Cardiovascular Reactivity and Proactive and Reactive Relational Aggression among Women With and Without a History of Sexual Abuse

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Abstract

This study examined the association between cardiovascular reactivity and proactive and reactive functions of relational aggression among women with and without a history of sexual abuse. Heart rate reactivity, blood pressure reactivity, and respiratory sinus arrhythmia reactivity while recounting a relational stressor (e.g., being left out) were assessed. Participants provided self-reports of relational aggression and a history of sexual abuse prior to age 16. Results indicated that cardiovascular reactivity was only associated with relational aggression among women with a history of sexual abuse. In addition, whereas blunted reactivity was associated with proactive relational aggression, exaggerated reactivity was associated with reactive relational aggression. These findings highlight the importance of considering contextual moderators of the association between cardiovascular reactivity and aggression; moreover, results highlight distinct cardiovascular correlates of different functions of aggression. Finally, the findings underscore the need for additional research examining the physiological correlates of aggressive behavior among women.

Cardiovascular Reactivity and Proactive and Reactive Relational Aggression among Women With and Without a History of Childhood Sexual Abuse

An emerging body of research has implicated physiological reactivity to stress in the development of aggression and antisocial behavior (Lorber, 2004; Scarpa & Raine, 1997). However, work in this area is limited in three important ways. First, most studies have either failed to include women (see Lorber, 2004, for a review) or have focused on forms of aggression that are more characteristic of men (i.e., physical aggression; Rappaport & Thomas, 2004). Second, few studies have examined the association between stress reactivity and proactive and reactive functions of aggression, despite the suggestion by a number of theorists that these relations may be distinct (Hubbard et al., 2002; Scarpa & Raine, 1997). Finally, although emerging research suggests that stressful contextual experiences may interact with physiological risk in the prediction of aggressive conduct (Scarpa & Raine, 1997), limited work has examined whether stressful experiences, such as a history of sexual abuse, moderates the association between stress reactivity and aggression. The goal of the present study was to address these limitations by investigating the moderating effect of a history of sexual abuse in the association between cardiovascular reactivity to stress and proactive and reactive relational aggression in a sample of adult women.

An important limitation of research examining the relationship between cardiovascular reactivity and aggression is that few studies have investigated this association in women. For example, a meta-analysis of the association between physiological arousal and antisocial behavior reported that most studies excluded women altogether, and those that included both men and women often failed to report findings separately by gender (Lorber, 2004). Compounding this problem, the vast majority of research in this area has focused on physical

forms of aggression. Physical aggression is defined as behaviors intended to hurt or harm others through physical harm (e.g., hitting, kicking, and punching), and is relatively uncommon among women (Crick, Ostrov, & Kawabata, 2007; see Card, Stucky, Sawalani, & Little, 2008 for a recent meta-analysis); thus, women with cardiovascular risk for aggression may be relatively unlikely to engage in these gender-atypical behaviors (Powch and Houston, 1996). Consistent with this perspective, one recent study reported that heart rate reactivity while viewing provocation situations was associated with antisocial behavior, externalizing problems, and reactive aggression among adolescent men but not adolescent women (Crozier et al., 2008). Among women, cardiovascular reactivity may instead predict socially manipulative aggressive behaviors, such as relational aggression, because these behaviors are relatively socially acceptable for females (Powch and Houston, 1996). Relational aggression is defined as behaviors intended to hurt or harm others through the manipulation of interpersonal relationships, and includes behaviors such as giving someone the "silent treatment," spreading malicious rumors, and purposefully excluding someone from an activity (Crick et al., 2007).

Cardiovascular reactivity, reflecting functioning of both the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS), may be involved in the development of relational aggression among women (Murray-Close & Crick, 2007; see Berntson, Quigley, & Lozano, 2007 for a detailed description regarding the role of autonomic nervous system in cardiovascular functioning). Moreover, as changes in the SNS and PNS in response to stressors can reflect coactivation, coinhibition, or uncoupled processes (Berntson, Cacioppo, & Quigley, 1991), it is important to include multiple measures of cardiovascular arousal. Thus, measures utilized in this study include HRR (reflecting both SNS and PNS functioning), systolic and diastolic blood pressure reactivity (SBPR and DBPR, respectively; reflecting both SNS and PNS

functioning), and respiratory sinus arrhythmia reactivity (RSAR, a measure of the ebbing and flowing of heart rate during respiration that is an index of PNS functioning; Beauchaine, 2001; Berntson et al., 2007; Erath, El-Sheikh, & Cummings, 2009; Hubbard et al., 2002).

Two distinct theoretical perspectives regarding the association between physiological reactivity and aggression have been proposed. On the one hand, blunted arousal in response to stress may be associated with aggression because it reflects fearlessness (Ortz & Raine, 2004), which may lead to a lack of concern about the repercussions of aggressive conduct (Kindlon et al., 1995; Raine, 2002) and impair socialization against aggression (Fung et al., 2005). In addition, blunted PNS withdrawal in response to stress may reflect problems with emotion regulation capabilities (Calkins, Graziano, & Keane, 2007), potentially resulting in aggressive conduct. Indeed, some studies have documented an association between aggression and blunted heart rate reactivity (HRR; Hubbard et al., 2002; Kibler, Prosser, & Ma, 2004; Ortiz & Raine, 2004; Schneider, Nicolotti, & Delamater, 2002) and blunted parasympathetic withdrawal (Calkins et al., 2007; Katz, 2007; Kibler et al., 2004; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996).

As preliminary research suggests that women are relatively physiologically reactive to relational stressors (e.g., being the target of social exclusion; Murray-Close & Crick, 2007), an emerging body of research has examined how reactivity to these stressors may place women at risk for relational aggression. In one recent study, blunted HRR and blunted RSA withdrawal in response to a relational stressor were associated with relational aggression in adolescent girls (Sijtsema, Shoulberg, & Murray-Close, 2011). In a study with young adults, blunted HRR and blunted RSA withdrawal predicted relational aggression against romantic partners among women exhibiting low levels of cognitive risk (i.e., low levels of hostile attributions, defined as the belief

that ambiguous provocations were enacted with hostile intent), although the findings for RSA only approached conventional levels of statistical significance (Murray-Close, 2011).

