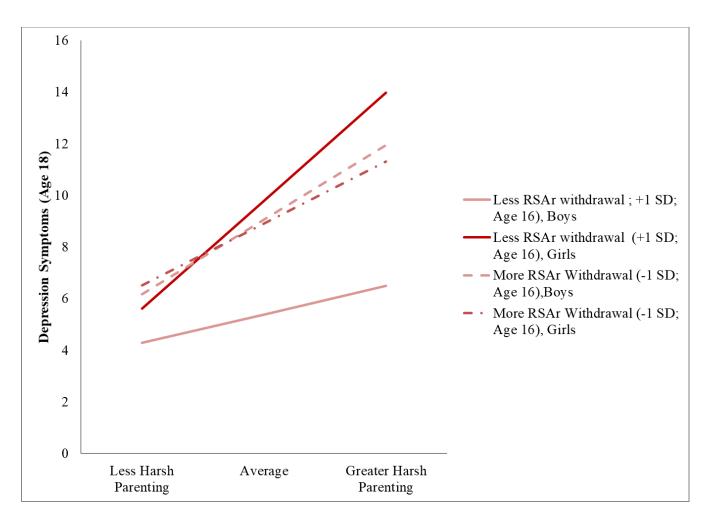


Interaction of Youth-Reported Harsh Parenting and Pre-Ejection Period Reactivity at 16 on Aggressive Behavior at Age 18

Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP in milliseconds (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Model accounts for the effects of sex, race, family SES, and baseline PEP.

Figure 3a.

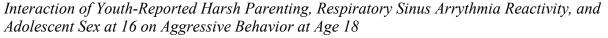
Interaction of Youth-Reported Harsh Parenting, Respiratory Sinus Arrythmia Reactivity, and Adolescent Sex at 16 on Depression Symptoms at Age 18

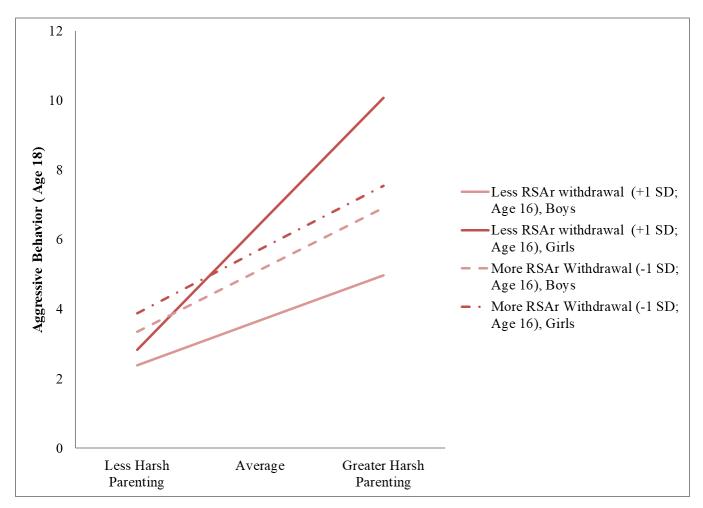


Note. RSAr = respiratory sinus arrhythmia reactivity. Higher RSAr is operationalized as increases or small decreases in RSA or less withdrawal (+1 SD, 0.63) and lower RSAr is operationalized as decreases in RSA or greater withdrawal (-1 SD, -1.05). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline RSA, and lower order two-way interactions.

Marginal Interaction.

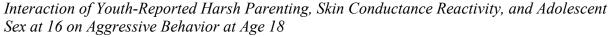
Figure 4a.

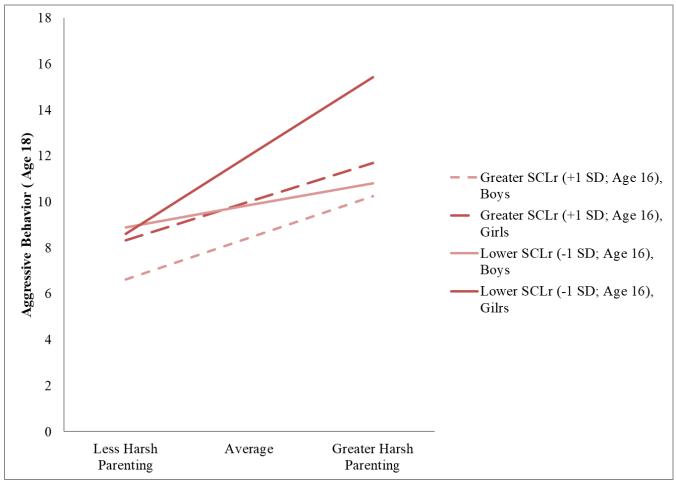




Note. RSAr = respiratory sinus arrhythmia reactivity. Higher RSAr is operationalized as increases or small decreases in RSA or less withdrawal (+1 SD, 0.63) and lower RSAr is operationalized as decreases in RSA or greater withdrawal (-1 SD, -1.05). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline RSA, and lower order two-way interactions.

Figure 5a.

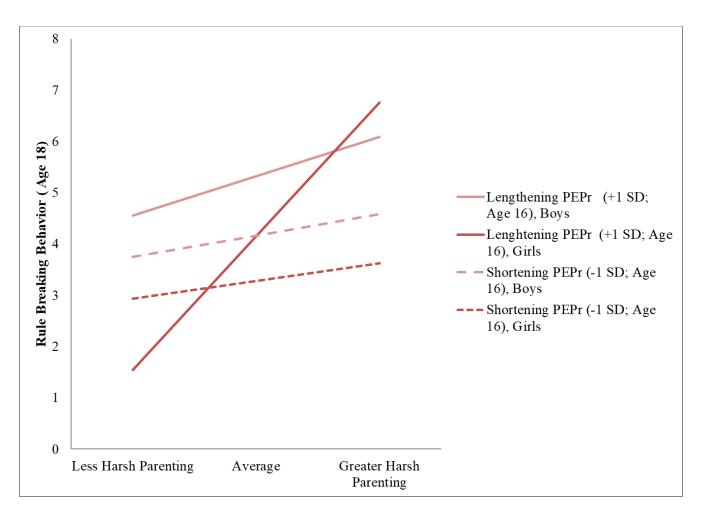




Note. SCLr = skin conductance level reactivity. Higher SCLr is operationalized as increases in SCL (+1 SD, 4.11) and lower SCLr is operationalized as small increases or decreases (-1 SD, - 0.37). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline SCL, and lower-order two-way interactions.

Figure 6a.

