An autonomic nervous system context of harsh parenting and youth aggression versus delinquency

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ABSTRACT

Harsh parenting is a significant predictor of youth aggression and delinquency. However, not every child exposed to adverse parenting develops such problem behaviors. Recent developmental evolutionary models suggest that variability in stress response reactivity to parenting, reflected by autonomic nervous system (ANS) functioning, may affect the impact of adverse parenting on youth behavioral adjustment. The aim of the present study was to investigate whether the parasympathetic and sympathetic branches of the ANS moderate the association between parenting and aggressive and delinquent behaviors. The study sample included low-income, ethnically diverse preadolescents ($M = 10.28$ years old; $N = 101$) and their caregivers. Direct effects were found from basal RSA to delinquent behaviors. In addition, harsh parenting predicted increased youths' aggressive and delinquent behaviors in the context of high RSA withdrawal and increased youths' delinquent behaviors in the context of shortened basal PEP. Implications for prevention and intervention are discussed.

1. Introduction

Harsh parenting behaviors are significantly associated with the development of a broad spectrum of externalizing behaviors in adolescence (Patterson, 2002a). However, a counterintuitive but empirically documented fact is that similar family environments seem to differentially affect youth adjustment, a phenomenon referred to as multifinality (Cicchetti & Rogosch, 1996). Growing evidence indicates that these varied effects may stem from individual neurobiological differences (Dyer et al., 2016; El-Sheikh & Whitson, 2006; Erath et al., 2011). The biological sensitivity to context (BSC) hypothesis suggests that youth physiological response to stress (e.g., stress reactivity) can magnify or diminish the effect of parenting on youth behavior, such that elevated stress reactivity heightens the effect of parenting on children's behavior (Boyce & Ellis, 2005). Given the role of the autonomic nervous system (ANS) in stress reactivity, a growing body of research has been modeling its moderating role in the effect of parenting on youth adjustment (Skibo et al., 2020; Sturge-Apple et al., 2016). BSC theory has been extensively tested among children and adolescents and has yielded mixed results (for review, see Sijtsema et al., 2013), thus requiring further study. Moreover, scarce research has examined the role of the autonomic nervous system in the connection between harsh parenting and youth externalizing behaviors (a) using both basal and reactivity data of the parasympathetic as well as

the sympathetic branches and (b) considering these measures in relation to two distinct aspects of the externalizing spectrum, aggressive and delinquent behaviors (Beauchaine, 2015; Dennis et al., 2012). Thus, the main aim of the present study was to test the link between harsh parenting behaviors and two distinct domains of youth externalizing behavior, aggression and delinquency, in the context of varying levels of autonomic reactivity.

1.1. Harsh parenting and externalizing behaviors in adolescence

Harsh parenting encompasses a wide range of behaviors, such as negative affect toward the child, coercion, aggression, and punitive punishments (Erath et al., 2011; Wiggins et al., 2015). Extant research implicates harsh parenting as a salient factor in the development of psychopathology, especially externalizing symptoms such as aggression and delinquency (Patterson, 2002a). Social learning theory posits that parents who exhibit harsh, negative, or aggressive behaviors socialize their children to exhibit similar behaviors during interpersonal interactions outside of the family (Bandura, 1978; Wiggins et al., 2015). Harsh or coercive parenting practices not only socialize aggression in children but also reduce parent attention toward their child's self-regulatory capacities (Saritaş et al., 2013). Moreover, child self-regulation abilities interact concurrently with parenting, such that poor self-regulation abilities elicits increased negativity from par-

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ents and confers greater risk for externalizing behaviors (Belsky et al., 2007).

The study of externalizing behavior problems is particularly relevant during the transition from childhood to adolescence, a period characterized by increased psychological and neurobiological changes, and the attendant vulnerability to environmental adversity (Mendle, 2014). On the one hand, normative transitions within social and academic contexts, such as that from elementary to middle school, occur during the time in which youth consolidate autonomy and increasingly attribute more salience to peers (Chan & Chan, 2013; Daddis, 2011). On the other hand, these psychosocial changes coincide with adolescent hormonal and neurobiological developments that are associated with orientation towards high arousal, emotionally driven behaviors, and heightened sensitivity to reward (McLaughlin et al., 2011). Together, preadolescence is a developmental phase of increased behavioral and biological plasticity as well as a heightened vulnerability for development of externalizing behaviors (Allwood et al., 2011). Because parent-child interactions are prolonged, frequent, and thus critically formative in youth socio-emotional development, parenting is one of the most potent shaping factors in the development of antisocial behaviors throughout the lifespan (Patterson et al., 2017).

1.2. Externalizing problems spectrum: aggression versus delinquency symptoms

Externalizing problem behaviors are the most common form of maladjustment during childhood and adolescence and are a robust determinant of later psychopathology (Dishion & Patterson, 2015; Reef et al., 2010). The spectrum of externalizing problem behaviors includes two main subtypes: *overt aggressive behavior* such as fighting, teasing, hitting, and arguing, and *covert delinquent behavior* such as stealing, lying, and cheating (Achenbach & Ruffle, 2000; Prinzie et al., 2006; Reef et al., 2011). Research indicates that aggression and delinquent behaviors are developmentally distinct in both etiology and trajectory (Stanger et al., 1997). For example, coercive parenting strategies, such as intrusiveness and coercion, are more frequently used among parents of aggressive children as compared to parents of delinquent children (Dishion & Patterson, 2015). Moreover, the average trajectory of aggressive behaviors typically decreases linearly from childhood to adolescence, whereas delinquency often decreases throughout childhood but re-emerges during preadolescence, between 10 and 12 years (Bongers et al., 2004). Longitudinal research shows adolescents who exhibit primarily aggressive behaviors are more likely to commit crimes as adults than those who exhibit delinquent behaviors (Oshri et al., 2018; Patterson, 2002b). Similarly, childhood physical aggression more consistently predicts later adolescent health risk behaviors such as substance use and risky sexual activity than delinquent behaviors (Timmermans et al., 2008). The etiological and developmental distinction of these externalizing subtypes points to the need for empirical research that can concurrently yet separately model overt aggressive and covert delinquent behavioral outcomes.

