

# BIOSOCIAL BASES OF REACTIVE AND PROACTIVE AGGRESSION: THE ROLES OF COMMUNITY VIOLENCE EXPOSURE AND HEART RATE

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*In order to more fully understand how individual differences influence adaptation to violence, this study examined the moderating influence of resting heart rate (HR) and HR variability (HRV) between community violence (CV) exposure and child reactive/proactive aggression. Forty 7-13-year-old community children self-reported CV exposure (i.e., victimization, witnessing, or hearing about violence) and were assessed for resting HR and HRV. Parents rated them on reactive/proactive aggression. CV victimization was positively related to proactive aggression only in conditions of low HR, and witnessed CV was positively related to reactive aggression only in conditions of high HRV. Main effects were not found for CV exposure or psychophysiological functioning, suggesting the importance of their interaction. Findings are discussed in terms of HR under-arousal, emotion dysregulation, fearlessness, and behavioral disinhibition as components that can increase aggression in response to violent contexts. Findings support a biosocial basis for childhood aggression and have implications for prevention and treatment. © 2008 Wiley Periodicals, Inc.*

Recently, researchers have noted the importance of examining interactions between biological and social variables that affect the risk of antisocial and aggressive behavior in order to obtain a comprehensive understanding of the problem (Raine, 2002b). One

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type of interaction is one in which the presence of both biological and social risk factors increases the rates of antisocial and violent behavior. In an adoption study, for example, crime rates in adoptees were highest when both environmental and heritable factors were present (Cloninger & Gottesman, 1987). As another example, Farrington (1997) found that boys with low resting HR who came from low socioeconomic status backgrounds were more likely to be rated as aggressive by their teachers.

The aim of this study was to assess such biosocial interactions in relation to aggression in a community sample of children. Specifically, we examined the influence of heart rate (HR) level and HR variability (HRV) on the relationship between community violence (CV) exposure with proactive and reactive aggression in children. The underlying assumption guiding this research is that such individual differences reflect personality processes that may influence children's adaptation to adverse social circumstances. The nature of these relationships is further described below.

## HEART RATE AND AGGRESSIVE BEHAVIOR

Research has found that biological variables contribute to the development of antisocial and aggressive behavior in children (Raine, 2002b; Scarpa & Raine, 2003). In particular, low autonomic arousal, especially in the form of low resting HR, is significantly associated with antisocial behavior in both children and adults. In a recent meta-analysis of 40 studies on antisocial behavior in children and adolescents, the average effect size for HR was  $-0.44$  at rest and  $-0.76$  during a stressor (Ortiz & Raine, 2004), leading the authors to conclude that low HR is the best-replicated biological correlate of antisocial behavior to date. In support of this conclusion, another recent meta-analysis of 19 studies found that both child and adult aggression was related to low resting HR with an average effect size of  $-0.38$  ( $d = -0.51$  in child samples and  $-0.30$  in adults; Lorber, 2004).

Personality theory has a history of research linking biological processes to certain dimensions of personality (e.g., Eysenck, 1967; Gray, 1970), and findings support the notion that such individual differences may predispose to violent behavior (Henry, Caspi, Moffitt, & Silva, 1996). HR under-arousal has been attributed to the personality trait of extraversion or stimulation-seeking; under-aroused individuals are thought to seek stimulation or reward to elevate their arousal (Eysenck, 1997; Quay, 1965; Raine, 1993, 1997; Raine, Reynolds, Venables, Mednick, & Farrington, 1998). One component of extraversion is impulsivity, or acting without forethought. Reinforcement Sensitivity Theory proposes that impulsive individuals may be oversensitive to reward due to an overactive behavioral activation system (BAS) coupled with an underactive behavioral inhibition system (BIS; Corr, 2002; Gray, 1982; Gray & McNaughton, 2000). Consistent with the notion of stimulation-seeking, Mathias & Stanford (2003) found that men characterized by high (versus normal) levels of impulsivity showed reduced resting HR and increased HR reactivity to a challenge. Knyazev, Slobodskaya, & Wilson (2002) similarly found evidence for cortical and autonomic under-arousal in relation to high BAS. In addition to reward sensitivity, under-arousal may also reflect low levels of fear during mildly stressful situations (Raine, 2002a, 2002b). In such cases of reduced fear, individuals are less likely to inhibit their behaviors in risky situations or to learn from cues of punishment. Taken together, these findings suggest that low HR may relate to personality profiles that are high in fearlessness, sensation-seeking, risk-taking, or impulsivity. Such personalities

may be channeled in socially appropriate ways (e.g., race car driving, sports, and recreational activities). Violence exposure, however, has the potential to socialize these personalities toward inappropriate aggressive behavior.

HR variability (HRV) has also been implicated in antisocial behavior, but the direction of the relationship is unclear. HRV reflects the normal variation of intervals that occur between heart beats as a function of respiration. Whereas HR has both sympathetic and parasympathetic influences, HRV is mediated by the vagus nerve and controlled primarily by the parasympathetic branch of the autonomic nervous system. The parasympathetic nervous system helps to slow the heart, which creates more beat-to-beat variability in HR, and this variability is thought to reflect vagal tone. Because of its parasympathetic influence, high vagal tone is thought to reflect emotion regulation capacity in the sense of being able to self-soothe when experiencing a strong emotion (Katz & Gottman, 1995; Porges, Doussard-Roosevelt, & Maiti, 1994); thus, it may reflect a protective factor that buffers children from adverse situations. For example, Katz and Gottman (1995) reported that increased HRV protected children exposed to marital conflict from developing increased externalizing behavior problems. By the same token, however, decreased HR and increased HRV have been found in children with a behaviorally disinhibited temperament (Kagan, 1989; Scarpa, Raine, Venables, & Mednick, 1997) and in impulsive adults (Barrett, 1972). Consistent with the HR literature described above, this type of bold, curious, and extraverted personality may increase the likelihood of being present in high-risk situations (e.g., parties, large social events) and may also increase impulsivity and risk-taking (e.g., use of drugs/alcohol) in children who are already vulnerable due to being exposed to violent homes and neighborhoods.