On the other hand, exaggerated, rather than blunted, physiological reactivity may be associated with aggression because it energizes aggressive responding (see Scarpa & Raine, 1997). In fact, researchers have reported a positive association between aggression and hostility and several measures of physiological reactivity, including HRR (Chida & Hamer, 2008; Lorber, 2004; Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998), blood pressure reactivity (Chida & Hamer, 2008), and heightened parasympathetic withdrawal (Beauchaine, 2001). In addition, accumulating evidence indicates that exaggerated cardiovascular reactivity to relational stress is associated with heightened engagement in relational aggression among women. For example, heightened SBPR was positively associated with relational, but not physical, forms of aggression among fifth grade girls (Murray-Close & Crick, 2007). Also, exaggerated HRR predicted relational aggression against romantic partners among women who exhibited additional risk factors for relational aggression (e.g., were frequent targets of relational aggression and exhibited hostile attributions; Murray-Close, 2011). Moreover, heightened RSA withdrawal has been found to predict relational aggression among women who exhibit additional risk factors for relational aggression, such as women with high levels of hostile attributions (Murray-Close, 2011) and women in low-quality romantic relationships (Murray-Close, Holland, & Roisman, in press).

The mixed findings regarding whether exaggerated or blunted stress responses place individuals at risk for aggression may reflect a second important limitation in this area of research. Specifically, to date few studies have empirically examined the distinct physiological profiles associated with proactive versus reactive functions of aggression (Hubbard et al., 2002;

Scarpa & Raine, 1997). Proactive aggression (relatively planful, non-emotional, and goal-directed aggressive behaviors) may be most consistent with the fearlessness theories of aggression and thus may be associated with blunted physiological arousal (e.g., Scarpa, Haden, & Tanaka, 2010; Sijtsema et al., 2011; van Goozen, Fairchild, Snoek, & Harold, 2007). In contrast, reactive aggression (aggressive displays enacted in anger following perceived negative experiences) may be related to increases in physiological arousal following negative experiences or situations (Hubbard et al., 2002; Scarpa & Raine, 1997). Preliminary research found that reactively aggressive children exhibited heightened HRR following a challenging task as compared to children high in both proactive and reactive aggression and non-aggressive controls (Pitts, 1997). In contrast, in a more recent study, reactive aggression was associated with relatively low levels of HRR to a provoking task, perhaps reflecting orienting and interest rather than emotional reactivity (Hubbard et al., 2002). These mixed findings highlight the need for additional research examining the physiological profiles associated with proactive versus reactive functions of aggression. Moreover, to our knowledge, no research has examined the association between physiological reactivity to stress and proactive versus reactive relational aggression.

A third limitation of research in this area is the paucity of studies that focused on contextual risk. Scholars have argued that physiological reactivity may be most strongly associated with aggressive behaviors among individuals who exhibit additional risk factors for aggression, such as child maltreatment, low social class, and rejection by peers (e.g., Raine, 2002; Sijtsema et al., 2011). In fact, several recent studies have demonstrated that patterns of SNS activation and/or PNS withdrawal are most strongly associated with relational aggression among individuals who exhibit additional risk factors for such conduct (e.g., hostile attribution

biases, social stressors such as low-quality relationships; e.g., Murray-Close, 2011; Murray-Close et al., in press; Sijtsema et al., 2011).

One potential contextual factor that may promote engagement in relational aggression and that has received little attention in previous research is a history of sexual abuse. Preliminary research indicates that a history of childhood maltreatment was associated with elevated relational aggression in girls but not boys (Cullerton-Sen et al., 2008). Moreover, when subtypes of abuse were examined, sexual abuse predicted heightened involvement in relational aggression among girls. These authors argue that the violations of trust and intimacy associated with sexual abuse may lead to dysfunctional views about relationships and may promote later perpetration of relational aggression. These findings highlight the possibility that cardiovascular reactivity to stress may only lead to elevated involvement in relational aggression among women who have additional risk factors for relational aggression, such as a history of sexual abuse.

In summary, a review of the extant literature identifies three important limitations in the literature: (1) limited research examining the relationship between cardiovascular reactivity and relational aggression in women; (2) lack of information regarding distinct associations between cardiovascular reactivity and proactive versus reactive functions of relational aggression, and (3) lack of information regarding whether contextual factors such as childhood sexual abuse moderate the association between cardiovascular reactivity and relationally aggressive conduct. Thus, the goal of the present study was to examine whether cardiovascular reactivity (i.e., heart rate, blood pressure, and RSA) to relational stress (e.g., experiences of exclusion) was associated with proactive and reactive functions of relational aggression in a sample of young women with and without a history of childhood sexual abuse. We hypothesized that blunted reactivity would be associated with proactive relational aggression whereas heightened reactivity would be

associated with reactive relational aggression. In addition, we expected that the association between cardiovascular reactivity and aggression would be strongest among women with a history of sexual abuse.

Method

Participants

The data analyzed in this study were drawn from a larger study investigating the association between exposure to childhood sexual abuse and relational/sexual health in adult women; however only the data on relational health is discussed in this study. Participants included 83 women aged 18-30 (*M* age = 22.02 years; *SD* = 2.71) recruited through newspaper advertisements and flyers, posted throughout the urban area of a medium-size town. The advertisement invited women with and without a history of unwanted sexual experiences to call for a confidential screening to assess whether they were eligible for a study on sexual health. We specifically recruited for women with a history of childhood sexual abuse and women with no history of sexual abuse because we wanted to focus on changes caused by early abuse on stress reactivity. Women were eligible if they were fluent in English and between the ages of 25 and 35 years. Because of data on sexual behaviors not relevant to this study, participants were also required to be sexually active with a partner (or partners) during the 4 weeks prior to the study. For this study, childhood sexual abuse was defined as self-reports of any unwanted sexual experience prior to the age of 16 in which the genitals were either touched or penetrated by somebody 5 or more years older (Finkelhor, Hotaling, Lewis, & Smith, 1989). Exclusion criteria for the women who were not abused included reports of any unwanted sexual experience (at any age) and a sexual experience prior to age 16 with someone five or more years older (even if wanted). Exclusion criteria for both groups included self-reported experience of a traumatic

event in the previous three months (i.e., an event that caused fear for one's life or integrity--or of those of a loved one--such as a car accident, witnessing the death of a significant other, a forced sexual experience, etc.), currently being in an abusive relationship (i.e., self report that her partner uses or used physical violence), taking medications known to affect cardiovascular function, and reporting a diagnosis of a psychotic disorder (e.g., schizophrenia, schizoaffective disorder, any delusional disorder and any bipolar disorder that required hospitalization). Women on antidepressants were included in the study as long as they reported being on the same medication and the same dose for 3 months or longer and having experienced no major side effects. Excluding individuals on antidepressants would severely affect the generalizability of the results to women with a history of childhood sexual abuse. Thirty-two of the participants (38.6% of the sample) had a history of sexual abuse, identified from the phone screening interview and an in-person semi-structured interview, the Clinician Administered PTSD Scale (Blake et al., 1990). Participants were asked to report on their race and allowed to check all racial backgrounds that were applicable; approximately 91% of the sample was Caucasian, 5% was Hispanic, 3% was Native American, 3% was Asian, and 1% was African-American.