Interaction of Youth-Reported Harsh Parenting, Pre-Ejection Period Reactivity, and Adolescent Sex at 16 on Rule Breaking Behavior at Age 18

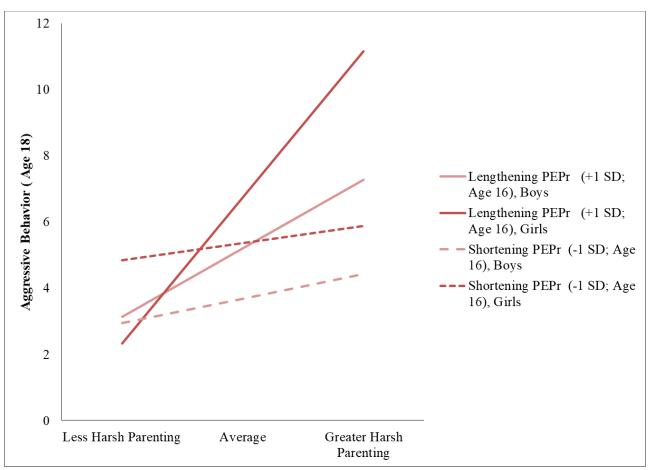


Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, - 11.29). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline PEP, and lower-order two-way interactions.

Marginal Interaction.

Figure 7a.

Interaction of Youth-Reported Harsh Parenting, Pre-Ejection Period Reactivity, and Adolescent Sex at 16 on Aggressive Behavior at Age 18



Note. PEPr = pre-ejection period reactivity. Higher PEPr is operationalized as lengthening of PEP (+1 SD, 3.27) and lower PEPr is operationalized as decreases or shortening of PEP (-1 SD, -11.29). Sex was coded as 0 = boys, 1 = girls. Model accounts for the effects of race, family SES, baseline PEP, and lower-order two-way interactions.

Table 1a

Fit Indices for Models examining Youth-Reported Harsh Parenting, RSAr, and Adolescent Sex

Models	χ^2	df	р	RMSEA	CFI
Depression Symptoms					
Unconditional	0.054	1	0.8158	0.000, p = .86	1.000
Harsh Parenting	7.042	5	0.2175	0.041, p = .51	0.993
Harsh Parenting x RSAr	13.109	8	0.1081	0.051, p = .43	0.983
Harsh Parenting x RSAr x Sex	15.692	11	0.1530	0.042, p = .57	0.985
Anxiety Symptoms					
Unconditional	4.291	1	0.0383	0.117, p = .10	0.989
Harsh Parenting	10.269	5	0.0680	0.066, p = .27	0.984
Harsh Parenting x RSAr	18.028	8	0.0210	0.072, p = .18	0.971
Harsh Parenting x RSAr x Sex	21.612	11	0.0276	0.063, p = .26	0.969
Rule-breaking Behavior					
Unconditional	1.208	1	0.2718	0.030, p = .40	.999
Harsh Parenting	2.746	5	0.7391	0.000, p = .90	1.000
Harsh Parenting x RSAr	3.801	8	0.8746	0.000, p = .98	1.000
Harsh Parenting x RSArx Sex	6.016	11	0.8723	0.000, p = .98	1.000
Aggressive Behavior					
Unconditional	11.067	1	0.0009	0.207, p = .01	0.966
Harsh Parenting	27.018	10	0.0026	0.084, p = .07	0.953
Harsh Parenting x RSAr	35.095	16	0.0039	0.070, p = .13	0.947
Harsh Parenting x RSAr x Sex	44.530	22	0.0030	0.065, p = .17	0.941

Table 2a

Fit Indices for Models examining Youth-Reported Harsh Parenting, SCLr, and Adolescent Sex

Models	χ^2	df	р	RMSEA	CFI
Depression Symptoms					
Unconditional	0.054	1	0.8158	0.000, p = .86	1.000
Harsh Parenting	7.042	5	0.2175	0.041, p = .51	0.993
Harsh Parenting x SCLr	17.698	8	0.0236	0.071, p = .19	0.969
Harsh Parenting x SCLr x Sex	36.340	11	0.0001	0.098, p = .01	0.924
Anxiety Symptoms					
Unconditional	4.291	1	0.0383	0.117, p = .10	0.989
Harsh Parenting	10.269	5	0.0680	0.066, p = .27	0.984
Harsh Parenting x SCLr	16.095	8	0.0410	0.065, p = .26	0.976
Harsh Parenting x SCLr x Sex	23.116	11	0.0170	0.067, p = .20	0.965
Rule-breaking Behavior					
Unconditional	1.208	1	0.2718	0.030, p = .40	.999
Harsh Parenting	2.746	5	0.7391	0.000, p = .90	1.000
Harsh Parenting x SCLr	3.878	8	0.8680	0.000, p = .97	1.000
Harsh Parenting x SCLr Sex	6.100	11	0.8666	0.000, p = .98	1.000
Aggressive Behavior					
Unconditional	11.067	1	0.0009	0.207, p = .01	0.966
Harsh Parenting	27.018	10	0.0026	0.084, p = .07	0.953
Harsh Parenting x SCLr	32.119	16	0.0096	0.065, p = .21	0.955
Harsh Parenting x SCLr x Sex	40.067	22	0.0106	0.058, p = .29	0.951

Table 3a

Fit Indices for Models examining Youth-Reported Harsh Parenting, PEPr, and Adolescent Sex

Models	χ^2	df	р	RMSEA	CFI
Depression Symptoms					
Unconditional	0.054	1	0.8158	0.000, p = .86	1.000
Harsh Parenting	7.042	5	0.2175	0.041, p = .51	0.993
Harsh Parenting x PEPr	15.295	8	0.0536	0.061, p = .30	0.976
Harsh Parenting x PEPr x Sex	19.569	11	0.0516	0.057, p = .35	0.972
Anxiety Symptoms				-	
Unconditional	4.291	1	0.0383	0.117, p = .10	0.989
Harsh Parenting	10.269	5	0.0680	0.066, p = .27	0.984
Harsh Parenting x PEPr	12.481	8	0.1310	0.048, p = .47	0.987
Harsh Parenting x PEPr x Sex	19.531	11	0.0522	0.057, p = .35	0.975
Rule-breaking Behavior					
Unconditional	1.208	1	0.2718	0.030, p = .40	.999
Harsh Parenting	2.746	5	0.7391	0.000, p = .90	1.000
Harsh Parenting x PEPr	7.897	8	0.4436	0.000, p = .79	1.000
Harsh Parenting x PEPr Sex	11.388	11	0.4114	0.012, p = .81	0.999
Aggressive Behavior					
Unconditional	11.067	1	0.0009	0.207, p = .01	0.966
Harsh Parenting	27.018	10	0.0026	0.084, p = .07	0.953
Harsh Parenting x PEPr	38.898	16	0.0011	0.077, p = .07	0.938
Harsh Parenting x PEPr x Sex	50.121	22	0.0006	0.073, p = .08	0.926

Appendix B

Additional Analyses

Results for Parent-Reported Harsh Parenting Interactions

Harsh Parenting as a Predictor of Adjustment (Aim 1).