Given the typical inverse relationship between HR and HRV, and the above-noted association between low resting HR and aggression, it may be predicted that aggression would be marked by increased levels of HRV. This pattern of low HR and increased HRV, reflecting vagotonia or high vagal tone, has been posited as a biological risk factor for antisocial behavior (Venables, 1988). Although this result was found by Scarpa, Fikretoglu, and Luscher (2000) in young adults, several studies have reported opposite effects, such that antisocial or disruptive behavior in youth was characterized by reduced respiratory sinus arrhythmia (RSA; i.e., another method of assessing vagal tone) and HRV (e.g., Mezzacappa, et al., 1997). Because of the relationship of low vagal tone to dysregulated emotional states, including anger, the findings of reduced HRV may pertain only to certain functions of aggression (e.g., reactive aggression versus proactive aggression), but functions were not distinguished in these studies. Given these discrepant findings, it is important to determine what other variables might impact this relationship, and indeed if findings may differ depending on the kind of aggression assessed.

## COMMUNITY VIOLENCE EXPOSURE AND AGGRESSION

Definitions for what constitutes CV exposure differ from study to study, but most often it refers to violence that is experienced as a victim or witness in or near homes, schools, and surrounding neighborhoods. CV exposure is associated with greater levels of aggression in both children and young adults (e.g., Eitle & Turner, 2002; Miller, Wasserman, Neugebauer, Gorman-Smith, & Kamboukos, 1999; Scarpa, 2001; 2003; Scarpa et al., 2002; Schwab-Stone et al., 1995, 1999; Widom, 1989). Margolin and

Gordis (2000) explain that violence exposure may result in the child interpreting the world as an unsafe place. Moreover, violent exposures have been linked to difficulties in emotional development, especially hypersensitivity to anger (e.g., Dodge, Petit, & Bates, 1997), as well as psychobiological effects such as increased catecholamine activity and variations in functioning of the hypothalamic-pituitary-adrenal axis (Van Voorhees & Scarpa, 2004). Thus, exposure to CV can affect various aspects of child development and it is important to examine how those processes may interact in a biosocial framework.

To our knowledge, only one study examined the role of biological variables in interaction with CV victimization (Scarpa & Ollendick, 2003). In this study of young adults, increased HRV was associated with aggression in non-victims, increased cortisol level was associated with aggression in victims, and reduced resting HR was associated with aggression in both victims and non-victims. The investigators speculated that the HR and HRV findings in non-victims may reflect fearlessness or over-control of emotions, whereas the cortisol findings in victims may reflect an attempt to actively cope with their stressful experiences (albeit in a maladaptive manner). These findings further implicate the potential importance of examining CV exposure in interaction with biological factors when studying aggression. Of note, indirect experiences with violence, such as witnessing, was not assessed in this study. Others have found that witnessing is more common than direct victimization in youth and young adults, with rates as high as 98% of respondents reporting that they have witnessed a violent event (see Scarpa, 2003 for a review). As such, it is unclear if the above-noted interaction effects applied uniquely to the victimization experience. Future studies on biosocial interactions may benefit from studying unique effects associated with indirect versus direct forms of violence exposure.

Based on the research, it seems that exposure to violence has serious cognitive, emotional, and psychobiological implications for children that may relate to the development of aggressive behavior. However, in addition to potential differences resulting from indirect and direct forms of CV exposure, it is also possible that biological and social risk factors differ depending on the function of aggression displayed. In particular, two functions of aggression that have been gaining increasing recognition in children are reactive and proactive aggression (Crick & Dodge, 1996; Dodge, 1991).

## **CHILDHOOD REACTIVE AND PROACTIVE AGGRESSION**

Reactive aggression is characterized by a highly emotional response, such as anger, fear, and frustration, and it is often preceded by a real or perceived threat or provocation. This type of aggression is rooted in the frustration-anger theory of aggression (see Vitaro, Brendgen, & Barker, 2006 for review) such that the motive is to react to the anger-frustration stimulus and injure the perpetrator of the threat or provocation, and it is often associated with hostile attribution biases. In contrast, proactive aggression is considered to be more instrumental in nature, as it is used in order to obtain a specific goal. This function of aggression is consistent with the social learning model of aggression (Bandura, 1973) in that aggressive behavior is regulated by reinforcement contingencies. As such, it is associated with valuing instrumental over social goals and believing aggression will lead to positive outcomes.

Although proactive and reactive functions of aggression are associated with one another, researchers have found support for their distinction (Merk, Orobio de Castro, Koops, & Mattys, 2005). Exploratory and confirmatory factor analyses have consistently yielded two factors of proactive and reactive aggression (Crick & Dodge, 1996; Day, Bream, & Paul, 1992; Poulin & Boivin, 2000). Research also suggests differences in associated risk factors. Raine et al. (2006) found that adolescents who engaged in reactive aggression were more anxious and impulsive, and also displayed information-processing abnormalities and reality distortions compared to proactively aggressive adolescents who were characterized as psychopathy-prone, seriously violent, and coming from a poor social background. Both functions were associated with excessive fighting, paranoid ideation, exposure to traumatic stress, and stimulation seeking.

Researchers have also suggested that reactive/proactive aggression have different psychophysiological correlates. Scarpa and Raine (2000) suggested that proactive and reactive aggression would be associated with physiological under- and over-arousal, respectively. The psychophysiological under-arousal of proactive aggression is consistent with stimulation-seeking (Eysenck, 1964; Quay, 1965) and fearlessness theory (Raine, 2002a; 2002b). In terms of reactive aggression, physiological responses are expected to be elevated due to the salience of real or perceived threat, thus reflecting an automatic stress response and impulsive emotional responses in reaction to anger or heightened emotionality (Lochman, Whidby, & FitzGerald 2000; Zillman, 1983). Such psychophysiological findings in relation to each function of aggression have been mixed, however. As would be expected, Hubbard et al. (2002) found that skin conductance (SC) and HR reactivity were positively associated to reactive, but not proactive, aggression in children placed in a frustrating situation. However, in Scarpa and Haden (2006), reactive aggression was related to *decreased* resting SC and HRV, while proactive aggression was associated with *elevated* resting SC and HRV, and they found no significant associations between resting HR and either function of aggression.

