Physiological Regulation, Psychosocial Adversity, and Proactive Versus Reactive Aggression: A Longitudinal Study

Wei Zhang

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Physiological Regulation, Psychosocial Adversity, and Proactive Versus Reactive Aggression: A Longitudinal Study

By

Wei Zhang

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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ABSTRACT

Physiological Regulation, Psychosocial Adversity, and Proactive Versus Reactive Aggression: A Longitudinal Study

By

Wei Zhang

Advisor: Yu Gao, PhD

Two types of aggression in children and adolescents have been identified: reactive aggression (RA) and proactive aggression (PA). Despite the accumulating evidence suggesting differential temperamental, behavioral, cognitive, social-environmental, and neurobiological correlates in relation to the two types of aggression, no study has examined emotion regulation in children with RA vs. PA using psychophysiological approaches. In this study a sample of eight to 10 years old children participated in an emotion regulation task in which they were required to either induce or inhibit their emotions. They also reported their aggressive behavior using the Reactive-Proactive Aggression questionnaire (RPQ; Raine et al., 2006). Electrocardiogram and respiration were assessed continuously during a 2-min rest period and the emotion regulation task and were used to derive the baseline respiratory sinus arrhythmia (RSA) and RSA reactivity, respectively. Both RSA measures and aggressive behavior were assessed again during the one-year follow-up visit. The aims of the study were threefold: First, we aimed to examine if emotion dysregulation, as reflected by low baseline RSA and/or reduced RSA reactivity, would be differentially associated with RA and PA. Second, the study examined the interaction effects between baseline RSA and RSA reactivity in relation to the two types of aggression. Finally, we explored the moderating effects of psychosocial risk factors on the RSA-aggression associations. Concurrent correlations
between RSA, aggression, and social adversity measures at each time point were first examined, and the predictive relations between these measures from Time 1 to Time 2 were then determined using multiple regressions and ANCOVAs. Results showed that after PA was controlled for, high RA was associated with autonomic hyperarousal, i.e., higher baseline RSA and increased RSA reactivity, in boys at Time 1 only. No such effect was found in girls. By contrast, high PA was associated with hypoarousal, i.e., reduced RSA reactivity, only in the conditions of low social adversity and no moderating effect of gender was found. Together, these findings provide initial evidence that the RA and PA are characterized by different patterns of the parasympathetic–mediated emotional regulation processes and that both neurobiological and psychosocial influences are important in understanding the etiology of aggressive behavior in children and adolescents.

Keywords: emotion regulation, respiratory sinus arrhythmia, social adversity, reactive aggression, proactive aggression
ACKNOWLEDGMENTS

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List of Abbreviations

ACC..................Anterior Cingulate Cortex
ACTH.................Adrenocorticotropic Hormone
ADHD.................Attention-Deficit and Hyperactivity Disorder
ANS...................Autonomic Nervous System
ASD.................Autism Spectrum Disorder
CRH...................Corticotrophin-releasing Hormone
CU....................Callous-Unemotional
DLFC.................Dorsolateral Frontal Cortex
EEG..................Electroencephalography
ERN..................Error-Related Negativity
ERP..................Event-Related Potential
HPA..................Hypothalamic-Pituitary-Adrenal
HR...................Heart Rate
HRV..................Heart Rate Variability
MFC..................Medial Frontal Cortex
OFC..................Orbitofrontal Cortex
PA....................Proactive Aggression
PAG..................Periaqueductal Gray
PEP..................Pre-ejection Period
PFC..................Prefrontal Cortex
PNS..................Parasympathetic Nervous System
RA................Reactive Aggression
RSA...............Respiratory Sinus Arrhythmia
RPQ..............Reactive-Proactive Aggression Questionnaire
SC................Skin Conductance
SNS...............Sympathetic Nervous System
tDCS...............Transcranial Direct Current Stimulation
vmPFC............Ventral Medial Prefrontal Cortex
CHAPTER 1 - REACTIVE AND PROACTIVE AGGRESSION

Introduction

Aggression in children and adolescents can have detrimental effects on school performance, parent and peer relationship, and future adjustment problems, and it constitutes a significant public concern for the society (Hubbard, McAuliffe, Morrow, & Romano, 2010). Prior research has suggested that aggression in children accounts for 50% of child referrals for psychological services as well as 25% of all special services in school settings (Nelson & Finch, 2000). In addition, children and adolescents with more aggressive behavior are more likely to commit crimes when they grow up as compared with children with or without other adjustment problems (Snyder & Sickmund, 1999). Since the 20th century, research in human aggression has flourished. Besides psychosocial risk factors, recent research has also examined psychophysiological abnormality, as a potential endophenotype, to better understand the etiology of aggressive and antisocial behavior (Hinnant & El-Sheikh, 2009; Patrick, 2008; Raine, 2002). However, the abnormality observed in adult population may be the consequences of the aggressive behavior itself or the manifest of environmental influences. Investigations of these abnormalities during the childhood may maximize the chance of identifying its genetics thus further minimizing the aggressive behavior itself (Sterzer & Stadler, 2009). In order to provide a more holistic picture, recent studies have started to examine the interaction effects between psychophysiological and psychosocial influences in predicting aggression (Raine, 2002; Wilson & Scarpa, 2012).

Although research on the associations between emotion dysregulation and externalizing behavioral problems has increased exponentially (Beauchaine, 2015a), no study has in particular differentiated the two subtypes of aggression: one is reactive and the other is proactive. Reactive
Aggression (RA) is characterized by impulsive and hostile responses to provocation and frustration, and is associated with higher levels of body arousal. Conversely, Proactive Aggression (PA) is instrumental, predatory, and goal directed, and has been linked to lower levels of arousal (Dodge & Coie, 1987; Scarpa & Raine, 1997). The main aim of the current study was to examine the effects of parasympathetic nervous system (PNS) - mediated emotion regulation process and psychosocial risk factors in predisposing children to either reactive or proactive subtypes of aggression using a longitudinal (repeated-measures) design.

**Reactive and Proactive Aggression**

Researchers have classified two subtypes of aggression in children and adolescents: RA (also called affective or impulsive aggression) and PA (also called instrumental or predatory aggression). According to the frustration-aggression theory, RA is the consequence of hostile attributional bias when an individual attends selectively to a hostile or threatening environmental cue during social interactions (Berkowitz, 1962; Crick, Dodge, Crick, & Dodge, 1996; Dodge & Coie, 1987; Dodge, Murphy, & Buchsbaum, 1984; Dodge, Pettit, McClaskey, Brown, & Gottman, 1986; Dollard, Miller, Doob, Mowrer, & Sears, 1939). Conversely, the social learning theory posits that PA is pulled by an anticipation of external rewards after the execution of violent behavior; thus it occurs mostly without provocation (Bandura, 1973; Crick et al., 1996; Crick & Dodge, 1994).

The social information-processing model of aggression (Crick et al., 1996; Crick & Dodge, 1994; Dodge et al., 1986) further suggests that RA and PA are associated with deficits related to different steps during the information-processing process. In particular, RA is con-
structured to relate to early stage problems; that is, reactively aggressive individuals “encode” en-
vironmental cues in a selective and inaccurate manner, attend to fewer and selectively to threat-
ening cues, and/or display a hostile attributional bias when “interpreting” others’ behaviors (de
Castro, Merk, Koops, Veerman, & Bosch, 2005; Dodge & Frame, 1982; Milich & Dodge, 1984).
In contrast, PA is associated with later stage problems; that is, proactively aggressive individuals
select the most positively evaluated responses to enact in the situation, expect more positive out-
comes for aggressive behaviors, feel more positively about their ability to perform aggressive be-
haviors, and make less negative evaluations of aggressive responses during the response deci-
sion–making process (Crick et al., 1996).

Multiple assessment tools have been developed to assess the two types of aggression and
most of the empirical studies have unitized one of the following two instruments. Dodge and
Coie (1987) first developed a 12-item teacher-rating instrument to measure RA and PA in ele-
mental school students. Respondents indicate how frequently each item applies to the child on a
1-to 5-point Likert scale, ranging from “never” to “almost always”. The scale contains three
items indexing RA (i.e., “when teased, strikes back”, “blames others in fights”, and “overreacts
angrily to accidents”), three items indexing PA (i.e., “use physical force to dominate”, “gets oth-
ers to gang up on a peer” and “threatens and bullies others”), and six items indexing unclassified
aggression (e.g., “teases and name-calls”, “gets into verbal arguments”). Multiple informant ver-
sions (i.e., teacher, parent, and self-report) of the Reactive-Proactive Aggression Questionnaire
(RPQ) that can be used in youth aged nine to 16 was later developed by Raine et al. (2006). This
scale contains 11 items of RA (e.g., “hit others to defend yourself”), and 12 items of PA (e.g.,
“used force to get money or things from others). Respondent rates each statement as 0 (never), 1
(sometimes), or 2 (often) for the frequency of occurrence (See Appendix A). Both of these scales had good psychometric characteristics, and factor analyses of these scales have identified RA and PA as two correlated but distinct subtypes of aggression (Crick & Dodge, 1994; Day, Bream, & Pal, 1992; Dodge & Coie, 1987; Poulin & Boivin, 2000a; Price & Dodge, 1989; Raine et al., 2006).

**Psychosocial Differences of RA and PA**

Although some argue against the distinctions between these subtypes of aggression given that aggressive individuals tend to show high levels of both RA and PA (Bobadilla, Wampler, & Taylor, 2012; Miller & Lynam, 2006), there is growing evidence suggesting that there are meaningful distinctions between RA and PA in the temperamental, behavioral, cognitive, social-environmental and developmental domains (Hubbard et al., 2010) (see Table 1).

**Temperamental and Behavioral Correlates**

Research has in general demonstrated that there are differential temperamental and behavioral correlates for RA and PA. For example, RA has been associated with anger rumination (Vitaro, Brendgen, & Tremblay, 2002; White & Turner, 2014), poor emotion regulation (Marsee & Frick, 2007), anxiety, and depression (Card & Little, 2006; McAuliffe, Hubbard, Rubin, Morrow, & Dearing, 2007; Raine et al., 2006). Conversely, PA has been associated with psychopathic personality (Raine et al., 2006) and callous-unemotional traits (Fanti, Frick, & Georgiou, 2008; Frick et al., 2003), referring to a specific set of affective and interpersonal styles (e.g., lack of guilt, fail to show empathy, and using others to get what they want; Frick et al., 2003; Kruh, 2005).

In addition, some researchers have found that RA is uniquely associated with attention
and impulsivity problems (Atkins & Stoff, 1993; Crick et al., 1996; Dodge & Coie, 1987; Raine et al., 2006), while others have linked both RA and PA to the attention-deficit and hyperactivity disorder (ADHD) (Card & Little, 2006; Connor, Steingard, Cunningham, Anderson, & Melloni, 2004). PA has been uniquely linked to delinquency and serious antisocial problems (Atkins & Stoff, 1993; Pulkkinen, 1996; Raine et al., 2006; Vitaro et al., 2002; Vitaro, Gendreau, Tremblay, & Olligny, 1998), and specifically to later externalizing and antisocial behaviors. For example, one longitudinal study showed that proactively, but not reactively aggressive children at age 12 years showed increased conduct problems in middle adolescence (Vitaro et al., 1998). In another study, PA at age 14 years was uniquely associated with adult criminality and externalizing behaviors in males, and internalizing behaviors and neuroticism in females, whereas RA was associated with better adjustment in adulthood (Pulkkinen, 1996).

**Cognitive Correlates**

Prior research has established the relationship between impaired intellectual functioning (i.e., IQ) and aggression and antisocial behaviors in children and adolescents (Archwamety & Katsiyannis, 2000; Dougherty et al., 2007; Loney, Frick, Ellis, & McCoy, 1998; McHale, Obrzut, & Sabers, 2003; Moffitt, Lynam, & Silva, 1994). Lower verbal IQ has been thought to impede an individuals’ ability to engage in private speech, which is important for behavioral regulation, and has also been associated with lower academic achievement and increased school rejection (Loney et al., 1998; Luria, 1980; Moffitt et al., 1994).

With regard to RA and PA, researchers have found that children with higher RA showed poor verbal skills, whereas those with higher PA were characterized by intact or better verbal abilities (Arsenio, Adams, & Gold, 2009; Loney et al., 1998; Luria, 1980; Moffitt et al., 1994). It
has been proposed that poor verbal skills found in reactively aggressive children may lead to failure in addressing potential interpersonal misunderstandings, thus further lead to an increase of provoked aggressive behaviors. In contrast, greater verbal abilities and thus better social skills may enable proactively aggressive youths to use aggression as a strategy to obtain external goals during social interactions (Arsenio et al., 2009). Similarly, although impaired executive functioning (EF) have often been found in the general antisocial population, RA, but not PA, has been uniquely related to deficits in EF and social information-processing (Crick et al., 1996; Dodge & Coie, 1987; Giancola, Moss, Martin, Kirisci, & Tarter, 1996; Raine et al., 2006).

Social and Environmental Correlates

Several social and environmental factors, including parenting styles and peer relationships, have been found to be predictive of the development of aggression and antisocial behaviors. With regard to RA and PA, research has shown that RA is associated with histories of physical abuse, harsh parenting, and peer rejection and victimization (Dodge, Lochman, Harnish, Bates, & al, 1997; Poulin & Boivin, 2000b). Conversely, PA is associated with parental substance abuse, family violence, and tolerance or even positive relationships with peers (Connor et al., 2004; Dodge et al., 1997; Poulin & Boivin, 2000b; Raine et al., 2006). These findings provide the empirical support for Dodge’s (Dodge, 1991) model, which proposes that RA and PA may originate from different causes. Briefly, it has been proposed that while RA develops in reaction to an abusive and harsh parenting or a negative and threatening environment (e.g., peer rejection and victimization), PA thrives in the “supportive” environments that foster the use of aggression as a means to achieve one’s goals (e.g., family violence and positive peer relationship). Furthermore, these different social and environmental correlates of RA and PA have
yielded great insights into learning about the developmental progression of the two subtypes of aggression.

**Developmental Trajectories**

Past research has examined the developmental course of the two subtypes of aggression (Fite & Colder, 2007; Fite, Colder, Lochman, & Wells, 2008; Lansford, Dodge, Bates, & Pettit, 2002; Vitaro et al., 2006). Fite and Colder (2007) have reported the 1-year stability of RA and PA to be .72 and .55, in a group of children between nine and 12 years old. Furthermore, findings have suggested that RA and PA both decline as children age (Fite et al., 2008; Lansford et al., 2002). In one study, Lansford et al. (2002) examined RA and PA using teacher’s report and found that both types of aggression declined from kindergarten through the 3rd grade, and from the 6th grade through the 7th grade. In a group of aggressive children, Fite et al. (2008) found that both RA and PA declined from 5th to 9th grade, and that the developmental trajectories were similar for both forms of aggression.

With regard to the long-term outcomes of children with the two subtypes of aggression, as previously reviewed, findings have suggested that children with higher RA will show relatively better adulthood adjustment (Pulkkinen, 1996), whereas those with higher PA are more likely to show severe later antisocial behavior problems (Atkins & Stoff, 1993; Pulkkinen, 1996; Raine et al., 2006). Researchers have proposed that the decreases in RA during late childhood and early adolescence may be due to improvement in self-regulation and temperament management. In contrast, PA has been proposed to remain stable or even increase throughout adolescence, as reinforced by the family and peer environment in using force to solve conflicts (Vitaro, Brendgen, & Barker, 2006; Vitaro et al., 2002).
Finally, there is some evidence showing that while RA is predictive of later violence during romantic relationships, PA is predictive of delinquency-related violence towards strangers (Brendgen, Vitaro, Tremblay, & Lavoie, 2001). According to the proposition by Hubbard et al. (2010), the progression from early RA to later violence towards the intimate partner may be due to negative parenting (e.g., lack of warmth and emotional involvement with the child), whereas the progression from early PA to later violence towards strangers may be due to a lack of parental monitoring, which in turn fosters violent behaviors.