Procedure

Institutional Review Board approval was obtained prior to the initiation of the present study. Eligible women were invited for a two-day assessment at our laboratory. Data from this study were collected during the second visit. Given some research suggesting that phase of the menstrual cycle is associated with cardiac autonomic regulation among women (McKinley et al., 2009; Sato, Miyake, Akatsu, & Kumashiro, 1995; Vallejo, Márquez, Borja-Aburto, Cárdenas, & Hermosillo, 2005), visits were scheduled to take place on consecutive days during the luteal phase of the participant's menstrual cycle, calculated by asking the woman to report the first day

of her last menstrual cycle. Visits were scheduled between 2 and 4pm. Participants were asked not to engage in biking, running, sexual activities, any sports activity and any activity that would be normally conducted in the gym, for 24 hrs before the study. Also participants were asked to abstain from alcohol and any recreational drug use for 24 hrs before the study. Adherence to this protocol was checked at the beginning of the study, and all participants reported that they had followed these directions. However, 24 participants reported having engaged in some level of exercise during the day (e.g., walking to work). Preliminary regression analyses controlling for amount of exercise (0 = none, 3 = high levels) indicated that the results were virtually identical. Finally, although participants were asked to abstain from smoking, 6 participants reported smoking between 0.5 – 4 cigarettes on the interview day. Preliminary regression analyses controlling for the number of cigarettes smoked indicated that this control did not alter the study findings. Thus, these variables were not included as covariates in the final analyses

On the first visit, a female experimenter collected informed consent and helped the participant complete a sexual psychophysiological assessment that activated sexual arousal responses (data not used for this study). It is important to note that studies have found that Caucasian women participating in studies utilizing this type of sexual assessment did not differ in age, depression, sexual knowledge, fear of negative evaluation and sexual function to women that refused to participate (Woo, Brotto, & Yule, 2010). For measures in the present study, women were asked to complete a series of questionnaires including the Demographics and Self-Report of Aggression and Social Behavior Measure. Then, the participant completed the Social Competence Interview, during which HRR, SBPR, DBPR, and RSAR were collected. Finally, participants completed a semi-structured interview with the second author (AR), to assess history

of traumatic events and corroborate the history of sexual abuse status reported in the phone screening. Each participant was compensated $50 for her time.

Measures

Self-Reports of Proactive and Reactive Relational Aggression

Participants provided self-reports of proactive and reactive functions of relational aggression using the Self-Report of Aggression and Social Behavior Measure (SRASBM; Murray-Close, Ostrov, Nelson, Crick, & Coccaro et al., 2010). This measure includes 5 items assessing proactive relational aggression (e.g., "I have threatened to share private information about my friends with other people in order to get them to comply with my wishes") and 6 items assessing reactive relational aggression (e.g., "When I am not invited to do something with a group of people, I will exclude those people from future activities"). Participants rated each item on a scale from 1 (not at all true) to 7 (very true). Scores for each subscale were calculated by averaging scores across items, with higher scores reflecting higher levels of relational aggression. Previous research has documented distinct factor structures, adequate reliability (αs > .68), moderate 8-month stability (*r*s > .58), and the predictive validity of these subscales in a large sample of adults (Murray-Close et al., 2010). Both proactive (α = .74) and reactive (α = .81) relational aggression were reliable in the present sample.

Social Competence Interview

To assess physiological reactivity to social stress, participants completed the Social Competence Interview (SCI; e.g., Ewart & Kolodner, 1991, 1994; Ewart, Jorgensen, & Kolodner, 1998), a semi-structured 8-12 minute interview developed to assess physiological reactivity to social stress. During the SCI, participants are provided with a stack of cards describing stressful situations with others and are asked to choose the stressor that has caused

them the most stress in recent months. Participants are then asked to describe the stressor in detail, including thoughts and emotions regarding the stressor. Previous research with children and adolescents has documented that the SCI is effective in eliciting a physiological stress response (e.g., Ewart et al., 1998), and that physiological reactivity to the SCI is associated with relational aggression among girls (Murray-Close & Crick, 2007).

The SCI was adapted in two ways for the purposes of the present study. First, given research suggesting that relationally aggressive young adults are especially likely to find relationally-toned stressors provoking (e.g., Bailey & Ostrov, 2008; Murray-Close et al., 2010) and preliminary research with children and adults indicating that physiological reactivity to relational stress is associated with relationally aggressive behavior (Murray-Close, 2011; Murray-Close & Crick, 2007; Sijtsema et al., 2011), the stressor cards for the present study all described relational stressors. In addition, the stressors were adapted to be appropriate for young adults, and included relational stressors involving peers and romantic partners (e.g., "Someone gossips about you behind your back"; "Someone flirts with or seduces your romantic partner or love interest").

Following the procedures developed by Ewart and Kolodner (1991), participants were asked to describe a specific instance when the problem arose (e.g., what happened, where and when it happened, who was present, what the participant said and did, what the others said and did, what happened as a result, etc.). The interviewer helped the participant reconstruct the event using standard probe questions, guided imagery, and reflective listening techniques (Ewart & Kolodner, 1991).

Clinician Administered PTSD Scale to Assess Childhood Sexual Abuse

A trained interviewer administered the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990), a scale that asks about past traumatic experiences and the associated symptoms of posttraumatic stress disorder (PTSD). The first part of the interview requires participants to briefly discuss events that were particularly distressing and threatening to one's life or integrity. For the purpose of this study, participants were asked also to disclose any sexual experience that was with someone at least 5 years older prior to age 16 or that they experienced as forced or coercive. Extra information about the age of the perpetrator, age of the participant at the time(s) of the event(s), and the type of sexual behaviors experienced was asked if this information was not offered spontaneously. This interviewer has shown in previous studies to be able to conduct reliable interviews, evidenced by a high correlations between the results of the CAPS and scores on other self-reported measures of traumatic events, such as the Distressing Events Questionnaire (r = .87) (Kubany, Leisen, Kaplan, & Kelly, 2000).