1.3. Autonomic nervous system functioning

How children respond to adverse rearing environments is significantly influenced by their ability to self-regulate (Blair, 2010). The ANS plays a foundational role in the neurobiological mechanisms that underlie stress reactivity and self-regulation (Holzman & Bridgett, 2017). According to the neurovisceral integration model, the ANS and CNS influence one another bi-directionally, such that cognitive and affective neural structures within the CNS mediate the ANS to affect behavioral self-regulation (Thayer & Lane, 2000). Accordingly, a growing body of research suggests that patterns of stress reactivity—specifically, the individual's psychobiological capacity for self-regulation un-

der acute stress—partially explain variability in parent influence on child adjustment (McQuade & Breaux, 2017; Obradović et al., 2010; Oshri et al., 2018).

1.4. Overview of sympathetic and parasympathetic nervous systems

The ANS is divided into two integrated but functionally unique parts: the sympathetic nervous system (SNS), responsible for energy expenditure, and the parasympathetic nervous system (PNS), responsible for energy conservation. The sympathetic nervous system coordinates behavioral processes that involve mobilization of bodily resources to meet environmental demands (Dennis et al., 2012). When a threat is perceived, the SNS increases cardiac ventricular contractility, and thus cardiac output, allowing the body to more efficiently carry out fight or flight behaviors. As such, the SNS has been shown to act as a proxy and predictor of individual variability in response to stress (Erath et al., 2009). Although no single measure can directly indicate SNS or PNS activity, study of autonomic functioning certainly reveals precise patterns that reflect the influence of the two branches (Jänig & Häbler, 2000). As such, the patterns in which the SNS influences cardiac contractility may be proxied by pre-ejection period (PEP).

The use of PEP, or the timing between the depolarization of the left ventricle and the release of blood from the aorta, is thought to be an effect proxy of sympathetic influence on cardiac contractility (Beauchaine, 2015; Cacioppo et al., 1994). PEP shortening indicates greater cardiac output and sympathetic influence on the heart. Variations in PEP occur during the stress response and have been shown to reflect individual variations in stress responsivity (Beauchaine et al., 2001). Shortened basal PEP, as well as increased PEP shortening during stress, indicate increased sympathetic activity. This augmentation in SNS influence over cardiac contractility is highly associated with decreases in self-regulation: Beauchaine et al. (2013) found that children with shorter basal PEP exhibited increased conduct problems and aggression. In turn, attenuated self-regulation proxied by PEP shortening often exacerbates risks conferred by early childhood adversity, leading to maladaptive behavioral outcomes such as aggression and impulsivity (Bubier et al., 2009; Erath et al., 2009). Bubier et al. (2009) found that in the context of shorter basal PEP, harsh parenting predicted increased child externalizing behaviors. Similarly, among children with elevated PEP reactivity (i.e., PEP shortening) high permissive parenting was associated with increased affiliation with deviant peers, which is closely linked to risk behaviors (Hinnant et al., 2016). These findings indicate that development of youth maladjustment is significantly associated with patterns of sympathetic functioning, namely increases in sympathetic influence on cardiac contractility as proxied by PEP shortening. Although these findings are certainly informative, investigation of parasympathetic influence is also necessary to further elucidate implications of autonomic functioning on development.

The parasympathetic nervous system (PNS) is associated with “resting and digesting” behaviors used to gather and save energy resources. Similar to the SNS, there is no general index for PNS function; however, measuring vagal regulation of cardiac rate allows us indirectly assess parasympathetic influence. The tenth cranial nerve, also known as the vagus nerve, is the mechanism by which parasympathetic influence is exerted on the heart. Vagal efferent pathways stemming from the nucleus ambiguus lead to cyclical variations in inter-heartbeat intervals known as resting sinus arrhythmia, or RSA (Beauchaine, 2001; Porges, 2007). Vagal activity during resting states, also known as baseline RSA, is a proxy of individual ability to self-regulate and maintain homeostasis when reacting to environmental stimuli, including those emanating from family interactions (El-Sheikh et al., 2001; Skibo et al., 2020; Wagner et al., 2017). Attenuated baseline RSA is linked to increased conduct problems, hostility, and internalizing be-

haviors (Balzarotti et al., 2017; Beauchaine et al., 2001; Dyer et al., 2016). Vagal activity during acute stress, as measured by RSA reactivity (RSA-R), includes both vagal withdrawal (decrease in RSA from baseline) and augmentation (increase in RSA from baseline). Typically, vagal withdrawal occurs during emotionally negative or stressful stimuli and leads to increased heart rate and metabolic output, possibly indicating individual ability to adaptively respond to stress (Perry et al., 2012; Porges, 2007). Vagal augmentation slows heart rate and suggests propensity toward immobilization in response to environmental demands (Erath et al., 2011). However, extreme levels of vagal withdrawal during acute stress may indicate decreased capacity for self-regulation (Hastings & Miller, 2014; Kahle et al., 2018). As such, patterns of heightened vagal withdrawal has been associated with maladaptive stress response patterns and consequent externalizing problems (Dennis et al., 2012; Fortunato et al., 2013; Miller et al., 2013; Obradović et al., 2010; Tabachnick et al., 2020).