Discrepancies between the Hubbard et al. (2002) and Scarpa and Haden (2006) findings may be explained by methodological differences. For example, the former measured autonomic reactivity during a frustrating task and used teacher ratings of aggression, while the latter measured baseline activity during a rest period and used parent ratings of aggression. Another explanation involves the interpretation of the psychophysiological processes. Perhaps the notion of general under-arousal is too simplistic and different autonomic patterns exist depending on context. Notably, neither of these studies took into account the psychosocial context of violence exposure. In particular, direct victimization may provide an environment whereby children learn that aggression can achieve goals of intimidation, power, or tangible goods as used in proactive aggression. However, witnessing or hearing about violence may increase a child's sense of general threat and lead to attributions of the world as a hostile place, thus leading to reactive aggression. In both cases, CV exposure is hypothesized to increase aggression, but the risk may be especially heightened in children with low HR and increased HRV if these biological processes reflect individual differences in personality that, in turn, affect adaptation to the violent context.

## AIMS OF STUDY

The current study will expand the literature in several ways. First, previous studies on CV exposure typically used high risk samples (e.g., boys from urban settings; Miller

et al., 1999; Schwab-Stone et al., 1999), while non-high risk samples have largely been ignored. Given that CV occurs in all environments, this study focuses on an under-researched rural population. Second, research on CV exposure has mainly concentrated on witnessing violence or overall exposure in which direct (i.e., victimization) and indirect (i.e., hearing about or witnessing) violence are not distinguished (Miller et al., 1999; Schwab-Stone et al., 1999). The present study examines effects of both indirect and direct forms of CV, under the assumption that unique patterns of relationships may be lost if all types of exposure are combined. Third, although studies have examined the main effects of psychophysiological and CV variables on aggression in children (e.g., Hubbard et al., 2002; Scarpa & Haden, 2006), they have not examined their interaction. The primary aim of the current study is to examine a biosocial interaction effect (i.e., CV exposure and HR or HRV) and its relation to childhood aggression. We focus on resting levels of HR and HRV since these values are the best-replicated psychophysiological correlates of aggressive behavior and point to individual differences in personality that can influence adaptation to the environment. Finally, when examining the antisocial outcome of CV exposure, reactive and proactive aggression often has not been differentiated (Miller et al., 1999; Schwab-Stone et al., 1995, 1999). Hence, the current study differentiates between these functions while examining the biosocial interaction. Moreover, since psychiatric disorder has been found to be associated with reactive and proactive aggression, diagnosis will be evaluated as a potential confound.

The primary objective of the study was to determine a biosocial effect for aggression by examining the moderating roles of resting HR and HRV in the relationships between CV exposure (i.e., victimization, witnessed, and heard about) with both reactive and proactive aggression. The primary hypothesis was that resting HR and HRV would moderate the relationship between CV exposure and proactive and reactive aggression. The interaction effect was expected to show that while all forms of CV exposure would be positively related to aggression, the relationships would be strongest under conditions of low resting HR or increased HRV. Because no previous study had incorporated different forms of violence exposure in examining biosocial interactions, no specific predictions were made about differences between victimization, witnessing, or hearing about violence. Nonetheless, it can be argued that direct victimization may have stronger relationships to proactive aggression, and indirect exposure (through hearing about or witnessing violence) may have stronger relationships to reactive aggression. Similarly, because previous studies did not differentiate functions of aggression, no predictions were made about differences between reactive and proactive aggression. However, previous notions of under-arousal, stimulation seeking, and fearlessness (Scarpa & Raine, 1997) would suggest a stronger biosocial interaction with low HR for proactive aggression, while studies of disinhibited and impulsive temperaments (Kagan, 1989; Porges, 2003) would suggest a stronger biosocial interaction with increased HRV for reactive aggression.

## **METHOD**

### ***Participants***

Children and their parents were recruited in a region of Southwestern Virginia. Advertisements were posted in the community schools and university, child medical

offices, and other area businesses working with children. In an effort to obtain a full range of aggression and behavioral problems, recruitment letters were also sent to all parents of children receiving special education services in the local schools. Advertisements and letters recruited children for a study researching social, emotional, and behavioral reactions and directed interested families to call the laboratory. Parents completed a phone screening questionnaire to determine eligibility. Inclusion criteria included a child between 7–13 years old with a parent who knew the child well and could come to the laboratory for testing. Exclusion criteria included a known diagnosis of a Pervasive Developmental Disorder, Mental Retardation, or a cardiac problem that would interfere with the measurement of HR or HRV (e.g., arrhythmia). The final sample included 23 boys and 17 girls (35 White and 5 Black), ranging from 7–13 years ( $M = 9.80$  years,  $SD = 1.81$ ). Complete data with resting HR, resting HRV, and all aggression and CV subscales were available for 38 of the participants which is considered to be a suitable number of participants for detecting significance of a large effect using an alpha level of .05 with four independent variables (Cohen, 1992).

### Measures

*Reactive and Proactive Aggression.* Reactive and proactive aggression was assessed using the Revised Parent Rating Scale for Reactive and Proactive Aggression measure (R-PRPA; Brown, Atkins, Osborne, & Milnanow, 1996). The R-PRPA asks parents to rate the frequency of behaviors reflecting proactive (10 items; e.g., takes things from others, has hurt others to win a game) and reactive aggression (6 items; e.g., gets mad when doesn't get his/her way, blames others) on a scale of 1 = *never* to 3 = *very often*. Items were summed to form continuous measures for each function of aggression.