In sum, a limited number of longitudinal studies have shown that the level of RA declines whereas PA remains stable or even increases from early childhood to adolescence, and their long-term outcomes may be qualitatively different.

**Gender Differences**

Studies in aggressive behavior have often shown that boys are generally more aggressive than girls. Of note, boys are found to be three times more likely to be diagnosed with conduct problems than girls (Zoccolillo, 1993). Relatively less research has examined gender differences in terms of RA and PA. Salmivalli and Nieminen (2002) found that boys scored higher on both RA and PA as compared to girls in a sample of 1,062 children aged 10 to 12 years. In contrast, Connor, Steingard, Anderson, and Melloni (2003) found no gender differences in RA or PA in a clinical sample of seriously emotional disturbed children and adolescents. Gender differences in correlates of RA and PA have also been reported. For example, in one study by Connor et al. (2003), RA was associated with hyperactivity/impulsivity in boys and early abuse experience and lower verbal IQ in girls, whereas PA was found to be associated with drug use, hostility, and negative parenting in both genders. In a recent study by Stickle, Marini, and Thomas (2012), it
was found that girls with both RA and PA showed more callous-unemotional (CU) traits compared with boys with both subtypes of aggression in a sample of adjudicated adolescents. Moreover, RA was found to be uniquely associated with poor emotion regulation and anger reaction, whereas PA was uniquely associated with CU traits in a sample of detained girls aged 12 to 18 (Marsee & Frick, 2007). Understanding the distinct correlates of RA and PA in boys and girls may shed light on future prevention and treatment efforts that target on each gender group.

**Table 1. Psychosocial Differences between RA and PA**

<table>
<thead>
<tr>
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<th>RA</th>
<th>PA</th>
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<tr>
<td>Temperamental/ Behavioral</td>
<td>Anger rumination, emotion dysregulation, anxiety, depression, attention, and impulsivity problems</td>
<td>Psychopathic personality, CU traits, and hyperactivity features</td>
</tr>
<tr>
<td>Cognitive</td>
<td>Lower verbal IQ and poor executive functioning</td>
<td>Better verbal skills and intact executive functioning</td>
</tr>
<tr>
<td>Social/ Environmental</td>
<td>Negative environment, harsh parenting, peer rejection, and peer victimization</td>
<td>“Supportive environment”, parental substance abuse, lack of parental monitoring, family violence, tolerant or even positive relationships with peers</td>
</tr>
<tr>
<td>Developmental</td>
<td>Decreases in late childhood and early adolescence</td>
<td>Chronic antisocial behaviors</td>
</tr>
<tr>
<td>Gender</td>
<td>In boys: RA was associated with hyperactivity and impulsivity.</td>
<td>In girls: PA was associated with CU traits.</td>
</tr>
<tr>
<td></td>
<td>In girls: RA was associated with lower verbal IQ, CU traits, and emotion dysregulation.</td>
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Neurobiological Differences of RA and PA

In addition, there is growing literature documenting the differences between RA and PA at the neurobiological levels (see Table 2).

Genetics

Findings from the behavioral genetic research have shown that genetic effects account for up to 50% of the variations in shaping aggressive behavior (Miles & Carey, 1997; Plomin, Nitz, & Rowe, 1990). Studies examining the genetic structure of RA and PA have suggested that heritability is slightly higher for PA than for RA and that there may be gender differences. For instance, in 172 male and female twin pairs who were six years old, Brendgen et al. (2006) found that genetic influences explained 39% and 41% of the variances of RA and PA, respectively, and that the genetic influences are highly correlated ($r = .87$) in both gender groups. In a sample of nine-to 10-year-old twins ($N = 596$ pairs), Baker et al. (2008) reported that in boys, the genetic influences on PA were higher than those on RA (50% and 38% for PA and RA, respectively), whereas in girls, environmental but not genetic influences almost entirely accounted for both subtypes of aggression. Furthermore, there is evidence that while the stability of RA may be due to both genetic and non-shared environmental influences (i.e., experiences that make siblings dissimilar), the stability of PA is primarily mediated by the genetic influences (Tuvblad, Raine, Zheng, & Baker, 2009). In this study, participants included nine- to 10-year-old twins ($N = 1,241$) who were assessed again at ages 11 to 14 years ($N = 874$). Taken together, findings suggest that the genetic influences may play a more significant role in PA and its development, in particular in males.
Neuroimaging

Very few neuroimaging studies have examined the structural and functional brain abnormality in children and adolescents with aggressive behavior (Connor, 2002). However, findings from the adult literature have in general shown the structural and functional brain deficits prominently in the prefrontal cortex (PFC; involved in inhibitory control, decision-making, and emotional processing), the temporal cortex (language and memory), the amygdala (fear conditioning and emotion), the anterior cingulate cortex (ACC; emotion regulation and autonomic functioning), and the hippocampus (emotion regulation and fear conditioning) are associated with higher antisocial and aggressive behaviors (Blair, 2003; Blair, 2001; Kiehl, 2006; Yang & Raine, 2009).

Concerning RA and PA, relatively less neuroimaging evidence has been documented, although several neurocognitive models have been proposed to conceptualize the neurobiological distinctions between the two subtypes of aggression. For example, White, Meffert, and Blair (2015) formulated the cognitive neuroscience models of aggression in the legal context. Specifically, their presumptions of the different neurological substrates of the two subtypes of aggression include: a) RA is associated with increased amygdala and periaqueductal gray (PAG) activation, and also with ventral medial PFC and striatum deficiency, implying an over-activated basic-threat system in response to social provocation and difficulties in solving frustration; and b) PA is associated with functional deficits in amygdala and ventral medial PFC, implying a lack of empathy and problems with moral decision-making. Similarly, Blair (2001) proposed a neurocognitive model of aggression, in which RA is postulated to be associated with impairments in the executive emotional system and orbitofrontal cortex (OFC) in particular, whereas PA is
linked to dysfunctions in the systems (e.g., amygdala) involved in socialization, which is partly reflected by deficits in fear conditioning.

Indeed, several preliminary empirical studies in adults have supported these conceptual neurocognitive models. For example, it was found that prefrontal dysfunction was specific to the affective (impulsive/reactive) but not the predatory (premeditated/proactive) subgroup of convicted murderers (Raine et al., 1998). Dolan, Deakin, Roberts, and Anderson (2002) found that impulsive–aggressive patients showed significant volume reduction in the temporal lobe. Coccaro, McCloskey, Fitzgerald, and Phan (2007) found an amygdala-OFC dysfunction in response to angry faces in individuals with impulsive/reactive aggression. Although neuroimaging research on PA is limited, researchers have found that individuals with psychopathy, a cluster of personality traits that are highly correlated with PA, showed reduced activity in the amygdala and the rostral ACC/ventral medial PFC (vmPFC) in response to facial emotional expressions (Kiehl et al., 2001) and during a fear conditioning task (Birbaumer et al., 2005). Similarly, among non-clinical individuals, researchers have found that people high on psychopathic traits showed reduced activation in the amygdala in response to emotional expressions (Gordon, Baird, & End, 2004).

In aggregate, prior theoretical and empirical literature has suggested that RA is associated with impairments in the brain areas related to information processing, emotion regulation and inhibitory control (i.e., prefrontal areas, temporal lobe, amygdala, and OFC). In contrast, PA is associated with impairment in the areas related to emotion responses and fear conditioning (i.e., amygdala, ACC, and vmPFC). Given that the empirical findings are exclusively from the adults, it will be important for future studies to replicate these findings in younger populations.
Psychophysiology

Studies using psychophysiological methods have been flourishing since the last decade and been used widely in the area of research in aggression and antisocial behaviors in children and adolescents.

**Autonomic nervous system activity.** In general, lower baseline or resting heart rate (HR) is one of the most robust biological correlates of aggressive and antisocial behaviors in youth (Lorber, 2004; Ortiz & Raine, 2004; Pitts, 1997). Lower baseline HR, indicating lower baseline levels of autonomic nervous system (ANS) activity, has been associated with both RA and PA (Raine, Fung, Portnoy, Choy, & Spring, 2014). In terms of ANS reactivity to social or cognitive stimuli, researchers have found RA to be associated with larger HR reactivity and higher skin conductance (SC) responses to stressful or threatening stimuli (see Lorber, 2004 for a review). For example, Pitts (1997) found that RA but not PA was associated with increased HR reactivity during a challenging situation in boys from the 3rd to 6th grades. Hubbard et al. (2002) found RA but not PA to be related to increased SC reactivity to provocation in a group of 2nd graders. In contrast, PA but not RA have been associated with reduced SC responses to punishments in youths (Gao, Tuvblad, Schell, Baker, & Raine, 2015).

Findings on the associations between PNS activity (i.e., vagal activity) and aggression, however, are less consistent. For example, prior research has shown that conduct problems are associated with either low (Beauchaine, Gatzke-Kopp, & Mead, 2007; Calkins & Dedmon, 2000) or high levels of baseline PNS activity (Dietrich et al., 2007; Scarpa, Fikretoglu, & Luscher, 2000). Low baseline PNS activity has been posited to relate to poor emotion regulation
(Beauchaine, 2001; Porges, Doussard-Roosevelt, & Maiti, 1994), though high baseline PNS activity has been related to a passive coping style to stressor or vagal sensitivity (Raine & Venables, 1984; Venables, 1988). With regard to RA and PA, low baseline PNS activity has been found in children with high RA, indicating their poor emotion regulation skills (Scarpa, Haden, & Tanaka, 2010; Xu, Raine, Yu, & Krieg, 2013). Conversely, high baseline PNS has been found in proactively aggressive children (Scarpa et al., 2010), which may reflect their better emotional control or regulation skills, and/or their insensitivity to negative effects (Fabes & Eisenberg, 1997; Scarpa et al., 2010). Relatively less is known about the direct effects of PNS reactivity in response to threats/stressors on the two subtypes of aggressive behavior. Evidence from adult populations has indicated in women with a sexual abuse history, blunted PNS reactivity during a stressful interview to be uniquely associated with proactively relational aggression (Murray-Close & Rellini, 2012). Zhang and Gao (2015) found that PNS reactivity during a moral decision-making task to be positively related to RA but negatively related to PA in college students from benign home environments.

Taken together, empirical evidence suggests that the two subtypes of aggression may be characterized by different ANS activity profiles. While hyperarousal may be more relevant to the reactive form of aggression, reflecting exaggerated responses to stress, provocation, and negative affect, autonomic hypoarousal seems more relevant to the proactive form aggression (Eysenck, 1997; Raine, Venables, & Mednick, 1997; Scarpa & Raine, 1997), indicating their fearlessness, callousness, and the tendency towards sensation seeking in attempt to maintain arousal to an optimal level.
EEG and ERP correlates. A large number of studies using electrocortical measures has shown that enhanced slow wave electroencephalographic (EEG) activity is associated with aggressive and antisocial behaviors in children and adolescents (Raine, Venables, & Williams, 1990; Scarpa & Raine, 1997). This predominance of the slow wave activities may suggest cortical immaturity that is linked to impaired inhibitive ability (Volavka, 1990).

Research has also examined the associations between left versus right frontal EEG asymmetry and aggressive behaviors. Based on the motivational direction model of frontal asymmetry, relatively greater left frontal activity is related to approach motivation, whereas greater right frontal activity is linked to withdrawal motivation (Harmon-Jones, 2004). Results from the line of work by Harmon-Jones and colleagues (Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Harmon-Jones, Vaughn-Scott, Mohr, Sigelman, & Harmon-Jones, 2004; Harmon-Jones, 2003, 2004; Peterson, Shackman, & Harmon-Jones, 2008) have shown that relatively increased left vs. right frontal activation during rest and anger induction is related to higher levels of anger and aggressive behaviors in adults. In addition, frontal EEG activity asymmetry has been found in children and adolescents with mood and disruptive behavior disorders (Rybak, Crayton, Young, Herba, & Konopka, 2006).

In terms of RA and PA, however, it is still unclear to what degree the frontal asymmetry findings may apply to the two subtypes of aggression. Only one recent study by Dambacher et al. (2015) has shown that proactively aggressive behavior could be reduced by inducing right hemispheric dominance through transcranial Direct Current Stimulation (tDCS) in men. Given that RA was more related to anger, hostility, and impulsivity (Raine et al., 2006; White & Turner,
2014), and that PA was thought to be predatory and goal oriented (Dodge & Coie, 1987), the enhancement of the right hemisphere activity may theoretically inhibit the approach motivation as mediated by the left hemisphere activity. Therefore, these preliminary data have provided evidence to support the notion that the increased left frontal activity may be more significantly associated with PA but not RA. Future studies are needed to replicate these findings.

Another line of research has focused on event-related potentials (ERPs) in relation to aggressive behaviors. Among these studies, abnormality in P3 (i.e., P300), a rather broad positivity that is maximal along the midline at the parietal recording sites and between 300 and 500 milliseconds following stimulus presentation (Sutton, Braren, Zubin, & John, 1965), has been found to be a robust correlate of aggressive and violent behavior. P3 component is a well-known and reliable marker of brain functioning related to information processing. In particular, lower amplitude and longer latency of P3 indicate limited neural resources and inefficient cognitive functioning (Donchin, 1979). Research on the P3 component in response to a variety of tasks (e.g., the visual and auditory oddball tasks, the Stroop task) has demonstrated that antisocial behaviors are associated with reduced P3 amplitudes and lengthened P3 latencies, reflecting their information processing impairments (Gao & Raine, 2009).

Further to this point, research has shown that P3 deficits are uniquely associated with the impulsive/reactive (Barratt, Stanford, Kent, Felthous, & Alan, 1997; Gerstle, Mathias, & Stanford, 1998) but not the instrumental/proactive form of aggression (Barratt et al., 1997; Stanford, Houston, Villemarette-Pittman, & Greve, 2003). Thus, impaired cognitive functioning, such as inefficient attention allocation, maybe specific to RA. Finally, since most studies have focused on P3, it is important for future studies to investigate other ERP components, such as P1
reflecting selective attention ability; Mangun & Hillyard, 1996) and N2 (response inhibition; Bekker, Kenemans, & Verbaten, 2005), in relation to the two subtypes of aggression.

**Endocrinology**

Cortisol is a type of hormone that indexes the arousal of the hypothalamic-pituitary-adrenal (HPA) system, which controls the neurophysiological processes in response to stressors (de Kloet, 1998; Johnson, Kamilaris, Chrousos, & Gold, 1992). The exposure to a stressor triggers the hypothalamus to communicate with the pituitary gland by secreting corticotrophin-releasing hormone (CRH), and the pituitary gland responds by secreting adrenocorticotropic hormone (ACTH), which further activates the adrenal gland to release more cortisol (Lopez-Duran, Hajal, Olson, Felt, & Vazquez, 2009). Cortisol levels have been reported to be reduced in aggressive children and adolescents (McBurnett, Lahey, Rathouz, & Loeber, 2000; Shoal, Giancola, & Kirillova, 2003), suggesting lower HPA arousal in these individuals. The associations between cortisol activity and aggression have not been reported consistently, however. For example, McBurnett et al. (1991) found elevated cortisol levels in conduct disordered children with a comorbid anxiety disorder.

