

Heart Rate as a Moderator between Child Abuse Potential
and Reactive and Proactive Aggression

Akiho Tanaka

This Thesis submitted to the faculty of the Virginia Polytechnic Institute and State University in
partial fulfillment of the requirements for the degree of

Master of Science
In
Psychology

APPROVED:

Angela Scarpa, Ph. D., Committee Chair
Bruce H. Friedman, Ph.D., Committee Member
Lee D. Cooper, Ph.D., Committee Member

April 28, 2006
Blacksburg, Virginia

Keywords: Aggression, Child Abuse, Heart Rate

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Abstract

Previous research regarding the biosocial approach to aggression suggests that the interaction between biological and environmental variables contribute to aggression. However, this particular relationship has not yet been fully explored in children. Therefore, this study examined the moderating influence of psychophysiological activity, particularly resting heart rate (HR) and HR variability (HRV), on the relationship between child abuse potential (CAP) and child reactive and proactive aggression.

Thirty-six children, between the ages of 7 and 13, and their parents were recruited from the local schools and community in Southwestern Virginia. Parents completed self-report measures for child abuse potential (CAP) and the type of aggression displayed by their children (i.e., reactive or proactive). Children were assessed for resting HR and HRV for four minutes during a rest period.

CAP was related to increased proactive (i.e., instrumental) and reactive (i.e., hostile) aggression in children with low levels of resting HR. CAP was also related to increased proactive aggression in children with high resting HRV. Significant main effects were not found for CAP or psychophysiological functioning, indicating the importance of examining the interaction of these variables.

Taken together, the findings suggest the existence of an interaction of these two biological and social variables above and beyond their individual influences as risk factors. The role of cardiovascular underarousal in relation to proactive and reactive aggression in abused

children is discussed. Overall, this study supports the notion of a biosocial interaction for aggression in children, and thus has implications for future research and treatment.

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Heart Rate as a Moderator between Child Abuse Potential and Reactive and Proactive Aggression

Introduction

Aggression is a major problem for both children and adolescents. According to the U.S. Department of Health and Human Services, between 30 to 40 percent of male teenagers and 16 to 32 percent of female teenagers reported having committed a serious violent offense (e.g., aggravated assault, gang fights, robbery, rape) by the age of 17. In as early as toddlerhood, children often bite, hit, push, and shove their peers when angry or frustrated. During their preschool years, children learn to express their aggression verbally (e.g., temper tantrums, yelling at other children) (Coie & Dodge, 1997). Aggressive behavior is associated with many different factors. These factors include psychophysiological variables, specifically heart rate (HR; Farrington, 1997; Raine, 1993, 2002b; Raine, Reynolds, Venables, & Mednick, 1997; Raine et al., 1998; Scarpa & Raine, 1997, in press; Wadsworth, 1976) and environmental variables (e.g., Bowser, 2002; Scarpa & Ollendick, 2003; Schwab-Stone et al., 1995), particularly experiencing childhood abuse. For a comprehensive understanding of the problem, these variables should be examined together to better inform prevention and intervention techniques.

As stated above, previous research has found that biological factors contribute to the development of childhood aggressive and antisocial behavior (Raine, 2002b; Scarpa & Raine, in press). Particularly, a correlation was found between low autonomic activity (in the form of low resting HR and skin conductance, SC) and antisocial, violent, and criminal behavior in both adults and children. Such findings have been explained through stimulation seeking theory. This theory posits that antisocial individuals seek to elevate their autonomic activity level to optimal

or normal levels through stimulation because they are uncomfortable with their low level of autonomic activity (Eysenck, 1997; Quay, 1965; Raine, 1993, 1997; Raine, et al., 1998). The findings could also be explained by fearlessness theory which suggests that low autonomic activity is a marker for low fear levels during mildly stressful situations (Raine, 2002a, 2002b). According to this theory, individuals with reduced levels of fear are less likely to inhibit their behaviors in risky situations or to learn from punishment cues.

The importance of researching biological and social factors in conjunction when studying antisocial and aggressive behavior has been emphasized in order to obtain a thorough understanding of the problem (Raine, 2002b). Specifically, one form of interaction suggests that presence of both social (e.g., family and socioeconomic) and biological (e.g., heritable and psychophysiological) variables increases the risk of antisocial and violent behavior. For example, according to an adoption study by Cloninger and Gottesman (1987), crime rates in adoptees were found to be highest when they had both heritable and environmental factors. In another study by Farrington (1997), boys with a low resting HR who came from a large family with a poor parent-child relationship were at greater risk to become violent adult offenders in comparison to boys with a low resting HR who did not come from such a family. Additionally, Scarpa et al. (1999) found that increased heart rate variability (HRV) was associated with aggression in young adults that had not been victims of violence, but the same results were not found in those who were victims of violence.

Low resting HR has been suggested to be the best-replicated biological correlate of antisocial behavior. The average effect size for HR was -0.44 at rest and -0.76 during a stressor in a recent meta-analysis of 40 studies on antisocial behavior in children and adolescents (Ortiz & Raine, 2004). Additionally, in a meta-analysis of 19 studies, the relationship between low

resting HR and child and adult aggression had an average effect size of -0.38 ($d = -0.51$ in child samples and -0.30 in adults; Lorber, 2004). Researchers on heritability of HR in children have presented a model including common environmental effects (20% of the total variance), unique environmental effects (48%), and additive genetic effects (32%) (Boomsma & Plomin, 1986). Schieken et al. (1989) found a heritability estimate of .61 for resting HR with additive genetic and unique environmental effects only. Similarly, Dubreuil and colleagues (2003) described sleeping HR variance with a model consisting of genetic effects (61%) and unique environmental effects (39%). Additionally, Healy (1992) suggested the existence of a strong genetic influence based on the difference in heritability estimates between monozygotic (MZ, $r = .72$) and dizygotic twins (DZ, $r = .06$) in infants. The implication is that an accumulation of biological and social risks increases the likelihood of an aggressive outcome.

Another known risk indicator for aggression is heart rate variability (HRV), or beat to beat differences over time in the length of the cardiac cycle. Unlike HR, which is influenced by both the sympathetic and parasympathetic branches of the autonomic nervous system, HRV is mediated by the vagus, the tenth cranial nerve, which provides the deceleratory parasympathetic component of cardiac activity (Beauchaine, 2001). Specifically, as the vagal efference increases during exhalation, HR decelerates, and vagal efference decreases during inhalation, accelerating HR, thus resulting in respiratory sinus arrhythmia (RSA). Vagal tone is estimated from RSA, and is the degree of ebbing and flowing of HR during the respiratory cycle (Bernston et al., 1997; Hayano et al., 1991; Katona & Jih, 1975). As opposed to heritability of HR, HRV seems to be affected more by environmental rather than genetic factors. Specifically, in young children between the ages of one and three, similar within-pair correlations were observed in MZ ($r = .70$) and DZ ($r = .64$), emphasizing the influence of common environmental influences (Healy, 1992).

There are inconsistencies in the literature of the relationship between HRV and psychopathology. In particular, both externalizing and internalizing disorders have been associated with reduced vagal tone (Beauchaine, 2001). Likewise, discrepant evidence exists for the directionality of HRV in relation to aggressive behavior. Despite findings of an association between high HRV and psychological well-being such as social competence (Eisenberg et al., 1995) and high performance on executive functioning tasks (Hansen, Johnsen, & Thayer, 2003), a high vagal tone, or vagotonia, has been hypothesized to be a risk factor for antisocial behavior (Venables, 1988). Specifically, low resting HR of antisocial individuals has been suggested to be a function of high vagal tone, indicating a passive coping response in mildly stressful situations (Raine, 2002; Raine & Venables, 1984). Consistent with this notion, low resting HR and high resting HRV were associated with aggression in young adults (Scarpa, Fikretoglu, & Luscher, 2000).