The parasympathetic and sympathetic systems are highly related during behavioral processes (Porges, 2007). Gray's motivational theory (Gray, 1982; Gray, 1987a, 1987b) identifies two interdependent dimensions of behavior: inhibition and activation. According to Gray, the SNS comprises the Behavioral Approach System (BAS), which is responsible for approach behaviors and active avoidance behaviors that respectively maximize reward and minimize aversive consequences during situations of threat. Conversely, the Behavioral Inhibition System (BIS) inhibits approach behaviors when aversive consequences are anticipated. In line with Gray's motivational theory is the association between maladaptive behavioral outcomes and imbalances in functioning of the SNS (behavioral activation) and PNS (behavioral inhibition). Considering the interdependence of both PNS and SNS as determinants of behavior, an investigation of the ANS as a contextual factor for psychopathology would benefit from increased methodological rigor in which activity of both branches is measured.

1.5. The ANS as a determinant of biological sensitivity to context

Higher stress response reactivity, as indicated by biomarkers of the SNS (PEP shortening) and PNS (vagal withdrawal), are associated with increased vulnerability to maladaptive outcomes (El-Sheikh et al., 2007; Hastings & Miller, 2014). According to BSC theory, physiological reactivity amplifies individual sensitivity to environmental influence such that some individuals physically experience the input of their context in a more acute way than others (Boyce & Ellis, 2005). Moreover, BSC theory posits that sensitivity to contextual factors may not only be quantified (by measures such as RSA and PEP) but also used to predict developmental outcomes throughout the lifespan. Indeed, child physiological stress reactivity has been shown to interact concurrently with environmental factors to predict child outcomes such that highly reactive physiological profiles prove maladaptive in unsupportive or adverse contexts but adaptive in supportive contexts (El-Sheikh et al., 2007; Erath et al., 2009; Obradović et al., 2010). Although multiple studies have corroborated the BSC theory with young children, studies among pre-adolescent samples have yielded mixed results and lack longitudinal samples as well as economic and ethnically diverse samples (for review, see Sijtsema et al., 2013). Moreover, measurement of parenting behaviors has often been based on parent- or child-report, which has been shown to predict outcomes less consistently as compared to direct observation of parenting behavior (Faith et al., 2012; Pritchett et al., 2010). Furthermore, studies of BSC theory are often limited to one branch of the autonomic nervous system, investigating basal activity or stress reactivity, but not both. Finally, extant research on BSC theory and its relevance to externalizing outcomes lacks specificity in the type of outcome (i.e. delinquency versus aggression), which is necessary in order to better understand the nuanced etiologies of problem behaviors.

1.6. The present study

The main goal of the present study was to investigate the differential effects of harsh parenting on child aggressive and delinquent behaviors across varying levels of ANS functioning. Thus, we aimed to test the role of autonomic functioning at rest and during acute stress in moderating associations between harsh parenting and pre-adolescent aggressive and delinquent outcomes. Based on BSC theory, we hypothesized that high (i.e., shortened) basal PEP would exacerbate the effects of harsh parenting behaviors on aggressive and delinquent behaviors. We hypothesized that high PEP reactivity (i.e. PEP shortening during stress) would exacerbate the effects of harsh parenting on aggressive and delinquent behaviors. Additionally, we hypothesized that low baseline RSA and high RSA withdrawal during stress would exacerbate the impact of harsh parenting on aggression and delinquent behaviors.

The current study offers methodological advances that may assist in addressing methodological gaps in the BSC literature in several ways. First, we employed a longitudinal, community sample in order to assess developmental outcomes. Second, we assess ANS functioning via proxies of both SNS and PNS influence (PEP and RSA, respectively) during a modified Trier Social Stress task. We also quantified residual change (controlling for expected intra-individual variability) from baseline to stress to more comprehensively measure the child's autonomic functioning (Bubier et al., 2009). We measured parenting via an observed interaction task between parent and child, which builds on extant literature that primarily employs parent or child report. Moreover, we made the distinction between aggressive and delinquent behavioral outcomes to increase the specificity of our study and extend the literature regarding implications of autonomic functioning on problem behaviors. Finally, we used a low-income, ethnically diverse sample to test biological sensitivity in the context of the various stressors that are related to low SES and status as a racial or ethnic minority.

2. Methods

2.1. Sample

The sample was drawn from a small city in the southeastern U.S. and consisted of 101 pre-adolescent youths ages 9–12 years ($M_{\text{age}} = 10.28$ years; 43 % male, 57 % female) and their primary caregivers, who were mostly mothers (90.1 %, $M_{\text{age}} = 35.30$ years, $SD = 6.51$). Other caregivers included fathers, grandparents, and other extended family members. All families came from economically impoverished backgrounds (at or below 200 % of poverty line), and as such, 42.5 % of caregivers had received food stamps and/or Women, Infants, and Children (WIC) benefits in the past year. The sample was diverse, with 72.5 % of participants identifying as African American, 12.8 % identifying as White, and 6.4 % as Hispanic/Latino.

2.2. Procedures

Study and procedures were approved by the University of Georgia Institutional Review Board (Parenting and Children's Decision Making, approval number 00003946). In order to connect with community liaisons, the research team engaged the assistance of a local community organization that works to promote well-being among youth and families and has many established relationships with community leaders. The community organization worked with researchers to identify individuals who resided in the community of interest and were socially well-connected who could serve as study liaisons to recruit study participants. Those who agreed to be liaisons were fully informed of the purpose and procedures of the study and were compensated \$100 for each family that participated in the complete study.