Physiological Measures

Systolic and diastolic blood pressure were assessed using a Dinamap Vital Signs Monitor (Model 8100, Critikon, Tampa, FL). The interviewer placed the standard occluding cuff on the participant's nondominant arm and BPR was measured at 2-minute intervals. Heart rate and RSA were assessed using an electrocardiogram (ECG; Biopac, Santa Barbarbara©). After cleaning the areas with alcohol, the research assistant placed 3 electrodes on the participant's right collar bone, between the third and the fourth left ribs, and on the inside of her right ankle (grounding lead). Samples were collected at 1000 Hz. In addition to a 50HZ bypass FIR filter, the saved signal was visually inspected for movement artifacts which were deleted at the time of signal reduction. The AcqKnowledge automated software for computation of heart rate variability was utilized. This software utilized three steps. First the RR intervals were extracted for the ECG

signal using a modified Pan-Tompkins QRS detector. Second, the RR intervals were re-sampled to a continuous sampling rate in order to extract frequency information using a cubic-spline interpolation to generate this continuous time-domain representation of the RR intervals. Third, the frequency information was extracted from the RR intervals and analyzed to produce standard ratios. A Welch periodogram was used to generate the Power Spectral Density (equivalent to Transform > Power Spectral Density). RSA was assessed as the spectral power at the high frequency band typically connected with respiration in adults (0.15-.40 Hz).

Physiological level was assessed during a 6-minute resting baseline prior to the SCI and then during the SCI. The baseline followed a 3-minute resting period so that participants could become comfortable with the physiological measures. During the baseline, participants were asked to sit quietly and relax. Physiological reactivity to the SCI was computed by subtracting mean level during baseline from level during the SCI. Thus, negative scores reflect a decrease in the measure in response to stress whereas positive scores reflect an increase in the measures in response to stress. Positive values for HRR, SBPR, and DBPR reflect increases in physiological arousal in response to stress. In contrast, positive values for RSAR reflect relatively blunted withdrawal (i.e., a failure to exhibit decreases in RSA during stress). Due to equipment malfunction or experimenter error, HRR were missing for 6 participants, RSAR scores were missing for 7 participants, and SBPR and DPBR scores were missing for 5 participants.

Results

Descriptives

Inspection of study variables indicated that proactive relational aggression and reactive relational aggression were positively skewed. As a result, these variables were log transformed, leading to a substantial reduction in variable skew. In addition, two outliers for RSAR and one

outlier for SBPR were identified and replaced with the next closest value. Following the removal of outliers, physiological predictor variables exhibited relatively normal distributions (skew ranged from -.65-.66) and were well below thresholds indicating substantial deviation from normality (Kline, 2005). All findings below are reported for the transformed variables (for proactive and reactive aggression) and variables where outliers (for RSAR and SBPR) were removed.

Correlations among study variables are presented in Table 1. Consistent with previous research, proactive and reactive functions of relational aggression were highly correlated (Murray-Close et al., 2010). In addition, HRR was positively associated with SBPR and DBPR, and negatively correlated with RSAR (although this only approached conventional levels of statistical significance). SBPR and DBPR were also positively correlated. Experience of sexual abuse was not correlated with proactive or reactive relational aggression or with physiological reactivity to stress.

Physiological Reactivity to the Stress Interview

A set of repeated-measures analysis of variance (ANOVA) analyses were conducted separately by physiological index to examine whether participants exhibited changes in physiological activity from baseline to the Social Competence Interview and to examine whether a history of sexual abuse moderated this change. Findings indicated that, on average, participants did not exhibit change in RSA from baseline (M = .26, SD = .12) to the interview (M = .27, SD = .06), $F(1, 74) = .24$, $p = .62$, suggesting that some participants exhibited RSA augmentation whereas others exhibited RSA withdrawal in response to the stress interview. However, participants did exhibit an increase in heart rate from baseline (M = 72.83, SD = 10.67) to the interview (M = 75.87, SD = 10.19), $F(1, 75) = 36.32$, $p < .001$, partial $\eta^2 = .33$. In addition,

participants exhibited increases in systolic blood pressure from baseline (M = 112.74, SD = 9.29) to the stress interview (M = 119.56, SD = 12.16), $F(1, 76) = 55.84$, $p < .001$, partial $\eta^2 = .42$. Finally, participants exhibited increases in diastolic blood pressure from baseline (M = 61.41, SD = 8.78) to the interview (M = 69.56, SD = 8.89), $F(1, 76) = 163.54$, $p < .001$, partial $\eta^2 = .68$. Reactivity to the SCI was not moderated by sexual abuse history. Taken together, these findings suggest that the Social Competence Interview elicited increases in cardiovascular activity but was not related to average changes in PNS functioning in both women with and without a history of sexual abuse.

Cardiovascular Reactivity and Proactive and Reactive Relational Aggression

A series of hierarchical regression analyses were conducted to examine whether cardiovascular reactivity to relational stress (i.e., RSAR, HRR, SBPR, and DBPR, respectively) was associated with proactive and reactive relational aggression. In addition, we investigated whether a history of sexual abuse moderated these relations. In each regression, the nonfocal function of relational aggression was entered at Step 1 as a covariate (e.g., we controlled for proactive relational aggression when predicting reactive relational aggression). Physiological reactivity and history of sexual abuse (0 = no abuse, 1 = history of abuse) were entered at Step 2, and the interaction between these predictors was entered at Step 3. Continuous predictors were mean-centered prior to analyses. Preliminary analyses controlled controlling for body mass index (BMI) indicated that the results were virtually identical to those without this covariate; thus BMI was not included in the final analyses.

The results for the RSAR analyses (see Table 2, Model 1) indicated that RSAR interacted with sexual abuse in the prediction of proactive relational aggression. Follow-up simple slope analyses (Aiken & West, 1991) indicated that RSAR was positively associated with proactive

relational aggression among women with a history of sexual abuse, $t(69) = 2.16$, $p < .05$, but not among women without a history of sexual abuse, $t(69) = -.71$, $p = .48$ (see Figure 1). In other words, women with a history of sexual abuse who exhibited blunted RSA withdrawal to relational stress exhibited heightened levels of proactive relational aggression. In contrast, RSAR was not associated with reactive relational aggression.

Results indicated that SBPR was not associated with proactive or reactive relational aggression (see Table 2, Model 2). However, the interaction between DBPR and sexual abuse history in the prediction of proactive relational aggression was in the expected direction and approached conventional levels of statistical significance, $p = .09$ (see Table 2, Model 3). Follow-up simple slope analyses indicated that blunted DBPR was associated with proactive relational aggression among women with a history of sexual abuse, $t(71) = -2.05$, $p < .05$, but not among women who had not been abused, $t(71) = .33$, $p = .74$ (see Figure 2). In addition, DBPR and sexual abuse history interacted in the prediction of reactive relational aggression; follow-up simple slope analyses indicated that heightened DBPR was associated with reactive relational aggression among women with a history of sexual abuse, $t(71) = 1.98$, $p = .05$, but not among women without a history of sexual abuse, $t(71) = -1.00$, $p = .32$ (see Figure 3).

Finally, HRR interacted with sexual abuse in the prediction of proactive but not reactive relational aggression (see Table 2, Model 4). Simple slope analyses indicated that blunted HRR was associated with proactive relational aggression among women who had been sexually abused, $t(70) = -2.90$, $p < .01$, but not among women without a history of sexual abuse, $t(70) = .33$, $p = .74$ (see Figure 4).