In contrast, other researchers have found an association between hostility and aggressive behavior with an attenuated vagal tone (Beauchaine & Kopp, in press; Beauchaine, 2001; Mezzacappa et al., 1996, 1997). Similarly, Pine and colleagues (1998) found a relationship between increased levels of externalizing psychopathology and reduced HRV, independent of the relationship between reduced HRV and familial hypertension. This study also found an association of externalizing behavior with reduced HR. This may be explained by the independent functions of the sympathetic and parasympathetic branches (see Berntson, Cacioppo, Quigley, & Fabro, 1994), as concurrent parasympathetic and sympathetic dysregulation could result in reduced HR as well as reduced RSA. More broadly, reduced RSA levels are characteristic of dysregulated emotional states, such as anger. However, excessive vagal reactivity is characteristic of emotional lability, such as impulsive aggression. Moreover,

Beauchaine and colleagues suggest a biosocial developmental model, in which emotion regulation skills are socialized within families (e.g., Calkins, 1997; Stifter & Fox, 1990; Shipman & Zeman, 2001). Specifically, the emotional lability characteristic of aggression seems to be influenced by coercive parent-child exchanges beginning in the first 5 years from birth (Campbell, Pierce, Moore, Marakovitz, & Newby, 1996; Cole & Zahn-Waxler, 1992; Patterson, Capaldi, & Bank, 1991). Conflict can be escalated in such interactions by matching and exceeding each other's level of autonomic activity (Snyder et al., 1994; Snyder, Schrepferman, & St. Peter, 1997), thus negatively reinforcing heightened autonomic activity and emotional lability.

As such, these studies indicate the importance of studying autonomic variables in conjunction with adverse familial experiences. Nonetheless, the direction of such interactions remains unclear. The aim of this study is to assess biosocial interactions in relation to aggression in a community sample of children. Specifically, it examines the influence of HR level and HRV on the relationship between childhood physical abuse and proactive and reactive aggression in children.

Child Maltreatment and Aggression

In previous studies, physical abuse in children has been associated with increased risk of developing behavioral problems in childhood and violent, antisocial behavior in adolescence and adulthood (Cicchetti & Manly, 2001; Gibbs, Wheeler, Alloy, & Abramson, 2001; Jaffee, Caspi, Moffit, & Taylor, 2004; Lansford et al., 2002; Widom, 1989a, 1989b), suggesting a cycle of violence perpetuated across generations (De Bellis, 2001; Dodge, Pettit, Bates, & Valente, 1995; McGee, Wolfe, & Olson, 2001; Straus & Yodanis, 1996; Weiss, Dodge, Bates, & Pettit, 1992). Particularly, when compared to non-abused children, abused children displayed lower levels of

adaptive functioning and higher levels of behavior problems (Feldman et al., 1995). Similarly, it was concluded that physically abused children exhibited more externalizing behaviors, such as aggression and delinquency, in comparison to their non-abused counterparts (Kolko, 1992). Moreover, maltreated children were found to display aggressive behavior at an early age (Widom, 1989b). Lastly, the reported levels of adolescent physical maltreatment were found to be directly related to Antisocial Personality Disorder (ASPD) in adulthood (Gibbs et al., 2001).

Research indicates that although there is a relationship between child abuse and later antisocial behavior, the development of antisocial behavior does not depend on maltreatment alone. For example, Horwitz and colleagues (2001) found that childhood abuse does not directly result in ASPD since the outcome is affected by broader social variables such as lifetime stressors and low SES.

In response to DiLalla and Gottesman's (1991) argument that genetic transmission plays a role in the association between child physical maltreatment and antisocial behavior, Jaffee and colleagues conducted a twin study to examine whether child physical abuse leads to the development of antisocial behavior through genetic transmission or environmental causal process (Jaffee, Caspi, Moffitt, & Taylor, 2004). Their findings indicated that although child abuse and victim antisocial behavior was partially genetically mediated, physical maltreatment plays a causal role in the development of antisocial behavior in children. Specifically, physical maltreatment victimization was not found to be heritable. Instead, it was found that parents with a past history of antisocial behavior were more likely to abuse their children and also had children who engaged in high levels of antisocial behavior. Caspi and colleagues (2002) found evidence for a functional polymorphism in the MAOA gene moderating the relationship between early childhood maltreatment and male antisocial behavior. In terms of the heritability of

aggressive behavior, Edelbrock and colleagues (1995) found a genetic variance of 50% and shared environmental variance of 15% as measured by the Child Behavior Checklist.

Such findings imply that physical maltreatment has serious implications for children, specifically in the development of aggressive and antisocial behavior. They also imply that social and biological factors may influence the relationship between child abuse and antisocial/aggressive behavior. It is important to note, however, that the biological and social risk factors may differ depending on the form of aggression displayed. Two types of aggression that have been gaining increasing recognition in children are *reactive* and *proactive* aggression (Crick & Dodge, 1996; Dodge, 1991), but these distinctions have been relatively ignored in biosocial research.

Childhood Reactive and Proactive Aggression

Proactive aggression is defined as aggression that is used in order to obtain a specific goal. On the other hand, reactive aggression seems to be associated with emotional responses, including anger, fear, and frustration, to a real or perceived threat or provocation. (Dodge, 1993). According to research, there are differences in terms of qualities and risk factors for reactive and proactive aggression (Raine et al., 2006; Scarpa et al., 2002, under review). Specifically, adolescents who engaged in reactive aggression were found to be more impulsive and anxious, as well as displaying information-processing abnormalities and reality distortions (Raine et al., 2006). Reactive aggression also was found to be predictive of later dating violence, with maternal warmth and caregiving behavior acting as a moderator (Brendgen, Vitaro, Tremblay, & Lavoie, 2001). Alternatively, adolescents who engaged in proactive aggression appeared to be more seriously violent, psychopathy-prone, and come from a poor social background (Raine et al., 2006). Also, proactive aggression in childhood was found to be predictive of later

delinquency-related violence, and this relationship was moderated by parental supervision (Brendgen et al., 2001). Similarities found between the two forms of aggression included excessive fighting, paranoid ideation, exposure to traumatic stress, and stimulation-seeking (Raine et al., 2006). Further, in a community sample of children, reactive aggression was found to be associated with mood and anxiety disorders, whereas proactive aggression was associated with disruptive behavior problems (Scarpa et al., 2002, under review). According to Hill (2003) those displaying reactive aggression were more likely to have been physically abused, but Conner and colleagues (2004) found that physical abuse was correlated with both proactive and reactive aggression.

Research suggests that reactive and proactive aggression also may differ in terms of psychophysiological reactions. According to Scarpa and Raine (2000), it is hypothesized that reactive aggression is associated with heightened autonomic activity where proactive aggression is associated with attenuated autonomic activity. The low autonomic activity characteristic of those engaging in proactive aggression is supportive of the previously discussed stimulation-seeking theory (Eysenck, 1964; Quay, 1965) and fearlessness theory (Raine, 2002a, 2002b). Physiological responses for reactive aggression are expected to be elevated due to the salience of a perceived or real threat, in turn demonstrating an automatic stress response and impulsive emotional responses to counteract anger or heightened emotionality (Lochman, Whidby, & FitzGerald, 2000; Zillman, 1983).

According to Hubbard and colleagues (2002), when children were placed in a frustrating situation, SC and HR reactivity were found to be positively associated to reactive, but not proactive aggression. In a different study examining aggression and psychophysiological variables, however, decreased baseline SC and HRV were related to reactive aggression while

increased baseline SC and HRV were related to proactive aggression (Scarpa et al., under review). These findings are inconsistent with the hypotheses of general under-arousal in relation to proactive aggression and over-arousal in relation to reactive aggression (Scarpa & Raine, 2000). The discrepancies between findings of Scarpa et al. (under review) with those of Hubbard et al. (2002) may be explained by the different situations in which the measurements of physiological reactions were taken, as Scarpa and colleagues (under review) measured baseline activity within a rest period whereas Hubbard and colleagues (2002) measured autonomic reactivity while subjects completed a frustrating task. Also, the differences in self-report measures for proactive and reactive aggression may explain the discrepancies, as Scarpa and colleagues (under review) administered parental ratings while Hubbard and colleagues (2002) used teacher ratings of child behavior. However, neither of these studies took the psychosocial risk factor of child physical maltreatment into consideration. It is possible that the relationships between autonomic activity with reactive and proactive aggression may differ depending upon the child's experiences with maltreatment.

Aims of Study

Previous studies have focused on the effect of child physical abuse on reactive and proactive aggression in psychiatrically referred youth (Connor et al., 2004), thus not examining non-psychiatrically referred children from the community in rural environments. Also, although social and biological variables on aggression has been examined separately (Hubbard et al., 2002; Raine & Venables, 1984; Scarpa et al., 2002; Scarpa & Raine, in press; Wadsworth, 1976), studies have not focused on the biosocial interaction of HR, HRV, and child physical maltreatment in relation to aggressive behavior. Specifically, although reactive and proactive aggression has been differentiated in terms of the antisocial outcome of child physical

maltreatment (Connor et al., 2004), abuse has not been examined in conjunction with psychophysiological reactions to predict different types of aggression.