In relation to RA and PA, researchers have found that higher levels of baseline cortisol and increased cortisol reactivity to stress are associated with RA, whereas the HPA-axis profile (both baseline cortisol and cortisol reactivity) of proactively aggressive children is no different from that of non-aggressive children (Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2009; van Bokhoven et al., 2005). Together, evidence suggests that RA is characterized by a more active HPA system associated with enhanced stress reactivity, whereas the HPA-axis profile of proactively aggressive children was equivalent to that of non-aggressive children.
Table 2. Neurobiological Differences between RA and PA

<table>
<thead>
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<th>RA</th>
<th>PA</th>
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<tr>
<td>Neuroimaging</td>
<td>Impairments in brain regions linked to information processing, emotion regulation, and inhibitory control</td>
<td>Impairments in brain regions linked to negative emotion responsivity and fear conditioning</td>
</tr>
<tr>
<td>EEG</td>
<td>N/A</td>
<td>Greater left vs. right frontal EEG asymmetry</td>
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<tr>
<td>ERP</td>
<td>Lower P3 amplitude and longer P3 latency</td>
<td>Normal P3</td>
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<tr>
<td>ANS</td>
<td>ANS hyper-arousal</td>
<td>ANS hypo-arousal</td>
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<td>HPA</td>
<td>More active HPA system</td>
<td>Normal HPA system</td>
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Chapter Summary

Taken together, prior studies have demonstrated that RA and PA are different in the psychological, behavioral, cognitive, developmental, psychosocial, and neurobiological domains. While RA has been associated with anger rumination, anxiety and depression, PA has been linked to callous-unemotional traits and psychopathic personality traits. Additionally, while RA has been related to poor verbal ability and executive functioning, PA is characterized by good verbal skills and intact cognitive functioning. Furthermore, RA has been thought to develop from harsh parenting or a threatening environment. In contrast, PA may develop in reaction to the “supportive” environments that promote the use of aggressive behavior. Moreover, gender differences have been observed in terms of the differential correlates of RA and PA. The two types of aggression seem to follow different developmental trajectories, and RA seems to be associated
with better adulthood adjustment than PA. Finally, while RA predicts adulthood violence towards romantic partner, PA is predictive of the later violent behaviors towards strangers.

Most of the neurobiological research, in particular the brain imaging and EEG/ERP studies, has focused on adult populations and relatively little has been done with children and adolescents. While the neurobiological abnormality observed in adults may be the consequences of the aggressive behavior itself or the manifest of the environmental influences, in children an early investigation of the neurobiological abnormalities may maximize the chance of identifying its etiology and minimize the effect of aggressive behavior itself (Sterzer & Stadler, 2009). Dozens of studies have suggested that both youths and adults displaying antisocial and aggressive behaviors show abnormally low autonomic system activity (Sterzer & Stadler, 2009). However, very few studies have systematically investigated the neurobiological differences or similarities between young and old populations. In terms of RA and PA, empirical evidence has suggested that RA is associated with brain abnormalities in regions linked to information processing, emotion regulation and control of inhibition, higher ANS arousal, reduced P3 amplitude, and a higher level of HPA activity. In contrast, PA is associated with impairments in the brain areas related to responsivity to negative emotions and fear conditioning, lower ANS arousal, greater left vs. right hemisphere activation, relatively intact cognitive functioning (i.e., normal P3), and a normal level of HPA activity. Future studies incorporating and comparing multiple measures of physiological responses (i.e., autonomic arousal, EEG, ERP, and brain imaging) within the same task will help further our understanding of the etiology of aggression.
CHAPTER 2 - EMOTION REGULATION

Emotion Regulation

Emotion regulation has emerged as a popular research topic as it plays an important role in understanding both typical and atypical childhood development (Beauchaine, 2015a; Cole, Marin, & Dennis, 2004; Keenan, 2006). It has been generally agreed that the process of emotion regulation is distinct from that of emotion generation (the formation of an emotional response) (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Cole et al., 2004; Gross & Thompson, 2007). Furthermore, although the definition and research methodologies of emotion regulation often vary across studies (Adrian, Zeman, & Veits, 2011; Cole et al., 2004; Eisenberg et al., 2001; Morris, Silk, Steinberg, & Robinson, 2009), there seems to be a common intuitive understanding of what is meant by emotion regulation (Cole et al., 2004; Morris et al., 2009; Thompson, 2008). Broadly speaking, emotion regulation refers to the extrinsic and intrinsic processes that an individual makes to monitor, evaluate, and modify emotional responses and experiences as to accomplish one’s goals (Thompson, 1990). An extended model of emotion regulation includes three major regulatory stages: identification (whether or not to regulate), selection (which general regulation categories to use), and implementation (which specific regulation tactic to implement). Meanwhile, each stage has three basic elements: perception, valuation, and action (Gross, 2015; Sheppes, Suri, & Gross, 2015). Emotion dysregulation can occur in any of the basic elements of the three major regulatory stages (Sheppes et al., 2015).

Effective emotion regulation is essential for a child’s development of adaptive functioning (Cole et al., 2004), and emotion dysregulation has been associated with various forms of psychopathology (e.g., depression, anxiety, bipolar disorders, ADHD) (Beauchaine, 2015b; Nancy
Eisenberg et al., 2001; Leibenluft, 2011; Shaw et al., 2012; Silk, Steinberg, & Morris, 2003; Sobanski et al., 2010; Suveg & Zeman, 2004; Zeman, Shipman, & Suveg, 2002), and aggressive and violent behaviors in particular (Rothbart, Ahadi, & Hershey, 1994; Zeman et al., 2002). Indeed, children who are unable to regulate emotion efficiently are more likely to show hostile reaction consequent to frustration and provocation, whereas individuals with superior emotion regulation skills show fewer problematic behaviors via better impulse or anger control (Rothbart et al., 1994; Zeman et al., 2002).

**Measurements of Emotion Regulation**

Many of the prior studies have used self- or informant-report (i.e., teachers and parents), laboratory observation, or behavioral paradigms to assess emotion regulation. Recent studies with implementation of a variety of neurophysiological methodologies (e.g., neuroimaging, vagal activity, EEG/ERPs) have helped us to disentangle the important mechanisms underlying the emotion regulation processes (Adrian et al., 2011; Beauchaine, 2015a; Davidson, 2000). In fact, multiple levels of analyses including at least one physiological measure have often been used in emotion regulation research papers published in the top tier journals (Adrian et al., 2011).

**Behavioral Approaches**

**Self-report.** Self-report measures are important instruments for assessing emotion regulation even in young children (Durbin, 2010). Several instruments, including the Difficulties with Emotion Regulation Scale (Gratz & Roemer, 2004; Weinberg & Klonsky, 2009), the Emotion Regulation Questionnaire for Children and Adolescents (Gross & John, 2003; Gullone, MacDermott, & Hughes, 2009), and the Emotion/Affect Regulation Interview (Zeman & Garber, 1996), have often been used to assess emotion regulation in children beyond 13 years, between
10 and 18 years, and as young as 1st grade, respectively. For example, the Difficulties with Emotion Scale consists of 36 items assessing a variety of problems associated with regulation of negative affect, including the Non-Acceptance, Goals, Impulse, Awareness, Strategies, and Clarity subscales. The Emotion Regulation Questionnaire for Children and Adolescents is a 10-item questionnaire designed to assess the emotion regulation strategies of Reappraisal (e.g., “When I want to feel happier, I think about something different”) and Suppression (e.g., “I keep my feelings to myself.”). Finally, the Emotion/Affect Regulation Interview assesses Emotion Regulation Decision, Display Rules, Reasons and Methods of Regulation, and Emotion Self-Efficacy from the semi-structure interview using vignettes and open-ended questions.

**Informant-report.** Informant reports (e.g., parent and teacher) are sometimes used to measure emotion regulation in youth, as they usually demonstrate better reliability and validity (Morris, Robinson, & Eisenberg, 2006). The most frequently used parent or teacher reports are the Emotion Regulation Checklist (Shields & Cicchetti, 1997) and the California Q-sort (Block & Block, 1980; Shields & Cicchetti, 1997). The Emotion Regulation Checklist is a 24-item parent- or teacher-report assessing Emotion Liability/Negativity (i.e., dysregulated negative affect) and Emotion Regulation (i.e., appropriate emotion expression) in children and adolescents between six and 18 years old. The California Q-sort scale is a 100-item parent-report, which assesses different types of personalities with 10 emotion regulation items in children aged six to 12 years old.

**Observational methods.** Observational methods are often used in measuring emotion regulation during child development, due to the fact that very young children may have difficulties in understanding and reporting their own emotional experiences (Cole et al., 2004). Research
of child temperament has yielded important evidence on how emotions are regulated in young populations (e.g., infants and toddlers). For example, the Infant-Toddler Frustration Paradigm (e.g., Stifter & Braungart, 1995) has been adopted by researchers to assess the ability to self-sooth, avoid, communicate, orientate to others, and redirect in infants to 18-month-old toddlers in a variety of tasks such as arm restraints and toy removal. Moreover, emotion regulation has been examined during early mother-and-child interaction (Cohn & Tronick, 1987; Field, 2009; Tronick, 1989), because the quality of this emotional exchange between mother and child is predictive of later emotion regulation development (Cole et al., 2004). In this paradigm, the emotional exchange between mother and child is recorded, coded, and analyzed in terms of the timing and sequencing of the changes in each partner of the dyad (Field, 2009). Finally, many studies focus on child’s self-initiated attempts and skills to regulate negative emotions under challenging conditions. For example, the Anger Induction: Interaction Paradigm (Cummings, 1987; Cummings, Pellegrini, Notarius, & Cummings, 1989; Maughan & Cicchetti, 2002) is designed to examine child’s emotion behavioral responses and emotion regulation strategies (i.e., appropriately concerned, undercontrol, and overcontrol) in response to the interadult conflicts between the research assistant and the mother. Children’s emotion regulation has also been examined during the frustration task in which they must tolerate waiting to get a cookie when there is little of interest in the surrounding environment (Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002). Similarly, children’s attempts to regulate their emotional behaviors have been assessed in the disappointing situation in which the researcher creates positive expectations for receiving a desirable reward for children but in fact giving an undesirable one (Cole, 1986; Saarni, 1984).
**Neurobiological Approaches**

Various behavioral measures, although widely used and reliable, do not tap into the related internal neurobiological processes of emotion regulation (Beauchaine, 2015a; Lewis, Granic, & Lamm, 2006). The most compelling evidence for emotion regulation has emerged from studies that use neurophysiological methodologies (e.g., neuroimaging, EEG/ERPs, psychophysiology), which are critically important for understanding the underlying mechanism of emotion regulation (Beauchaine, 2015a; Jackson, Malmstadt, Larson, & Davidson, 2000).

Neuroimaging techniques can provide insights into the neural circuitries implicated in emotion processing and regulation (e.g., Davidson, 2000). Although as noted previously, neuroimaging research in children and adolescents has been far less than that of adults (Connor, 2002). EEG/ERPs studies are well-suited to identify possible neural markers of emotion dysregulation (Dennis, 2010; Lewis & Stieben, 2004). Autonomic activity, in particular the PNS activity, has been studied as the peripheral biomarker of emotion regulation (Beauchaine, 2001, 2015a, 2015b). Of note, autonomic activity is used routinely in emotion regulation research in children and adolescents. Compared to the ERP measures that capture the short-term self-regulation of emotion and require a very limited amount of active control, the ANS activity targets a relative long-term regulation process.

**Neurobiological Mechanisms of Emotion Regulation**

**Cortical and Subcortical Neural Network**

Evidence from neurobiological research indicates that emotion regulation is a top-down process where the cortical neural network regulates the emotion that is processed from the sub-
cortical neural network (Beauchaine, 2015a; Davidson, 2000; See Figure 1). The cortical network mediates the top-down deliberated regulation of emotion; in contrast, the subcortical network mediates the bottom-up autonomic process of generating emotion. The cortical network includes brain regions such as the medial frontal cortex (MFC), dorsolateral frontal cortex (DLFC), ACC, vmPFC and OFC, whereas the subcortical network includes brain regions such as the amygdala (Davidson, 2000). For example, the ACC, located in the MFC, is an essential cortical structure in modulation of cognition and emotion responses (Bush, Luu, & Posner, 2000; Casey et al., 1997; Dennis, 2010; Reed & Warner-Rogers, 2009). The amygdala, located in the medial temporal lobes of the brain, is a crucial subcortical structure for emotional perception and generation, including determining the motivational significance of the emotional cues (e.g., threat, fear, and anger), sending messages to the lower brain regions involved in initiating motor movement and release of neurochemicals, and receiving feedback from the PFC for either prompting or inhibiting emotion (Zeman, Cassano, Perry-Parrish, & Stegall, 2006).

Effective communications between the cortical and the subcortical areas are important for successful emotion regulation. In contrast, deficiencies in emotion regulation have been related to reduced functional connectivity between the PFC and amygdala, implying deficient top-down modulation of the amygdala by the PFC regions (Beauchaine, 2015a). In addition, it is found that the subcortical networks mature during early development, while the cortical networks continue to develop in the 20s (Beauchaine & McNulty, 2013; Beauchaine, 2015a). Thus, deficits in regulating emotion often found in children and adolescents may be partly due to the competition between the heightened activity in the subcortical network involved in processing emotional information and the immature cortical network involved in emotion regulation (Hare et al., 2008).
Researchers have also examined the cortical-subcortical activity during emotion regulation in relation to a child’s internalizing/externalizing behaviors (Davidson, 2000; Hare et al., 2008). Neuroimaging studies have revealed that children with anxiety disorders and trait of anxiety show less functional connectivity in the amygdala-ventrolateral PFC connections in response to negative facial expressions in a variety of tasks (e.g., attention task, Go/No-Go task) (Hare et al., 2008; Monk et al., 2008). Shannon, Sauder, Beauchaine, and Gatzke-Kopp (2009) found that adolescent males with higher externalizing behaviors showed reduced functional connectivity in the striatal-prefrontal connections during a Reward/No-Reward task as compared to the controls.

Note: OFC = orbitofrontal cortex; vmPFC = ventral medial prefrontal cortex; MFC = medial frontal cortex; DLFC = dorsolateral frontal cortex; ACC = anterior cingulate cortex.

Figure 1. Main Cortical (e.g., MFC, DLFC, ACC, vmPFC, OFC) and Subcortical Regions (e.g., amygdala) Involved in Emotion Regulation
Ventral and Dorsal Systems

Two distinct neural systems, ventral and dorsal, have been identified to be involved in regulating emotion automatically and effortfully, respectively (Dennis, Buss, & Hastings, 2012; Dennis, 2010; Derryberry & Rothbart, 1997; Drevets & Raichle, 1998; Henderson & Wachs, 2007; Ochsner, Bunge, Gross, & Gabrieli, 2002; Reed & Warner-Rogers, 2009). The ventral system, consisting of brain areas such as the limbic, brainstem region, and the orbitofrontal and medial structure of the PFC (Dennis, 2010; Reed & Warner-Rogers, 2009), is sensitive to emotionally significant information, and is responsible for executing autonomic evaluation and regulating emotion responses rapidly and defensively (Critchley, 2005; Dolan, 2002; Rolls, 2004). In contrast, the dorsal system, including the dorsolateral and the medial frontal areas (Dennis, 2010), utilizes motivationally relevant information from the ventral system to consciously formulate decisions among a number of courses of actions based on the anticipated consequents so as to regulate emotion in an effortful, proactive and deliberate way (Dennis, 2010; Ochsner et al., 2004; Reed & Warner-Rogers, 2009).

Abnormalities in the ventral and dorsal systems have been associated with maladaptive functioning related to emotional dysregulation. For example, neuroimaging evidence has shown that anxious and depressed individuals from seven to 32 year old have greater ventral activation of the PFC as compared to the non-depressed/anxious controls (Hare et al., 2008), implying highly and rigidly regulating/responding to the urging emotional value of the stimuli in the environment. In contrast, researchers have found that aggressive adults show deficits in both the OFC and other inter-connected core structures underlying emotion regulation, such as the ACC and the amygdala (Davidson, 2000; Hoptman, 2003), implying under-regulation of emotion with
both the ventral and dorsal systems. In his review article on the neurobiological bases of emotion regulation and violence, Davidson (2000) proposed that deficits in the core structures underlying emotion regulation, such as the OFC, the ACC, and the amygdala, would increase individuals’ vulnerability towards aggressive behavior (impulsive and affective aggression in particular).