This measure was adapted from the original teacher form of the same scale, where convergent validity was supported (Brown et al., 1996). In a recent study, our lab also supported validity of this measure, showing that parent and teacher reports both possessed high internal consistency, were significantly related across informants and showed construct validity in relation to aggressive behavior (Ollendick, Jarrett, Wolff, & Scarpa, 2007). Coefficient alpha was .79 and .78 for the items in the proactive and reactive scales, respectively. For our study, 74% of the parents who completed the R-PRPA were mothers and 26% fathers. Scores for reactive aggression ranged from 6 to 16 ( $M = 10.76$ ,  $SD = 2.48$ ), and scores for proactive aggression ranged from 10 to 16 ( $M = 12.19$ ,  $SD = 1.81$ ).

*CV Exposure.* CV exposure was assessed with the Children's Report of Exposure to Violence (CREV; Cooley, Turner, & Beidel, 1995), using scales for having heard about (e.g., "Has anyone ever told you about a stranger being beaten up?"), witnessing (e.g., "Have you seen somebody you know beaten up?"), and being a direct victim of CV (e.g., "Have you ever been beaten up?"). Children indicated how often they perceived being exposed to different violent incidents in their community ranging from 1 (*no, never*) to 4 (*everyday*), which were summed for each form of exposure. Scales for heard about and witnessed CV each included ten items assessing five types of incidents (beaten up, chased/threatened, robbed/mugged, shot/stabbed, and killed) involving either a stranger or someone familiar as the victim (standardized Cronbach alpha = .80 and .64, for heard about and witnessed, respectively). The direct victim scale consisted

of four items (beaten up, chased/threatened, robbed/ mugged, shot). Given the small number and diversity of these items the Cronbach alpha was low at .27.

*Psychophysiological Measurements.* HR and HRV were measured using the VU-AMS ambulatory monitoring system (Vrije Universiteit, Amsterdam) during 4 minutes of rest, after a 10-minute acclimation period. HR was measured in beats per minute (bpm), based on the average of inter-beat intervals (IBI; RR-wave intervals), and reflects both sympathetic and parasympathetic control of the heart. IBIs were continuously measured from a 3-lead electrocardiogram attached to the sternum and ribs, and average IBI (in milliseconds) were calculated over 30-second intervals and then converted to average bpm. The R-R time accuracy using the VU-AMS is one millisecond. The average resting HR for the present sample was 85.66 bpm ( $SD = 10.08$ ) ranging from 64.44 to 108.96 bpm.

HRV was derived by the AMS software using the Root Mean of the Squared Successive Differences (MSSD; Groot, de Geus, de Vries, 1998). The MSSD provides a sample of the short-term variance of the IBIs, which reflects vagal control of the heart and is an index of parasympathetic tone. Larger mean successive differences indicate increased HRV. This measure of HRV has been shown to strongly relate to high frequency power bands in spectral analyses (Friedman, Allen, Christie, & Santucci, 2002), and to cardiac vagal tone as assessed by pharmacological blockade (Hayano et al., 1991), thus providing evidence for its use as an index of vagal activity. Like HR, HRV was calculated and averaged over 30 second intervals during 4 minutes of rest. The average resting HRV was 64.70 ms ( $SD = 43.26$ ) ranging from 9.94 to 202.75 ms.

*Psychiatric Diagnoses.* The Anxiety Disorders Interview Schedule for DSM-IV: Parent Version (ADIS-IV:P; Silverman & Albano, 1996) was used to assess for children's symptoms of DSM-IV (APA, 2000) psychiatric diagnoses. The ADIS-IV:P is a structured diagnostic interview designed to assess for anxiety, mood, and externalizing disorders in youth ages 6 to 17. For this study, we assessed for attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), conduct disorder, anxiety disorders, and major depression or dysthymia. Parents were interviewed by a trained doctoral level student clinician. Diagnoses were determined via symptom count and the impairment rating given by the parent (ranging from 0 to 8, with a rating of 4 or more indicative of a diagnosis). The ADIS-IV has good inter-rater reliability and test-retest reliability (Silverman, Saavedra, & Pina, 2001). Full ADIS-IV:P data were available for 37 of the 38 participants.

### ***Procedure***

Parents and children were assessed in one session, where both completed measures in separate rooms after obtaining informed consent from parents and assent from children together. Parents were interviewed with the ADIS-IV:P and then completed the R-PRPA on their child's aggressive behavior. Meanwhile, the child completed the CREV. Because this was part of a larger study, the child also completed other measures at this time that are not reported here. To allow the children to acclimate to the setting, they were asked to complete some questionnaires and sit quietly for a total of 10 minutes. Electrodes were placed on the child and he/she was asked to sit quietly while resting HR and HRV were measured. Although not analyzed for this article, the children then were asked to complete a computerized continuous performance task followed by a computer game against a fictitious opponent and several additional

questionnaires before leaving the session. The total session lasted approximately one hour and a half. The dyads were compensated \$30 for their time.

### *Analytic Plan*

Descriptive information on demographics and CV exposure are presented first, followed by gender analyses. Gender analyses include t-tests comparing boys and girls on all measures as well as hierarchical regressions to test the potential interaction between gender and CV exposure in predicting aggressive behavior. Effects of psychiatric diagnoses as a possible confound are then tested by t-tests that compare children with and without diagnoses on the variables of interest, and by using diagnosis as a covariate in regression analyses. Correlations among all the variables of interest in this study are presented, including correlations with age and gender. Finally, the primary hypotheses are tested using hierarchical regressions to test for main effects of CV exposure, HR or HRV, and the interactions of HR/HRV with CV exposure. All analyses are repeated for both reactive and proactive aggression as the dependent variables, and the corresponding measure of aggression is used as a control variable. Supplemental analyses repeat the hierarchical regressions with all CV exposure variables entered simultaneously, so as to control for their inter-relationships.