In contrast to previous studies, this study focused on a population of children between the ages of 7 – 13. The subjects consisted of a community sample of children from a rural area in Southwest Virginia, including both girls and boys. Child maltreatment focused specifically on physical abuse, as assessed by the potential for abuse within the family (i.e., child abuse potential or CAP). Lastly, both proactive and reactive forms of aggression were measured. This study's primary objective was to determine a biosocial interaction effect for aggression by examining the role of HR and HRV in moderating the relationships between child physical abuse potential with both reactive and proactive aggression.

Hypotheses

It is hypothesized that the psychophysiological measures of HR and HRV will moderate the relationship between CAP and aggression, such that (1) CAP is associated with increased proactive aggression only in conditions of low HR, (2) CAP is associated with increased reactive aggression only in conditions of high HR (3) CAP is associated with increased proactive aggression only in conditions of high HRV, (4) CAP is associated with increased reactive aggression only in conditions of low HRV. If these results are found, it would imply that those with a high potential for experiencing physical abuse and have low autonomic activity are more likely to engage in instrumental/proactive aggression, whereas those who have a high autonomic activity are more likely to engage in hostile/reactive aggression. These expected results would underscore the notion that child maltreatment experiences do not directly lead to an aggressive outcome, but may be affected by the biological vulnerability of the child. Further, those biosocial interactions may differentially influence the type of aggression that is displayed.

Method

Participants

Children were recruited from local schools and the community in Southwest Virginia through convenience sampling. The participants were included in the final sample if they had complete data for resting HR, HRV, and all aggression and CAP subscales, thus forming 36 children between the ages of 7 and 13 years old ($M = 10.11$ years, $SD = 1.801$) and their parents. The children consisted of 25 boys and 11 girls (32 White and 4 Black). According to Cohen (1992), approximately 34 participants are necessary for a large effect using an alpha level of .05 with three independent variables. Therefore, 36 participants should be appropriate to detect a significant effect.

Assessment Measures (See Table 1 for Descriptive Information)

Revised Parent Rating Scale for Reactive and Proactive Aggression measure (R-PRPA, Brown, Atkins, Osborne, & Milnamow, 1996; Appendix A). Adapted from the original Teacher Report Form, this 28-item scale asks parents to rate the level of aggression and other antisocial behaviors in their children. Six of these items index reactive aggressive behaviors, and 10 items index proactive aggressive behaviors. Parents indicate the frequency of occurrence on a scale of 1 to 3 (e.g., never, sometimes, very often) of specific behaviors displayed by their children (e.g., “deliberately plays mean tricks on other students,” “says mean things about other children behind their back”). Separate scores are calculated for reactive (range = 6 to 18) and proactive (range = 10 to 30) aggressive behavior by summing their respective items.

The R-PRPA has demonstrated moderate to good validity and reliability. Through factor analysis, 6 items were found indexing reactive aggression and 10 items indexing proactive aggression, loading on separate factors. Although there is evidence of correlation between the

factors (i.e., $r = 0.67$; Brown et al., 1996), there is evidence of discriminant validity for each factor as well (Poulin & Boivin, 2000). Internal consistency, as measured by Cronbach's alpha coefficient alpha was .738 for the items in the proactive scale, and .797 for items in the reactive scale in the current study. The score for reactive aggression ranged from 6 to 17 ($M=10.61$, $SD=2.71$), while the score for proactive aggression ranged from 10 to 21 ($M=12.19$, $SD=2.32$).

Child Abuse Potential Inventory, Form VI (CAPI, Milner, 1986). This 160-item self-report questionnaire assesses the potential for physical child abuse by parents. Written at a third-grade reading level, items are presented in an agree/disagree, forced-choice format. This inventory contains a 77-item Physical Abuse scale to be used in the current study, including six descriptive factors (i.e., distress, rigidity, unhappiness, problems with child and self, problems with family, and problems with others) adding up to a total score. In terms of reliability, the split-half reliabilities range from 0.93 to 0.98 and KR-20 coefficients range from 0.85 to 0.96 for different age, gender, ethnic groups, and educational levels. Strong support for the predictive and concurrent validity of this measure was found (Milner, 1986, 1990). Specifically, there was a significant correlation (0.34) found between abuse scores and subsequent confirmed reports of neglect and abuse. In terms of construct validity, the amount of childhood physical abuse is positively correlated with the CAP abuse score (0.48). However, given the diverse nature of the items, the standardized Cronbach alpha was low at .55 for the current sample. Mean levels for the CAPI were 91.80 ($SD = 102.30$) for boys and 88.82 ($SD = 64.95$) for girls. For the total sample, the mean was 90.89 ($SD = 102.30$).

Psychophysiological Measures

Heart rate (bpm). As measured by the VU-AMS ambulatory monitoring system (Vrije University, Amsterdam), children's resting HR is assessed in beats per minute (bpm). A

three-lead electrocardiogram, attached to the ribs and sternum, is used to continuously measure r-wave intervals during a 4-minute rest period. HR, as assessed here, has both sympathetic and parasympathetic nervous system influences. Specifically, the sympathetic system speeds HR, and “brakes” are applied to HR by the parasympathetic system mediated by the vagus nerve. The average resting HR for this sample was 84.83bpm ($SD=9.82$) and ranged from 65.44 to 105.80bpm.

Heart rate variability. AMS software using the Root Mean of the Squared Successive Differences (MSSD) was used to derive HRV, according to the following formula: $MSSD = \sqrt{1/n * \sum (IBI_i - IBI_{i-1})^2}$ (Groot et al., 1998). The MSSD consists of a sample of the short-term variance of the IBIs used to index HRV. One index of parasympathetic tone is vagal control of the heart, expressed through HRV. Specifically, increased HRV, as indicated by larger mean successive differences, has been shown to be strongly associated with cardiac vagal tone as measured by pharmacological blockade (Hayano et al., 1991), and high frequency power bands in spectral analyses (Bigger et al., 1992; Friedman, Allen, Christie, & Santucci, 2002). This provides evidence for its use as an index of vagal activity. As with HR, HRV was calculated and averaged over 30 second intervals during four minutes of the rest period. The average resting HRV was 64.67 ($SD=41.64$) and ranged from 20.38 to 202.75.

Procedures

After parents gave consent for their child’s participation, assent from the children was obtained. Parents were administered the R-PRPA and the CAPI. Electrodes were attached to the children, and their HR and HRV was measured during a 4-minute rest period. A research assistant administered the measures and was available to answer any questions concerning the measures. This was part of a larger study including other measures completed by children and

parents not discussed in this paper. The total session took about 90 minutes. Upon completion of the session, participants were thanked and compensated with a modest stipend for their time.

Results

Demographic Effects

Effects for subjects' age, sex, and body mass index (BMI = height x weight) with CAP, HR, HRV, as well as reactive and proactive aggression were examined (see Table 1 for sex analyses and Table 2 for age analyses). Age and BMI were significantly correlated with each other ($r=0.66$, $p<0.001$). None of these demographic or somatic variables were shown to have systematic relationships with CAP and HR or HRV.

Child Abuse Potential, Resting Physiology, and Aggressive Behavior

Zero-order correlations between CAP (total and subscale scores), HR, HRV, and proactive/reactive aggression scores were computed (See Table 2). Reactive and proactive aggression were significantly positively correlated, $r=.57$, $p<.001$. CAP was significantly positively correlated with proactive aggression, $r=.37$, $p=.027$, and there was a trend for a positive association between CAP and reactive aggression, $r=.33$, $p=.05$. HR and HRV were significantly negatively correlated, $r=-.65$, $p<.001$. No other significant correlations among CAP, HR, HRV, and aggression were found.

After screening for normality, linearity, homogeneity, and multicollinearity, sets of hierarchical regressions were run in order to test for main effects of CAP and child's resting HR and HRV, and their interactions, on reactive/proactive aggression scores. Resting HRV was found to be non-normal, and thus was transformed using log transformations to meet regression assumptions. Separate regressions were conducted for proactive and reactive aggression, and with either HR or HRV in the equation. Both of the models examined the main effect for CAP in

the first block, the main effect for baseline HR or HRV in the second block, and the interaction effect between CAP and the physiological variable (i.e., CAP X HR or CAP x HRV) in the third block.