Furthermore, the ACC, located in the medial frontal cortex, is an essential structure of the dorsal system, and plays a significant role in regulating cognition and emotion behaviors (Bush et al., 2000; Casey et al., 1997; Dennis, 2010; Reed & Warner-Rogers, 2009). Research into the effortful regulatory control of emotion behaviors as generated by the ACC has focused on several ERP components. For example, the Nc component is a frontocentral negative deflection peaking around 200-700 milliseconds following the presentation of the stimulus. It is generated from the areas of the medial frontal cortex, including the ACC, and is sensitive to facial emotions (Batty & Taylor, 2006; Lewis, Todd, & Honsberger, 2007; Nelson & Nugent, 1990; Todd, Lewis, Meusel, & Zelazo, 2008). In a normative sample, Dennis, Malone, and Chen (2009) found that larger Nc amplitudes in response to facial expressions are associated with greater attention performance and better parent-reported emotion regulation. In addition, N2 and Error-related negativity (ERN) have also been examined, and both reflect the cognitive control processing over emotion as mediated by the dorsal ACC. Specifically, the N2 reflects conflict monitoring and response inhibition, whereas the ERN reflects recognition of a mismatch error following incorrect responses (Bekker et al., 2005; Bokura, Yamaguchi, & Kobayashi, 2001). Preliminary findings using ERP components in relation to emotion regulation as regulated by the ACC have demonstrated that anxious children show larger N2 amplitudes towards both positive and
negative facial cues and greater ERN in the Go/No-Go task, indicating increased deliberate control of the proponent responses and more error monitoring (Hum, Manassis, & Lewis, 2013).

**Autonomic Network**

There is growing evidence for the significant role of the autonomic nervous systems in a wide variety of cognitive and emotional functions. In general, there are two branches of the ANS: the parasympathetic and the sympathetic nervous systems (PNS and SNS), which function to mediate the direction and the magnitude of HR change. In general, HR decreases and increases in response to the activation of PNS and SNS influences, respectively. In addition, investigators have examined respiratory sinus arrhythmia (RSA) to measure PNS activity and pre-ejection period (PEP) to measure SNS activity (Berntson, Cacioppo, Binkley, et al., 1994; Cacioppo, Uchino, & Berntson, 1994). Specifically, PNS-linked cardiac activity, indexed by RSA or heart rate variability (HRV), has been linked with emotion regulation, whereas SNS-linked cardiac activity is associated with the behavioral approach system and reflects reward reactivity (Beauchaine et al., 2007; Beauchaine, 2001, 2015b; Brenner, Beauchaine, & Sylvers, 2005).

**PNS activity.** PNS-linked cardiac activity is a critical psychophysiological component used to reflect the emotion regulation process (Beauchaine et al., 2007; Beauchaine, 2001, 2015b; Brenner et al., 2005). PNS activity (i.e., vagal activity), as indexed by RSA or HRV, reflects the periodic fluctuations in the HR that is related to respiration, and is determined largely by the vagal influences on the heart (Beauchaine, 2001; Grossman, Beek, & Wientjes, 1990). Many studies have demonstrated that low baseline RSA and abnormal RSA reactivity are related to emotion dysregulation (Beauchaine, 2015b).
According to the neurovisceral integration theory (Thayer, Hansen, Saus-Rose, & Johnsen, 2009), the PNS branch of the cardiovascular activity serves as a peripheral index of the medial PFC function that governs goal-oriented behavior. As stated by Thayer et al. (2009), the medial prefrontal, the insular, and the cingulate cortices form the central neural network that mediates activation of the amygdala and in turn provides inhibitory input to the heart via the PNS connection with the sinoatrial node (Porges, 1995). Given the existence of inhibitory neural efferent pathways from the medial PFC to the PNS (Barbas et al., 2003; Lane et al., 2009a; Ter Horst et al., 1997; Wong et al., 2007), efficient prefrontal function produces higher baseline RSA, and moderate levels of the RSA reactivity (Crowell et al., 2006). Consistent with this proposition, emotion regulation deficit has been related to the deficiency in the prefrontal brain regions (Davidson, 2000; Hare et al., 2008), which in turn is associated with lower RSA and an abnormal RSA reactivity (excessive RSA withdrawal or RSA augmentation) (Beauchaine, 2015b).

In addition, more convincing evidence that RSA is related to PFC has been found in lesion studies (e.g., Buchanan et al., 2010), and studies using positron emission tomography (e.g., Lane et al., 2009) or fMRI (see a review by Thayer et al., 2012). For example, Buchanan et al. (2010) found that medial PFC damage is related to altered PNS activity. Similarly, Lane et al. (2009a) found that blood flow in the medial PFC and the ACC during emotion induction is associated with RSA activity. Together, these findings demonstrate that the PFC and the ACC contribute, at least in part, to RSA activities.

**PNS activity and emotion regulation.** Many researchers have examined baseline RSA and RSA reactivity in attempts to understand the neurobiological mechanisms underlying emotion regulation. Higher baseline RSA is believed to reflect adaption to the environment and better
emotion regulation capacity (Beauchaine, 2015b; Gyurak & Ayduk, 2008; Marcovitch et al., 2010). In the context of threat or challenge, however, vagal influence of the heart is expected to withdraw, thus HR and the sympathetic responses (fight or flight) may increase (Berntson, Cacioppo, Quigley, & Fabro, 1994). The removal of the vagal influence is referred to as vagal withdrawal (i.e., decline in RSA from baseline to task, or increased RSA reactivity), whereas increased vagal influence is referred to as vagal augmentation (increase in RSA from baseline to task or reduced RSA reactivity). Higher baseline RSA under normal condition as well as moderate RSA reactivity (i.e., efficient vagal withdrawal) when regulation is required have been posited to relate to effective PFC functioning and adaptive coping and self-regulation (Beauchaine, 2001, 2015b). Conversely, dysregulation of emotion and behaviors is characterized by lower baseline RSA and excessive RSA reactivity. Furthermore, vagal augmentation in response to challenges may deaccelerate HR, suggesting immobilization responses and a failure to cope efficiently with environmental demands (Calkins & Dedmon, 2000; Calkins, 1997).

Empirical studies have documented the relationship between reduced baseline RSA and abnormal RSA reactivity and increased youth psychopathology, including autism spectrum disorder (ASD) (Patriquin, Scarpa, Friedman, & Porges, 2013), depression (Rottenberg, 2007), anxiety (Hastings, Sullivan, et al., 2008), ADHD (Musser, Galloway-Long, Frick, & Nigg, 2013), and elevated aggressive and antisocial behavior (Beauchaine et al., 2007; Beauchaine, Katkin, Strassberg, & Snarr, 2001; Boyce et al., 2001; Calkins & Dedmon, 2000; Calkins, Graziano, & Keane, 2007; de Wied, van Boxtel, Matthys, & Meeus, 2011). For example, de Wied, van Boxtel, Matthys, and Meeus (2011) found the most severe subtype of aggressive behavior (indexed by higher callous-unemotional traits) was related to low baseline RSA in 12- to 15-year-
old boys recruited from the special school for adolescents with behavior problems. Beauchaine, Katkin, Strassberg, and Snarr (2001) found that 12- to 17-year-old boys with both comorbid conduct problems and ADHD showed significant lower baseline RSA than both the ADHD only and the control groups. Pang and Beauchaine (2012) found that children aged eight and 12 years old with concurrent depression and conduct problems showed excessive RSA reactivity while watching movie clips. Boyce et al. (2001) and Calkins et al. (2007) found that higher externalizing behavior was related to reduced RSA reactivity or even RSA augmentation in a variety of cognitive and stress paradigms in children between five and seven years old from the community.

However, findings in this field are mixed. Some studies have shown that higher but not lower RSA activity is associated with more aggressive behavioral problems (Dietrich et al., 2007; Scarpa et al., 2000). For example, Dietrich et al. (2007) found that 10- to 13-year-old children with higher vagal tone (and lower HR) during a rest period in the supine position exhibited elevated externalizing problems. This result is not surprising given that PNS activity is negatively associated with HR, and lower HR is robustly associated with antisocial and aggressive behaviors (Ortiz & Raine, 2004). Theoretically, a predominant PNS over SNS activity or excessive vagal sensitivity has been argued to reflect a passive vagal coping response to stress, which may further contribute to both lower HR and more antisocial behaviors (Raine & Venables, 1984; Venables, 1988). In addition, some studies have found that reduced PNS reactivity (i.e., vagal augmentation) in response to a number of challenges were associated with fewer externalizing problems and better self-regulation (Crowell et al., 2006; Hastings, Nuselovici, et al., 2008).
Finally, other research has failed to establish a significant direct relationship between the PNS activity and externalizing behaviors (El-Sheikh, Harger, & Whitson, 2001; El-Sheikh & Whitson, 2006; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010). For example, El-Sheikh et al. (2001) did not find significant correlations between externalizing behavior and baseline RSA activity/RSA reactivity. Instead they found that higher baseline RSA may buffer children against developing externalizing behaviors from exposure to marital conflicts in a community sample of children aged between eight and 12 years old. Similarly, in a sample of nine- to 16-year-old children and adolescents, Gordis et al. (2010) found that higher baseline RSA (suggesting better emotion regulation) protected against the effects of maltreatment on developing aggressive behavior, although this effect was moderated by skin conductance reactivity among girls.

Chapter Summary

Emotion regulation has been broadly studied in the area of child development. Difficulties in regulating emotional behavior have been related to a variety of youth psychopathology and externalizing behavioral problems in particular. Multiple methods, including structured and semi-structured interviews, self-administered scales, behavioral observation, and neurobiological assessments, have been designed to assess emotion regulation in the young population. Specifically, neurobiological methodologies (e.g., imaging, EEG/ERP, and ANS measures) have made tremendous contributions for the understanding of the neural mechanisms underlying emotion regulation deficits in youths with emotional and behavioral disorders.

Neurobiological studies have revealed that emotion regulation is a top-down process, in which the cortical neural network regulates the emotion that is generated from the subcortical neural network. Moreover, efficient emotion regulation requires both autonomic and effortful
regulatory control of emotion as mediated by the brain regions involved in the ventral and dorsal neural systems. Lower baseline PNS activity and abnormal PNS reactivity, reflecting emotion dysregulation and partly contributed by deficits in brain regions including the PFC and the ACC, have been related to aggressive and antisocial behavior.
CHAPTER 3 - PHYSIOLOGICAL REGULATION AND SUBTYPES OF AGGRESSION

PNS Activity, Emotion Regulation and Subtypes of Aggression

One possible reason for the mixed findings regarding the relations between PNS activity and aggression is that most of the prior studies have not differentiated subtypes of aggressive behaviors; that is, instead of measuring RA and PA separately, researchers assessed aggressive behavior in a more general manner (e.g., the aggression subscale of the Child Behavior Checklist; Achenbach, 1991). Considerable findings from prior literature have shown that the two subtypes of aggressive behavior have distinct neurobiological correlates, as described previously. Moreover, understanding the relationship between emotion dysregulation and reactive-proactive aggression is crucial for better identification and treatment of youth with aggressive behaviors. Indeed, teaching better emotion regulation skills have been the essential elements of the cognitive-behavioral therapy used in youth with aggressive behaviors (Kazdin, Bass, Siegel, & Thomas, 1989; Sukhodolsky, Kassinove, & Gorman, 2004).

Although researchers have found RA to be negatively related to baseline RSA (Scarpa et al., 2010; Xu et al., 2013), and PA to be positively associated with baseline RSA (Scarpa et al., 2010), direct evidence for the relations between reactive-proactive aggression and the PNS-mediated emotion regulation process, as indexed by RSA reactivity, is limited. Based on theoretical and empirical evidence mentioned in prior sections, we expect that RA would be related to lower baseline RSA and increased RSA reactivity, whereas PA is related to higher baseline RSA and reduced RSA reactivity. This hypothesis is formulated based on the following reasons.

Firstly, prior research has demonstrated that RA is related to increased ANS activity such as elevated HR and skin conductance reactivity to stress (Hubbard et al., 2002; Pitts, 1997). It
has been proposed that RA is “hot-headed” and characterized by autonomic over-arousal (Dodge, 1991; Scarpa & Raine, 1997). It is therefore possible that RA is characterized by hyper-active ANS activity, as reflected by higher RSA reactivity. Secondly, prior studies have shown that low baseline RSA (Dietrich et al., 2007; Forbes et al., 2006) and excessive RSA reactivity (Boyce et al., 2001; Calkins et al., 2007), or both (Hinnant & El-Sheikh, 2009), are associated with internalizing symptoms in children and adolescents. Given that RA rather than PA shows stronger correlations with internalizing behaviors (Card & Little, 2006; Scarpa et al., 2010; Dodge et al., 1997; Raine et al., 2006), it is conceivable that RA would be related to low baseline RSA and increased RSA reactivity, which reflect limited capacity of emotion regulation and the tendency of vigilance and over-sensitivity to emotional stimulation when confronted.

In contrast, previous studies have shown that PA is related to reduced ANS activity such as blunted skin conductance response to punishment (Gao et al., 2015; Lorber, 2004; Scarpa et al., 2010). Individuals with higher PA are “cold-blooded” and are characterized by autonomic under-arousal (Dodge, 1991; Scarpa & Raine, 1997). Therefore, it is possible that PA is related to a hypoactive ANS, as indicted by lower RSA reactivity. In addition, PA has been associated with psychopathic and callous-unemotional traits that are characterized by lack of guilt and empathy (Fanti et al., 2008; Raine et al., 2006), and reduced empathy and lower level of prosocial behavior are associated with reduced RSA reactivity (Gill & Calkins, 2003; Liew et al., 2011). Taken together, it is expected that PA would be associated with reduced RSA reactivity, reflecting a failure to engage with the environmental demands and immobilization responses in a challenging situation (Calkins, 1997). Evidence from adult literature has provided some indirect evidence for this proposition. For example, in women with a history of sexual abuse, proactive but
not reactive relationship aggression was found to be related to blunted RSA reactivity in response to a stressor (Murray-Close & Rellini, 2012).

**Joint Effects of Baseline RSA and RSA Reactivity**

Previous studies have focused mainly on the baseline RSA or RSA reactivity as independent predictor of aggressive behavior (e.g., de Wied et al., 2011; Pang & Beauchaine, 2013; Scarpa et al., 2008), as both measures reflect important aspects of the PNS-mediated emotional processes (Porges, 2007). However, one study has suggested that considering the joint effects of baseline RSA and RSA reactivity is important in predicting behavioral problems (Hinnant & El-Sheikh, 2009). In this study, researchers found that internalizing behavior is highest among children who showed low baseline RSA in conjunction with excessive RSA reactivity and that externalizing behavior is highest among those showing both low baseline RSA in conjunction with reduced RSA reactivity in a group of eight- to nine-year-old children from the community. It is suggested that future studies examine the independent effect of baseline RSA and RSA reactivity as well as their joint effects to further understand the etiology of aggressive and antisocial behavior (Hinnant & El-Sheikh, 2009).

**Biosocial Interaction**

Both neurobiological and psychosocial risk factors are important for understanding the etiologies of aggressive and antisocial behaviors (Gao, Baker, Raine, Wu, & Bezdjian, 2009; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010; Raine et al., 2014). There is growing evidence that the relationship between physiological activity and antisocial behaviors can be moderated by psychosocial factors, such as family adversity and history of abuse (Gao et al., 2009; Maliphant, Hume, & Furnham, 1990; Raine et al., 1997; Raine & Venables, 1984; Scarpa &
One line of this work has shown that the relationship between neurobiological risk factors and antisocial behaviors is particularly stronger in individuals without the psychosocial adversity background. For example, Raine and Venables (1984) found that low baseline HR is a risk factor for antisocial behaviors particularly in adolescents with higher socioeconomic status. Gao et al. (2009) found that the positive relationship between risky decision-making and callous-unemotional/psychopathic traits was only significant in children from benign home environments.