Eligibility requirements for potential participants included availability of the primary caregiver, ability of caregiver and child to speak English, and family income level being below 200 % of the poverty line. Enrolled families took part in data collection in a lab setting on the university campus. After consent and assent for caregiver and child were obtained, both were attached to an electrocardiogram via dermal electrodes for the duration of the survey. One electrode was placed on each side of the bottom rib cage, on each side of the clavicle, below the sternum, on the upper spine, and the lower spine. Biometric data were acquired using the BioLab software provided through MindWare Technologies. In order to stimulate participants' autonomic response to a simulated acute social-cognitive stressor, child participants were asked to perform a five-minute mental arithmetic stress task shown in prior research to produce a significant physiological reaction (Berntson et al., 1996).

2.3. Measures

2.3.1. PEP reactivity

Sympathetic nervous system activity, proxied by basal PEP and PEP-R, was collected using a mobile impedance cardiograph (MindWare Technologies, Ltd., Gahanna, OH). To measure PEP, impedance cardiography analysis was conducted by isolating the time interval between the initial electrical stimulation of the heart (onset of the R peak) and the opening of the aortic valve (B point of the dZ/dT wave; (Lozano et al., 2007)). Using the MindWare IMP 3.1.4 Software module, impedance data were ensemble-averaged in 30-second epochs and combined with R waves that were obtained from the electrocardiogram. Research assistants were extensively trained to cross-inspect and correct abnormal R-R intervals such as severe fluctuations, inadvertent cardiac fluctuations, and ectopic beats due to physical movement or breathing. Mean values of PEP across the 30-second epochs were calculated for the baseline and stress interaction task.

To account for within-person variability in PEP-R, a residualized change score was created using the mean level of PEP during the rest period and during the stress task ((Cacioppo et al., 1994). A lower PEP residualized change score indicates a decrease from baseline to the stress task, and thus more sympathetic influence over the heart.

$$\Delta PEP = \frac{PEP_{Stress} - PEP_{Baseline}}{SD(PEP_{Baseline}) \times \sqrt{1 - r(PEP_{Stress}, PEP_{Baseline})}}$$

2.3.2. Basal RSA and RSA reactivity

RSA and RSA-R were utilized to assess caregiver and child parasympathetic physiological activity (Thayer & Lane, 2000). Baseline RSA was measured 3 min after watching a relaxation video, and RSA-R was measured during the acute stress task. Procedures were in accordance with current standards for measuring RSA in psychophysiological research (Gary G Berntson et al., 1996). RSA was measured using MindWare Technologies BioLab software (Version 3.0.6; MindWare Technologies, Ltd., Gahanna, OH). Data were digitized with the MindWare 3.1.0 software module at a sampling rate of 1000 Hz. High-frequency components of RSA (0.12 to 0.42 Hz) were obtained via power spectrum analysis (Akseelrod et al., 1981) to target parasympathetic neural activity. Past research has indicated that high-frequency components of RSA are indicative of more effective self-regulation (Akseelrod et al., 1981; Appelhans & Luecken, 2006). Because accurate assessment of RSA depends on correct specification of respiration rates, and these rates decrease significantly from childhood to adulthood, two different respiratory bands were employed for parents and children ((Fleming et al., 2011) (Shader et al., 2018);). Therefore, the frequency threshold of RSA for adults was 0.15 Hz, while for youths it was .25 Hz. To estimate and correct for individual differences in respi-

ration, spectral analysis of thoracic impedance was utilized (Ernst et al., 1999). Derived from the ECG signal, interbeat intervals (IBIs) were converted into 120-second segments using an interpolation algorithm, and physically improbable IBIs were detected using a MindWare Minimum Artifact Deviation and Maximum Expected Deviation (MAD/MED) algorithm that places some bounds on the variability of the IBI from beat to beat, and flags beats which exceed these limits (Berntson et al., 1990). Data were also manually inspected by trained researchers to detect abnormal R-R intervals such as inadvertent cardiac fluctuations and ectopic beats in RSA due to participants' physical movement or breath. Abnormal R-R intervals were manually corrected by deleting extra beats and inserting mid-beats, according to MindWare Technologies instructions. A residualized change score (equation below) was calculated for RSA reactivity using participants' mean RSA during the baseline period and during the stress period (arithmetic task). The use of a residualized change score allows for the adjustment of within-person variability in baseline RSA (Berntson et al., 1997). A lower RSA residualized change score indicates a decrease from baseline to the stress task (i.e., more vagal withdrawal), and thus suggests a higher level of RSA reactivity.

$$\Delta HRV = \frac{HRV_{Stress} - HRV_{Baseline}}{SD(HRV_{Baseline}) \times \sqrt{1 - r(HRV_{Stress}, HRV_{Baseline})}}$$

2.3.3. Child stress task

The task was constructed based on the Trier Social Stress Task (Allen et al., 2014; Allen et al., 2017; Kirschbaum et al., 1993). At the beginning of the task, children were asked to imagine that they were in a math competition taking place in front of their teachers, parents, and peers in which they would be completing a series of backward digit counting tasks. Though caregivers sat next to the child during the task, they were advised by the researchers not to participate or intervene during the task. It should be noted, however, that this instruction was not given during the initial stages of the study, and as such, a small number of caregivers (less than 5% of the sample) did intervene during the task. When reviewing task instructions, the research assistant led the child through an example trial by demonstrating how to start at 100 and count down by 1. Once the research assistant determined the child understood the task, the five-minute timer began. Children were asked to start at 100 and count down by 2 until they were told to stop. In order to maintain the level of stress for the child, the difficulty increased throughout the length of the task according to the competency displayed by the child (e.g., "Now start at 100 and count down by 6").