Results for proactive aggression and HR can be found in Table 3, reactive aggression and HR in Table 4, proactive aggression and HRV in Table 5, and reactive aggression and HRV in Table 6. The model predicting proactive aggression was significant for CAP and HR $F(3,35)=5.367, p=.004$, and HRV, $F(3,35)=3.949, p=0.017$. For the model predicting reactive aggression, CAP and HR, $F(3,35)=4.381, p=0.011$, and HRV, $F(3,35)=3.001, p=0.045$ were significant. Interaction terms were significant in block three for the interaction of CAP with HR predicting proactive aggression, unstandardized $\beta=-.001$, partial $t(35)=-3.06, p=.004$, partial $r = .579$, as well as reactive aggression, unstandardized $\beta=-.001$, partial $t(35)=-2.657, p=.012$, partial $r = .540$. The interaction term for CAP and HRV was also significant for the model predicting proactive aggression, unstandardized $\beta=-.035$, partial $t(35)=2.197, p=.035$, partial $r = .520$. However, when the models controlled for the effect of the other form of aggression, the results were no longer significant.

In order to confirm the significant interactions, post hoc probing as recommended by Aiken and West (1991) and Holmbeck (2002) was conducted. First, regressions were run such that CAP was entered in block one, HR or HRV in block two, and their respective interactions (i.e., CAP x HR or CAP x HRV) in block three. A trend remained for the interaction between CAP and HR in predicting reactive aggression, unstandardized $\beta= -.001$, partial $t(35)=-2.657, p=.012$. For proactive aggression, the interaction between CAP and HR, unstandardized $\beta=-.001$, partial $t(35)=-3.06, p=.004$, and HRV, unstandardized $\beta=.035$, partial $t(35)=2.197, p=.035$, remained significant.

Therefore, in order to determine how resting HR influences the relationship between CAP and proactive/reactive aggression, as well as how HRV influences the CAP – proactive aggression relationship, separate simultaneous regressions were conducted testing the effect for CAP, a conditional resting HR or HRV moderator (i.e., high or low HR, high or low HRV), and their respective interaction terms on either proactive or reactive aggression. As recommended by Aiken and West (1991), the low moderator was computed by adding one standard deviation and the high moderator was computed by subtracting one standard deviation to the original values. Regression lines were then plotted for high and low resting HRV using the centered values for CAP predicting reactive aggression (see Figure 1 and Figure 2 respectively). As illustrated, CAP was differentially related to aggression dependent upon levels of resting HR and HRV. In particular, CAP was positively associated with proactive ($b=.016, p=.001$) and reactive aggression ($b=.018, p=.002$) when resting HR was low, but CAP was negatively associated with proactive ($b=-.006, p=.335$) and reactive aggression ($b=-.005, p=.494$) when resting HR was high. Regression lines were also plotted for high and low resting HRV using the centered values for CAP predicting proactive aggression (see Figure 3). As illustrated, CAP was differentially related to aggression dependent upon levels of resting HRV. Specifically, CAP and proactive aggression were positively associated when resting HRV was high ($b=.016, p=.003$), but negatively associated when resting HRV was low ($b=-.001, p=.861$).

Supplemental Analyses

Criteria were not met for a mediator relationship as HR/HRV was not correlated with aggression nor CAP. Multiple regression analyses were conducted separately for the 6 subscales (see Table 2) of the CAPI (i.e., Distress, Rigidity, Unhappiness, Problems with Child and Self, Problems with Family, and Problems with Others) to examine whether they contributed to the

relationship between CAP, HR/HRV, and aggression. Interaction terms were significant for the Distress, Problems with Child and Self, and Problems with Others subscales of the CAPI with HR and HRV for predicting proactive and reactive aggression (see Tables 7 – 14) and these interactions remained significant after post hoc probing was conducted. The Distress subscale is made up of items related to distress from interactional problems between the child and parent. This dimension represents a pattern of sadness, frustration, worry, anger, worthlessness, depression, fear, confusion, and misunderstanding. The dimension for Problems with Child and Self assesses the parent's perception of having a problem child as well as the child's ability and competency. An elevation in this dimension may indicate a parent who perceives his/her child as bad, slow, having special problems, or getting into trouble often. The Problems with Others subscale measures the parent's attitude toward relationships and interpersonal problems. Elevated scores in this subscale would suggest that the responder perceives social relationships as a cause of his/her unhappiness, pain, and personal difficulties.

Thus, in order to determine how resting HR and HRV influences the relationship between subscales (i.e., Distress, Problems with Child and Self, Problems with Others) of CAP and proactive/reactive aggression, as well as how HRV influences the relationship between the CAP subscales and proactive aggression, separate simultaneous regressions were conducted testing the effect for the subscales, a conditional resting HR or HRV moderator (i.e., high or low HR, high or low HRV), and their respective interaction terms on either proactive or reactive aggression. Then, regression lines were plotted for high and low resting HR using the centered values for the CAP subscales predicting proactive and reactive aggression. The three subscales were differentially related to aggression dependent upon levels of resting HR and HRV. In particular, they were positively associated with proactive and reactive aggression when resting

HR was low, but were negatively associated with proactive and reactive aggression when resting HR was high. Also the subscales were positively associated with proactive aggression when resting HRV was high, but negatively associated when HRV was low.

Finally, even when controlling for the other type of aggression the relationships between some of the subscales, psychophysiological correlates, and aggression were significant. In particular, Distress was positively associated with proactive aggression when resting HRV was high, but negatively associated with proactive aggression when resting HRV was low. Also, Problems with Child and Self was positively associated with proactive aggression when resting HR was low, but negatively associated with proactive when resting HR was high. Additionally, Problems with Others was positively associated with proactive aggression when resting HR was low, but negatively associated with proactive aggression when resting HR was high. Finally, Problems with Others and proactive aggression were positively associated when resting HRV was high, but negatively associated when resting HRV was low. In sum, the supplemental findings indicate that the interactions between CAP and aggression may be specific to problems associated with family distress, child/self, and others.

Discussion

Although the influence of biological and social factors on aggression have been studied independently (Hubbard et al., 2002; Raine & Venables, 1984; Scarpa et al., 2002; Scarpa & Raine, in press; Wadsworth, 1976), there is a paucity of literature on the interaction effect of these variables. The present study explored the role of autonomic activity in the form of HR and HRV as a moderator between CAP and proactive and reactive aggression.

The hypotheses of this study were partially supported. The results of this study support a biosocial interaction between CAP and HR/HRV in relation to childhood aggression.

Specifically, CAP is related to increased proactive (i.e., instrumental) and reactive (i.e., hostile) aggression in children with low levels of resting HR. CAP is also related to increased proactive aggression in children with high resting HRV. Taken together, this suggests the existence of an interaction of these two biological and social variables above and beyond their individual influences as risk factors.

Further, the results indicate that as levels of CAP increase, low resting HR is a risk factor for both types of aggression in children. This replicates previous findings on relationships between low resting HR and aggression in children and adults (Lorber, 2004, Ortiz & Raine, 2004). Also, it is consistent with Farrington (1997), in which boys with low resting HR were at greater risk for future violent acts if they came from a large family with a poor parent-child relationship in comparison to boys who came from a more favorable home environment. Thus, this study extends previous findings of a biosocial interaction effect, suggesting that psychophysiological variables influence the effect of child abuse on aggression.

In terms of HRV, CAP is related to increased proactive aggression in children with high levels of resting HRV. This finding is consistent with the theory of vagotonia (Venables, 1988), but is specific to proactive aggression. However, this finding is contrary to other studies in which childhood and adolescent aggression was related to attenuated vagal tone (Beauchaine, 2001; Beauchaine & Kopp, in press; Mezzacappa et al., 1996, 1997). It is possible that the findings in other studies were obscured because multiple types of conduct problems were included in the samples (e.g., overall externalizing behavior; delinquency) or because the different types of aggression were not separated.

Limitations

The main limitations of this study consist of the small sample size of 36 children and the

use of a predominantly Caucasian community sample. Specifically, the small sample size decreases power and the ability to explore more fine-grained interactions. Also, the use of a community sample compromises generalizability to children with more serious levels of aggressive behavior and child abuse. Taking these factors into consideration, these results may differ between girls and boys, and may not apply to more high risk populations.