Follow-up analyses were run to examine whether symptoms of PTSD, which were positively associated with a history of sexual abuse ($r = .35$, $p < .01$), served as a moderator of

the association between cardiovascular reactivity and proactive and reactive relational aggression. We re-ran all regression analyses with PTSD symptoms, rather than history of sexual abuse, as our moderator variable. Findings (not shown) indicated that PTSD symptoms were not associated with proactive or reactive relational aggression. In addition, PTSD symptoms did not interact with the cardiovascular reactivity in the prediction of aggressive conduct. Although the interaction between PTSD symptoms and heart rate reactivity in the prediction of reactive relational aggression approached conventional levels of statistical significance (p = .09), follow-up simple slope analyses were not significant.

Discussion

The purpose of the present study was to examine the association between cardiovascular reactivity to a relational stressor and proactive and reactive functions of relational aggression in a sample of women with and without a history of childhood sexual abuse. Consistent with emerging theory and research (Hubbard et al., 2002; Scarpa & Raine, 1997; van Goozen et al., 2007), we hypothesized that blunted cardiovascular reactivity would be associated with proactive functions of relational aggression whereas exaggerated reactivity would be associated with reactive functions of relational aggression. Moreover, given evidence indicating that physiological reactivity may be most strongly associated with relationally aggressive conduct among individuals who exhibit additional risk factors for such conduct (e.g., Murray-Close, 2011; Sijtsema et al., 2011), we expected that cardiovascular reactivity to relational stress would be most strongly associated with relational aggression among women with a history of sexual abuse. Consistent with hypotheses, blunted cardiovascular reactivity (i.e., blunted RSA withdrawal, DBPR, and HRR) was associated with proactive relational aggression among women with a history of sexual abuse (although the interaction between DBRP and sexual abuse

in the prediction of proactive relational aggression only approached conventional levels of statistical significance). In contrast, women with a history of sexual abuse who displayed exaggerated DBPR exhibited elevated levels of reactive relational aggression.

Despite the proposal by a number of theorists that proactive aggression is associated with underarousal whereas reactive aggression is associated with exaggerated arousal following stressors (e.g., Hubbard et al., 2002), limited research to date has examined this hypothesis empirically, and, to our knowledge, no research has examined these relations with respect to relational forms of aggression. Nonetheless, evidence indicates that distinct functions of relational aggression emerge early in childhood (Murray-Close & Ostrov, 2009) and have distinct correlates in childhood and adolescence (Little, Jones, Henrich, & Hawley, 2003) as well as adulthood (Bailey & Ostrov, 2008; Murray-Close et al., 2010). Our findings provide some of the first evidence that distinct physiological profiles may place women at risk for proactive versus reactive functions of relational aggression.

In addition, despite an important body of research documenting an association between physical abuse and physically aggressive conduct (e.g., Teisl & Cicchetti, 2008), limited research to date has examined the association between childhood abuse and relational aggression in adulthood (although see Murray-Close and colleagues, 2010). Consistent with previous research (Cullerton-Sen et al., 2008), our findings highlight the possibility that experiences of sexual abuse may serve as an important risk factor for the development of relationally aggressive behaviors. However, it is important to note that in the present study, a history of sexual abuse alone did not place women at risk for relational aggression, as the zero-order correlations between a history of abuse and aggression were not significant. Instead, a history of sexual abuse interacted with cardiovascular reactivity to relational stress to predict elevated levels of relational

aggression. These findings suggest that many women with a history of sexual abuse do not engage in relational aggression as adults. Rather, women who exhibit atypical responses to relational stress and also have a history of sexual abuse appear to be most at risk for such conduct. These findings underscore the importance of considering potential risk factors that may place women with a history of sexual abuse at risk for aggressive conduct. For example, cognitive and emotional processes that have been implicated in aggressive behavior in maltreated youth, such as emotion dysregulation and impaired social cognitive processes (Teisl & Cicchetti, 2008), may also interact with experiences of abuse and cardiovascular reactivity to predict aggression. Of note, symptoms of PTSD did not moderate the association between cardiovascular reactivity and aggression, suggesting that other processes related to a history of sexual abuse may be most relevant to these findings. Results also contribute to a growing body of research suggesting that physiological reactivity may only translate into aggression among individuals who exhibit additional risk factors for such conduct (e.g., Raine, 2002; Sijtsema et al., 2011).

Interestingly, cardiovascular reactivity to relational stressors was more strongly associated with proactive than reactive functions of relational aggression among women with a history of sexual abuse. In fact, several indices of blunted cardiovascular reactivity (i.e., blunted RSAR withdrawal, blunted DBPR and HRR) predicted proactive relational aggression whereas only one measure of exaggerated cardiovascular reactivity (i.e., DBRP) predicted reactive relational aggression among women with a history of sexual abuse. This finding is surprising given evidence that a history of abuse is often found to more strongly predict reactive, rather than proactive, functions of aggression (e.g., Dodge, Lochman, Harnish, Bates, & Pettit, 1997; Ford, Fraleigh, & Connor, 2010) and that emotional lability may partially explain the association

between a history of abuse and reactive aggression (Shields & Cicchetti, 1998). Moreover, although most previous research in this area tends to focus on physical abuse and/or physically aggressive behavior (e.g., Dodge et al., 1997), one recent study did find that a history of maltreatment (including sexual abuse) was more strongly associated with reactive than proactive relational aggression (Murray-Close et al., 2010). However, very limited research to date has examined the association between sexual abuse and relational aggression in women, and Cullerton-Sen and colleagues (2008) argue that sexual abuse may be associated with relational aggression because it violates relational trust and intimacy with close others. It is possible that a history of sexual abuse, such as having a relative who exploited his or her relationship with the participant for personal sexual gratification, leads to maladaptive views regarding how relationships can be manipulated for personal gain. Given this possibility, future research should attend to how distinct subtypes of abuse relate to both forms and functions of aggressive conduct.

An important implication of the findings from this study is that different measures of cardiovascular reactivity may show distinct associations with relational aggression. For example, RSAR, HRR, and DBPR were associated with proactive relational aggression among women with a history of sexual abuse whereas SBPR was not. In addition, only DBPR predicted reactive relational aggression among women with a history of sexual abuse. The one previous study to assess the association between blood pressure reactivity and relational aggression reported that exaggerated SBPR, but not DBPR, predicted relational aggression in 5th grade girls (Murray-Close & Crick, 2007). These findings highlight the possibility that distinct psychophysiological processes may be involved in relational aggression among a normative sample of girls versus women with a history of sexual abuse. Moreover, although all four indices of cardiovascular reactivity assessed in the present study reflect functioning of the autonomic nervous system, each

index reflects distinct physiological processes (e.g., RSAR reflects PNS functioning whereas HRR, SBPR, and DBPR reflect both SNS and PNS functioning), and our findings are consistent with several meta-analyses indicating that different measures of cardiac activity are often differentially associated with aggression and antisocial behavior (Kibler et al., 2004; Lorber, 2004).