## **RESULTS**

### *Community Violence Exposure Descriptives and Demographic Effects of Gender and Age*

Approximately 10.5% of the sample reported no CV exposure by hearing about it, witnessing it, or being directly victimized, indicating that the vast majority of children had some form of violence exposure. Children endorsed hearing about violence most often ( $M = 4.36$ ,  $SD = 3.69$ , ranging from 0 to 13), followed by witnessing ( $M = 1.11$ ,  $SD = 1.57$ , ranging from 0 to 5), and direct victimization ( $M = .97$ ,  $SD = 1.08$ , ranging from 0 to 4). The most common type of CV exposure involved someone being beaten (17.4–43.4%), regardless of whether the beating was witnessed, heard about, or directly experienced by the child. Hearing about and witnessing violence also were more commonly endorsed than direct victimization and had similar rates for violence that occurred towards a stranger or familiar person. Children also commonly reported that they had heard that a stranger was killed (29%), shot/stabbed (23.2%), or robbed/mugged (23.2%), or had witnessed someone they knew being chased/threatened (25%). Regarding the four types of direct victimization, the children most often reported being beaten (36.2%) and being chased/threatened (30.4%). The least common forms of victimization were being robbed/mugged (5.8%) and shot/stabbed (1.4%).

Effects for participants' gender and age with the three forms of CV exposure (heard about, witnessed, and direct victimization), HR, HRV, as well as proactive and reactive aggression were evaluated. Older participants tended to report hearing more reports of CV ( $r = .36$ ,  $p = .03$ ); age was not significantly associated with any other variable. Significant ( $p < .05$ ) gender differences for each of the CV exposure variables indicated that boys reported hearing about ( $M = 5.32$ ,  $SD = 3.87$ ), witnessing ( $M = 1.55$ ,  $SD = 1.79$ ), and being a direct victim ( $M = 1.27$ ,  $SD = 1.16$ ) of CV more often than girls (hearing about:  $M = 2.46$ ,  $SD = 2.60$ ; witnessing:  $M = 0.38$ ,  $SD = 0.65$ ;

hearing about:  $M = 0.50$ ,  $SD = 0.76$ ),  $t(39) = 5.89$ , 2.44, and 2.61, respectively. Relative to boys, girls received significantly higher scores on proactive aggression (girls:  $M = 13.00$ ,  $SD = 1.96$ ; boys:  $M = 11.70$ ,  $SD = 1.55$ ;  $t(39) = 42.33$ ,  $p = .03$ ) and showed a non-significant trend toward greater HRV (girls:  $M = 81.93$ ,  $SD = 57.59$ ; boys:  $M = 54.33$ ,  $SD = 29.76$ ;  $t(37) = 8.96$ ,  $p = .06$ ). Full descriptive statistics are available from the authors upon request.

In order to test for interactions of gender with CV exposure, hierarchical regressions were conducted, as per guidelines in Aiken and West (1991) and Holmbeck (2002). As recommended, variables were centered and screened for normality, linearity, homogeneity, and multicollinearity. Each type of violence exposure (victimization, hearing, or witnessing) was included in Block one, gender in Block two, and the interaction of gender with each form of CV exposure in Block three. The analyses were repeated to predict each function of aggression (reactive or proactive). The overall models predicting reactive aggression were not significant for direct victimization ( $F(3,37) = .86$ ,  $p = .47$ ), witnessing ( $F(3,37) = .16$ ,  $p = .34$ ), or hearing about CV ( $F(3,37) = .83$ ,  $p = .49$ ). For the prediction of proactive aggression, the overall model that included direct CV victimization was significant ( $F(3,37) = 3.15$ ,  $p = .04$ ) due to the main effect of victimization ( $t(37) = 3.02$ ,  $p = .01$ ), while the models that included witnessing ( $F(3,37) = 2.47$ ,  $p = .08$ ) and hearing about CV ( $F(3,37) = 2.21$ ,  $p = .11$ ) were not significant. Importantly, no significant interactions were found, indicating that the relationships of CV exposure to reactive and proactive aggression did not differ by gender.

### ***Psychiatric Disorder as a Potential Confound***

Regarding psychiatric diagnoses, based on the ADIS-IV:P, 32.4% ( $n = 12$ ) of the sample met criteria for one or more disorder. The total number of diagnoses ranged from 0 to 6 with a mean of .70 ( $SD = 1.29$ ). Out of the 12 participants who met criteria for a diagnosis, most had two diagnoses ( $n = 5$ ), followed by one ( $n = 4$ ), three ( $n = 2$ ), and six diagnoses ( $n = 1$ ). Regarding primary diagnoses, 27% ( $n = 10$ ) met criteria for an externalizing disorder and 21.6% ( $n = 8$ ) for an internalizing disorder. Specifically, for internalizing disorders, eight participants were diagnosed with an anxiety disorder and one child with a mood disorder (major depressive disorder). For externalizing disorders, seven children were diagnosed with ADHD (three inattentive type, one hyperactivity-impulsivity type, three combined) and five children with ODD.

Systematic relationships between psychiatric diagnoses with HR, HRV, proactive and reactive aggression were also evaluated by conducting independent sample t-tests. Although sample sizes were small and unequal for children diagnosed ( $N = 12$ ) and not diagnosed ( $N = 25$ ), Levene's test for equality of variances was not violated. No significant differences were found between those children with and without a diagnosis for HR and HRV, though significant effects were found for proactive and reactive aggression. Children meeting criteria for any disorder were rated as showing increased proactive ( $M = 13.17$ ,  $SD = 2.04$ ) and reactive aggression ( $M = 12.67$ ,  $SD = 2.15$ ) than those with no diagnoses (proactive:  $M = 11.72$ ,  $SD = 2.04$ ,  $t(35) = 2.43$ ,  $p = .02$ ; reactive:  $M = 9.84$ ,  $SD = 2.10$ ,  $t(35) = 3.81$ ,  $p < .01$ ). Children diagnosed with an externalizing disorder (ADHD or ODD) also received higher ratings of proactive ( $M = 13.60$ ,  $SD = 1.90$ ) and reactive aggression ( $M = 12.90$ ,  $SD = 2.29$ ) than those with no externalizing disorder diagnosis (proactive:  $M = 11.67$ ,  $SD = 1.49$ ,  $t(35) = 3.25$ ,  $p < .01$ ; reactive:  $M = 9.96$ ,  $SD = 2.07$ ,