Another limitation of this study is the nature of the self-report measures used. Such instruments are subject to inaccuracies, including lack of knowledge, inaccurate reports, and poor recall. Also, social desirability (e.g., responding in a socially appropriate way), could result in inaccurate or unreliable responses. In particular, the correlation between the CAPI and the R-PRPA may be suggestive of a parental perception bias, in which parents who are more likely to abuse their children are also more likely to perceive their children as more aggressive. Finally, cross-informant measures or behavioral observations may have been used to counteract potential biases from self-report.

In addition, it should be noted that this study assessed for CAP, thus actual child abuse was not directly measured. Further, the CAPI had a relatively low internal consistency as measured by Cronbach's alpha, which may be reflective of the diverse nature of the measure consisting of interpersonal styles and personality attributes of the parents. With the use of a more reliable measure of child maltreatment, stronger relationships may be found between child abuse and aggression. Also, there is ambiguity surrounding the definition and assessment of aggression. While some define aggression in terms of physical violence, others include nonphysical acts (i.e., interactional or relational). For this study, aggression was categorized as reactive and proactive. However, it should be noted that the R-PRPA measured aggressive styles and did not ask for reports of actual acts. Nevertheless, the R-PRPA was found to be psychometrically sound.

Additionally, this study did not include independent verification of aggressive behavior since only parent-report measures were used to assess aggression. More research is needed in the area of defining and assessing aggression.

Theoretical, Research, and Treatment Implications

Despite such limitations, this study provides useful information about the influence of child abuse potential and autonomic activity. Additionally, these findings could help to explain the underlying mechanisms of aggression in children, having implications for prevention and intervention. Thus, the significant biosocial interactions obtained through this study can not be easily dismissed and add to previous findings in the field.

Much research has been conducted on psychophysiological correlates of antisocial behavior in children and adults, including aggression, conduct disorder, crime, delinquency, psychopathy, antisocial personality disorder, hostility, and violence. Particularly, low resting HR is one of the best-replicated psychophysiological measures associated with antisocial behavior. This relationship is suggestive of a fearless/disinhibited temperament and/or a stimulation-seeking tendency, which have been found to predict antisocial outcomes (Raine et al., 1998).

Also, low resting HR could be reflective of poor functioning in the right hemisphere of the brain. Specifically, the right hemisphere plays a dominant role in controlling autonomic mechanisms such as HR (Lane & Jennings, 1995) and low HR has been found to be associated with decreased functioning in the right hemisphere (Zamrini et al., 1990; Yokoyama, Jennings, Ackles, Hood, & Boller, 1987). Thus, even though the left hemisphere has often been associated with violent and criminal behavior (Raine, 1993), research findings also support a relationship between right (Raine et al., 2001a) hemisphere dysfunction and antisocial behavior and violence.

In terms of HRV, studies have found that infants who have a high vagal tone are more

reactive to both negative and positive events as well as being emotionally expressive (Field, Woodson, Greenberg, & Cohen, 1982; Fox, 1989). However as they grow older, they develop an increasing ability to self-soothe (Huffman et al., cited in Porges et al., 1994), which suggests an increased capacity of emotion regulation. Thus, one hypothesis is that these individuals may develop better control of their emotions, so much to the effect that they fail to develop conditioned emotional responses to punishment, which typically deters antisocial behavior (Eysenck, 1977). Such individuals may be at greater risk for becoming proactively aggressive. In other studies, high vagal tone has also been found in infants displaying irritability, fussiness, and poor self-calming by the age of nine-months, who lack the typical vagal withdrawal in response to stress usually seen in normal infants (Portales, Doussard-Roosevelt, Lee, & Porges, cited in Porges et al., 1994). This “fussy” or difficult infant behavior has been related to antisocial behavior later on (Thomas, Chess, & Birch, 1968). Based on the assumption that this behavior continues to young adulthood, another hypothesis is that high vagal tone is representative of a biological risk for inadequate attempts of emotional expression and/or difficult temperament. Consistent with this hypothesis, Scarpa and colleagues (2000) found that increased HRV was primarily associated with verbal forms of aggression, indicating maladaptive attempts of emotional expression. Alternatively, the developmental trajectory of aggressive behavior may be context-dependent as high HRV may be a risk factor for maltreated children but not for non-maltreated children. This is supported by findings of this study, as increased proactive aggression was positively associated with CAP, but only under conditions of high HRV.

Although there is evidence of associations between psychophysiological correlates and aggression, recent literature suggests that there is a biosocial interaction between the psychophysiological and psychosocial risk factors which may result in increased aggressive

behavior in comparison to either risk factor alone. This supports transactional models of development which suggests that the intrinsic and extrinsic risk factors of behavior problems make a contribution together that cannot be teased apart (Sameroff, 1995). Additionally, this is consistent with other studies involving biological variables such as genetics, birth complications and obstetric, hormones, neurotoxins, and neurotransmitters (see Raine, 2002a, 2002b for a review). This study supports such a biosocial interaction between CAP, HR/HRV and aggression.

Since there was a relationship between CAP, HR/HRV, and proactive and reactive aggression, it is important to consider and examine each variable in research and treatment of aggressive children. In terms of research, future studies could investigate specific components that make up CAP in more depth and explore their various developmental trajectories. Specifically, researchers could focus on the Distress, Problems with Child and Self, and Problems with Others subscale of the CAPI in relation to aggression, as these dimensions were shown to have significant relationships in this study. By examining such personality characteristics and psychological difficulties of the parent, they may be able to better predict aggressive outcomes. As for treatment, therapists could explore the role of CAP and HR/HRV in the development of aggressive behavior to facilitate the creation and implementation of treatment plans for the children and families. For children with a biological vulnerability, it would be especially important to include the families, particularly parents, in the treatment process, as their role was implicated in this study's findings. For children with psychosocial adversity, prevention efforts may also do well to focus on early developmental influences of autonomic functioning. For example, a recent study found that an environmental preschool enrichment program similar to Head Start, resulted in significant increases in SC and brain-wave activity and attention 8

years later (Raine et al., 2001b). In addition, the findings of this study could be incorporated in developing psychophysiological assessment measures for psychopathology in children. Overall, these results should encourage members in this field to examine both biological and social variables in their future endeavors.

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Appendix B

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Informed Consent for Participants of Investigative Projects (Parent for Self and Child)

I. The Purpose of this Research/Project

If your child is between 7 and 13 years old, you are invited to participate in a research project run through the Child Study Center, Department of Psychology at Virginia Tech. The project is designed to study emotional reactions in 7-13 year-old children. Approximately one hundred eighty five families are being asked to participate.

As part of this study, you are asked to complete an interview and questionnaires about your child and some questionnaires about your perceptions of yourself and your family. A questionnaire packet will also be sent home for the other parent to complete, when applicable. Your child will be asked to complete several computerized tasks and questionnaires. Physiological responses in your child that reflect his/her emotional reaction to a series of computer tasks will also be measured. The entire session will last approximately 3 hours.

Physiological reactivity is measured by changes in your child's heart rate, skin sweat, and in the activity of the hormone cortisol. Cortisol is a natural chemical in the body that helps guide certain body functions such as energy and metabolism, and it can be measured through the saliva in the mouth. Changes in heart rate, sweat, and cortisol reactivity occur when children feel they are stressed or anxious, but some children can manage these reactions more easily than others. Physiological reactivity is important to measure because it is thought to affect social behavior and emotions in potentially stressful situations that children encounter.

Because puberty can also affect your child's physiological responses and behavior, this study will also include an assessment of your child's level of healthy maturational development. This will be done through a questionnaire completed by your child. We also ask that you inform us if your child has asthma, heart problems, or any other physical illness because these can affect physiological reactivity. Your child may be ineligible to participate if he/she has a severe chronic illness.