Although the findings from the present study provide some of the first evidence indicating that distinct cardiovascular profiles are associated with proactive versus reactive relational aggression among women with a history of sexual abuse, a number of limitations must be acknowledged. First, we focused on sexual abuse given research suggesting that this subtype of abuse may be more strongly associated with relational aggression among females than other forms of maltreatment (Cullerton-Sen et al., 2008); however, as subtypes of abuse often co-occur (Manly, Kim, Rogosch, & Cicchetti, 2001; Teisl & Cicchetti, 2008), it is likely that many of the maltreated women in the present study had also experienced other subtypes of childhood maltreatment. Future research would benefit from examining whether the association between cardiovascular reactivity to relational stress and relational aggression is moderated by other forms of maltreatment, such as physical or emotional abuse. In addition, in the present study, sexual abuse was measured via retrospective reports, which may be limited by problems such as recall errors and false reporting (DeLillo et al., 2006). Future research would benefit from prospective designs and/or cases of documented experiences of sexual abuse (e.g., records from Child Protective Services).

Second, only females were recruited in the present study given evidence that the associations between relational aggression and both cardiovascular reactivity (Murray-Close & Crick, 2007) and history of abuse (Cullerton-Sen et al., 2008) differ for men versus women. As a

number of previous studies have examined the association between cardiovascular reactivity and aggression among men (Kibler et al., 2004; Lorber, 2004), the purpose of the present investigation was to investigate whether physiological processes may be associated with forms of aggression relatively typical among women. However, as some previous research suggests that cardiovascular reactivity may be more strongly associated with physical aggression for men and relational aggression for women (Murray-Close & Crick, 2007), future research would benefit from examining whether gender moderates the association between physiological reactivity to stress and proactive and reactive forms of physical and relational aggression.

Third, the number of participants in the present study was relatively small (N = 83), perhaps resulting in underpowered tests of interaction effects. In fact, follow-up power analyses using G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009) indicated that the power to detect an effect from a predictor in multiple regression based on parameters similar to those in our regression analyses (i.e., 4 predictors, .025 variance in the outcome explained by the predictor of interest, and .40 residual variance in the outcome) with a sample size of 83 is .61 (although see Hoenig & Heisey, 2001, for further discussion on the controversy and limitations surrounding post-hoc power analysis). Given research suggesting the interaction effects tend to be difficult to detect statistically in nonexperimental research designs (McClelland & Judd, 1993), these findings suggest that our analyses may have been underpowered and suggest that future research in this area should include larger sample sizes. However, the emergence of several significant interactions despite the relatively small sample size suggests that our findings may be relatively robust.

Future research would also benefit from inclusion of additional indices of physiological reactivity, including measures of SNS activity such as preejection period and skin conductance,

so that the distinct associations between SNS and PNS functioning and relational aggression can be assessed. In fact, findings from one recent study highlight the possibility that SNS activity may be more strongly associated with relational aggression against romantic partners in women whereas PNS activity may be more strongly associated with such conduct among men (Murray-Close et al., in press). In addition, including measures of the processes that influence SBPR and DBPR, such as cardiac output and vascular resistance (Berntson et al., 2007; Ewart & Kolodner, 1993) may highlight potential physiological mechanisms that link SBPR and DBPR with relational aggression. Such approaches would also provide important insights regarding the autonomic regulatory processes involved in these associations; for example, evidence of vascular resistance would highlight the role of sympathetic alpha-adrenergic receptors whereas cardiac output would highlight beta-adrenergic receptors (Berntson et al., 2007). Evidence of sympathetic versus parasympathetic influence on cardiovascular reactivity would also implicate distinct neurotransmitters, such as acetylcholine (a primary neurotransmitter for postganglionic parasympathetic fibers) and norepinephrine (a primary neurotransmitter for postganglionic sympathetic fibers) (Berntson et al., 2007). In addition, skin conductance reactivity would highlight the role of sympathetic cholinergic processes (Berntson et al., 2007). Thus, future research would benefit from examination of the underlying processes that may give rise to distinct cardiovascular responses to stress among aggressive versus nonaggressive women.

It would also be useful to examine whether the neurobiological models that have been developed to examine physically aggressive behaviors are relevant to the emergence of relational aggression in females. For example, Davidson, Putnam and Larson (2000) have argued that impulsive (i.e., reactive) aggression results in part from a failure to inhibit negative emotional reactions, reflecting dysfunctions in the inhibitory connections between the prefrontal cortex and

the amygdala. In contrast, proactive may reflect amygdala dysfunction, impairing the processes of aversive conditioning and instrumental learning involved in moral socialization and conscience development (e.g., Blair, 2004; Frick & Morris, 2004). Berntson and Cacioppo (2007) argue that a primary mechanism by which psychosocial stressors result in increases in heart rate and blood pressure is via activation of subcortical structures such as the amygdala and the resulting inhibition of the baroreflex response. It is possible that the association between heightened DBP reactivity and reactive relational aggression reflects dysfunction of neural emotion regulation circuitry. In contrast, the association between blunted cardiovascular reactivity and proactive relational aggression may reflect amygdala dysfunction when encountering stressors, resulting in impaired moral socialization. Although almost no research has examined the neural correlates of relational aggression, emerging evidence suggests that experiences of being the target of relational aggression are associated with activation of amygdala and prefrontal areas associated with emotional distress and emotion regulation, respectively (Sebastian et al., 2011). These findings, combined with the results from the present study, highlight the importance of considering the neural processes and concomitant cardiovascular responses that may be involved in proactive and reactive relational aggression, and suggest that applying models that have to date focused on physical forms of aggression to more relational forms of such conduct may serve as a good starting point in this new area of research.

Finally, future research may benefit from assessing physiological reactivity to several different types of stressors. Obradović and colleagues (2011) recently demonstrated that the interactions between marital conflict and RSAR in the prediction of externalizing problems among kindergarten students differed depending on whether RSAR was measured in response to

a cognitive versus interpersonal challenge. In the present study, we assessed cardiovascular reactivity to a relational stressor given research suggesting that women tend to find these types of stressor particularly distressing (e.g., Crick, Grotpeter, & Bigbee, 2002; Murray-Close & Crick, 2007) and that cognitive and emotional sensitivity to these stressors tend to be associated with relational forms of aggression (e.g., Bailey & Ostrov, 2008; Crick, Grotpeter, & Bigbee, 2002). However, future research would benefit from investigating whether the associations between cardiovascular reactivity and relational aggression differ depending on the type of stressor used.