One proposed explanation for this interaction effect is the “social push” theory (Raine, 2002). According to this theory, if an individual is not exposed to social risk factors that “push” him/her towards antisocial behaviors, the neurobiological risk factors will likely be more meaningful in predicting antisocial behaviors. In contrast, if an individual is exposed to social risk factors, then those influences will better account for his/her antisocial tendencies. The “social push” theory provides important insights into the interplay between the biological and psychosocial influences. Alternatively, some studies have illustrated a different kind of biosocial interacting effect. For example, Obradović et al. (2010) found that children with excessive RSA reactivity to stress showed more externalizing behavior symptoms in the context of higher levels of family stress. Likewise, in a group of Hong Kong schoolchildren, Raine et al. (2014) found that low baseline HR in combination with high social adversity predicted high RA but not PA. Taken together, these findings contribute to the growing body of research that examines both biological and psychosocial factors associated with antisocial and aggressive behavior, and highlight the importance of investigating their interaction effects to obtain a more holistic picture of the development of antisocial behavior.
A few studies have examined the differential biosocial interaction effects in relation to RA and PA. For example, in a group of undergraduate participants ($N = 84$), researchers measured RSA activity during rest and an emotion-evoking decision-making task. It was found that high RA was related to high baseline RSA at the levels of high social adversity and elevated RSA reactivity at the levels of low social adversity; in contrast, high PA was related to reduced RSA reactivity at the low levels of social adversity (Zhang & Gao, 2015). Raine et al. (2014) found that the interaction effect between low baseline HR and high social adversity was specific to RA, suggesting that psychosocial influences may play a more critical role in the development of reactive than in proactive aggression (Card & Little, 2006; Polman, Orobio de Castro, Koops, van Boxtel, & Merk, 2007).

**Moderating Effect of Gender**

Research relating physiological regulation deficits and aggressive behaviors has been mostly conducted with boys or male adults, partly due to the fact that boys are more likely to develop aggressive and antisocial behaviors than girls (Beauchaine, Hong, & Marsh, 2008; Keenan, 2006; Lorber, 2004). Recent studies suggest there may be different ANS mechanisms of externalizing behavior for boys versus girls (Beauchaine et al., 2008; Gordis et al., 2010). For example, among 175 eight to 12 years old children, Beauchaine et al. (2008) found that boys with more aggression and conduct problems showed lower baseline RSA and reduced PEP reactivity towards reward (suggesting reward insensitivity in this individuals), whereas aggressive girls showed similar physiological responses as the girls from the control group. Furthermore, as previously reviewed in Chapter 1, boys and girls show differential correlates of the two subtypes of
aggression. Therefore, it is possible that the relationships between PNS activity and subtypes of aggression are moderated by gender. No studies have been conducted to address this issue.

**Chapter Summary**

Based on prior literature, it was hypothesized that RA would be associated with low baseline RSA and increased RSA reactivity, and that PA be characterized by high baseline RSA and reduced RSA reactivity. Evidence has also suggested that baseline RSA and RSA reactivity may interact in predicting antisocial behavior. Finally, the moderating effect of social adversity and gender should also be examined. Addressing these research gaps may provide important insights into better understanding of the etiology of aggressive and antisocial behavior, and may have implications for development of treatment and intervention programs.
CHAPTER 4 – CURRENT STUDY

No study has examined the effect of emotion dysregulation on RA and PA in children using psychophysiological approaches. The current study examined the relationships between patterns of both baseline RSA and RSA reactivity and the two different types of aggressive behavior, and the moderating effect of social adversity. In an attempt to address these research questions, data from a longitudinal study with eight to 10 years old children at the initial assessment and the follow-up visit one-year later were analyzed. Hypotheses were tested by examining the concurrent relations between measures of RSA, aggression, and social adversity (SA) at each time point, and the predictive relations between these measures from Time 1 (T1) to Time 2 (T2). The moderating effect of gender was also explored. Specifically, the following aims and hypotheses were tested:

**Aim 1: The concurrent relations between RSA, aggression, and SA (for T1 and T2 separately)**

Hypothesis 1.1. RA would be characterized by low baseline RSA and increased RSA reactivity, whereas PA would be linked to high baseline RSA and reduced RSA reactivity.

Hypothesis 1.2. The combination of low baseline RSA and high RSA reactivity would result in highest score on RA, and the combination of high baseline RSA and low RSA reactivity would lead to highest PA.

Hypothesis 1.3. The above associations would be stronger at the lower compared to higher levels of SA.

**Aim 2: Predictive relations between RSA, aggression, and SA**
Hypothesis 2.1. Individuals with low baseline RSA and/or increased RSA reactivity at both time points would show highest scores on RA at T2.

Hypothesis 2.2. Individuals with high baseline RSA and/or reduced RSA reactivity at both time points would have highest scores on PA at T2.

Hypothesis 2.3. The above relations are more salient under the condition of low levels of SA.

Method

Participants

The original sample consisted of eight- to 10-year-old children (mean age = 9.06, SD = 0.60) that were drawn from a longitudinal study that examined the social and neurobiological risk factors for children and adolescents living in Brooklyn, New York. Within the study area, fliers soliciting enrollment were placed in public areas and targeted mailings were sent to parents. Children with a diagnosed psychiatric disorder, mental retardation, or a pervasive developmental disorder were excluded. At T1, there were 340 participants, 164 of whom were male (48.2%), with ethnic breakdown as follows: 52% Black, 21% Caucasian, 11% Hispanic, 2% Asian, and the remaining 14% of mixed/other. At T2 (one year later), there were 254 participants, 122 of whom were male (48%), with ethnicity breakdown as 47.3% Black, 22.9% Caucasian, 8.6% Hispanic, and the remaining 17.9% mixed or other.

Data from the T1 and T2 assessment were used in the current study. Participants and their primary caregivers were invited to the university for a 2-hour laboratory visit including behavioral interviews, neurocognitive testing, psychophysiological recording, as well as social risk factor assessment. Monetary compensation and transportation reimbursement were provided to
the participating families at the end of the visit. The university Institutional Review Board approved all procedures. Parental consent and child assent were obtained for all participants.

**Measures**

**Reactive and Proactive Aggression.** The self-report Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006) (Appendix A) was used to assess the two types of aggression at both T1 and T2. There are 11 items (e.g., “Reacted angrily when provoked by others?”) assessing RA and 12 items (e.g., “Had fights with others to show who was on top”) assessing PA. Participants rated the frequency of occurrence on a 3-point Likert scale (0 = “never”, 1 = “sometimes”, and 2 = “often”), with higher total scores denoting higher aggression. Prior studies showed that the correlation between RA and PA range from .67 to .83 (Dodge & Coie, 1987; Poulin & Boivin, 2000a; Price & Dodge, 1989; Raine et al., 2006). In the current study, the Cronbach’s alpha internal reliability for RA and PA were .815 and .808 at T1 and .792 and .782 at T2.

**Social Adversity.** Following prior literature, a social adversity index was created based on caregiver’s report on the psychosocial information questionnaire at T1 (Choy et al., 2015; Gao, Raine, Chan, Venables, & Mednick, 2010; Raine, Reynolds, Venables, & Mednick, 2002) (Appendix B). The social adversity index was created by adding 1 point for each of the following 10 items: Divorced Parents (single parent family or living with guardians other than parents), Foster Home, Public Housing, Welfare Food Stamps, Parent Ever Arrested (either parent has been arrested at least once), Parents Physically Ill, Parents Mentally Ill, Crowded Home (five or more family members per house room), Teenager Mother (aged years 19 or younger when child
was born), and Large Family (sibling order fifth or higher by age 3 years). All items are scored either 0 = “no” or 1 = “yes”, with a higher total score denoting higher level of social adversity.

**Psychophysiological Data Acquisition and Reduction**

At both T1 and T2, children’s RSA activity was recorded during a 2-min rest period (baseline) and an emotion-regulation paradigm (Musser et al., 2011).

**Emotion Regulation Task.** The emotion regulation task (Musser et al., 2011, 2013) consisted of four 2-min-long film clips taken from the movie Homeward Bound, the story of three pets who are left behind when their family goes on vacation and try to find their way home, were presented. Children were instructed to either induce or suppress their emotion while their physiological activities were measured. The first two movie clips elicited negative emotions and the last two elicited positive emotions. In the induction condition, children were asked to show the emotion they experience of the main characters, and in the suppression condition, children were told to think about how the characters were feeling but not to show the emotion on their faces. The same sequence of the clips and instruction was given to each child: 1) negative induction, 2) negative suppression, 3) positive induction, and 4) positive suppression. A 2-min rest condition was present for helping restore emotion between the negative and the positive conditions.

**RSA.** All psychophysiological data were collected continually using a MP150 system and analyzed offline with AcqKnowledge 4.2 software (Biopac Systems Inc., Goleta, CA). RSA was derived from the ECG100C amplifier with a band pass filter of 35 Hz and 1.0 Hz and a RSP100C respiration amplifier with a band pass filter of 1.0 Hz and 0.05 HZ. ECG was recorded using the ECG100C amplifier with two pre-jelled Ag-AgCl disposable vinyl electrodes placed at a modified Lead II configuration. Respiration was recorded using RSP100C amplifier by putting
a respiration belt around the abdomen of the subject at the point of complete exhalation. The AcqKnowledge automated function for RSA analysis was utilized. This software followed the well-validated peak-valley method (Grossman et al., 1990), in which RSA was computed in milliseconds as the difference between the minimum and the maximum R-R intervals during respiration. Higher values reflect greater PNS activity while lower values indicate lower PNS activity (Gruber, Harvey, & Johnson, 2009). As all RSA variables are positive skewed, log transformations were applied before further analysis.

Baseline RSA was calculated as the average of the two 2-min rest periods at the beginning and the end of the 40-min long psychophysiological protocol. Following prior literature (Calkins et al., 2007; Musser et al., 2013), RSA reactivity was computed by subtracting the baseline RSA value from the average RSA value during each movie clip, with negative values reflecting RSA withdrawal or increased RSA reactivity and positive values reflecting RSA augmentation or reduced RSA reactivity. RSA reactivity across the four movie conditions was highly correlated \( r = 0.80 - 0.92 \), therefore, the average of the four RSA reactivity measures was used in following analyses.

**Missing Data, Skewness, and Outliers**

At each time point some RSA data were missing due to acquisition or scoring problems, such as equipment malfunction, extraneous movement (e.g., too much noise to detect R-waves), and electrode misplacement or displacement (e.g., improper placement of the respiration belt). In addition, a few participants have missing data in aggression and social adversity measures. Missing data were assessed by the Little’s MCAR test, which showed that data were missing completely at random (Little & Rubin, 1989) (all \( p > .600 \), non-significant \( p \) values indicate data are
missing completely at random). Pairwise deletion was therefore used to maximize all data available on an analysis by analysis basis. Furthermore, given that at T1 RSA reactivity data from 40% of participants (N=136) were missing due to technical issues, independent samples t-tests were performed to determine if the remaining participants (N = 202) differed significantly from the rest on gender, ethnicity, and the main study variables. Results indicated that these two groups did not differ on any of these measures (all ps >. 272).

Prior to analyses, univariate outliers (±3SDs from the mean) were removed. For highly skewed variables (e.g., RSA and PA), outliers were removed after a log-transformation was applied. A full description of missing data and outliers for the primary study variables of the individual and combined T1 and T2 datasets is presented in Table 3. T1 dataset (N = 340) and T2 dataset (N = 254) were each used independently to examine the concurrent relationships. The combined T1 & T2 dataset (N = 254) with children participated in both assessments was used in the longitudinal (repeated-measures) analyses.
Table 3. Missing Data and Outliers for Each Time Point Separately and for the Combined Dataset.

<table>
<thead>
<tr>
<th></th>
<th>RSA</th>
<th>RSA-R</th>
<th>RA</th>
<th>PA</th>
<th>SA</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>48 (14%)</td>
<td>136 (40%)</td>
<td>11 (3%)</td>
<td>11 (3%)</td>
<td>5 (1%)</td>
</tr>
<tr>
<td>N = 340</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outliers</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Available Cases</td>
<td>290</td>
<td>202</td>
<td>329</td>
<td>328</td>
<td>334</td>
</tr>
<tr>
<td>Means</td>
<td>127.96</td>
<td>-3.18</td>
<td>6.40</td>
<td>1.59</td>
<td>2.95</td>
</tr>
<tr>
<td>SDs</td>
<td>61.14</td>
<td>26.48</td>
<td>4.37</td>
<td>2.63</td>
<td>2.00</td>
</tr>
<tr>
<td>Range</td>
<td>[24.75, 319.22]</td>
<td>[-88.78, 78.25]</td>
<td>[0,20]</td>
<td>[0,15]</td>
<td>[0,8]</td>
</tr>
<tr>
<td>Skewness</td>
<td>0.90</td>
<td>0.064</td>
<td>0.59</td>
<td>2.53</td>
<td>0.50</td>
</tr>
<tr>
<td>T2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>4 (2%)</td>
<td>31 (12%)</td>
<td>7 (3%)</td>
<td>7 (3%)</td>
<td>5 (2%)</td>
</tr>
<tr>
<td>N = 254</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outliers</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Available Cases</td>
<td>249</td>
<td>221</td>
<td>247</td>
<td>246</td>
<td>249</td>
</tr>
<tr>
<td>Means</td>
<td>125.43</td>
<td>-1.62</td>
<td>7.47</td>
<td>1.44</td>
<td>2.74</td>
</tr>
<tr>
<td>SDs</td>
<td>60.64</td>
<td>36.32</td>
<td>3.92</td>
<td>2.24</td>
<td>1.88</td>
</tr>
<tr>
<td>Range</td>
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<td>[-118.77, 115.66]</td>
<td>[0,19]</td>
<td>[0,13]</td>
<td>[0,8]</td>
</tr>
<tr>
<td>Skewness</td>
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<td>0.16</td>
<td>0.41</td>
<td>2.55</td>
<td>0.51</td>
</tr>
<tr>
<td>T1&amp;T2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missing from T1</td>
<td>35 (16%)</td>
<td>103 (40%)</td>
<td>8 (3%)</td>
<td>8 (3%)</td>
<td>5 (2%)</td>
</tr>
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<td>N = 254</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outliers from T1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Outliers from T2</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Available Cases</td>
<td>215</td>
<td>133</td>
<td>239</td>
<td>239</td>
<td>249</td>
</tr>
</tbody>
</table>

Note. RSA-R = RSA reactivity; RA = reactive aggression; PA = proactive aggression; SA = social adversity.

Statistical Analyses

**Aim 1- Concurrent relations between RSA, aggression, and SA.** At each time point, hierarchical multiple regressions were conducted to examine the relationships between RSA and aggression, and the moderating effect of SA. In all regression models, RA or PA was the dependent variable. First, baseline RSA, RSA reactivity, and SA were entered to test for their main effects. Second, the two-way interaction terms were added (i.e., baseline RSA x RSA reactivity, baseline RSA x SA, and RSA reactivity x SA). Finally, the three-way interaction term (i.e., baseline RSA x RSA reactivity x SA) was entered. All variables were mean centralized before analyses. Collinearity diagnostic tests were conducted and showed that the regression analyses were
not affected by multicollinearity (i.e., tolerance >.10 and variance inflation factor <10; Tabachnick & Fidell, 2013).

Significant two-way interaction effect would be probed by conducting the simple effect analysis of baseline RSA/RSA reactivity on aggression at high (1 +SD) and low (1 –SD) level of RSA reactivity or SA. Should the three-way interaction be significant, the two-way interaction between baseline RSA and RSA reactivity would be examined at the low (1 –SD) vs. high (1 +SD) levels of SA (Aiken, Leona, & West, 1991).