2.3.4. Conflict discussion

During a six-minute conflict discussion task, caregivers and children were presented with a stack of notecards, each listing a different topic about which parents and children often disagree (e.g., chores, completing homework, fighting with siblings). The dyad was asked to work together to select the three topics they most frequently disagree about. Once they chose their three topics, dyads were told they would have six minutes to discuss the selected issues and try to achieve possible solutions. Dyads were instructed to try and talk about all three of the problems, and if they finished before the time was up, they were asked to go through the remaining notecards and discuss any additional problems. The research assistant then left the room and returned when the 6 min were over. Parenting behaviors were coded using a 7-point Likert scale (1 = *very low*, 7 = *very high*) based on an existing global coding system (Martin et al., 2008). Coded behaviors for harsh parenting included negativity toward child, intrusiveness, and coercion. Inter-rater reliability was estimated using an intra-class correlation (ICC) based on double coding 24.5 % of the videos. Intra-class correlations were .84, .88, and .79 for negativity, intrusiveness, and coercion, respectively.

2.3.5. Child psychopathology symptoms

Caregivers completed the Child Behavior Checklist (CBCL; (Achenbach & Ruffle, 2000) to report on their child's externalizing problems (Cronbach's $\alpha = .91$). The CBCL contains 118 items rated on a 3-point scale ranging from 0 (*not very true*) to 2 (*very true or often true*) and yields a broadband externalizing measure as well as Delinquent and Aggressive subscales, which have been shown to be reliable and valid in similar samples (Dutra et al., 2004). Whereas T-scores between 65 and 70 represent borderline clinical levels, T-scores greater than 70 indicate clinical levels of symptoms (Achenbach & Ruffle, 2000). In the current sample, 5% of children met criteria for borderline clinical levels of aggressive symptoms and 5.9 % met criteria for borderline delinquent symptoms at T1. Additionally, 2.8 % of children demonstrated clinical levels of delinquent symptoms and 2.8 % demonstrated clinical levels of aggressive symptoms at T1. T-scores for these domains were used in the analyses.

2.4. Analytic plan

Distributions of all variables were checked for normality, and skewed distributions of externalizing subtypes were logarithmically transformed before analysis. A structural equation model (SEM) in Mplus version 7.31 (Muthén & Muthén, 2014) was used to test study hypotheses. The SEM was estimated using maximum-likelihood estimation (Klein & Moosbrugger, 2000). Missing data at T1 ranged from 0 to 6.9 %, as follows: 3.9–4.9% for physiological measures, 6.9 % for parenting measures, and 0% for T1 child behavioral measures. Due to an attrition rate of 33 % from T1 to T2, during which 10–13 months passed, missing data for T2 child behavioral measures was 33 %. Little's Missing Completely at Random (MCAR) test suggested that missing data patterns met the MCAR assumption, $\chi^2(172) = 162.120, p = .694$. A CFA was then performed to test a latent construct of harsh parenting using three indicators: negative affect, intrusiveness, and coercion (see Fig. 1). Next, SEM was used to model the interaction effect of the latent parenting construct and autonomic activity—basal RSA, RSA-R, basal PEP, and PEP-R—on child aggressive and delinquent outcomes. Finally, the Johnson and Neyman (1936) technique was employed to probe and interpret the regions of significance at differing values of the moderator.

3. Results

3.1. Descriptive analysis

Descriptive statistics and bivariate correlations for all study variables are displayed in Table 1. Children with lower basal RSA exhibited higher levels of delinquent behaviors at T1 ($r = -.254, p < .05$). Children with lengthened PEP at baseline experienced increased parental intrusiveness ($r = .234, p < .05$). Children with elevated (shortened) PEP reactivity measured during the acute stress task also experienced increased parental negative affect ($r = -.209, p < .05$). Parental intrusiveness was also significantly associated with youths' increased delinquent behaviors at T1 ($r = .239, p < .05$). Older children exhibited more aggressive behaviors at T1 as compared to younger children ($r = .250, p < .01$). Boys were found to experience greater parent coercive behaviors as compared to girls ($r = -.266, p < .01$) and exhibited decreased basal RSA ($r = -.341, p < .01$).

3.2. Confirmatory factor analysis

A CFA of harsh parenting was conducted, with three indicators used: parent negative affect toward child, parent intrusiveness, and parent coercion (see Fig. 1). All factor loadings were significant and above .50, as suggested by Brown (2014). Model fit was excellent (see Fig. 1).

3.3. Moderation analyses

3.3.1. PEP baseline & reactivity

Moderation analyses were conducted with a harsh parenting latent factor to determine how basal PEP and PEP-R interacted with parenting to predict pre-adolescent aggression and delinquent behaviors in Time 2, after adjusting for variance in T1. No significant direct effect was found between harsh parenting and either aggression or delinquency. The interaction effect of harsh parenting and basal PEP was also not significantly associated with youths' aggression at T2. However, the interaction effect of harsh parenting and basal PEP was positively associated with delinquency at T2 after adjusting for variance at T1 ($\beta = -.562; SE = .143; p = .000; 95\% CI [-.797, -.327]$). Post-hoc probing of the moderation, depicted in Fig. 3, show that increased levels of harsh parenting predicted increased delinquent behavior among

Fig. 1. Measurement model for mothers' harsh parenting latent factor in relation to externalizing subtypes. Note. All values are standardized. All factor loadings are significant ($p < .000$). Model fit is excellent: $\chi^2(4) = 0.377, p = 0.984; RMSEA = 0.000; CFI = 1.00; TLI = 1.151; SRMR = 0.012$.