$t(35) = 3.74, p < .01$ ). This was also the case for children diagnosed with internalizing disorders (i.e., anxiety disorder or major depression), whereby they received increased ratings of proactive ( $M = 13.50, SD = 2.33$ ) and reactive aggression ( $M = 13.38, SD = 1.85$ ) than those with no internalizing disorder diagnosis (proactive:  $M = 11.83, SD = 1.49, t(35) = 2.48, p = .02$ ; reactive:  $M = 10.03, SD = 2.13, t(35) = 4.03, p < .01$ ). The fact that diagnosis was not related to HR or HRV suggests that psychiatric diagnosis could not account for the findings. However, because having a diagnosis did relate to increased scores for both reactive and proactive aggression, it was included as a covariate in the regression analyses presented below.

### ***CV Exposure, Resting Psychophysiology, and Aggressive Behavior***

Zero-order correlations between CV exposure (heard about, witnessed, and directly victimized), HR, HRV, and proactive/reactive aggression scores were computed. Reactive and proactive aggression were significantly positively correlated,  $r = .54, p < .001$ . Witnessing CV was significantly positively correlated with hearing about CV,  $r = .45, p = .007$ , and being directly victimized,  $r = .42, p = .01$ , and there was a trend for a positive association between victimization and hearing about CV,  $r = .28, p = .10$ , suggesting that the three forms of CV exposure often cooccur. As expected, HR and HRV were significantly negatively correlated,  $r = -.62, p < .001$ .

Main effects and interactions were tested according to the guidelines in Aiken and West (1991) and Holmbeck (2002), and variables were centered and screened for normality, linearity, homogeneity, and multicollinearity. Six hierarchical regressions were run in order to test for main effects of CV exposure and resting HR and HRV, and their interactions, on the reactive or proactive aggression scores. Separate regressions were conducted for each of the three types of CV exposure and repeated for each of the two functions of aggression. For each of the regressions, Block one controlled for the effect of the other function of aggression in light of their significant inter-correlation, as well as the effect of meeting criteria for a psychiatric diagnosis on the ADIS-IV:P (1 = diagnosis, 0 = no diagnosis). Block two tested for the effects of one of the three types of CV exposure (i.e., witnessed, heard about, or victimization). Block three tested for HR and HRV effects. Last, Block four tested the interaction terms between the CV exposure variable with HR and HRV (i.e., witnessed x HR and HRV, heard about x HR and HRV, victimization x HR and HRV).

Results for proactive aggression can be found in Table 1 and for reactive aggression in Table 2. Because gender was significantly associated with proactive aggression, it was also controlled in Block one in the model predicting proactive aggression. No significant main effects were found for the psychophysiological or CV variables. For proactive aggression, the interaction of victimization and HR was significant: unstandardized  $\beta = -.78$ , partial  $t(35) = -3.41, p = .003$ , and partial  $r = -.60$ . For reactive aggression, the interaction of witnessed CV and HRV was significant: unstandardized  $\beta = .90$ , partial  $t(35) = -2.73, p = .012$ , and partial  $r = .50$ .

As recommended by Aiken and West (1991) and Holmbeck (2002), post-hoc probing was conducted for the significant interaction effects noted above. To characterize the victimization-HR interaction, a regression tested the relationship between CV victimization and proactive aggression using a conditional HR moderator variable (i.e., plus or minus one standard deviation). To characterize the witnessed-HRV interaction, a regression tested the relationship between witnessed CV and reactive aggression using a conditional HRV variable (i.e., plus or minus one SD).

**Table 1. Hierarchical Regressions With Each CV Exposure Term Predicting Proactive Aggression (Controlling for Participant's Gender, Psychiatric Diagnosis, and Reactive Aggression)**

<i>CV Exposure</i>	<i>B</i>	<i>SE B</i>	<i>B</i>
Participant's Gender	.97	.53	.26
Psychiatric diagnosis	.30	.61	.08
Reactive aggression	.34	.09	.49**
Witnessed	.08	.19	.08.
Heard about	.13	.08	-.25
Victimization	.43	.25	.25
HR	.00	.03	.14
HRV	.00	.01	.02
Witnessed X HR	-.01	.04	-.06
Witnessed X HRV	-.01	.01	-.32
Heard about X HR	.00	.01	.05
Heard about X HRV	.00	.01	.33
Victimization X HR	-.15	.05	-.78**
Victimization X HRV	-.03	.02	-.48

Note: Results are combined for ease of presentation, but note that each form of violence exposure (i.e., witnessed, heard about, or victimization) and its respective interaction terms were analyzed in a separate regression. CV = Community Violence, HR = heart rate, HRV = heart rate variability

\*\* $p < .01$ ; \* $p < .05$ .