Independent samples t-tests were performed to examine gender effects. Should any significant gender difference be observed for either aggression measure, gender would be entered in the first step in the regression models to control for its effect, and regression analyses would then be conducted again separately for boys and girls.

Given that RA and PA are highly correlated, many researchers control for the non-focal subtype of aggression or use a residual score (i.e., RA score is regressed on PA score as to create a “pure” measure of RA) in their analyses (Cima & Raine, 2009; Fanti et al., 2008; Lynam, Hoyle, & Newman, 2006; Miller & Lynam, 2006; Poulin & Boivin, 2000b; Raine et al., 2006). However, it has been argued that the interpretation of the findings after partialling might be problematic due to the high proportion of error variances (Lynam et al., 2006). In contrast, some researchers have suggested that the removal of the common variances between RA and PA could be helpful for detecting the unique differences between the two subtypes of aggressive behavior (Miller & Lynam, 2006; Raine et al., 2006). It is therefore recommended that results for both the “raw” score relationships and the partial relationship shall be reported (Miller & Lynam, 2006; Raine et al., 2006). Following this suggestion, in addition to using the “raw” scores, the unique
variance of each form of aggression would be evaluated by controlling for the other form of aggresive behavior in the regression models. In line with prior studies (Fanti et al., 2008; Poulin & Boivin, 2000b; Smithmyer, Hubbard, & Simons, 2000), all regression analyses were performed again with the non-focal type of aggression added as a covariate in the first step.

**Aim 2: Predictive relations between RSA, aggression, and SA.**

**Baseline RSA.** To test the hypothesis that individuals with low baseline RSA at both time points would show higher T2 RA score, and that individuals with high baseline RSA at both time points would score highest on T2 PA, three discrete groups were formed on the basis of whether participants fell into the top or bottom 50% cutoffs on two waves of baseline RSA measures. Persistently high baseline RSA participants were defined as those who fell into the top 50% of baseline RSA at both time points, persistently low baseline RSA group were those who fell into the bottom half of baseline RSA at both time points, and the other participants were in the mixed baseline RSA group. F tests (for continuous variables) and Fisher’s Exact tests (for categorical variables) were conducted to examine group differences on RSA measures, socio-demographic data (ethnicity, sex, and SA) and aggression.

Two-way ANCOVAs with RSA group (persistently low baseline RSA, persistently high baseline RSA, and mixed group) and SA (high vs. low; median split, median = 3) were conducted for RA and PA separately when controlling for covariates and T1 RA/PA measure. Should the interaction between RSA group and SA be significant, one-way ANCOVAs would be conducted to examine the effect of RSA group separately for individuals with higher and those with lower levels of social adversity.
Hierarchical regression analyses were also conducted with T1 and T2 baseline RSA, SA, and their two-way and three-way interactions as predictors. The T2 aggression measure was the predicted variable with the T1 aggression measure as a covariate.

**RSA Reactivity.** Similarly, three groups were formed: the “persistently increased RSA reactivity” group consists of individuals who showed increased RSA reactivity at both time points, the “persistently reduced RSA reactivity” group are those showing reduced RSA reactivity at both T1 and T2, and the rest are in the “mixed group”. Two-way ANCOVAs were first performed, and then one-way ANCOVAs were conducted if the interaction between RSA reactivity group and SA was significant.

Finally, a regression was performed with T2 RA/PA as the predicted variable, T1 aggression measure as a covariate, and the T1 RSA reactivity, T2 RSA reactivity, SA, and their two-way and three-way interactions as predictors.

## Results

**Aim 1: Concurrent Relations between RSA, Aggression, and SA**

**Correlations.** Correlations between the main study variables at T1 and T2 are presented in Table 4. As expected, at both waves, RA and PA were highly correlated ($r = .60$ at T1 and .63 at T2, both $p < .01$). Increased RSA reactivity was correlated to PA at T1 ($r = -.15$, $p < .05$) but not T2 ($r = .02$, n.s.). Baseline RSA was not associated with either RA or PA at both time points. SA was positively associated with RA ($r = .11$ at T1 and .23 at T2, $p < .05$) at both time points, and also with PA ($r = .25$, $p < .05$) at T2. Since boys showed significantly higher RA scores than girls at both T1 and T2 ($p < .022$), when predicting RA gender was entered in the first step to
control for its effect, and separate regression analyses were conducted to predict RA in boys and girls. Gender differences were not found for PA, ethnicity, SA, baseline RSA, or RSA reactivity.

Table 4. Correlations between Main Study Variables at Each Time Point

<table>
<thead>
<tr>
<th></th>
<th>T 1</th>
<th>RSA†</th>
<th>RSA-R†</th>
<th>RA</th>
<th>PA†</th>
<th>SA</th>
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</thead>
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<tr>
<td>RSA†</td>
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</tr>
<tr>
<td>RSA-R†</td>
<td>-.32**</td>
<td>1.00</td>
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<td></td>
</tr>
<tr>
<td>RA</td>
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<td>-.07</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PA†</td>
<td>.04</td>
<td>-.15*</td>
<td>.60**</td>
<td>1.00</td>
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<td></td>
</tr>
<tr>
<td>SA</td>
<td>.16**</td>
<td>.00</td>
<td>.11*</td>
<td>.07</td>
<td>1.00</td>
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<table>
<thead>
<tr>
<th></th>
<th>T 2</th>
<th>RSA†</th>
<th>RSA-R†</th>
<th>RA</th>
<th>PA†</th>
<th>SA</th>
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</thead>
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<td></td>
</tr>
<tr>
<td>RSA-R†</td>
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<td>1.00</td>
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<tr>
<td>RA</td>
<td>.04</td>
<td>-.03</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PA†</td>
<td>-.03</td>
<td>.02</td>
<td>.63**</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SA</td>
<td>.02</td>
<td>-.02</td>
<td>.23**</td>
<td>.25**</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Note. †Log transformed variables. *p<.05, **p<.01

T1. Regression analyses showed that the models with main effects (baseline RSA, RSA reactivity, and SA) did not reach significance when predicting either RA or PA (for RA, p = .485; for PA, p = .156), although RSA reactivity remained a significant predictor for PA (p = .034). See Table 5 for the summary of regression results. Similarly, adding the two-way interactions to the model did not add significant predictive ability (RA, p = .221; PA, p = .860), although the baseline RSA x RSA reactivity interaction effect remained significant when predicting RA (p = .029). Finally, the models including the three-way interactions did not significantly predict RA or PA (for RA, p = .313; for PA, p = .325). When analyses were conducted for boys and girls separately for RA, results were substantially the same.
### Table 5. Summary of Hierarchical Regressions for RA and PA: Predicting Effects of Baseline RSA, RSA Reactivity, Social Adversity and Their Interactions

<table>
<thead>
<tr>
<th>T1</th>
<th></th>
<th>RA</th>
<th></th>
<th></th>
<th></th>
<th>PA†</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>B</td>
<td>SE</td>
<td>t</td>
<td>∆R²</td>
<td>B</td>
<td>SE</td>
<td>t</td>
<td>∆R²</td>
</tr>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
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<td>-2.39*</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
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<td>0.07</td>
<td>0.47</td>
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</tr>
<tr>
<td>Baseline RSA†</td>
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<td>-0.16</td>
<td>0.08</td>
<td>-2.14*</td>
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</tr>
<tr>
<td>RSA Reactivity†</td>
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<td>0.08</td>
<td>0.45</td>
<td>0.02</td>
<td>0.08</td>
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</tr>
<tr>
<td>Baseline RSA† x RSA Reactivity†</td>
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<td>0.08</td>
<td>-0.91</td>
<td>0.00</td>
<td>0.08</td>
<td>0.02</td>
<td></td>
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</tr>
<tr>
<td>Baseline RSA† x SA</td>
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<td>0.07</td>
<td>-0.07</td>
<td>-0.06</td>
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<td>-0.80</td>
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<tr>
<td>RSA Reactivity† x SA</td>
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<td>-0.08</td>
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<td>-1.01</td>
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<table>
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<th></th>
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<th></th>
<th>PA†</th>
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<tr>
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<td></td>
<td>B</td>
<td>SE</td>
<td>t</td>
<td>∆R²</td>
<td>B</td>
<td>SE</td>
<td>t</td>
<td>∆R²</td>
</tr>
<tr>
<td>Step 1</td>
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<td>0.07</td>
<td>-3.31**</td>
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<td>Gender</td>
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<td>0.07</td>
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<tr>
<td>Baseline RSA†</td>
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<td>0.07</td>
<td>3.00**</td>
<td>0.22</td>
<td>0.07</td>
<td>3.20**</td>
<td></td>
<td></td>
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<tr>
<td>RSA Reactivity†</td>
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<td>0.07</td>
<td>1.02</td>
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<td>0.07</td>
<td>-0.58</td>
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</tr>
<tr>
<td>Baseline RSA† x RSA Reactivity†</td>
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<td>0.07</td>
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<td>0.01</td>
<td>0.07</td>
<td>0.07</td>
<td></td>
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</tr>
<tr>
<td>Baseline RSA† x SA</td>
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<td>0.06</td>
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<td>-0.16</td>
<td>0.07</td>
<td>-2.50*</td>
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<td></td>
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<tr>
<td>RSA Reactivity† x SA</td>
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<td>Step 4</td>
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<td>0.07</td>
<td>-0.40</td>
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<td></td>
</tr>
</tbody>
</table>

Note. †Log transformed variables. *p<.10, *p<.05, **p<.01.

Regression analyses were also conducted when the non-focal aggression was controlled for in the first step. Similar results were obtained in terms of the main effects of baseline RSA, RSA reactivity, and SA as well as their two- and three-way interactions for the overall sample (RA, ps > .160; PA, ps > .225). Finally, when analyses were examined separately for boys and
girls, after PA was controlled for, adding the two-way interactions was significant in predicting RA in boys only ($p = .034$), with the baseline RSA x RSA reactivity interaction effect being significant ($p = .009$). Simple effect analysis showed that after controlling for PA, higher baseline RSA was related to higher RA in those with increased RSA reactivity ($p = .039$), whereas lower baseline RSA was marginally associated with higher RA in boys with reduced RSA reactivity ($p = .076$) (Figure 2). No such effects were found in girls.

![Figure 2. The Interaction Effect between Baseline RSA and RSA Reactivity in Predicting Time 1 RA (controlling for PA) in Boys Only](image)

**T2.** Regression analyses showed that the models including main effects were significant for both RA and PA (for RA, $p = .018$; for PA, $p = .017$). In particular, higher social adversity was related to higher RA and PA (for RA, $p = .003$; for PA, $p = .002$), although neither baseline RSA nor RSA reactivity was a significant predictor for RA or PA (all $ps > .602$). See Table 3.
For RA, adding the two-way interaction terms did not reach significance \( (p = .190) \), although the RSA reactivity x SA interaction was significant \( (p = .034) \). After controlling for the main effects, the model with two-way interactions was marginally significant for PA \( (p = .053) \), with the RSA reactivity x social adversity interaction being significant \( (p = .013) \). Simple effect analysis showed that reduced RSA reactivity was marginally related to higher PA at low levels of adversity \( (p = .053) \), whereas RSA reactivity was not associated with PA at high levels of adversity \( (p = .150) \) (Figure 3). Finally, when regression analyses were performed separately for girls and boys for RA and when the non-focal aggression was controlled for, results were substantially the same.

*Figure 3. The Interaction Effect between RSA Reactivity and Social Adversity in Predicting Time 2 PA (Combining Boys and Girls)*
Summary. Hierarchical regression models were conducted to examine the effects of RSA measures and SA on the two types of aggression at each time point. It was found that neither baseline RSA nor RSA reactivity was significantly associated with RA or PA. However, after controlling for PA, the joint effect of higher baseline RSA and increased RSA reactivity was associated with higher RA in boys only at T1. In addition, reduced RSA reactivity was marginally related to higher PA in the overall sample at T2, but this effect was only significant in the condition of low levels of SA.

Aim 2: Predictive Relations between RSA, Aggression, and SA

Correlations. From T1 to T2, the autocorrelations of baseline RSA and RSA reactivity were .50 (p < .01) and .14 (n. s.), respectively (see Table 6). Figures 4 and 5 illustrate the individual differences in the change pattern of RSA measures from T1 to T2.

Table 6. Correlations between Time 1 and Time 2 Variables

<table>
<thead>
<tr>
<th></th>
<th>T1 RSA†</th>
<th>T2 RSA†</th>
<th>T1 RSA-R†</th>
<th>T2 RSA-R†</th>
<th>T1 RA</th>
<th>T2 RA</th>
<th>T1 PA†</th>
<th>T2 PA†</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 RSA†</td>
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<td>T1 RSA-R†</td>
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<td>-.07</td>
<td>1.00</td>
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<td></td>
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<tr>
<td>T2 RSA-R†</td>
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<td>-.30**</td>
<td>.14</td>
<td>1.00</td>
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<td>.14*</td>
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<td>-.05</td>
<td>1.00</td>
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<tr>
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<td>-.01</td>
<td>-.03</td>
<td>.42**</td>
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<td>-.09</td>
<td>-.05</td>
<td>.59**</td>
<td>.26**</td>
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<tr>
<td>T2 PA†</td>
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<td>-.08</td>
<td>.02</td>
<td>.31**</td>
<td>.63**</td>
<td>.33**</td>
<td>1.00</td>
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</table>

Note. †Log transformed variables. *p<.05, **p<.01.
Figure 4. Individual Differences in the Change Pattern of Baseline RSA from Time 1 to Time 2 ($r = .50, p < .01$)

Figure 5. Individual Differences in the Change Pattern of RSA Reactivity from Time 1 to Time 2 ($r = .14, p > .05$).
Baseline RSA. Three baseline RSA groups were created (i.e., persistently high baseline RSA, \( n = 78 \); mixed baseline RSA, \( n = 57 \); persistently low baseline RSA, \( n = 80 \)) based on whether participants fell into the top or bottom half on baseline RSA at T1 and T2. Means, SDs, and ranges for the three groups on these measures are listed in Table 7. Fisher’s Exact test showed that the three groups differed on ethnicity (\( p = .001 \)): there were more Caucasian and Hispanic but fewer African American participants in the “persistently low baseline” group, and more African American participants in the high baseline group than expected.