In conclusion, the findings from the present study suggest that cardiovascular reactivity to relational stressors is associated with heightened involvement in relational aggression among women with a history of childhood sexual abuse. The results provide some of the first evidence supporting the theoretical claim that distinct patterns of physiological reactivity are associated with proactive versus reactive functions of aggression (Scarpa & Raine, 1997), and, to our knowledge, provide the first test of this hypothesis with respect to relational forms of aggression. Our results have important implications for clinicians working with women who have a history of sexual abuse. Specifically, women with early maltreating experiences may be at risk for engaging in relationally aggressive conduct if they also exhibit dysregulated responses to stress. Blunted versus exaggerated physiological reactivity to stress may have important implications for treatment; for example, women who exhibited exaggerated stress responses may benefit most from anger management training (see Murray-Close, 2011). In contrast, treatment for women with blunted physiological reactivity to stress may benefit from an emphasis on the negative outcomes associated with aggressive behavior, as previous research has documented that cognitive biases regarding outcome expectancies are associated with proactive functions of relational aggression in adolescent girls (Marsee & Frick, 2007). Finally, the findings also

highlight the importance of additional research examining the psychophysiological correlates of non-physical forms of aggressive and aggressive behaviors perpetrated by females.

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Table 1. *Correlations among study variables.*

	1.	2.	3.	4.	5.	6.
1. ProRel	1.00					
2. ReRel	.77***	1.00				
3. RSAR	.13	.09	1.00			
4. HRR	-.14	-.09	-.21†	1.00		
5. SBPR	-.01	-.05	.14	.29*	1.00	
6. DBPR	-.08	-.05	-.17	.32**	.26*	1.00
7. Abuse	.04	.01	-.02	.07	.13	-.08
Mean	1.39	1.90	.02	3.04	7.39	8.15
SD	.60	.89	.10	4.30	5.15	5.43

†$p < .10$ *$p < .05$ **$p < .01$ ***$p < .001$

Note. ProRel = proactive relational aggression; ReRel = reactive relational aggression; RSAR = Respiratory sinus arrhythmia reactivity; SBPR = systolic blood pressure reactivity, DBPR = diastolic blood pressure reactivity; HRR = Heart rate reactivity; Abuse = experience of sexual abuse (0= no, 1 = yes).

Table 2. *Regression Analyses of Physiological Reactivity,*

			Dependent Variable					
Model	Step		*Proactive Relational Aggression*			*Reactive Relational Aggression*		
			β	t-value	ΔR^2	β	t-value	ΔR^2
1	1	Nonfocal Aggression	.77***	10.33	.59***	.77***	10.33	.59***
	2	RSAR	.06	.83	.01	-.01	-.15	.00
		Sexual Abuse (SA)	.03	.45		-.03	-.43	
	3	RSAR X SA	.20*	2.09	.02*	-.13	-1.31	.01
2	1	Nonfocal Aggression	.76***	10.12	.57***	.76***	10.12	.57***
	2	SBPR	.03	.40	.00	-.05	-.60	.00
		Sexual Abuse (SA)	.04	.52		-.03	-.36	
	3	SBPR X SA	-.11	-1.16	.01	.01	.11	.00
3	1	Nonfocal Aggression	.76***	10.12	.57***	.76***	10.12	.57***
	2	DBPR	-.04	-.56	.00	.01	.15	.00
		Sexual Abuse (SA)	.04	.50		-.03	-.41	
	3	DBPR X SA	-.16†	-1.73	.02†	.20*	2.15	.03*
4	1	Nonfocal Aggression	.78***	10.64	.60***	.78***	10.64	.60***
	2	HRR	-.07	-.93	.01	.02	.22	.00
		Sexual Abuse (SA)	.03	.39		-.03	-.44	
	3	HRR X SA	-.19*	-2.13	.02*	.13	1.39	.01

†$p < .10$, *$p < .05$, **$p < .01$ ***$p < .001$

Note. RSAR = Respiratory sinus arrhythmia reactivity; SBPR = systolic blood pressure reactivity, DBPR = diastolic blood pressure reactivity; HRR = Heart rate reactivity; Sexual abuse, 0 = No abuse, 1 = Abuse. Nonfocal aggression = proactive relational aggression when reactive relational aggression is the dependent variable and reactive relational aggression when proactive relational aggression is the dependent variable.

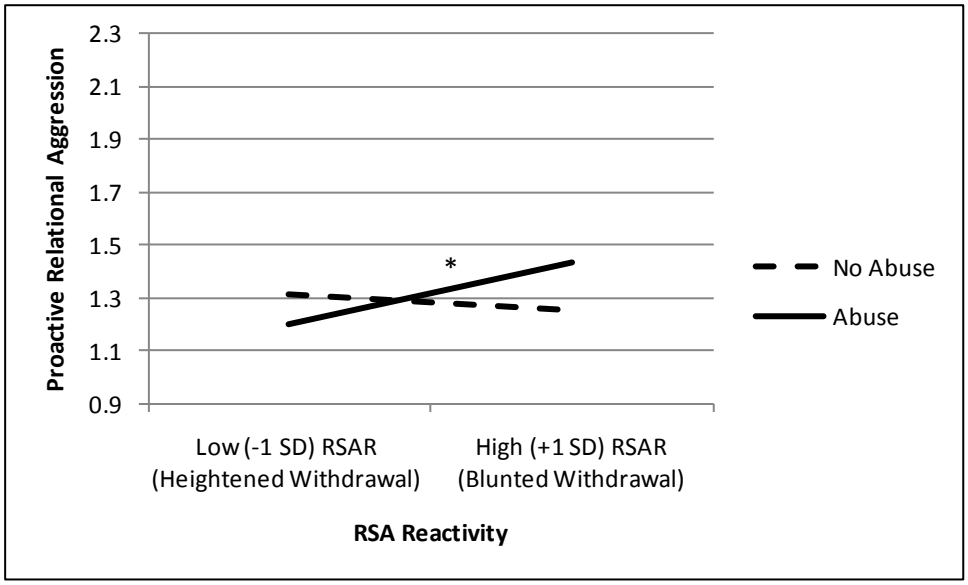
Figure Captions.