II. Procedures

Your child will be asked to complete several questionnaires and computer tasks. The first game is a card game where your child tries to earn the most points associated with different cards. In the second computer game, your child believes that he/she is playing against another child for points and that the child earning the most points wins the game. Throughout the game, your child may give or receive consequences that consist of a startling burst of noise or removing points on the computer. This makes the game similar to other computer games that are challenging and slightly stressful for children. In reality, there is no other child playing the

game with your child. Your child will simply play against the computer, although he/she believes that another child is also playing in the next room. It is necessary for us to pretend that another child is there in order to make the competition seem realistic and challenging for your child. This is necessary in order to obtain physiological reactivity measurements that might occur in similar social situations that children have with other children. **We ask that you do not speak to your child about this game before he/she participates.**

Your child's heart rate and sweating will be monitored before and after the second computer game from an electrode placed on the skin over the collarbone, below the lower ribs, and on his/her hand. This is similar to procedures used in hospitals and involves no pain or risk for your child, although the adhesive on the electrodes may hurt like a bandaid when removed from the skin. Cortisol will be measured in your child's saliva three times – once before and twice after the computer game. Saliva is collected by having him/her drop saliva from his/her mouth through a straw and into a small tube. Cortisol levels are measured from the saliva collected in the tube.

As a general indicator of pubertal development, your child will be asked to complete a questionnaire about how children change as they grow and become teenagers. Your child will be asked to report in the questionnaire if he/she has noticed any of these changes in him/herself. His/her height and weight will be measured. Before and after the game your child will be asked some questions about his/her mood and thoughts regarding the game. Your child's responses will be audio-taped so that we may review them at a later time.

You will be interviewed about your child's behavior to determine possible diagnoses he/she may have. You will then be asked to complete several questionnaires about your child's medical history and health habits, behavior, emotions, and family. You will also be asked to complete two questionnaires regarding your perceptions of your own emotional experiences and parenting attitudes. A packet of these questionnaires will also be sent to your child's other parent, when applicable. Appropriate schoolteachers will also be sent two questionnaires about the child's behavior in school. Lastly, we will obtain school records regarding your child's intellectual and achievement testing through Special Education services. This is necessary so we can take into account how cognitive functioning affects emotional and behavioral problems.

III. Risks

The only risk of participating in this study is the possibility that your child might experience emotional discomfort during the computer task, which is designed to be mildly stressful. To minimize this possibility, this study uses only procedures that have been successfully used before with no adverse effects on participants. Furthermore, you are free to withdraw your child from this study at any time with no penalties. Your child is also told that he/she does not have to participate or may stop playing the game at any time if he/she wishes to do so. The child is also told that he/she does not have to answer any questions if he/she does not want to.

IV. Benefits of this Project

The benefit of this project will be a better understanding of emotional and behavioral

problems and development in children. Note that there is no guarantee of benefits made to encourage you or your child to participate. The participation of you and your child is completely voluntary.

V. Extent of Anonymity and Confidentiality

Any information obtained during this research will be kept confidential. Situations where confidentiality must be broken include 1) if a previously unreported incident of child abuse is known or strongly suspected, 2) if a participant is believed to be a threat to him/herself or others, or 3) if the information is subpoenaed by court order. In these cases, the investigator must notify appropriate authorities, and you will be informed of the need to report.

All information is coded after it is obtained so there is no way to associate you or your child by name with the information. Any information that carries identifying material will be kept in locked files. Only designated research personnel will have access to this information. Furthermore, all audio-tapes will be destroyed after the study is completed. At no time will information obtained from this study be released to anyone without your written consent, except under the conditions noted above. Results may be published or presented for scientific purposes, but your or your child's identity will not be revealed in any description or publication of this research.

VI. Compensation

You will receive \$40 for participation in this study, which you can share with your child if you like. Your child will be entered into a drawing to win a \$25 gift certificate for a local store. The drawing will be held for every fifty participants until the study is completed.

VII. Freedom to Withdraw

You are free to withdraw your child from this study at any time without penalty. Parents and children have the right to refuse to answer any questions or respond to any part of this study, and there would be no penalty.

There may be circumstances under which the investigator may determine that your child can not continue in the study. In these cases, you will be notified and the session will end.

VIII. Approval of Research

This project has been approved, as required by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute and State University, by the Department of Psychology and the Child Study Center, and by the Montgomery County Public School System.

IX. Subject's Responsibilities

I voluntarily agree to participate in this study, and give my consent for my child to participate. I have the following responsibilities:

1. Complete an interview and questionnaires.
2. Give my permission for my child to complete psychological and physiological assessments, consisting of computer tasks and questionnaires.
3. Do not tell my child that there is no real competitor in the computer game before he/she plays.
4. Give my permission to access school records for measures of intellectual and achievement testing.
5. Give my permission for questionnaires to be sent to the child's schoolteachers and other parent.

X. Permission

I have read the preceding Informed Consent and conditions of this project, or it has been read to me, and I understand its contents. Any questions I have pertaining to the study have been answered. I hereby acknowledge the above and give my voluntary consent for my child's participation in this project. A copy of this consent form has been given to me.

Should I have any questions about this research or its conduct, I may contact any of the persons named below:

| | |
|---|----------|
| Dr. Angela Scarpa, Investigator, Department of Psychology | 231-2615 |
| Dr. Thomas H. Ollendick, Director, Child Study Center | 231-6451 |
| Dr. D.W. Harrison, Psychology Human Subjects Committee | 231-4422 |
| Dr. David M. Moore, Chair, IRB, Research Division | 231-4911 |

If my child participates, I understand that he/she may withdraw at any time without penalty. I agree to abide by the rules of this project.

Parent/Guardian's Name (Printed)

Parent's/Guardian's Signature

Date

Appendix C

CHILD ASSENT TO PARTICIPATE IN A RESEARCH STUDY

DR. SCARPA HAS ASKED ME TO BE IN A STUDY OF BEHAVIOR IN CHILDREN. SHE WANTS TO SEE HOW MY HEART, SWEAT, AND A HORMONE CALLED CORTISOL CHANGE WHEN I PLAY A COMPUTER GAME WITH OTHER CHILDREN. HORMONES ARE CHEMICALS IN THE BODY THAT CAN BE MEASURED THROUGH MY SALIVA (SPIT). I AM ASKED TO PLAY A COMPUTER GAME WITH ANOTHER CHILD. I WILL DROP SALIVA THROUGH A STRAW AND INTO A SMALL TUBE ONCE BEFORE AND TWICE AFTER THE GAME. I WILL ALSO HAVE A MONITOR PLACED ON THE SKIN OVER MY COLLARBONE AND LOWER RIB AND ON MY HAND THAT WILL MEASURE MY HEART RATE AND SWEATING. THE MONITORS MIGHT HURT LIKE A BANDAID WHEN THEY ARE REMOVED. I WILL BE ASKED QUESTIONS ABOUT THE GAME, AND MY ANSWERS WILL BE TAPED. I AM ALSO ASKED TO FILL OUT A QUESTIONNAIRE ABOUT HOW CHILDREN CHANGE AS THEY BECOME TEENAGERS TO SEE IF I HAVE NOTICED ANY OF THOSE CHANGES IN MYSELF. I AM ALSO ASKED TO FILL OUT OTHER QUESTIONNAIRES ABOUT MYSELF AND TO PLAY A FEW COMPUTERIZED GAMES. IT WILL TAKE ABOUT THREE HOURS TO DO ALL THIS.

I UNDERSTAND THAT I WILL NOT RECEIVE ANYTHING FOR COMPLETING THESE THINGS, BUT I WILL BE ENTERED INTO A DRAWING WHERE I CAN WIN A \$25 GIFT CERTIFICATE. IF I DO NOT WANT TO BE IN THE STUDY, I DO NOT HAVE TO. IF I AM UNCOMFORTABLE OR FEEL ANY PAIN I SHOULD TELL THE EXPERIMENTER SO WE CAN FIX IT OR STOP THE STUDY.

ALSO, IF I DO NOT WANT TO ANSWER ANY QUESTIONS THAT IS OKAY TOO. I UNDERSTAND THAT ANYTHING I TELL THE TESTER IS PRIVATE, BUT THERE MAY BE A TIME WHEN SOMETHING I SAY MUST BE TOLD TO A COUNSELOR OR MY PARENTS TO HELP ME OR SOMEONE IN MY FAMILY, SPECIFICALLY IF I AM BEING HURT OR IF I AM GOING TO HURT SOMEONE ELSE.

IF I HAVE ANY QUESTIONS NOW, I CAN ASK THE TESTER. IF I HAVE ANY QUESTIONS AFTER THE STUDY IS FINISHED, I MAY CALL ANGELA SCARPA AT 231-2615.