In models with parent-reported harsh parenting as a predictor of adjustment, few associations were found (Table 6). Parent-reported harsh parenting did not predict the intercept or slope of depression symptoms (χ^2 (5) = 6.545, p = .27; χ^2/df = 1.31; RMSEA = .04, p = .56; CFI = 0.99). No significant associations emerged for the intercept or slope of anxiety symptoms in models with parent-reported harsh parenting (χ^2 (5) = 12.836, p = .02; χ^2/df = 2.56; RMSEA = .08, p = .15; CFI = 0.98).

Among externalizing behavior outcomes, parent-reported harsh parenting was not a significant predictor of the intercept or slope of rule-breaking behavior (Table 6; χ^2 (5) = 3.907, p = .56; $\chi^2/df = 0.78$; RMSEA = .00, p = .81; CFI = 1.00). However, greater parent-reported harsh parenting at age 16 was marginally associated with greater aggressive behavior at age 18 (χ^2 (5) = 13.351, p = .20; $\chi^2/df = 1.33$; RMSEA = .04, p = .62; CFI = 0.99).

Physiological Reactivity as a Moderator (Aim 2)

RSAr as a moderator. All models that examined parent-reported harsh parenting and RSAr as predictors of adjustment outcomes fit adequately (Appendix B: Table 7b). RSAr and RSAb were not significant predictors of the intercepts or slopes of any adjustment outcomes (Appendix B: Table 1b). RSAr also did not moderate any associations between parent-reported harsh parenting and adjustment outcomes.

SCLr as a moderator. All models fit the data adequately and SCLb was included as a covariate (Appendix B: Table 8b). In models with parent-reported harsh parenting, SCLb was a significant predictor of the slope of depression, such that greater baseline SCL was associated with increases in depression symptoms across adolescence (Appendix B: Table 2b). SCLr was

also a marginally significant predictor of the intercept of anxiety symptoms such that greater SCLr was associated with marginally fewer anxiety symptoms.

An interaction between parent-reported harsh parenting and SCLr approached statistical significance for depression symptoms at age 18 (intercept). The prototypical plot (Appendix B: Figure 1b) illustrates that all adolescents had stable levels of depressive symptoms across ages 16 to 18, and youth with lower parent-reported harsh parenting and low SCLr (solid blue line) had the highest predicted means for depression at age 18 (M = 9.93). These youth had a significant 0.56 *SD* difference in depressive symptoms compared to their counterparts with higher SCLr at age 18 (dashed blue line, $\Delta \chi^2$ [1] = 9.28, p < 0.01). Youth with high levels of harsh parenting had similar levels of depression and no significant differences were noted in the intercepts at age 18 ($\Delta \chi^2$ [1] = 0.11, *ns*). Youth with low harsh parenting and high SCLr had the lowest levels of depression symptoms at age 18 (M = 5.86). Additionally, the prototypical plot in Appendix B Figure 3b also depicts the interaction effect on the intercept of depression at age 18.

A significant two-way interaction emerged where SCLr moderated relations between harsh parenting and rule-breaking behavior at age 18 (intercept). Similar to depression symptoms, adolescents with low harsh parenting and low SCLr had the highest predicted scores for rule-breaking behavior at age 18 (Figure 2b; blue solid line; M = 4.93). These youth had a 0.68 *SD* difference in scores with higher SCLr youth who also experienced lower levels of harsh parenting (blue dashed line; $\Delta \chi^2$ [1] = 15.11, p < 0.001). Additionally, low SCLr and less harsh parenting had a 0.39 *SD* from youth with high harsh parenting and lower SCLr (orange solid line; $\Delta \chi^2$ [1] = 5.19, p < 0.01). Higher SCLr youth with less harsh parenting also had the lowest levels of rule-breaking behavior (M = 2.96). No significant differences in rule-breaking behavior were detected for youth with greater parent reported harsh parenting, regardless of SCLr. The

173

prototypical plot in Appendix B Figure 4b illustrates the specific interaction effect on rulebreaking behavior at age 18.

PEPr as a moderator. All models fit the data adequately (Appendix B: Table 9b) and PEPb was included as a covariate in all models. Among models with parent-reported harsh parenting, PEPr and PEPb did not predict the intercepts of any adjustment symptoms at age 18 (Appendix B: Table 3b). Similarly, baseline and reactivity did not predict trajectories for depression, anxiety, or rule-breaking behavior across late adolescence. No two-way interactions between harsh parenting and PEPr emerged.

Physiological reactivity and Sex as Conjoint Moderators (Aim 3)

Sex and RSAr as moderators. All models fit the data adequately (Appendix B: Table 7b). No significant three-way interactions with parent-reported harsh parenting emerged for adjustment intercepts and slopes (Appendix B: Table 4b).

Sex and SCLr as moderators. All models fit the data adequately (Appendix B: Tables 8b). No significant three-way interactions predicted the intercepts and slopes of any adjustment outcomes in models with parent reported harsh parenting (Appendix B: Table 5b).

Sex and PEPr as moderators. Data fit the models well (Appendix B: Table 9b). No significant three-way interactions predicted the intercepts and slopes of any adjustment outcomes in models with parent reported harsh parenting (Appendix B: Table 6b).

Table 1b

Interaction Effects of Parent-Reported Harsh Parenting and RSAr on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depres	ssion			Any	kiety		-	Rule B	reaking		Ag	gressive	Behavior	
	Interce	pt	Slop	е	Interce	pt	Slop	е	Interce	ept	Slop	е	Interce	ept	Slope	е
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.43***	0.37	-0.56***	0.16	-1.23**	0.47	-0.61**	0.22	-0.50**	0.16	-0.08	0.08	-0.83***	0.22	-	-
Race	-0.52	1.05	-0.37	0.46	-0.40	1.33	-0.19	0.64	-0.52	0.44	-0.11	0.22	-0.62~	0.60	-	-
Sex	-1.55	0.94	0.12	0.42	-4.34***	1.19	0.01	0.56	1.18**	0.40	0.13	0.20	-1.34**	0.55	-	-
Harsh Parenting (parent)	-0.07	0.16	-0.04	0.07	0.05	0.20	0.09	0.09	0.02	0.05	-0.01	0.03	0.15	0.09	-	-
RSAb	-0.44	0.46	-0.09	0.20	-0.75	0.56	-0.30	0.27	-0.13	0.19	-0.02	0.09	-0.30	0.26	-	-
RSAr	-0.55	0.62	-0.24	0.28	-0.14	0.76	-0.02	0.37	-0.17	0.25	-0.16	0.12	-0.12	0.34	-	-
HP x RSAr	-0.01	0.18	0.03	0.08	0.21	0.22	0.04	0.11	-0.02	0.07	0.03	0.04	0.10	0.10	-	-
Means (µ)	8.09***	0.46	0.18	0.20	9.33***	0.59	-0.27	0.27	4.07***	0.19	0.17~	0.10	5.11***	0.29	-0.54***	0.15
Variances (σ^2)	41.53***	6.18	4.65*	2.23	61.68***	8.79	8.44**	3.34	6.49***	1.13	0.76†	0.47	13.26***	1.58	1.16*	0.56
I/S correlation	7.73**				11.()9**			0.8	30			-			
R^2	9.1%		11.09	6	11.1	%	9.0)%	11.4%	,)	4.6%	1	12.1%	<u></u> 0	-	