Table 1
Bivariate Correlations, Means, and Standard Deviations of Study Variables (N = 101).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. RSA_B	--													
2. Δ RSA	-.250*	--												
3. PEP_B	-.091	-.071	--											
4. Δ PEP	.090	-.031	-.577**	--										
5. RBK_T1	-.197	.143	.087	-.004	--									
6. RBK_T2	-.254*	.149	.020	.028	.391**	--								
7. AGG_T1	-.164	.048	.190	-.012	.771**	.274**	--							
8. AGG_T2	-.191	.179	-.004	-.056	.341**	.502**	.445**	--						
9. P_NEG	.001	.115	.165	-.209*	.164	.130	.085	.276*	--					
10. P_INT	-.043	-.026	.234*	-.166	.239*	.055	.109	.120	.611**	--				
11. P_CRS	.174	.051	-.041	.139	.048	-.005	.067	.110	.539**	-.368*	--			
12. SEX	-.341**	.102	.111	-.041	-.302	.211	.017	.133	-.05	-.077	-.266**	--		
13. AGE	-.195	.163	.079	-.013	.141	.077	.250**	.106	-.002	-.052	.011	.143	--	
14. INC	.025	-.036	-.007	-.001	-.098	.008	-.002	-.109	-.032	-.241	-.185	.199	0.24	--
Mean	6.95	-1.35	77.42	-.06	53.86	53.00	52.84	52.90	2.34	2.95	1.71	.52	10.28	21.74
SD	1.19	1.41	24.75	1.38	5.77	4.06	5.60	5.14	1.37	1.39	1.39	.502	1.19	12.8

Note. RSA_B = Basal resting sinus arrhythmia, Δ RSA = Residualized change RSA during acute stress task, Δ PEP = Residualized change PEP during acute stress task, RBK_T1 = Time 1 rule-breaking T score, RBK_T2 = Time 2 rule-breaking T score, AGG_T1 = Time 1 aggression T score, AGG_T2 = Time 2 aggression T score, P_NEG = Parent negative affect toward child, P_INT = Parent intrusiveness toward child, P_CRS = Parent coercion toward child, INC = Family median annual income (in thousands). * $p < .05$, ** $p < .01$, *** $p < .001$.

youths with shortened PEP (and thus increased SNS activity) at rest. The proportion of variance in youths' delinquent behaviors explained by this moderation was 68 %. Moderation did not significantly differ based on sex, income, or age. Interaction terms involving PEP-R did not significantly predict youths' aggressive or delinquent behaviors (Fig. 2).

3.3.2. RSA baseline & reactivity

Basal RSA negatively predicted youths' delinquent behaviors at T2 after adjusting for variance in T1, such that attenuated RSA at rest predicted increased delinquency ($\beta = -.426$; $SE = .145$; $p = .003$; 95 % CI $[-.040, -.009]$). No significant effect was found for the interaction between harsh parenting and basal RSA. The interaction effect of harsh parenting and RSA-R at T1 was significantly associated with youths' delinquent and aggressive behaviors at T2, indicating moderation (respectively: $\beta = -.550$; $SE = .201$; $p = .006$; 95 % CI $[-.881, -.218]$; $\beta = -.590$; $SE = .157$; $p = .000$; 95 % CI $[-.848, -.332]$). Post-hoc probing of the moderation, as seen in Figs. 5 and 6, showed that among youths' who exhibited high RSA withdrawal during stress, increased levels of harsh parenting predicted increased aggressive and delinquent behaviors. The proportion of variance in youths' delinquent and aggressive behaviors explained by these pathways was 58.3 %

and 77.3 %, respectively. Moderation did not significantly differ based on sex, income, or age.

All values are standardized. Dashed lines are insignificant. Delinquency and aggression at T1, household income, child sex, and child age were controlled for. * $p < .05$, ** $p < .01$, *** $p < .001$ (Fig. 4).

4. Discussion

Parenting behaviors are key in shaping youth socio-emotional adjustment and risk for psychopathology (Saritaş et al., 2013). Recent theory and research suggest the effect of parenting on youths' adjustment can vary significantly depending on individual psychobiological reactivity to the environment (Boyce & Ellis, 2005). Specifically, research suggests that youths' reactivity to parenting is modulated by autonomic functioning, which can increase or decrease risk for externalizing psychopathology. Therefore, in the present study we used indirect indices of both sympathetic and parasympathetic functioning to investigate the moderating role of the ANS in the effects of harsh parenting on child aggressive and delinquent behaviors. Our findings corroborate and advance knowledge on the differential effects of harsh parenting and externalizing outcomes due to variations in autonomic functioning. This study provides insight into the roles of parasympathetic influence of cardiac rate and sympathetic influence on car-

Fig. 2. Moderation model with sympathetic activity at rest. Note. All values are standardized. Dashed lines are insignificant. Delinquency and aggression at T1, household income, child sex, and child age were controlled for. * $p < .05$, ** $p < .01$, *** $p < .001$.