Table 7. Baseline RSA Group Comparisons on RSA, Sociodemographic and Aggression Measures

<table>
<thead>
<tr>
<th>T1 &amp; T2 Dataset</th>
<th>Persistently Low Baseline RSA ( (n = 80) )</th>
<th>Mixed Baseline RSA ( (n = 57) )</th>
<th>Persistently High Baseline RSA ( (n = 78) )</th>
<th>( F/ \chi^2 )</th>
<th>( p )</th>
<th>( \eta^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 Baseline RSA ( \dagger )</td>
<td>1.86 (0.18) [1.39, 2.07]</td>
<td>2.03 (0.15) [1.60, 2.47]</td>
<td>2.24 (0.12) [2.08, 2.50]</td>
<td>126.71 ( a )</td>
<td>0.000</td>
<td>0.54</td>
</tr>
<tr>
<td>T2 Baseline RSA ( \dagger )</td>
<td>1.86 (0.11) [1.53, 2.02]</td>
<td>2.07 (0.17) [1.59, 2.41]</td>
<td>2.22 (0.12) [2.03, 2.49]</td>
<td>145.45 ( a )</td>
<td>0.000</td>
<td>0.58</td>
</tr>
<tr>
<td>T1 RSA Reactivity ( \dagger )</td>
<td>-0.00 (0.10) [-0.20, 0.21]</td>
<td>0.00 (0.11) [-0.23, 0.20]</td>
<td>-0.04 (0.07) [-0.24, 0.15]</td>
<td>3.20 ( a )</td>
<td>0.044</td>
<td>0.04</td>
</tr>
<tr>
<td>T2 RSA Reactivity ( \dagger )</td>
<td>0.01 (0.11) [-0.24, 0.34]</td>
<td>0.02 (0.12) [-0.27, 0.36]</td>
<td>-0.02 (0.11) [-0.31, 0.26]</td>
<td>1.84 ( a )</td>
<td>0.141</td>
<td>0.02</td>
</tr>
<tr>
<td>Ethnicity (%)</td>
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<td></td>
<td></td>
<td>25.67 ( b )</td>
<td>0.001</td>
<td>-</td>
</tr>
<tr>
<td>Caucasian</td>
<td>25 (32%)</td>
<td>10 (18%)</td>
<td>13 (26%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>24 (31%)</td>
<td>27 (47%)</td>
<td>49 (61%)</td>
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<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>11 (14%)</td>
<td>1 (2%)</td>
<td>5 (6%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>3 (4%)</td>
<td>2 (4%)</td>
<td>2 (3%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed or other</td>
<td>17 (21%)</td>
<td>17 (30%)</td>
<td>9 (12%)</td>
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</tr>
<tr>
<td>Male (%)</td>
<td>38 (48%)</td>
<td>31 (54%)</td>
<td>38 (49%)</td>
<td>0.69 ( b )</td>
<td>0.710</td>
<td>-</td>
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<td>SA</td>
<td>2.56 (1.92)</td>
<td>2.93 (2.04)</td>
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<td>0.77 ( a )</td>
<td>0.466</td>
<td>0.01</td>
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<tr>
<td>T1 RA</td>
<td>6.67 (4.27)</td>
<td>6.11 (4.23)</td>
<td>7.53 (4.69)</td>
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<tr>
<td>T2 RA</td>
<td>7.60 (4.08)</td>
<td>7.27 (4.31)</td>
<td>8.14 (3.49)</td>
<td>0.83</td>
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<tr>
<td>T1 PA ( \dagger )</td>
<td>0.29 (0.34)</td>
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<td>T2 PA ( \dagger )</td>
<td>0.30 (0.31)</td>
<td>0.27 (0.26)</td>
<td>0.28 (0.32)</td>
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<td>0.890</td>
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Note. \( a \) F test, \( b \) Fisher’s Exact test (Fisher exact test was used where at least 1 cell count was less than 5). \( \eta^2 \) is reported as an index for the effect size. \( \dagger \) Log transformed variables. \( *p<.05, **p<.01 \)
Two-way ANCOVAs with ethnicity as a controlled variable showed that after T1 RA/PA was controlled for, the three baseline RSA groups did not differ on either RA or PA at T2 (for RA, \( F = .32, p = .725, \eta^2 = .003 \); for PA, \( F = .479, p = .620, \eta^2 = .005 \)). See Table 5 for means and SDs. The interaction between baseline RSA groups and SA was also non-significant (for RA, \( p = .370 \); for PA, \( p = .348 \)).

Regression yielded similar results. After T1 RA/PA was controlled for, the interaction between T1 and T2 baseline RSA was not related to either RA or PA at T2, and that the moderating effect of SA was non-significant. See Table 8 for statistics.

Table 8. Summary of Hierarchical Regression for T2 RA/PA: Predicting Effects of T1 and T2 Baseline RSA, and SA and Their Interactions

<table>
<thead>
<tr>
<th></th>
<th>T2 RA</th>
<th></th>
<th>T2 PA†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( B )</td>
<td>( SE )</td>
<td>( t )</td>
</tr>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>-0.04</td>
<td>0.06</td>
<td>-0.65</td>
</tr>
<tr>
<td>T1 RA/PA†</td>
<td>0.39</td>
<td>0.07</td>
<td>6.06**</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 Baseline RSA†</td>
<td>-0.03</td>
<td>0.08</td>
<td>-0.38</td>
</tr>
<tr>
<td>T2 Baseline RSA†</td>
<td>-0.02</td>
<td>0.08</td>
<td>-0.29</td>
</tr>
<tr>
<td>SA</td>
<td>0.12</td>
<td>0.07</td>
<td>1.61</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 Baseline RSA† x T2 Baseline RSA†</td>
<td>0.01</td>
<td>0.07</td>
<td>0.18</td>
</tr>
<tr>
<td>T1 Baseline RSA† x SA</td>
<td>-0.02</td>
<td>0.09</td>
<td>-0.22</td>
</tr>
<tr>
<td>T2 Baseline RSA† x SA</td>
<td>-0.07</td>
<td>0.09</td>
<td>-0.79</td>
</tr>
<tr>
<td><strong>Step 4</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 Baseline RSA† x T2 Baseline RSA† x SA</td>
<td>0.00</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. †Log transformed variables. *\( p < .10 \), *\( p < .05 \), **\( p < .01 \).

**RSA Reactivity.** Similarly, three RSA reactivity groups were created (i.e., persistently increased RSA reactivity, \( n = 42 \); mixed group, \( n = 67 \); persistently reduced RSA reactivity, \( n = \) ...
The means, SDs, and ranges of the three groups are listed in Table 9. The three groups differed significantly on their baseline RSA and RSA reactivity at each time point. Group differences were not observed for ethnicity, sex, or social adversity.

Table 9. RSA Reactivity Group Comparisons in RSA, Sociodemographic Data, and Aggression Measures

<table>
<thead>
<tr>
<th>T1 &amp; T2 Dataset</th>
<th>Persistently Increased RSA Reactivity</th>
<th>Mixed RSA Reactivity</th>
<th>Persistently Reduced RSA Reactivity</th>
<th>F/χ²</th>
<th>p</th>
<th>η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 Baseline RSA †</td>
<td>(n = 42)</td>
<td>(n = 67)</td>
<td>(n = 27)</td>
<td>4.53 a</td>
<td>0.013</td>
<td>0.07</td>
</tr>
<tr>
<td>T2 Baseline RSA †</td>
<td>[1.50, 2.47]</td>
<td>[1.45, 2.50]</td>
<td>[1.45, 2.38]</td>
<td>5.05 a</td>
<td>0.008</td>
<td>0.07</td>
</tr>
<tr>
<td>T1 RSA Reactivity †</td>
<td>[-0.08 (0.06)</td>
<td>-0.02 (0.08)</td>
<td>0.09 (0.06)</td>
<td>48.76 a</td>
<td>0.000</td>
<td>0.43</td>
</tr>
<tr>
<td>T2 RSA Reactivity †</td>
<td>[-0.09 (0.07)</td>
<td>0.02 (0.10)</td>
<td>0.10 (0.10)</td>
<td>39.69 a</td>
<td>0.000</td>
<td>0.38</td>
</tr>
<tr>
<td>Ethnicity (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>12 (29%)</td>
<td>16 (24%)</td>
<td>5 (19%)</td>
<td>7.99b</td>
<td>0.381</td>
<td>-</td>
</tr>
<tr>
<td>Black</td>
<td>17 (40%)</td>
<td>34 (51%)</td>
<td>10 (37%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>3 (7%)</td>
<td>3 (4%)</td>
<td>1 (4%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>0 (0%)</td>
<td>1 (1%)</td>
<td>1 (4%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed or other</td>
<td>10 (24%)</td>
<td>13 (19%)</td>
<td>10 (37%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21 (50%)</td>
<td>29 (43%)</td>
<td>19 (70%)</td>
<td>4.86 b</td>
<td>0.088</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Social Adversity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.81 (1.73)</td>
<td>2.61 (1.94)</td>
<td>2.41 (1.80)</td>
<td>0.40 a</td>
<td>0.673</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>T1 RA</td>
<td>6.95 (4.74)</td>
<td>6.29 (4.56)</td>
<td>6.30 (3.37)</td>
<td>0.32</td>
<td>0.730</td>
<td>0.01</td>
</tr>
<tr>
<td>T2 RA</td>
<td>7.52 (3.71)</td>
<td>7.62 (3.93)</td>
<td>7.77 (4.11)</td>
<td>0.03</td>
<td>0.970</td>
<td>0.00</td>
</tr>
<tr>
<td>T1 PA †</td>
<td>0.67 (0.34)</td>
<td>0.30 (0.33)</td>
<td>0.24 (0.27)</td>
<td>0.36</td>
<td>0.700</td>
<td>0.01</td>
</tr>
<tr>
<td>T2 PA †</td>
<td>0.25 (0.29)</td>
<td>0.30 (0.31)</td>
<td>0.31 (0.30)</td>
<td>0.49</td>
<td>0.616</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Note. a F test, b Fisher’s Exact test. η² is reported as an index for the effect size. †Log transformed variables. *p<.05, **p<.01

Two-way ANCOVAs showed that the three RSA reactivity groups did not differ significantly on either T2 RA or PA (for RA, F = .31, p = .737, η² = .005; for PA, F = .76, p = .470, η² = .013). See Table 7 for means and SDs. The interaction between RSA reactivity group and SA was non-significant (for RA, p = .958; for PA, p = .714).
Regression analyses also indicated that after controlling for T1 RA/PA, the interaction effects between T1 and T2 RSA reactivity were not significant in predicting T2 RA/PA, and the moderating effects of SA was non-significant. See Table 10 for statistics.

**Table 10. Summary of Hierarchical Regression for T2 RA/PA: Predicting Effects of T1 and T2 RSA reactivity, and SA and Their Interactions**

<table>
<thead>
<tr>
<th></th>
<th>T2 RA</th>
<th></th>
<th></th>
<th>T2 PA†</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE</td>
<td>t</td>
<td>∆R²</td>
<td>B</td>
</tr>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>-0.01</td>
<td>0.08</td>
<td>-0.11</td>
<td>No gender effect</td>
<td></td>
</tr>
<tr>
<td>T1 RA/PA†</td>
<td>0.39</td>
<td>0.09</td>
<td>4.56**</td>
<td>0.36</td>
<td>0.09</td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 RSA Reactivity†</td>
<td>0.05</td>
<td>0.09</td>
<td>0.54</td>
<td>0.01</td>
<td>0.09</td>
</tr>
<tr>
<td>T2 RSA Reactivity†</td>
<td>0.00</td>
<td>0.09</td>
<td>-0.04</td>
<td>0.07</td>
<td>0.10</td>
</tr>
<tr>
<td>SA</td>
<td>0.18</td>
<td>0.08</td>
<td>2.17*</td>
<td>0.21</td>
<td>0.09</td>
</tr>
<tr>
<td>Step 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 RSA Reactivity† x T2 RSA Reactivity†</td>
<td>-0.05</td>
<td>0.08</td>
<td>-0.62</td>
<td>-0.06</td>
<td>0.09</td>
</tr>
<tr>
<td>T1 RSA Reactivity† x SA</td>
<td>0.08</td>
<td>0.09</td>
<td>0.92</td>
<td>0.13</td>
<td>0.09</td>
</tr>
<tr>
<td>T2 RSA Reactivity† x SA</td>
<td>0.00</td>
<td>0.1</td>
<td>0.01</td>
<td>-0.08</td>
<td>0.10</td>
</tr>
<tr>
<td>Step 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 RSA Reactivity† x T2 RSA Reactivity† x SA</td>
<td>0.11</td>
<td>0.11</td>
<td>0.96</td>
<td>0.05</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Note. †Log transformed variables. *p<.10, *p<.05, **p<.01.

**Summary.** To examine the predictive relations between RSA measures and aggression, both ANCOVAs and hierarchical regressions were conducted. Results showed that having higher or lower baseline RSA on both time points were not related to RA or PA at T2. Similarly, showing increased or reduced RSA reactivity across the two time points were not associated with either type of aggression at T2. Finally, the moderating effect of SA was not significant in all models.
Discussion

This is the first study to examine both the concurrent and longitudinal (repeated-measures) relationships among physiological regulation (e.g., RSA measures), social adversity, and reactive-proactive aggression in children. The major findings are: 1) after controlling for PA, high RA was related to high baseline RSA in conjunction with increased RSA reactivity during emotion regulation, although these findings were only significant in boys at T1; no such effects were found for girls; 2) high PA was associated with reduced RSA reactivity during emotion regulation in both boys and girls at the low levels of social adversity at T2, and 3) longitudinal (repeated-measures) investigations showed non-significant relationship between RSA activity and the two subtypes of aggression.

Inconsistent with our prediction, high RA was found to relate to high but not low baseline RSA. Furthermore, we documented an interaction effect between baseline RSA and RSA reactivity in predicting RA. Specifically, higher RA was related to higher baseline RSA in conjunction with increased RSA reactivity in boys at T1. Both the baseline RSA and RSA reactivity relate to the PNS-mediated emotion regulation process (Hinnant & El-Sheikh, 2009) and have been associated with problems behaviors in youth (Beauchaine et al., 2007; Boyce, 2001; Calkins et al., 2007). Elevated vagal tone may indicate disinhibited temperament (Kagan, 1989) and may reflect a predominance PNS over SNS activity or excessive vagal sensitivity, which may further contribute to lower HR and higher aggressive behaviors (Raine & Venables, 1984; Venables, 1988). This finding is consistent with our prior work with adult population showing that high baseline RSA and increased RSA reactivity in an emotion-provoking decision-making task was related to high RA but not PA, although it was only significant in the condition of higher social adversity.
(Zhang & Gao, 2015). It is also worth noting that in the current and in Zhang and Gao’s study, the interaction effect was only significant after partialling out the effects of the non-focal subtype of aggression. These findings suggest that the combination of high baseline RSA and increased RSA reactivity may be particularly helpful for detecting the unique differences between RA and PA (Miller & Lynam, 2006; Raine et al., 2006). Taken together, findings indicate that ANS hyperarousal, as indexed by high baseline RSA and increased RSA reactivity, may reflect emotional liability (Beauchaine et al., 2007; Beauchaine, 2001; Sloan et al., 1994), which in turn contributes to the development of reactive/impulsive aggression that is characterized by irritable and hostile response to stress or provocation.

Interestingly, when predicting RA we found moderating effect of gender: the interaction effect of baseline RSA and RSA reactivity was only significant in boys. In addition, boys were found to show significantly more RA but not PA than girls. The restricted range of RA scores (particularly after controlling for PA) in girls may partly contribute to the null findings in this gender group. Alternatively, our finding may indicate differential underlying etiology for RA in the two gender groups. Recent research suggests that there are distinct biological mechanisms of externalizing behavior for boys versus girls (Beauchaine et al., 2008; Gordis et al., 2010). For example, Beauchaine and colleagues (2008) found that compared to the normal controls, boys with aggression and conduct problems showed reduced autonomic activity, whereas girls showed elevated electrodermal activity. Our finding of the relationship between RSA activity and RA only in boys provide further support that the neurobiological mechanisms underlying aggression may be different for the two gender groups.
We found that children with higher PA exhibited reduced RSA reactivity during emotion regulation in the conditions of low social adversity at T2. This is consistent with the notion that an under-aroused ANS is associated with PA specifically. Reduced RSA reactivity may reflect deficits in regulation when encountering stressor (Murray-Close & Rellini, 2012), ineffectively coping with the environmental demands, and immobilization response to challenges (Calkins & Dedmon, 2000; Calkins, 1997). Taken together, reduced ANS responses, reflecting impairments in emotion regulation and coping skills, may at least in part contribute to PA that is associated with psychopathic traits and blunted affect (Fanti et al., 2008; Frick et al., 2003).

More importantly, we found that reduced RSA reactivity was associated with higher PA at low but not high levels of social adversity. This adds to the prior literature indicating that the links between neurobiological activity and aggressive and antisocial behavior might be stronger in participants from a benign social background (Gao et al., 2009; Raine & Venables, 1984). As argued by the “social push” theory (Raine, 2002), without the “push” towards aggressive behavior, neurobiological risk factors (i.e., reduced RSA reactivity) may have stronger effect on the emerge of the antisocial behavior.