Table 2b

Interaction Effects of Parent-Reported Harsh Parenting and SCLr on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depre	ssion			Anx	riety			Rule E	Breaking		Ag	gressive	Behavior	
	Interce	pt	Slop	е	Interce	pt	Slope	2	Interce	ept	Slop	е	Interce	ept	Slope	е
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.41***	0.38	-0.63***	0.16	-1.31**	0.48	-0.68**	0.22	-0.45**	0.16	-0.09	0.08	-0.79***	0.23	-	-
Race	0.21	1.14	0.12	0.50	-0.38	1.44	0.11	0.68	-0.59	0.47	0.05	0.23	-0.88	0.66	-	-
Sex	-1.37	0.96	0.12	0.42	-4.54***	1.22	- 0.04	0.58	1.34***	0.40	0.12	0.20	-1.19*	0.56	-	-
Harsh Parenting (parent)	0.03	0.16	-0.002	0.07	0.15	0.21	0.15	0.09	0.02	0.07	0.002~	0.03	0.15	0.09	-	-
SCLb	0.19	0.14	0.16**	0.06	0.09	0.17	0.11	0.08	-0.01	0.06	0.04	0.03	-0.05	0.08	-	-
SCLr	-0.41	0.33	0.11	0.14	-0.69~	0.36	-0.21	0.17	-0.16	0.12	-0.04	0.06	-0.25	0.16	-	-
HP x SCLr	0.16~	0.09	0.03	0.04	0.03	0.11	0.02	0.05	0.09**	0.04	0.01	0.02	0.07	0.05	-	-
Means (µ)	7.99***	0.46	0.21	0.20	9.37***	0.59	-0.23	0.28	4.01***	0.20	0.18~	0.10	5.05***	0.29	-0.54***	0.15
Variances (σ^2)	37.58***	6.20	3.27	2.22	59.23***	8.87	7.42*	3.39	6.32***	1.12	0.82~	0.47	12.89***	1.57	1.16*	0.56
I/S correlation	6.42*				9.7	76*			0	.88			-			
R^2	16.0%	, D	23.7%	6	14.4	1%	15.7	7%	16.9%	6	8.4%	0	14.7%	Ó	-	

Table 3b

Interaction Effects of Parent-Reported Harsh Parenting and PEPr on Growth in Internalizing Symptoms and Externalizing Behaviors in Adolescence (N = 242)

		Depres	ssion	I	Anxiety					Rule F	Breaking		Aggressive Behavior				
	Intercept Slo			е	Interce	pt :	Slope		Intercept		Slope		Intercept		Slope	е	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	
SES	-1.42***	0.38	-0.58***	0.16	-1.28**	0.47	-0.64**	0.22	-0.47**	0.16	-0.05	0.08	-0.82***	0.22	-	-	
Race	-0.68	1.03	-0.44	0.46	-0.74	1.32	-0.7	0.63	-0.53	0.43	-0.08	0.21	-0.72	0.60	-	-	
Sex	-1.37	0.97	0.05	0.43	-4.16***	1.22	0.01	0.58	1.30***	0.41	0.23	0.20	-1.51*	0.56	-	-	
Harsh Parenting (parent)	-0.07	0.16	-0.05	0.07	0.07	0.20	0.09	0.09	0.03	0.07	-0.01	0.03	0.16~	0.09	-	-	
PEPb	0.01	0.05	0.02	0.02	0.02	0.07	0.03	0.03	-0.01	0.02	-0.02	0.01	-0.02	0.03	-	-	
PEPr	0.11	0.09	0.02	0.04	0.10	0.11	0.07	0.05	0.04	0.04	0.02	0.02	0.04	0.05	-	-	
HP x PEPr	-0.002	0.03	0.01	0.01	-0.02	0.04	0.003	0.02	-0.01	0.01	0.01	0.01	-0.01	0.02	-	-	
Means (μ)	8.08***	0.46	0.19	0.20	9.40***	0.59	-0.25	0.28	4.08***	0.19	0.17~	0.10	5.12***	0.29	-0.54***	0.15	
Variances (σ^2)	40.00***	6.12	3.74~	2.25	62.59***	8.84	8.48**	3.30	6.45***	1.14	0.79~	0.48	13.19***	1.58	1.06	0.56	
I/S correlation	7.00*				11.37**				0	0.82			-				
R^2	10.0% 14.5%			/0	10.8% 11.0%				12.7%	/ ₀	8.9%	ó	12.2% -				

Table 4b

Interaction Effects of Parent-Reported Harsh Parenting, J	RSAr, and Adolescent Sex on Growth in Internalizing Symptoms
and Externalizing Behaviors in Adolescence ($N = 242$)	

	Depression					Any	xiety			Rule B	reaking		Aş	gressive	Aggressive Behavior		
	Intercept		Slope		Intercept		Slope		Intercept		Slope		Intercept		Slope		
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	
SES	-1.60***	0.38	-0.66***		-1.32**	0.48	-0.71**	0.23	-0.54***	0.16	-0.09	0.08	-0.87***	0.22	-	-	
Race	-0.24	1.05	-0.37	0.47	-0.14	1.35	-0.19	0.65	-0.29	0.44	-0.04	0.22	-0.40	0.61	-	-	
Sex	-1.54~	0.93	0.10	0.41	-4.33***	1.18	-0.03	0.56	1.22**	0.37	0.15	0.19	-1.33**	0.54	-	-	
Harsh Parenting (parent)	-002	0.16	-0.00	0.07	0.01	0.21	0.12	0.10	0.04	0.07	-0.02	0.03	0.18*	0.09	-	-	
RSAb	-0.51	0.45	-0.10	0.20	-0.88	0.56	-0.32	0.27	-0.16	0.18	-0.04	0.09	-0.31	0.25	-	-	
RSAr	-0.53	0.61	-0.25	0.27	-0.13	0.76	-0.03	0.37	-0.19	0.24	-0.18	0.12	-0.10	0.34	-	-	
HP x RSAr	0.02	0.18	0.05	0.08	0.20	0.22	0.05	0.11	-0.02	0.07	0.03	0.03	0.11	0.10	-	-	
HP x Sex	-0.44	0.32	-0.34*	0.14	0.08	0.41	-0.24	0.19	-0.11	0.13	0.03	0.07	-0.19	0.19			
RSAr x Sex	-2.67*	1.19	-0.64	0.54	-2.34	1.49	-0.77	0.73	-1.53***	0.48	-0.43~	0.24	-1.49*	0.66			
HP x RSAr x Sex	-0.17	0.36	0.07	0.16	0.40	0.45	0.19	0.22	0.02	0.14	0.08	0.07	-0.12	0.20			
Means (μ)	8.23***	0.46	0.27	0.20	9.35***	0.59	-0.20	0.28	4.13***	0.19	0.17~	0.10	5.20***	0.29	-0.54***	0.15	
Variances (σ^2)	39.67***	6.08	4.36*	2.09	59.34***	8.64	7.60*	3.36	5.76***	1.09	0.63	0.47	12.71***	1.53	1.33*	0.57	
I/S correlation		7.12	/** -		9.99*				0.53					-			
R^2	13.2% 17.8%			1⁄0	13.1	1%	13.	.2%	18.3%	<u>ə</u>	12.8%		15.3%				