I CAN STOP BEING IN THE STUDY AT ANY TIME. EVEN IF I DO SAY I WANT TO BE IN THE STUDY NOW, I CAN CHANGE MY MIND AFTER I START. I UNDERSTAND WHAT I AM ASKED TO DO AND WHO TO ASK IF I HAVE A QUESTION.

I WANT TO SAY "YES" TO BE IN THE STUDY.

Child's Signature

Date

Witness's Signature

Date

Table 1. Means (SD in Parentheses) and Ranges for Total, and Means, T Values, and Significance Levels By Sex of Participant.

| Variables | Total | Range | Male | Female | t | p |
|------------------------------|------------------|---------|-------------------|------------------|------|-----|
| CAP | 90.89 (91.56) | 9 – 356 | 91.80 (102.30) | 88.82 (64.95) | .09 | .93 |
| Distress | 54.72 (64.86) | 0 – 232 | 55.96 (70.69) | 51.91 (52.13) | .17 | .87 |
| Rigidity | 7.28 (8.76) | 0 – 37 | 6.56 (8.20) | 8.91 (10.13) | -.74 | .47 |
| Unhappiness | 14.03 (12.34) | 0 – 42 | 13.08 (11.96) | 16.18 (13.50) | -.69 | .50 |
| Problems with Family | 5.19 (9.45) | 0 – 36 | 6.48 (11.01) | 2.27 (3.17) | 1.24 | .23 |
| Problems with Child and Self | 4.36 | 0 – 26 | 4.44 | 4.18 | .09 | .92 |

| | | | | | | |
|----------------------|---------|----------------|---------|---------|-------|-----|
| | (7.62) | | (7.65) | (7.90) | | |
| Problems with Others | 5.58 | 0 – 24 | 5.68 | 5.36 | .11 | .91 |
| | (7.96) | | (8.48) | (6.99) | | |
| Resting HR | 84.83 | 65.44 – 105.80 | 85.91 | 82.35 | 1.00 | .32 |
| | (9.82) | | (10.47) | (8.05) | | |
| Resting HRV | 64.67 | 20 – 202.75 | 57.07 | 81.96 | -1.70 | .10 |
| | (41.64) | | (31.01) | (57.34) | | |
| Proactive Aggression | 12.19 | 10 – 21 | 11.92 | 12.82 | -1.08 | .29 |
| | (.23) | | (2.45) | (1.94) | | |
| Reactive Aggression | 10.61 | 6 – 17 | 10.44 | 11.00 | -0.57 | .58 |
| | (2.71) | | (2.90) | (2.28) | | |

Note. Non-centered values are shown and reflect actual values reported. $N = 36$. * $p < .05$.

Table 2.

Zero-order Correlations between Proactive/Reactive Aggression, HR, HRV, CAP, and Age.

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
|----------------------------------|-------|-------|---------|-------|-------|-------|-------|-------|-------|------|------|-------|----|
| 1. Proactive Aggression | 1 | | | | | | | | | | | | |
| 2. Reactive Aggression | .57** | 1 | | | | | | | | | | | |
| 3. Heart Rate | -0.13 | 0.1 | 1 | | | | | | | | | | |
| 4. Heart Rate Variability | 0.2 | -0.11 | -0.65** | 1 | | | | | | | | | |
| 5. Child Abuse Potential | .37* | 0.33 | -0.18 | 0.12 | 1 | | | | | | | | |
| 6. Distress | 0.33* | 0.38* | -0.11 | 0.06 | .98** | 1 | | | | | | | |
| 7. Rigidity | 0.26 | 0.24 | -0.1 | 0.19 | 0.25 | 0.16 | 1 | | | | | | |
| 8. Unhappiness | 0.08 | -0.13 | -0.35 | 0.17 | .64** | .59** | -0.1 | 1 | | | | | |
| 9. Problems with Family | 0.3 | 0.21 | -0.14 | 0.03 | .79** | .76** | 0.04 | .48** | 1 | | | | |
| 10. Problems with Child and Self | .45** | 0.17 | -0.16 | 0.18 | .61** | .56** | 0.03 | 0.24 | .62** | 1 | | | |
| 11. Problems with Others | .34* | 0.27 | -0.22 | 0.26 | .79** | .74** | .55** | .40* | .43** | .40* | 1 | | |
| 12. Age | 0.091 | 0.12 | -0.03 | -0.18 | 0.081 | 0.05 | 0.14 | 0.01 | 0.08 | 0.08 | 0.22 | 1 | |
| 13. Body Mass Index | 0.04 | -0.02 | -0.06 | -0.29 | 0.2 | 0.18 | 0.06 | 0.21 | 0.03 | 0.12 | 0.29 | .66** | 1 |

Note. Non-centered values are shown and reflect actual values reported. $N = 36$. * $p < .05$. ** $p < .01$.

Table 3.

Hierarchical regressions with CAP and HR predicting proactive aggression

| Variable | B | SE B | β | ΔR^2 |
|----------|------|------|---------|--------------|
| Block 1 | | | | .14* |
| CAP | .01 | .00 | .37* | |
| Block 2 | | | | .01 |
| CAP | .01 | .00 | .36* | |
| HR | -.02 | .04 | -.07 | |
| Block 3 | | | | .20** |
| CAP | .01 | .00 | .19 | |
| HR | -.03 | .04 | -.12 | |
| CAP X HR | -.00 | .00 | -.47** | |

Note: ** $p < .01$. * $p \leq .05$.

Table 4.

Hierarchical regressions with CAP and HR predicting reactive aggression

| Variable | B | SE B | β | ΔR^2 |
|----------|------|------|---------|--------------|
| Block 1 | | | | .11* |
| CAP | .01 | .01 | .33* | |
| Block 2 | | | | .03 |
| CAP | .01 | .01 | .36* | |
| HR | .05 | .05 | .17 | |
| Block 3 | | | | .16* |
| CAP | .01 | .01 | .21 | |
| HR | .03 | .04 | .13 | |
| CAP X HR | -.00 | .00 | -.42* | |

Note: ** $p < .01$. * $p \leq .05$.

Table 5.

Hierarchical regressions with CAP and HRV predicting proactive aggression

| Variable | B | SE B | β | ΔR^2 |
|-----------|------|------|---------|--------------|
| Block 1 | | | | .14* |
| CAP | .01 | .00 | .37* | |
| Block 2 | | | | .03 |
| CAP | .01 | .00 | .35* | |
| HRV | 1.51 | 1.53 | .16 | |
| Block 3 | | | | .11* |
| CAP | .01 | .00 | .29 | |
| HR | 1.34 | 1.45 | .14 | |
| CAP X HRV | .04 | .02 | .34* | |

Note: ** $p < .01$. * $p \leq .05$.

Table 6.

Hierarchical regressions with CAP and HRV predicting reactive aggression

| Variable | B | SE B | β | ΔR^2 |
|-----------|-------|------|---------|--------------|
| Block 1 | | | | .11* |
| CAP | .01 | .01 | .33* | |
| Block 2 | | | | .02 |
| CAP | .01 | .01 | .35* | |
| HRV | -1.70 | 1.82 | -.15 | |
| Block 3 | | | | .09 |
| CAP | .01 | .01 | .31 | |
| HR | -1.88 | 1.76 | -.17 | |
| CAP X HRV | .04 | .02 | .30 | |

Note: ** $p < .01$. * $p \leq .05$.

Table 7.

Hierarchical regressions with CAP Distress and HR predicting proactive aggression

| Variable | B | SE B | β | ΔR^2 |
|---------------|------|------|---------|--------------|
| Block 1 | | | | .11* |
| Distress | .01 | .01 | .33* | |
| Block 2 | | | | .01 |
| Distress | .01 | .01 | .33* | |
| HR | -.02 | .04 | -.10 | |
| Block 3 | | | | .19** |
| Distress | .01 | .01 | .13 | |
| HR | -.04 | .04 | -.16 | |
| Distress X HR | -.00 | .00 | -.47** | |

Note: ** $p < .01$. * $p \leq .05$.

Table 8.

Hierarchical regressions with CAP Distress and HR predicting reactive aggression

| Variable | B | SE B | B | ΔR^2 |
|---------------|------|------|-------|--------------|
| Block 1 | | | | .15* |
| Distress | .02 | .01 | .39* | |
| Block 2 | | | | .03 |
| Distress | .02 | .01 | .40* | |
| HR | .05 | .05 | .15 | |
| Block 3 | | | | .15* |
| Distress | .01 | .01 | .22 | |
| HR | .03 | .04 | .10 | |
| Distress X HR | -.00 | .00 | -.42* | |

Note: ** $p < .01$. * $p \leq .05$.