Fig. 3. Interpretation of the moderating role of basal PEP on the link between harsh parenting and delinquent behaviors. The upper panel presents an adapted simple slope interpretation, and the lower panel presents the Johnson-Neyman plot. In both figures, the shadowed areas indicate regions of significance. The solid line represents an extremely shortened basal PEP level that is the lowermost boundary of the left significant region (PEP = 18.26, slope $b = .067$, $p = .006$). The dashed-dotted line represents the moderately shortened basal PEP level that is the uppermost boundary of the left significant region (PEP = 42, slope $b = .043$, $p = .003$). The dashed line represents the moderately lengthened basal PEP level that is the lowermost boundary of the right significant region (PEP = 78, slope $b = .007$, ns). The dotted line represents the extremely lengthened basal PEP that is the uppermost limit of the significant region (PEP = 128, slope $b = -.043$, ns).

diac contractility, during baseline and stress, as a biological context for child externalizing outcomes. Specifically, the link between harsh parenting and youths' delinquent behaviors was exacerbated by shortened basal PEP, whereas the link between harsh parenting and both delinquent and aggressive behaviors was exacerbated by RSA withdrawal during stress. Because patterns of the autonomic nervous system, especially stress response reactivity, are subject to change via interventions such as mindful meditation, understanding the moderating role of the ANS as a protective or risk factor in the association between harsh parenting and externalizing outcomes may lead to important prevention implications (Chen, Brody, & Miller, 2017).

Our first hypothesis was partially supported. Among youths with shortened basal PEP, harsh parenting predicted increased delinquent behaviors at T2. Shortened basal PEP has been shown to be a risk factor for conduct problems in both children and adolescents, and is thought to be indicative of temperament-level emotional-

ity (Beauchaine et al., 2001). Our findings partially replicate those by Bubier et al. (2009), who found that among children with shortened basal PEP, harsh parenting and low neighborhood cohesion predicted increased externalizing behaviors. Our findings are also in line with BSC theory, indicating that children with elevated baseline sympathetic functioning show increased sensitivity to environmental adversity and thus increased propensity for maladaptation in the context of harsh parenting.

Importantly, the moderating effect of basal PEP did not extend to aggressive behaviors, which may support extant research that distinguishes aggressive and delinquent behaviors as developmentally unique (Prinz et al., 2006). Indeed, in their seminal study of child development from ages 4–18, Bongers et al. (2004) found a divergence in the trajectories of aggressive and delinquent behaviors. Although aggressive behaviors normatively declined in a linear fashion, delinquent behaviors took a curvilinear shape, decreasing linearly until ages

Fig. 4. Moderation model with parasympathetic reactivity. Note. RSA-R = Resting sinus arrhythmia reactivity.

10 through 12, and increasing again thereafter. This trend is arguably at work within the current sample, which encompasses the critical period in which delinquent behaviors often re-emerge (rates of delinquent behavior are indeed marginally higher than rates of aggressive behaviors in the current sample). Although basal PEP may indeed be a salient and unique contributor to youth delinquent, and not aggressive, behaviors, further replication is necessary before this conclusion is drawn.

Our second hypothesis was not supported in that the effect of harsh parenting on child aggression was not influenced by PEP reactivity (shortening and/or lengthening) during acute stress. As such, we may conclude that sympathetic influence over cardiac contractility during acute stress may be less indicative of BSC, and less formative in the development of maladjustment, when compared to sympathetic influence over cardiac contractility at rest. Once again, this finding is similar to that of Bubier et al. (2009), who found that PEP reactivity during stress did not moderate the link between harsh parenting and externalizing behaviors among children. The present finding diverges from those of Hinnant et al. (2016), which implicated PEP shortening during acute stress as a salient contributor to adolescent risk behaviors. This divergence may be due to assessment of PEP at differing periods of development, with Hinnant et al. employing a sample of middle adolescents rather than pre-adolescents. Although nascent findings have pointed to developmental increases of PEP during childhood, as well as stabilization of PEP during adolescence (Alkon et al., 2003; Matthews et al., 2002), a more recent study has yielded conflicting findings (Hinnant et al., 2011). As such, additional study is necessary to further clarify the role of PEP reactivity in the link between harsh parenting and externalizing behaviors across differing stages of child and adolescent development.

Our third hypothesis was supported, in that the effect of harsh parenting on youths' aggressive and delinquent behaviors was exacerbated in the context of high RSA reactivity (vagal withdrawal). Moreover, the effect of harsh parenting on aggressive, but not delinquent, behaviors was attenuated in the context of low RSA reactivity (vagal augmentation). This finding partially supports a BSC framework, in which high and low autonomic reactivity act respectively as risk and protective factors in adverse contexts such as harsh parenting. Once again, replication of these results is necessary before further delving into the distinction between aggressive and delinquent behaviors and their association to vagal augmentation. Our results also highlight the necessity to distinguish between community and clinical samples when studying autonomic functioning. Blunted parasympathetic reactivity has been shown to increase risk for conduct problems among clinical samples (Brenner & Beauchaine, 2011). In contrast, the opposite trend is seen in community samples: low reactivity is shown to mitigate risk. This may be due to the less frequent incidence of maltreatment in com-

munity samples as compared to clinical samples (Dennis et al., 2012; Obradović et al., 2010).