Table 5b

Interaction Effects of Parent-Reported Harsh Parenting, SCLr, and Adolescent Sex on Growth in Internalizing Symptoms	
and Externalizing Behaviors in Adolescence ($N = 242$)	

		Depres	ssion		Anxiety					Rule Bı	reaking		Aggressive Behavior			
-	Intercept		Slope		Intercept		Slope		Intercept		Slope		Intercept		Slope	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
SES	-1.53***	0.39	-0.72***	0.16	-1.30**	0.49	-0.74***	0.23	-0.49**	0.17	-0.08	0.08	-0.82***	0.23	-	-
Race	0.10	1.15	0.00	0.49	-0.40	1.45	0.03	0.68	-0.65	0.48	0.05	0.24	-0.82	0.66	-	-
Sex	-1.42	0.99	0.22	0.43	-4.43***	1.26	0.17	0.58	1.31**	0.42	0.12	0.21	-1.17*	0.58	-	-
Harsh Parenting (parent)	0.10	0.19	-0.01	0.08	0.11	0.23	0.12	0.11	0.03	0.08	-0.01	0.04	0.20*	0.11	-	-
SCLb	0.20	0.14	0.16**	0.06	0.09	0.17	0.11	0.08	-0.00	0.06	0.04	0.03	-0.05	0.08	-	-
SCLr	-0.48	0.34	0.09	0.15	-0.70~	0.37	-0.22	0.17	-0.14	0.12	-0.02	0.06	-0.30~	0.17	-	-
HP x SCLr	0.19	0.11	0.06	0.05	0.07	0.14	0.08	0.06	0.08~	0.04	0.01	0.02	0.09	0.06	-	-
HP x Sex	-0.55	0.35	-0.25~	0.15	0.01	0.44	-0.17	0.20	-0.12	0.15	0.02	0.07	-0.26	0.12		
SCLr x Sex	0.01	0.65	-0.21	0.28	-0.23	0.73	-0.22	0.32	-0.15	0.24	-0.05	0.12	0.29	0.32		
HP x SCLr x Sex	-0.09	0.22	-0.14	0.10	-0.08	0.26	-0.20	0.12	0.03	0.08	0.02	0.04	-0.08	0.11		
Means (µ)	8.07***	0.50	0.14	0.22	9.33***	0.63	-0.33	0.29	4.04***	0.21	0.18~	0.11	5.09***	0.33	-0.54***	0.15
Variances (σ^2)	36.98***	6.23	2.76	2.25	59.27***	8.87	6.78*	3.39	6.26***	1.12	0.81~	0.47	12.63***	1.56	1.22*	0.57
I/S correlation		5.94	1*		9.59*				0.8	88		-				
R^2	18.0% 35.		35.7%	%	14.9	<i>}</i> %	22.5	5%	17.3%	ó	8.3%		16.6% -			

Table 6b

Interaction Effects of Parent-Reported Harsh Parenting, PEPr, and Adolescent Sex on Growth in Internalizing Sy	mptoms
and Externalizing Behaviors in Adolescence ($N = 242$)	

	Depression				Anxiety				-	Rule B	reaking		Aş	Aggressive Behavior			
	Intercept		Slope		Intercept		Slope		Intercept		Slope		Intercept		Slope	?	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE	
SES	-1.45***	0.38	-0.64***	0.16	-1.20**	0.48	-0.65**	0.22	-0.47**	0.16	-0.04	0.08	-0.83***	0.22	-	-	
Race	-0.58	1.04	-0.42	0.46	-0.55	1.34	-0.23	0.63	-0.46	0.44	-0.04	0.22	-0.66	0.60	-	-	
Sex	-1.40	0.96	0.03	0.43	-4.15***	1.22	0.01	0.57	1.34***	0.41	0.26	0.20	-1.16*	0.55	-	-	
Harsh Parenting (parent)	0.01	0.16	-0.01	0.07	0.04	0.21	0.12	0.10	0.04	0.07	-0.02	0.03	0.19*	0.09	-	-	
PEPb	0.01	0.05	0.01	0.02	0.02	0.07	0.03	0.03	-0.01	0.02	-0.02	0.01	-0.02	0.03	-	-	
PEPr	0.07	0.09	0.02	0.04	0.06	0.12	0.09	0.05	0.04	0.04	0.03	0.02	0.02	0.05	-	-	
HP x PEPr	-0.01	0.03	0.02	0.04	-0.03	0.04	0.01	0.05	-0.01	0.01	0.01	0.01	-0.02	0.02	-	-	
HP x Sex	-0.45	0.32	-0.32	0.14	0.10	0.41	-0.20	0.19	-0.13	0.13	0.02	0.07	-0.21	0.19			
PEPr x Sex	0.22	0.18	0.01	0.08	0.19	0.23	0.01	0.11	0.09	0.07	0.01	0.04	0.15	0.10			
HP x PEPr x Sex	-0.01	0.06	-0.04	0.03	0.01	0.08	-0.06	0.04	-0.01	0.02	-0.01	0.01	0.01	0.03			
Means (μ)	8.22***	0.46	0.27	0.20	9.40***	0.60	-0.18	0.28	4.15***	0.20	0.18~	0.10	5.22***	0.29	-0.53***	0.15	
Variances (σ^2)	38.95***	6.09	3.27	2.25	62.06***	8.83	7.78*	3.30	6.24***	1.13	0.78†	0.48	12.78***	1.57	1.10*	0.56	
I/S correlation		6.53	3*		11.17**					0.′	78		-				
R^2	0.119** 0.227			7	0.11	.1*	0.15	~4م	0.155*	*	0.109		0.144*	:*			