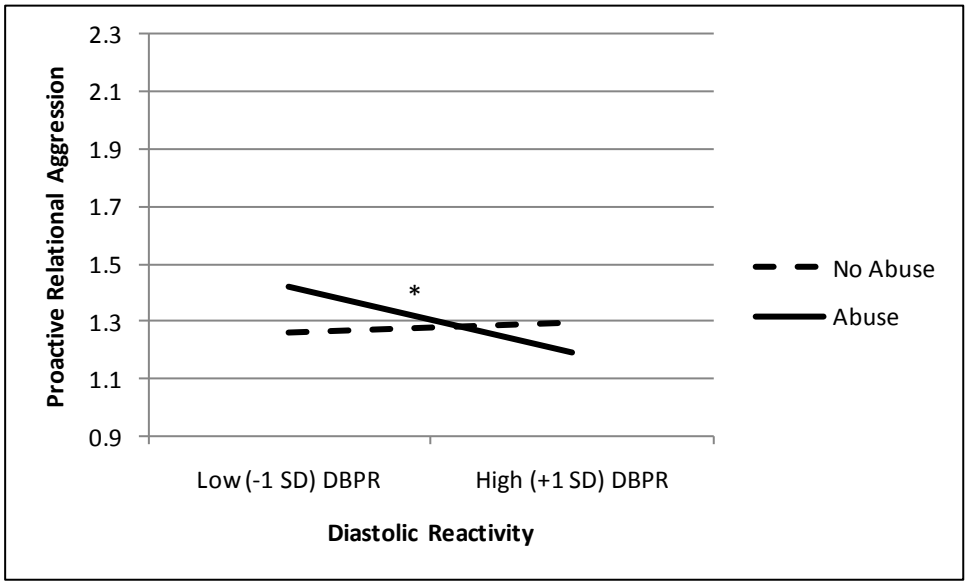
Figure 1. Interaction between RSAR and History of Sexual Abuse Predicting Proactive Relational Aggression.

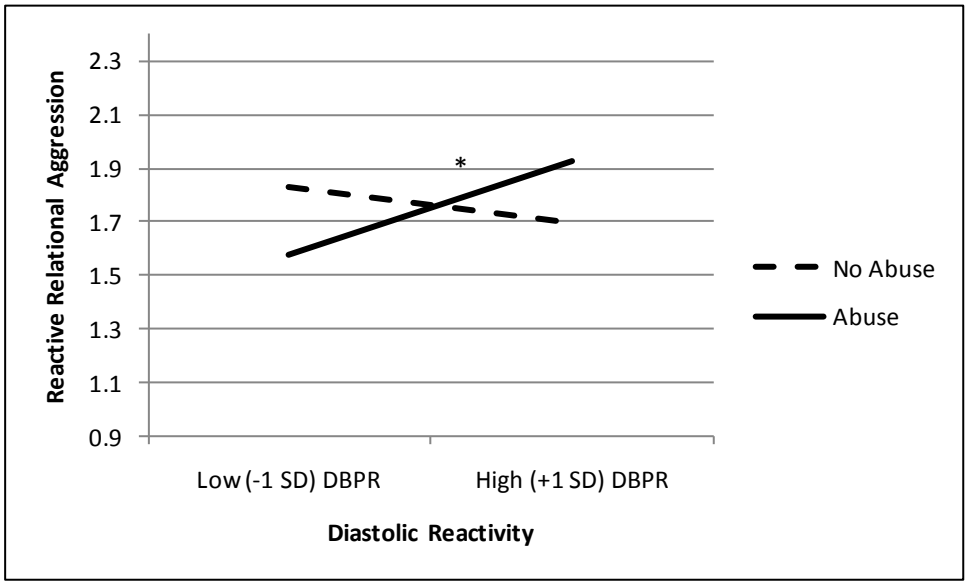
Figure 2. Interaction between DBPR and History of Sexual Abuse Predicting Proactive Relational Aggression.

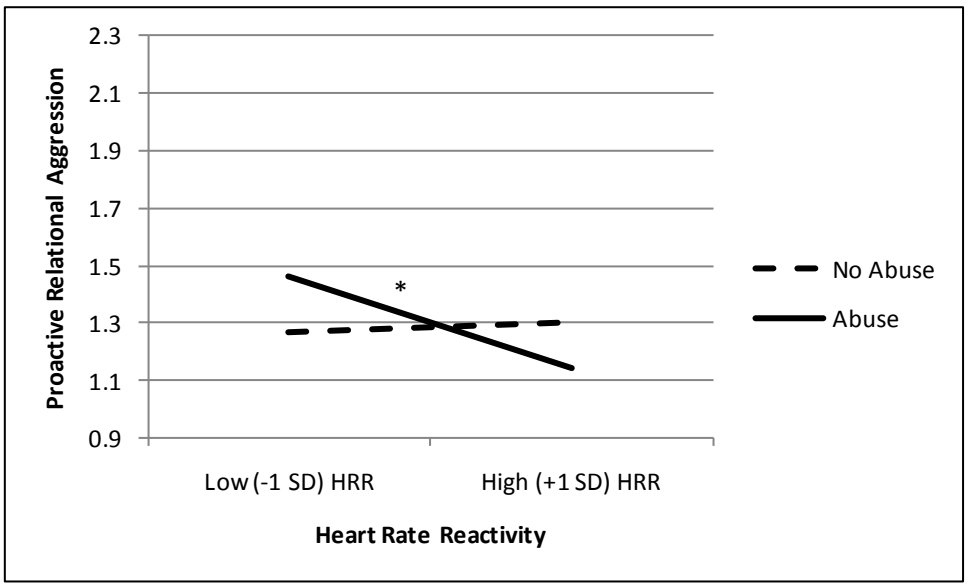
Figure 3. Interaction between DBPR and History of Sexual Abuse Predicting Reactive Relational Aggression.

Figure 4. Interaction between HRR and History of Sexual Abuse Predicting Proactive Relational Aggression.


Proactive Relational Aggression
2.3
2.1
1.9
1.7
1.5
1.3
1.1
0.9
*
No Abuse
Abuse
Low (-1 SD) RSAR
(Heightened Withdrawal)
High (+1 SD) RSAR
(Blunted Withdrawal)
RSA Reactivity


2.3
2.1
1.9
1.7
1.5
1.3
1.1
0.9
Proactive Relational Aggression
*
No Abuse
Abuse
Low (-1 SD) DBPR
High (+1 SD) DBPR
Diastolic Reactivity


Reactive Relational Aggression
2.3
2.1
1.9
1.7
1.5
1.3
1.1
0.9
*
No Abuse
Abuse
Low (-1 SD) DBPR
High (+1 SD) DBPR
Diastolic Reactivity


2.3
2.1
1.9
1.7
1.5
1.3
1.1
0.9
Proactive Relational Aggression
*
No Abuse
Abuse
Low (-1 SD) HRR
High (+1 SD) HRR
Heart Rate Reactivity