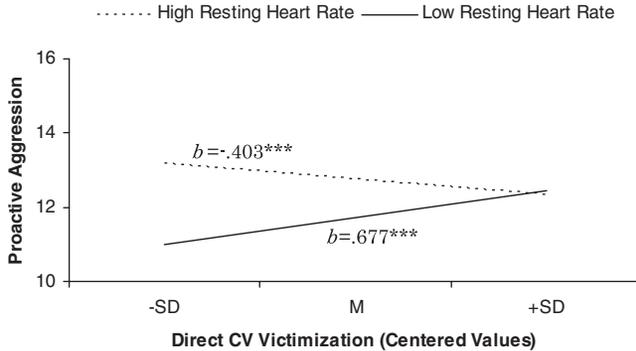
**Table 2. Hierarchical Regressions With Each CV Exposure Term Predicting Reactive Aggression (Controlling for Psychiatric Diagnosis and Proactive Aggression)**

<i>CV Exposure</i>	<i>B</i>	<i>SE B</i>	<i>B</i>
Psychiatric diagnosis	2.05	.76	.39**
Proactive aggression	.70	.12	.52**
Witnessed	-.32	.21	-.19
Heard about	.12	.09	.21
Victimization	-.14	.30	-.04
HR	.02	.05	.05
HRV	-.00	.01	-.03
Witnessed X HR	.07	.06	.38
Witnessed X HRV	.05	.02	.90*
Heard about X HR	-.04	.02	-.49
Heard about X HRV	-.01	.01	-.47
Victimization X HR	-.05	.03	-.30
Victimization X HRV	-.03	.03	-.49

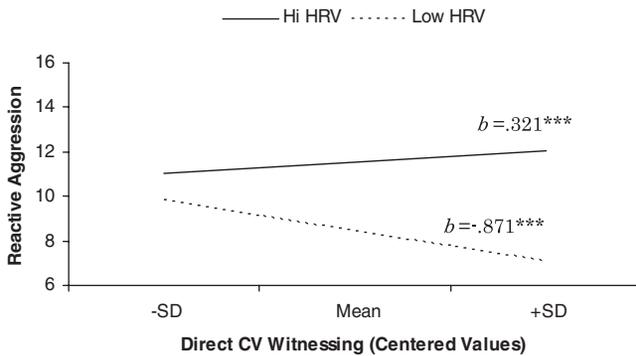
Note: Results are combined for ease of presentation, but note that each form of violence exposure (i.e., witnessed, heard about, or victimization) and its respective interaction terms were analyzed in a separate regression. CV = Community Violence, HR = heart rate, HRV = heart rate variability

\*\* $p < .01$ ; \* $p < .05$ .

Regression lines for CV exposure and aggression were then plotted for high and low resting HR or HRV (see Figure 1 for HR and Figure 2 for HRV). As illustrated in Figure 1, CV victimization and proactive aggression were positively associated when resting HR was low ( $b = .68, p < .001$ ) but negatively associated when resting HR was high ( $b = -.40, p < .001$ ). As illustrated in Figure 2, witnessed CV and reactive aggression were positively associated when resting HRV was high ( $b = .32, p < .001$ ) but negatively associated when resting HRV was low ( $b = -.87, p < .001$ ).



**Figure 1.** Regression lines for relationship between CV Victimization and proactive aggression as moderated by resting heart rate.  $b$  = unstandardized coefficient.  $***p < .001$ .



**Figure 2.** Regression lines for relationship between CV Witnessing and reactive aggression as moderated by resting heart rate variability.  $b$  = unstandardized coefficient.  $***p < .001$ .

### Supplemental Analyses

Additional regression analyses were conducted in which all forms of CV exposure (i.e., witnessed, heard about, or victimization) were entered simultaneously to control for one another. Separate regressions were conducted for proactive and reactive aggression such that Block one controlled for the effect of the other function of aggression and psychiatric diagnosis (and gender was entered in this block for the proactive aggression model), Block two tested for the effects of each type of CV exposure simultaneously, Block three tested for HR and HRV effects, and Block four tested the interaction terms between each of the CV exposure variables with HR and HRV.

The findings were identical to those of the main analyses which separated each form of CV exposure. No significant main effects were found for the CV exposure or psychophysiological variables; however, two interaction terms were significant. The interaction between CV victimization and HR was significant for proactive aggression: unstandardized  $\beta = -.86$ , partial  $t(35) = -4.60$ ,  $p < .01$ , partial  $r = -.54$ , while the interaction of witnessed CV and HRV was significant for reactive aggression: unstandardized  $\beta = .51$ , partial  $t(35) = -3.59$ ,  $p < .01$ , and partial  $r = .51$ .

## DISCUSSION

The primary findings of this study support a biosocial interaction whereby resting HR and HRV moderate the relationship between CV exposure and aggression in children. Specifically, violence victimization was related to increased proactive aggression in children with low levels of resting HR but to decreased proactive aggression in children with high levels of resting HR. On the other hand, witnessed CV was related to increased reactive aggression in children with high levels of resting HRV but to decreased reactive aggression in children with low levels of HRV. These results were found even when the effects of all forms of CV exposure were tested together in an effort to control for one another and occurred above and beyond the effects of gender or psychiatric diagnosis on aggression. Last, significant main effects were not found for CV exposure or psychophysiological functioning, indicating that it is the interaction of these two biological and social variables that is important and not simply an additive effect of risk factors.

The notion of HR underarousal as a risk factor for aggression is consistent with multiple studies that have now found a relationship between low HR and aggression in youth (see Lorber, 2004, and Ortiz & Raine, 2004 for reviews) and in boys from adverse backgrounds (Farrington, 1997). The current findings add to this literature by suggesting that this biosocial effect is particularly heightened under conditions of increased violence victimization and may be specific to proactive aggression. Perhaps high levels of CV provide a learning environment whereby child victims are taught to value aggression as an instrumental tool. Low HR may reflect a fearless or impulsive personality style that increases the child's vulnerability to such aggressive socialization.

HRV was found to moderate the relationship between witnessed CV exposure and reactive, but not proactive, aggression. This finding is interesting in light of the findings of Scarpa & Ollendick (2003) that HRV was related to increased aggression in young adults only if they reported no victimization experiences. In that study, witnessed CV was not assessed, so it is possible that non-victims could have witnessed CV. Our results are consistent with the notion of vagotonia posited by Venables (1988) but seem to apply only to reactive aggression and only in interaction with witnessed CV. Perhaps those who witness violence in their community are more likely to attribute hostile intent to others and react defensively. It is unclear why vagotonia would raise this risk, however, if increased HRV reflects better emotion regulation (e.g., Katz & Gottman, 1995; Porges, et al., 1994). Although speculative, we suggest that high HRV also reflects a disinhibited temperament (Kagan, 1989), which may increase impulsivity and risk-taking in these already vulnerable children. Future studies are needed to explore such explanatory mechanisms, however, in that the current study did not directly assess components of personality.