Table 7b

Models	χ^2	df	р	RMSEA	CFI
Depression Symptoms			-		
Unconditional	0.054	1	0.8158	0.000, p = .86	1.000
Harsh Parenting	6.545	5	0.2568	0.036, p = .57	0.994
Harsh Parenting x RSAr	10.341	8	0.2419	0.035, p = .62	0.991
Harsh Parenting x RSAr x Sex	22.202	11	0.0229	0.065, p = .23	0.961
Anxiety Symptoms				_	
Unconditional	4.291	1	0.0383	0.117, p = .10	0.989
Harsh Parenting	12.836	5	0.0250	0.080, p = .15	0.975
Harsh Parenting x RSAr	16.482	8	0.0360	0.066, p = .24	0.973
Harsh Parenting x RSAr x Sex	20.656	11	0.0371	0.060, p = .30	0.970
Rule-breaking Behavior					
Unconditional	1.208	1	0.2718	0.030, p = .40	.999
Harsh Parenting	3.907	5	0.5629	0.000, p = .81	1.000
Harsh Parenting x RSAr	4.813	8	0.7774	0.000, p = .95	1.000
Harsh Parenting x RSArx Sex	7.431	11	0.7632	0.000, p = .95	1.000
Aggressive Behavior					
Unconditional	11.067	1	0.0009	0.207, p = .01	0.966
Harsh Parenting	13.351	10	0.2047	0.037, p = .62	0.989
Harsh Parenting x RSAr	21.209	16	0.1706	0.037, p = .68	0.983
Harsh Parenting x RSAr x Sex	28.963	22	0.1459	0.036, p = .72	0.978

Fit Indices for Models examining Parent-Reported Harsh Parenting, RSAr, and Adolescent Sex

Table 8b

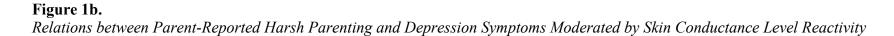
Models	χ^2	df	р	RMSEA	CFI
Depression Symptoms					
Unconditional	0.084	1	0.8158	0.000, p = .83	1.000
Harsh Parenting	6.545	5	0.2568	0.036, p = .57	0.994
Harsh Parenting x SCLr	17.812	8	0.0227	0.071, p = .19	0.966
Harsh Parenting x SCLr x Sex	23.891	11	0.0132	0.070, p = .18	0.957
Anxiety Symptoms				-	
Unconditional	4.238	1	0.0383	0.116, p = .10	0.989
Harsh Parenting	12.836	5	0.0250	0.080, p = .15	0.975
Harsh Parenting x SCLr	30.372	8	0.0002	0.107, p = .01	0.933
Harsh Parenting x SCLr x Sex	30.640	11	0.0013	0.086, p = .05	0.941
Rule-breaking Behavior					
Unconditional	0.752	1	0.2718	0.000, p = .51	1.000
Harsh Parenting	3.907	5	0.5629	0.000, p = .81	1.000
Harsh Parenting x SCLr	6.995	8	0.5372	0.000, p = .85	1.000
Harsh Parenting x SCLr x Sex	7.508	11	0.7566	0.000, p = .96	1.000
Aggressive Behavior					
Unconditional	11.067	1	0.0009	0.207, p = .01	0.966
Harsh Parenting	13.351	10	0.2047	0.037, p = .62	0.989
Harsh Parenting x SCLr	18.720	16	0.2834	0.027, p = .79	0.991
Harsh Parenting x SCLr x Sex	26.049	22	0.2496	0.028, p = .83	0.987

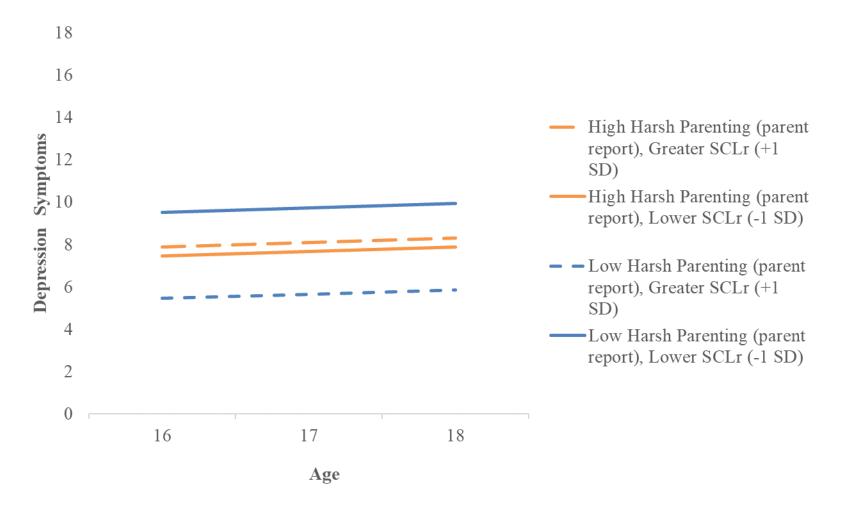
Fit Indices for Models examining Parent-Reported Harsh Parenting, SCLr, and Adolescent Sex

Table 9b

Models	χ^2	df	р	RMSEA	CFI
Depression Symptoms					
Unconditional	0.084	1	0.8158	0.000, p = .83	1.000
Harsh Parenting	6.545	5	0.2568	0.036, p = .57	0.994
Harsh Parenting x PEPr	18.671	8	0.0167	0.074, p = .16	0.962
Harsh Parenting x PEPr x Sex	21.879	11	0.0253	0.064, p = .16	0.962
Anxiety Symptoms					
Unconditional	4.238	1	0.0383	0.116, p = .10	0.989
Harsh Parenting	12.836	5	0.0250	0.080, p = .15	0.975
Harsh Parenting x PEPr	14.298	8	0.0743	0.057, p = .35	0.980
Harsh Parenting x PEPr x Sex	15.703	11	0.1525	0.042, p = .57	0.985
Rule-breaking Behavior					
Unconditional	0.752	1	0.2718	0.000, p = .51	1.000
Harsh Parenting	3.907	5	0.5629	0.000, p = .81	1.000
Harsh Parenting x PEPr	8.126	8	0.4212	0.008, p = .77	0.999
Harsh Parenting x PEPr Sex	8.145	11	0.7003	0.000, p = .94	1.000
Aggressive Behavior					
Unconditional	11.067	1	0.0009	0.207, p = .01	0.966
Harsh Parenting	13.351	10	0.2047	0.037, p = .62	0.989
Harsh Parenting x PEPr	16.365	16	0.4278	0.010, p = .88	0.999
Harsh Parenting x PEPr x Sex	22.294	22	0.4425	0.001, p = .92	0.999