It is interesting that the moderating effect of social adversity was only found significant for PA in our study. In contrast, a recent investigation with 11- to 17-year-old children and adolescents from Hong Kong showed that the interaction between baseline HR and social adversity was significant for RA but not for PA (Raine et al., 2014). Differences in sample characteristics and the measure of ANS functioning may partly contribute to this discrepancy. Raine et al.’s study was with older population from Asia, whereas ours were younger (eight to 10 years old).
and from the U.S. In addition, Raine et al. examined HR, which is controlled by both parasympathetic and sympathetic branches of the ANS, whereas ours focused on the parasympathetic-influenced cardiac activity in particular. The latter is believed to be a better indicator of physiological regulation. Future studies are needed to further investigate the biosocial effects in relation to the subtypes of aggression. Nonetheless, our results have documented the differential biosocial interaction effect for RA and PA, and again highlight the importance of examining both neurobiological activity and psychosocial factors to better understand the distinct etiologies for the development of antisocial behavior.

Different concurrent relationships between RSA measures, SA, and aggression were found at the two time points. Furthermore, when examining the longitudinal (repeated-measures) relationships among RSA activity, aggression, and social adversity, we found that the baseline RSA was relatively stable from T1 to T2 whereas the measures of RSA reactivity were not significantly correlated. Prior findings have suggested that vagal activity increases significantly during infancy and childhood, then gradually decreases or stays unchanged with development (Korkushko, Shatilo, Plachinda, & Shatilo, 1991; Umetani, Singer, McCraty, & Atkinson, 1998). However, it is still unclear at what age vagal activity reaches maximum. For example, some research suggests that the baseline RSA peaks between four and six years old (Finley & Nugent, 1995; Yeragani, Pohl, Berger, Balon, & Srinivasan, 1994), whereas others suggest between 10 and 14 years (Korkushko et al., 1991; Massin & von Bernuth, 1997). In addition, there is some evidence that baseline RSA showed better stability as compared to the RSA reactivity (Bornstein & Suess, 2000; Hinnant & El-Sheikh, 2009; Pang & Beauchaine, 2013). For instance, in a recent study in eight- and 12-year-old children and adolescents with conduct problems
and/or depression, baseline RSA was found relatively stable (inter-correlation ranges from .35 to .54) across the three time points (each separated by one year), whereas developmental increases were observed in RSA reactivity in response to emotional film clips across time (not significantly correlated) (Pang & Beauchaine, 2013). The null findings of the longitudinal (repeated-measures) relationships among RSA, psychological risk, and reactive-proactive aggression in our sample, may be partly due to the large individual differences in the developmental patterns of RSA reactivity during this age period.

Finally, to the author’s knowledge, the present study is the first to use the paradigm of emotion induction and suppression in response to both negative and positive emotion film in children in attempt to study the relations between physiological regulation and aggression. The same paradigm has been used in children with ADHD. It was found that children with ADHD, especially those with both ADHD and low prosocial behavior, exhibited a stable pattern of reduced RSA activity across all task conditions, suggesting deficits in the physiological functioning of emotion regulation in general (Musser et al., 2011, 2013). Similarly, we found that the RSA reactivity measures were highly correlated when inducing or suppressing positive or negative emotions, suggesting that the physiological regulation deficits observed in children with aggressive behavior was not specific to the valance (positive or negative) or the direction (inducing or suppressing) of emotion regulation.

**Limitations and Future Direction**

**Limitations**

There are several strengths to the current study. Emotion dysregulation has been consistently reported in children with aggressive behavior. In the current study, we have expanded on
these findings by investigating the two distinct subtypes of aggression (i.e., RA and PA) with regard to the PNS-mediated emotion regulation process. Additionally, the current study used data from a longitudinal (repeated-measures) design with a relatively large sample size, and focused on the change during the early years of development. Moreover, the potential moderating roles of psychosocial influences as well as the effect of gender were examined. Despite the strengths of this study, several limitations need to be acknowledged.

First, although the current study was conducted using a longitudinal (repeated-measures) design, due to the funding limit only two-time points were available. This may not be sufficient to fully capture the developmental change over time and it was impossible to investigate the non-linear relationship among variables (Burchina, Nelson, & Poe, 2006; Rogosa, Brandt, & Zimowski, 1982). In order to make stronger causal inferences, longitudinal designs with three or more time points are required. In addition, since the social adversity was only assessed at T1, we could not determine whether the change of psychosocial influences (or lack of) may contribute to the relationship between autonomic abnormality and aggression over time. To address this issue, hierarchical linear modeling is preferred as it measures both within individual (change over time) and between individuals (RSA, social adversity, and RSA x social adversity) variations (Raudenbush & Bryk, 2002).

Second, the aggression measures of RA and PA were limited to the child’s self-report, although our data exhibited good reliability across the one-year period, ranging from .782 to .815. Previous studies have shown that the correlation between the parent-report and the self-report of antisocial behaviors are low to moderate (Loeber, Green, Lahey, & Stouthamer-Loeber,
Further research may benefit from integrating data from multiple informants, including parents, teachers, and children, to assess children aggression (e.g., Raine et al., 2006). Similarly, the current study has measured emotion regulation using the psychophysiological measure (RSA) only, although it has been considered a valid and reliable measure of emotion regulation (Beauchaine, 2015b). Research using multiple methodologies to assess emotion regulation, such as self-reports (e.g., Gratz & Roemer, 2004; Ochsner et al., 2002; Zeman & Garber, 1996), observation methods (e.g., Cole, 1986; Cummings, 1987; Cummings et al., 1989), neuroimaging (e.g., Davidson, 2000), EEG/ERP (e.g., Dennis, 2010; Lewis & Stieben, 2004), and RSA, are needed for the future research to fully understand the emotion regulation deficits in relation to aggressive behavior. Structural equation modeling with latent variables may help mitigate these problems by integrating multiple informants and measures.

Lastly, it is possible that the missing RSA data could have affected our findings, although the Little’s MCAR test (Little & Rubin, 1989) was conducted to ensure that data were missing completely at random. In addition, a series of independent samples t-tests indicated that those included and excluded from analyses did not differ significantly on any of the key variables. Nonetheless, future studies need to pay extra attention to the experimental processes involving psychophysiological data collection, to maximize the amount of usable data and thus increase statistical power.
Future Research

Identifying the neurobiological mechanisms underlying physiological regulation deficits has the potential to advance our understanding of how the brain processes and modulates emotional experience as well as the neural circuitry involved in emotional dysfunction in relation to RA and PA. The neural changes associated with emotional suppression have been of interest given that the inability to suppress negative emotion is linked to psychopathology and aggressive behaviors (Schaefer et al., 2002). Emotional suppression has been shown to be accompanied by increased HR and SC reactivity (Gross & Levenson, 1997; Gross, 1998; Gross, 2002; Ohira et al., 2006; Richards & Gross, 2000), and neuroimaging studies have found that several prefrontal regions including the left lateral prefrontal cortex, the adjacent medial prefrontal cortex, and the medial orbitofrontal cortex, play critical roles in inhibiting activation of limbic regions during emotion suppression among healthy adults (Davidson, 2000; Ohira et al., 2006). In contrast, relatively little is known about the neural substrates of emotion induction. Furthermore, in practice, individuals may wish to up-regulate positive affect and down-regulate negative affect. Research has shown that different neural networks involved are in positive and negative emotion regulation in adults. The ANS reacts differentially to specific emotions (such as happiness, sadness, fear, etc.) during emotion regulation (Driscoll, Tranel, & Anderson, 2009; Ohira et al., 2006; Reynaud, El-Khoury-Malhame, Blin, & Khalfa, 2012). For example, when comparing an emotion suppression task to an emotion attending task, suppressing emotion leads to a significantly lower HR when viewing a happy film, and a higher SC response when viewing a fear-inducing film (Reynaud et al., 2012). Additionally, neuroimaging studies have shown that regulation of positive and negative emotions is associated with both shared (e.g., the left superior and lateral
frontal regions) and distinct neural regions (positive emotion: the prefrontal regions and the left insula; negative emotion: the left orbitofrontal gyrus, the left superior frontal gyrus, and the anterior cingulate gyrus) (Mak, Hu, Zhang, Xiao, & Lee, 2009). Future studies are needed to disentangle the neural subtrahends of regulation of specific emotions in children and adolescents.

Future research should also investigate at what age the effect of physiological dysregulation on aggression can be observed. The transition from infancy to toddlerhood has been argued to be a critical period for the development of emotion regulation (Beauchaine, 2001). In the infant literature, higher baseline RSA is considered as a psychophysiological marker of negative emotionality and difficulty (Fox & Field, 1989; Porges et al., 1994; Stifter & Fox, 1990). However, higher baseline RSA indicates positive emotionality and social competence in later childhood (Beauchaine, 2001; Porges et al., 1994). In terms of RSA reactivity, moderate levels of RSA reactivity have been consistently associated with more adaptive responding and better engagement during challenging tasks in from infancy to adulthood (see Beauchaine, 2001 for a review). For example, even in 3-month-old infants, those showing RSA withdrawal were rated higher on soothability and had increased duration of orienting than those who showed RSA augmentation (Huffman et al., 1998). Previous studies have suggested that researchers must take caution in interpretation of the relationship between RSA and aggression in the developmental context. More research is needed to further understand the complex relation between physiological regulation and aggressive behavior in early childhood.

Future research should also benefit from examining the possible moderating effects of puberty stages on the relationship between psychophysiological regulation, psychosocial factors
and reactive-proactive aggression. A failure to analyze this may partially explain the missing predictive effects of RSA activity in relation to RA or PA from the longitudinal (repeated-measures) perspective in the current study. The development of the secondary sexual characteristics is important, and can signal the onset of the physiological and psychological changes of profound important perspectives to the individual, family and society (Herman-Giddens et al., 1997). It has been found that in the U.S. children are developing pubertal characteristics at younger ages as compared with the earlier generations (Herman-Giddens, Wang, & Koch, 2001; Sun et al., 2002). In addition, significant variations of pubertal development between genders and among different ethnicity groups have been documented. The average age of entering puberty in girls is approximately around 11, while in boys is around 12 (Sun et al., 2002). The timing of onset of sexual maturation is earlier for both black girls and boys than for children from other racial groups (Herman-Giddens et al., 1997, 2001; Sun et al., 2002). For example, non-Hispanic black girls showed earlier sexual development for pubic hair and breast development than Hispanic and non-Hispanic white girls (while no significant difference was observed between Hispanic and Non-Hispanic white girls) (Sun et al., 2002). Herman-Giddens et al. (2001) found that the average ages for onset of hair growth was 11.2 and 12.0 for black boys and White boys, respectively.

The timing of puberty has been associated with different adjustment problems, such as depression and externalizing behavior (Negriff & Susman, 2011). Additionally, early maturing girls and late maturing boys have been posited to display the highest risks of adjustment problems (Petersen & Taylor, 1980). Elevated testosterone levels have been found in the early maturing boys, and higher testosterone levels are linked to increased physical aggression, delinquency
and negative emotion (Archer, 2006). However, testosterone may lead to aggression via the aromatization of testosterone into estrogen in girls, and that higher estrogens is related to more aggressive behavior (Finkelstein, 1997). Finally, prior research has shown that pubertal hormones may influence brain development, both structurally and functionally (Blakemore, Burnett, & Dahl, 2010). For example, previous literature has shown that puberty stages are positively associated with the growth of the amygdala volume in boys, though these finding are less consistent in girls (Bramen et al., 2011; Neufang et al., 2009). Since puberty may exert a complex and profound impact on changes of brain and behaviors, in the future studies to investigate aggressive behavior in children and adolescents, consideration should be given to the pubertal timing. According to the prior literature, puberty stages could be measured using a golden standard called the Tanner stages in which puberty development has been classified in five different stages (Tanner, 1962). Although it is unknown how puberty may affect the development of RA or PA, or RSA changes, future studies should include this measure to assess its effect.

**Conclusion and Implications**

In conclusion, the present study provides further evidence that there are different etiologies for RA and PA. In particular, our findings indicate that RA is related to ANS over-arousal that contributes to emotion liability and difficulties in regulating behavior and cognition, whereas PA is associated with ANS underarousal that contributes to lack of emotional awareness and difficulties engaging. Although further replication is needed, our findings highlight the importance of investigating both the baseline and task measures of RSA, as well as the moderating effects of social adversity and gender.
Further understanding of the distinct neurobiological and psychosocial profiles for RA and PA may provide important practical implication for the design of more effective intervention and treatment techniques to the specific needs for the at-risk individuals (Kempes, Matthys, De Vries, & Van Engeland, 2005). As described by Kempes et al. (2005), reactively aggressive behavior might be influenced by the intervention that focuses on anger management and is designed to remedy higher level of arousal and anger reaction towards stressful life events. In contrast, in order to reduce PA, intervention shall take the form of program that emphasizes on teaching children to become more attentive to the positive and the negative outcome of their own behaviors. In addition, future studies may consider using RSA as a non-invasive and reliable biological measure to assess the efficacy of treatment programs that focus on improving emotion regulation in children and adolescents.
APPENDIX A: RPQ

Self-report Reactive-Proactive Aggression Questionnaire (RPQ) (Raine et al., 2006)

Instructions: There are times when most of us feel angry, or have done things we should not have done. Rate each of the items below by putting a circle around 0 (never), 1 (sometimes), or 2 (often). Do not spend a lot of time thinking about the items—just give your first response. Make sure you answer all the items (see below).

How often have you…

1. Yelled at others when they have annoyed you
2. Had fights with others to show who was on top
3. Reacted angrily when provoked by others
4. Taken things from other students
5. Gotten angry when frustrated
6. Vandalized something for fun
7. Had temper tantrums
8. Damaged things because you felt mad
9. Had a gang fight to be cool
10. Hurt others to win a game
11. Become angry or mad when you don’t get your way
12. Used physical force to get others to do what you want
13. Gotten angry or mad when you lost a game
14. Gotten angry when others threatened you
15. Used force to obtain money or things from others
16. Felt better after hitting or yelling at someone
17. Threatened and bullied someone
18. Made obscene phone calls for fun
19. Hit others to defend yourself
20. Gotten others to gang up on someone else
21. Carried a weapon to use in a fight
22. Gotten angry or mad or hit others when teased
23. Yelled at others so they would do things for you

Scoring: RA items (1, 3, 5, 7, 8, 11, 13, 14, 16, 19, 22) and PA items (2, 4, 6, 9, 10, 12, 15, 17, 18, 20, 21, 23). Items are summated to form reactive and proactive subscale scores.
**APPENDIX B: SOCIAL ADVERSITY INDEX**

*These questions are part of the demographic questionnaire that the caregiver filled out during the initial lab visit between 2012 and 2014.

References: (Choy et al., 2015; Gao et al., 2010; Raine et al., 2002)

1. Current marital status of biological parents (select one from next line)__________:
   (Married =1; Widowed = 2; Separated = 3; Divorced = 4; Never Married = 5)

2. From ages 6 months to two and a half years of age, how many times was your child separated from the mother (for any reason: illness, hospitalization, institutionalization, orphaned) __________?
   If so, for how long (in weeks) did this last? __________
   Was your child separated from both parents?  YES   NO

3. Do you live in Government home/apartment?  YES  NO

4. Were you ever on welfare or food stamps from the government?  YES  NO

5. Has the father or mother ever been arrested?  YES  NO
   How many times? __________

6. Has the father or mother had a physical illness (e.g., heart, lung problems, etc.) that impaired their functioning at some time during the child’s lifetime? YES  NO

7. Has the father or mother had a significant mental illness (e.g., alcoholism, major depression, schizophrenia, anxiety) that impaired their functioning at some time during the child’s lifetime? YES  NO

8. Number of people living with family (including immediate and extended family): __________
   Number of rooms (include bedroom, living room, dining room, kitchen): __________
9. Mother’s age at birth of child: ___________

10. Birth order of the child: ___________

   Number of brothers: ___________ full   ___________ half

   Number of older brothers: ___________ full   ___________ half

   Number of sisters: ___________ full   ___________ half

   Number of older sisters: ___________ full   ___________ half
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