Though not hypothesized, it is interesting that victimization was *inversely* related to aggression in the condition of high resting HR, and witnessed CV was inversely related to reactive aggression in the condition of low resting HRV. This suggests that high HR and low HRV may actually protect against aggression as CV exposure increases. This unexpected finding may be a result of chance due to the small sample size. Nevertheless, heightened sympathetic and reduced parasympathetic activity may reflect a responsive autonomic nervous system that serves a protective function by aiding individuals to avoid maladaptive and risky behaviors, as suggested by Bechara, Tranel, Damasio, and Damasio (1996). In support of this contention, some studies have found increased autonomic arousal (i.e., both HR and SC) in at-risk males who

desisted from engaging in later crime (Brennan et al., 1997; Raine, Venables, & Williams, 1995), indicating that high arousal can protect some children from a criminal outcome. By the same token, an inspection of Figure 1 indicates that high resting HR in conditions of *low* violence exposure may actually be a risk for increased aggression. Again, we find that autonomic patterns differ according to context. Perhaps in benign environments, low resting HR reflects the typical healthy autonomic nervous system and, in fact, high resting HR would represent risk. These roles may switch at high levels of CV exposure. Such various interpretations need to be tested in future research.

The primary limitations of this study involve the small sample size, use of a community sample, and cross-sectional design. Although, the small sample size reduces power, it was sufficient to capture large effects, which arguably may be of more interest. The finding of significant interactions, despite a small sample, suggests that effects may become larger when subgroups of children are analyzed.

Proactive aggression was greater in girls in our sample, whereas previous research indicates either no gender differences or greater aggression in boys (e.g., Kempes, Matthys, van Goozen, & van Engeland, 2006). Perhaps our sample differed in that others used children from foreign, urban, and high-risk samples. Still, girls showed lower CV exposure scores than boys in our sample (also consistent with prior CV work). Also, despite the gender effect for proactive aggression, we found no significant gender interactions with CV exposure. Finally, the biosocial interactions remained after controlling for gender. For these reasons, it seems that gender was not systematically influencing our results.

The use of a community sample also limits generalizability to children with more serious violence experiences and levels of aggressive behavior. One goal of this study was to assess CV outcomes in a rural low-risk sample, and so this sample is necessarily unique from samples used in prior CV work. The mean exposure scores are lower than those reported in other studies that used the same CV scale in school-based populations with largely minority and urban participants (Cooley et al., 1995; Cooley-Quille, Boyd, Frantz, & Walsh, 2001). As such, readers are cautioned that these results may not apply to nationally representative samples where violence exposure is likely to be more frequent and severe or to samples where the behaviors are more serious. In such cases, the effects may have been stronger and we may have seen the predicted main effects for violence exposure as well as for the psychophysiological measures. Despite the non-clinical nature of this sample, a substantial minority did obtain a clinical diagnosis using a structured diagnostic interview and the effect remained significant after controlling for diagnosis. Moreover, it is disheartening to note the high number of children who reported some history of violence exposure and victimization, in particular. As such, these findings can not be simply accounted for by the low-risk nature of the sample.

The cross-sectional design limits causal inferences that can be made from this study. Because the study is not longitudinal, the direction of effects is not determined. It is possible, for example, that having low resting HR leads one to engage in more proactive aggression, which places the child at higher risk of being victimized. Indeed, it is likely that all these variables transact and lead to a vicious cycle of victimization and aggression.

Finally, this study is limited by its reliance on self-report measures. Such measures may be subject to inaccuracies, including poor recall, lack of knowledge, and social desirability issues. In particular, the CREV was completed by the children and therefore may be inaccurate based on their level of cognitive ability to understand the

questions. To help circumvent this, research assistants were available to assist the children in completing the measures. Also related to measurement issues, parents completed the aggression scale and may have been unaware of some of the aggression their children exhibited in other contexts. Though cross-informant measures and behavioral observations would have been useful, the findings of the current study still seem meaningful in the context of parent-observed aggression and warrant further investigation of aggression in other contexts (e.g., school).

In conclusion, our findings support a biosocial interaction between HR and HRV with violence exposure in the community; this is consistent with transactional models of development which posit that behavior problems arise out of contributions from both intrinsic and extrinsic risk factors that cannot be readily separated from each other (Sameroff, 1995) and may represent individual differences in how children adapt to adverse contexts. These findings converge with biosocial interactions involving many other biological systems including genetics, obstetric/birth complications, hormones, and neurotransmitters (see Raine, 2002b for a review) that have implications for prevention and intervention. They suggest, for example, that treatment to reduce family conflict or create a more stable environment that prevents additional violence exposure could have a protective influence on children with biological deficits. One study found that a stable home environment protected children with fetal alcohol syndrome from developing antisocial behavior (Streissguth, Barr, Kogan, & Bookstein, 1996). Other programs that empower communities to reduce social disorganization, eliminate child maltreatment, or redirect stimulation seeking to more prosocial activities may similarly suppress a biological predisposition toward antisociality. Alternatively, early intervention might benefit children who experience psychosocial hardship, but do not yet show behavioral difficulties, by changing their psychophysiological functioning. A preschool enrichment program similar to Head Start, for example, resulted in significant increases in SC and brain-wave activity and attention 8 years later (Raine et al., 2001). Directly altering biological functioning through interventions like prenatal care, nutrition/health programs, biofeedback, or psychotropic medication may also have utility in this regard.

It is clear that the most effective prevention and intervention and the greatest understanding of antisocial development will include multiple modes of treatment and information from both biological and psychosocial perspectives. The findings from this study should encourage researchers to examine multiple functions of aggression and to consider both biological and social variables in their work.

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