Fit Indices for Models examining Parent-Reported Harsh Parenting, PEPr, and Adolescent Sex

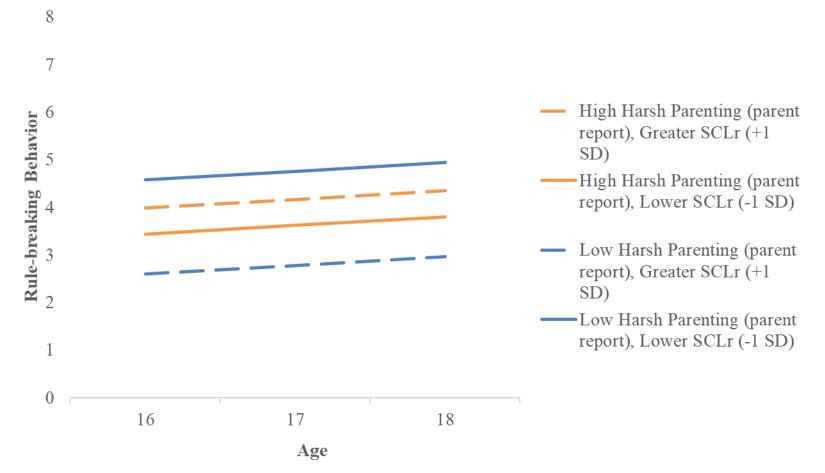




Note. SCLr = skin conductance level reactivity. Higher SCLr is operationalized as increases in SCL (+1 SD, 4.11) and lower SCLr is operationalized as small increases or decreases (-1 SD, -0.37). Model accounts for the effects of sex, race, family SES, and baseline SCL. *Marginal Interaction*.



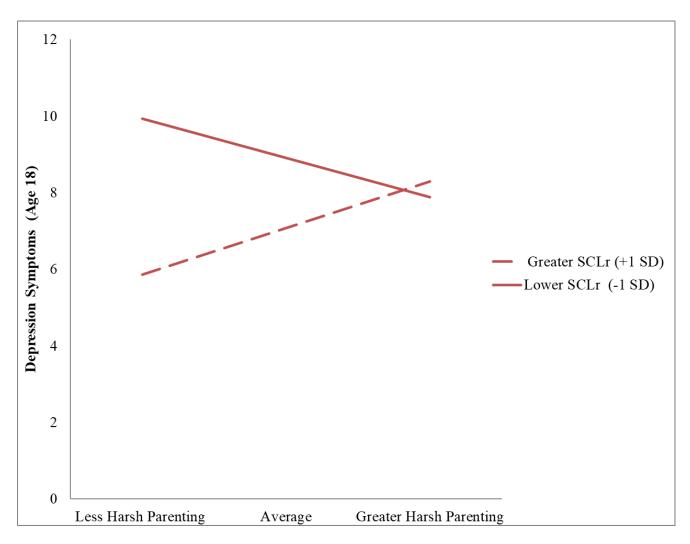
Relations between Parent-Reported Harsh Parenting and Rule-breaking Behavior Moderated by Skin Conductance Level Reactivity



Note. SCLr = skin conductance level reactivity. Higher SCLr is operationalized as increases in SCL (+1 SD, 4.11) and lower SCLr is operationalized as small increases or decreases (-1 SD, -0.37). Model accounts for the effects of sex, race, family SES, and baseline SCL.

Figure 3b.

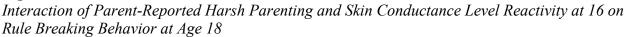
Interaction of Parent-Reported Harsh Parenting and Skin Conductance Level Reactivity at 16 on Depression Symptoms at Age 18

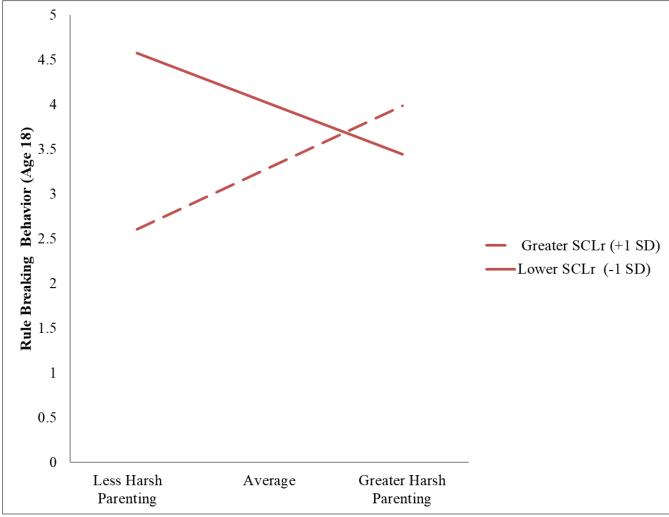


Note. SCLr = skin conductance level reactivity. Higher SCLr is operationalized as increases in SCL (+1 SD, 4.11) and lower SCLr is operationalized as small increases or decreases (-1 SD, -0.37). Model accounts for the effects of sex, race, family SES, and baseline SCL.

Marginal Interaction.

Figure 4b.





Note. SCLr = skin conductance level reactivity. Higher SCLr is operationalized as increases in SCL (+1 SD, 4.11) and lower SCLr is operationalized as small increases or decreases (-1 SD, - 0.37). Model accounts for the effects of sex, race, family SES, and baseline SCL.

Appendix C

Measures

Parent Child Behaviors (CTS-PC; Child Version)

Children often do things that are wrong, disobey, or make their parents angry. We would like to know what your parents have done when you, did something wrong or made them angry or upset.

This is a list of things your might have done in the past year and I would like you to answer whether your parents have: done it once in the past year, done it twice in the past year, 3-5 times, 6-10 times, 11-20 times, or more than 20 times in the past year.

If you haven't done it in the past year but it has happened before that, I would like to know this, too.