Table 9.

Hierarchical regressions with CAP Distress and HRV predicting proactive aggression

| Variable | B | SE B | B | ΔR^2 |
|----------------|------|------|------|--------------|
| Block 1 | | | | .11* |
| Distress | .01 | .01 | .33* | |
| Block 2 | | | | .03 |
| Distress | .01 | .01 | .33* | |
| HRV | 1.74 | 1.54 | .18 | |
| Block 3 | | | | .10* |
| Distress | .01 | .01 | .24 | |
| HRV | 1.93 | 1.47 | .20 | |
| Distress X HRV | .05 | .02 | .33* | |

Note: ** $p < .01$. * $p \leq .05$.

Table 10.

Hierarchical regressions with CAP Problems with Child and Self and HR predicting proactive aggression

| Variable | B | SE B | B | ΔR^2 |
|------------|------|------|--------|--------------|
| Block 1 | | | | .21** |
| Child | .14 | .05 | .45** | |
| Block 2 | | | | .00 |
| Child | .14 | .05 | .44** | |
| HR | -.02 | .04 | -.07 | |
| Block 3 | | | | .23** |
| Child | -.03 | .07 | -.08 | |
| HR | -.03 | .03 | -.11 | |
| Child X HR | -.01 | .00 | -.71** | |

Note: ** $p < .01$. * $p \leq .05$.

Table 11.

Hierarchical regressions with CAP Problems with Child and Self and HR predicting reactive aggression

| Variable | B | SE B | B | ΔR^2 |
|------------|------|------|--------|--------------|
| Block 1 | | | | .03 |
| Child | .06 | .06 | .17 | |
| Block 2 | | | | .02 |
| Child | .07 | .06 | .19 | |
| HR | .04 | .05 | .13 | |
| Block 3 | | | | .19** |
| Child | .05 | .06 | .15 | |
| HR | .03 | .04 | .09 | |
| Child X HR | -.02 | .01 | -.44** | |

Note: ** $p < .01$. * $p \leq .05$.

Table 12.

Hierarchical regressions with CAP Problems with Child and Self and HRV predicting proactive aggression

| Variable | B | SE B | B | ΔR^2 |
|-------------|-----|------|-------|--------------|
| Block 1 | | | | .21** |
| Child | .14 | .05 | .45** | |
| Block 2 | | | | .02 |
| Child | .13 | .05 | .43** | |
| HRV | .17 | 1.49 | .12 | |
| Block 3 | | | | .11* |
| Child | .12 | .05 | .40* | |
| HRV | .79 | 1.41 | .08 | |
| Child X HRV | .35 | 0.15 | .34* | |

Note: ** $p < .01$. * $p \leq .05$.

Table 13.

Hierarchical regressions with CAP Problems with Others and HR predicting proactive aggression

| Variable | B | SE B | B | ΔR^2 |
|------------|------|------|--------|--------------|
| Block 1 | | | | .12* |
| Other | .10 | .05 | .34* | |
| Block 2 | | | | .00 |
| Other | .10 | .05 | .33* | |
| HR | -.02 | .04 | -.06 | |
| Block 3 | | | | .31** |
| Other | .04 | .04 | .13 | |
| HR | -.04 | .03 | -.19 | |
| Other X HR | -.02 | .01 | -.60** | |

Note: ** $p < .01$. * $p \leq .05$.

Table 14.

Hierarchical regressions with CAP Problems with Others and HR predicting reactive aggression

| Variable | B | SE B | B | ΔR^2 |
|------------|------|------|-------|--------------|
| Block 1 | | | | .07 |
| Other | .10 | .06 | .27 | |
| Block 2 | | | | .03 |
| Other | .10 | .06 | .30 | |
| HR | .05 | .05 | .17 | |
| Block 3 | | | | .13* |
| Other | .06 | .06 | .18 | |
| HR | .03 | .05 | .09 | |
| Other X HR | -.02 | .01 | -.38* | |

Note: ** $p < .01$. * $p \leq .05$.

Table 15.

Hierarchical regressions with CAP Problems with Others and HRV predicting proactive aggression

| Variable | B | SE B | B | ΔR^2 |
|-------------|------|------|-------|--------------|
| Block 1 | | | | .12* |
| Other | .10 | .05 | .34* | |
| Block 2 | | | | .01 |
| Other | .10 | .05 | .31 | |
| HRV | 1.16 | 1.60 | .12 | |
| Block 3 | | | | .18** |
| Other | .05 | .05 | .18 | |
| HRV | 1.20 | 1.45 | .12 | |
| Other X HRV | 0.63 | .20 | .44** | |

Note: ** $p < .01$. * $p \leq .05$.

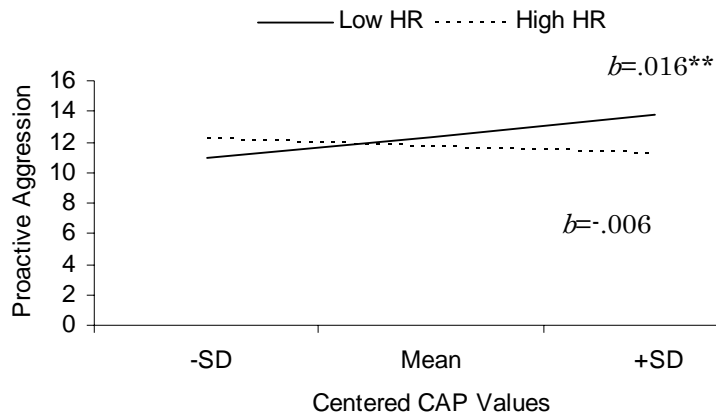


Figure 1. Regression lines for relationship between CAP and proactive aggression as moderated by resting heart rate (HR). b = unstandardized coefficient. $** p < .01$. $* p < .05$.

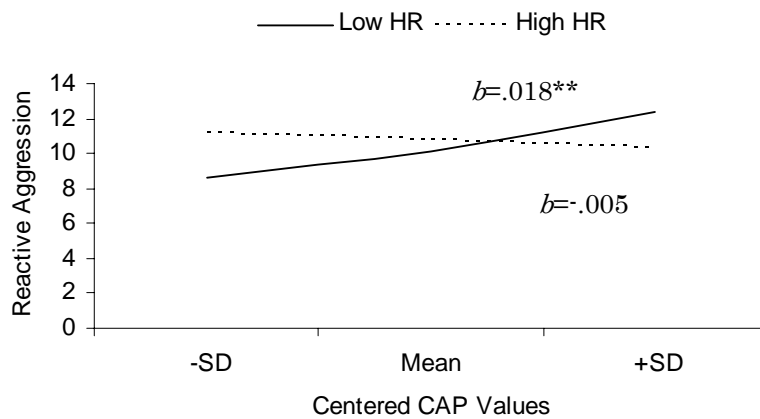


Figure 2. Regression lines for relationship between CAP and reactive aggression as moderated by resting heart rate (HR). b = unstandardized coefficient. $** p < .01$. $* p < .05$.

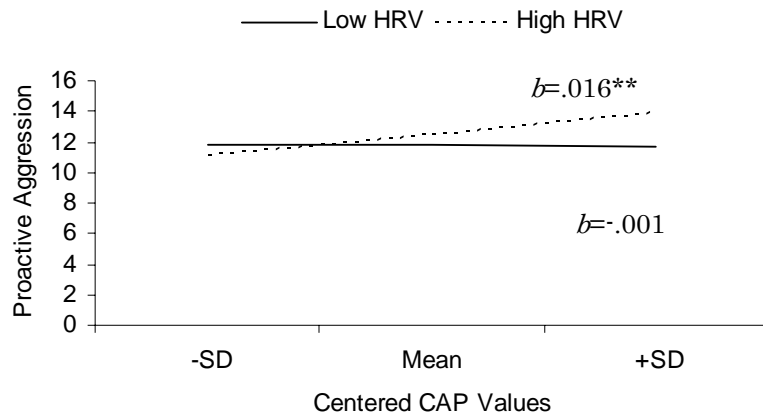


Figure 3. Regression lines for relationship between CAP and proactive aggression as moderated by resting heart rate variability (HRV). b = unstandardized coefficient. $** p < .01$.

* $p < .05$.