In contrast to our last hypothesis, the effect of harsh parenting on externalizing behaviors did not significantly differ based on levels of basal RSA. However, we found that low basal RSA longitudinally predicted youths' delinquent behaviors, thus corroborating similar findings on the associations between blunted parasympathetic functioning and behavioral dysregulation (Dyer et al., 2016; Eisenberg et al., 2012). Low basal RSA has been linked to increased negative emotionality and thus may indicate a lack of self-regulation, effects of which are seen in heightened risk behaviors (Beauchaine, 2001; Belsky & Pluess, 2009). This pathway may be further informed by extant links between emotion regulation and Theory of Mind (ToM; Frith & Frith, 1999), or the ability to attribute mental states to others in order to understand and predict their behavior. Deficits in ToM have been closely linked poor emotion regulation (Hudson & Jacques, 2014) (Lagattuta et al., 2015) and subsequent antisocial behaviors such as delinquency, aggression, and callous-unemotional traits due to an attenuated capacity for prosocial behaviors (Imuta et al., 2016; Sebastian et al., 2012). Therefore, children with low basal RSA are at a resting state that may increase their risk for development of maladaptive behaviors in adolescence, possibly via deficits in Theory of Mind (James Benjamin Hinnant & El-Sheikh, 2009; Quintana et al., 2012). Although nascent research has pointed to connections between physiological arousal and aspects of ToM (Gower & Crick, 2011; Zhang & Wang, 2020), more investigation is necessary to elucidate the nuances of these links.

In addition to elucidating nuances within autonomic functioning and its moderating context of the impact of parenting on youth adjustment, this study further highlights the theoretical and methodological importance of distinguishing externalizing subtypes as unique. As suggested by a developmental psychopathology perspective, environmental conditions can usher in dynamic developmental processes that eventuate in multiple developmental outcomes. This is expressed by the concept of multifinality and equifinality (Cicchetti & Rogosch, 1996). According to the principle of multifinality, individuals exposed to similar types of adversity may still exhibit varied outcomes due to the multi-level influences of the systems in which each individual operates. As such, harsh parenting has been shown to confer risk for aggression and delinquency for many, but certainly not all, children and adolescents. Variations in parasympathetic and sympathetic functioning, at rest and during acute stress, lead to differential outcomes despite the shared context of harsh parenting. Consequently, these findings serve as evidence for the framework of multifinality, in which shared context leads to an array of outcomes based on individual differences. However, equifinality is also evidenced, in that varying patterns of auto-

Fig. 5. Interpretation of the moderating role of RSA reactivity on the link between harsh parenting and delinquent behaviors. The upper panel presents an adapted simple slope interpretation, and the lower panel presents the Johnson-Neyman plot. In both figures, the shadowed areas indicate regions of significance. The solid line represents a level of high RSA withdrawal that is the lowermost boundary of the significant region (Δ RSA = -4.2, slope $b = .064$, $p = .03$). The dashed-dotted line represents a level of moderate RSA withdrawal that is the uppermost boundary of the significant region (Δ RSA = -2.6, slope $b = .029$, $p = .047$). The dashed line represents a level of moderate RSA augmentation that is one standard deviation above the mean (Δ RSA = .245, slope $b = -.033$, ns). The dotted line represents a level of high RSA augmentation that is two standard deviations above the mean (Δ RSA = 1.72, slope $b = -.066$, ns).

conomic functioning predicted similar outcomes of aggression and delinquency. Finally, our study supports the biological sensitivity to context (BSC) theory, in which patterns of high physiological activity and reactivity indicate greater sensitivity to environmental input and thus greater vulnerability to the risks conferred by adversity.

5. Limitations

This study has several limitations that should be acknowledged. First, our limited sample size and power would be helped by replication among studies with increased sample sizes and similar design. Moreover, due to the circumscribed sample of low-income families and primarily mothers, our findings may not be generalizable across differing demographics and caregiver arrangements. However, it is necessary to study ethnic minorities and impoverished families to comprehensively study resilience, and thus this specificity of sample is also a strength. The study would also be better served to have an increased number

of time points to study development of externalizing behaviors across adolescence. This limitation is mitigated by the study's employment of multiple reporters and data collection methods (parent report, physiological data, and coded video interaction) as well as two time-points that allow for longitudinal insights into development of externalizing behaviors.

This study extends literature on the functioning of the autonomic nervous system in the context of adolescent externalizing behavior. Pre-adolescence is a significant period in which both physiological and socioemotional development occurs, and these changes are salient contributors to externalizing behaviors in later adolescence and adulthood. Our findings corroborate and extend present study on the functioning of the autonomic nervous system in the development of problem behaviors. Moreover, our findings support current efforts to conceptually differentiate externalizing behaviors by implicating the parasympathetic and sympathetic branches as unique contributors to the long-

Fig. 6. Interpretation of the moderating role of RSA reactivity on the link between harsh parenting and delinquent behaviors. The upper panel presents an adapted simple slope interpretation, and the lower panel presents the Johnson-Neyman plot. In both figures, the shadowed areas indicate regions of significance. The solid line represents a level of high RSA withdrawal that is the lowermost boundary of the left significant region ($\Delta\text{RSA} = -4.2$, slope $b = .10$, $p = .004$). The dashed-dotted line represents a level of moderate RSA withdrawal that is the uppermost boundary of the left significant region ($\Delta\text{RSA} = -2.8$, slope $b = .045$, ns). The dashed line represents a level of moderate RSA augmentation that is the lowermost boundary of the right significant region ($\Delta\text{RSA} = -1.1$, slope $b = -.023$, ns). The dotted line represents a level of high RSA augmentation that is the uppermost limit of the significant region ($\Delta\text{RSA} = 1.7$, slope $b = -.136$, $p = .000$).

term development of aggression and delinquency. Although our community sample limits a discussion of clinical implications, these findings may inform practitioners who directly treat children and adolescents with aggressive and delinquent behaviors. This work was partially supported by the National Center for Advancing Translational Sciences of the National Institutes of Health (Award Number UL1TR002378, PI: Assaf Oshri). Additional support for this work was provided through the National Institute on Drug Abuse (Award Number K01DA045219, PI: Assaf Oshri).

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