- 1 =Once in the past year
- 2 =Twice in the past year
- 3 = 3-5 times in the past year
- 4 = 6-10 times in the past year
- 5 = 11-20 times in the past year
- 6 = More than 20 times in the past year
- 7 =Not in the past year, but it happened before
- 0 = This has never happened

	Father	Mother
1. Explained why something was wrong.	1234567 0	1234567 0
2. Put you in time out	1234567 0	1 2 3 4 5 6 7 0
3. Shook you.	1234567 0	1234567 0
4. Hit you on the bottom with something like a belt,	1234567 0	1234567 0
hairbrush, a stick or some other hard object		
5. Gave you something else to do instead of what you were	12345670	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$
doing.		
6. Shouted, yelled or screamed at you.	1234567 0	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$
7. Hit you with a fist or kicked you hard	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$
8. Spanked you on the bottom with his/her bare band.	1234567 0	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$
9. Swore or cursed at you.	1234567 0	1234567 0
10. Said he/she would send you away or you out of the house.	1234567 0	1234567 0
11. Threatened to hit you but did not actually do it.	1234567 0	1234567 0
12. Hit you on some other pan of the body besides the bottom	1234567 0	1234567 0
with something like a belt, hairbrush, a stick or some other		
hard object		
13. Slapped you on the hand, arm, or leg.	1234567 0	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$
14. Took away privileges or grounded you.	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$	$1\ 2\ 3\ 4\ 5\ 6\ 7\ 0$
15. Pinched you.	1234567 0	1234567 0
16. Threw or knocked you down.	1234567 0	1234567 0
17. Called you dumb or lazy or some other name like that.	1234567 0	1234567 0
18. Slapped you on the face or head or ears.	1234567 0	1234567 0

Child Depression Inventory

Kids have different feelings and ideas. On these next pages, we have listed groups of sentences and we want you to pick **one** sentence from each group that describes you <u>best</u> for the past two weeks. There is no right answer or wrong answer. Just pick one sentence from each group that best describes the way you have been recently.

When you pick the sentence you want, put a mark like this ' \underline{X} in the box next to your answer.

Think about your fee lings and ideas for the PAST TWO WEEKS.

- 1. I am sad once in a while. I am sad m any times. I am sad all the time.
- Nothing will ever work out for me. I am not sure if things will work out for me. Things will work out for me O.K.
- I do most things O.K. I do many things wrong. I do everything wrong.
- 4. I have fun in many things. I have fun in some things. Nothing is fun at all.
- 5. I am bad all the time. I am bad many times. I am bad once in a while.
- I think about bad things happening to me once in a while. I worry that bad things will happen to me. I am sure that terrible things-will happen.
- I hate myself.
 I do not like myself.
 I like myself.
- All bad things are my fault. Many bad things are my fault. Bad things are not usually my fault.
- I feel like crying every day.
 I feel like crying many days.
 I feel like crying once in a while.

- 10. I feel like crying every day.I feel like crying many days.I feel like crying once in a while.
- 11. Things bother me all the time. Things bother me many times. Things bother me once in a while.
- 12. I like being with people.I do not like being with people.I do not want to be with people at all.
- 13. I cannot make up my mind about things. It is hard to make up my mind about things. I make up my mind about things easily.
- 14. I look O.K. There are some bad things about my looks. I look ugly.
- 15. I have to push myself all the time to do my schoolwork. I have to push myself many times to do my schoolwork. Doing schoolwork is not a big problem.
- 16. I have trouble sleeping every night.I have trouble sleeping many nights.I sleep pretty well.
- 17. I am tired once in a while. I am tired many days. I am tired all the time.
- Most days I do not feel like eating. Many days I do not feel like eating. I eat pretty well.
- 19. I do not worry about aches and pains.I worry about aches and pains many times.I worry about aches and pains all the time.
- 20. I do not feel alone. I feel alone many times. I feel alone all the time.

- 21. I never have fun at school.I have fun at school only once in a while.I have fun at school many times.
- 22. I have plenty of friends.I have some friends but I wish I had more.I do not have any friends.
- 23. My schoolwork is all right. My schoolwork is not as good as before. I do very badly in subjects I used to be good in.
- 24. I can never be as good as other kids. I can be as good as other kids if I want to. I am just as good as other kids.
- 25. Nobody really loves me.I am not sure if anybody loves me.I am sure that somebody loves me.
- 26. I usually do what I am told. I do not do what I am told most times. I never do what I am told.
- 27. I get along with people.I get into fights many times.I get into fights all the time.

	Yes	No
1. Often I feel sick in my stomach.		
2. I am nervous.		
3. I often worry about something bad happening to me.		
4. I fear other kids will laugh at me in class.		
5. I have too many headaches.		
6. I worry that other do not like me.		
7. I wake up scared sometimes.		
8. I get nervous around people.		
9. I feel someone will tell me I do things the wrong way.		
10. I fear other people will laugh at me.		
11. I have trouble making up my mind.		
12. 1 get nervous when things do not go the right way for me.		
13. Others seem to do things easter than I can.		
14. I like everyone I know.		
15. Often I have trouble getting my breath.		
16. I worry a lot of the time.		
17. I feel bad If people laugh at me.		
18. I am afraid of a lot of things.		
19. I am always kind.		
20. I get mad. easily.		
21. I worry about what my parents will say to me.		
22. I feel that others do not like the way I do things.		
23. I am afraid to give a talk to my class.		
24. I always have good manners.		
25. It Is hard for me to get sleep at night.		
26. I worry about what other people think about me.		
27. I feel alone even when there are people with me.		
28. I get teased at school.		
29. I am always good.		
30. My feelings get hurt easily.		
31. My hands feel sweaty.		
32. I worry about making mistakes in front of people.		
33.1 am always nice to everyone.		
34. I am tired a lot.		
35. I worry about what is going to happen.		
36. Other people are happier than I am.		
37. I am afraid to speak up in a group.		
38. I tell the truth every single time.		
39. I have bad dreams.		
40. I get angry sometimes.		
41. I worry about being called on In class.		
42. I worry when I go to bed at night.		

43. It Is hard for me to keep my mind on my schoolwork.	
44. I sometimes say things I should not say.	
45. I worry about someone beating me up.	
46. I wiggle in my seat a lot.	
47. A lot of people are against me.	
48. I have told a lie.	
49. I worry about saying something dumb.	

Youth Self-Report

0 - Not True	1 – Somewhat or Sometimes True	2 – Very True or Often True
--------------	--------------------------------	-----------------------------

Rule I	Breaking Behavior
2	I drink alcohol without my parents' approval
26	I don't feel guilty after doing something I shouldn't
28	I break rules at home, school, or elsewhere
39	I hang around with kids who get in trouble
43	I lie or cheat
63	I would rather be with older kids than kids my own age
67	I run away from home
72	I set fires
81	I steal at home
82	I steal from places other than home
90	I swear or use dirty language
<i>96</i>	I think about sex too much
<i>99</i>	I smoke, chew, sniff tobacco, or use e-cigarettes
101	I cut classes or skip school
105	I use drugs for nonmedical purposes (don't include alcohol or tobacco)

Aggree	ssive Behavior
3	I argue a lot
16	I am mean to others
19	I try to get a lot of attention
20	I destroy my own things
21	I destroy things belonging to others
23	I disobey at school
37	I get in many fights
57	I physically attack people
68	I scream a lot
86	I am stubborn
87	My moods or feelings change suddenly
89	I am suspicious
<i>94</i>	I tease others a lot
<i>95</i>	I have a hot temper
9 7	I threaten to hurt people
104	I am louder than other kids