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Biological Sensitivity to Context in Couples: Why Partner Aggression Hurts Some More Than Others

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Abstract

Objective—Cardiovascular reactivity to laboratory stressors was investigated as (a) a moderator of associations of partner aggression with affective functioning, alcohol problems, and parenting, and (b) a consequence of partner aggression.

Method—Cohabiting adult couples ($N = 453$) with 3- to 7-year-old children were recruited by random digit dialing and completed questionnaires assessing couple physical aggression, discipline practices, anger, stress, depressive symptoms, and problem alcohol use. Heart rate and blood pressure were measured at rest and in response to laboratory stressors (mental arithmetic and video presentations of family conflict).

Results—Males' physical aggression more strongly predicted women's affective functioning and alcohol problems when the women had greater cardiovascular reactivity. This pattern did not extend to men. Physical victimization was associated with lower cardiovascular reactivity.

Conclusions—The present results provide partial support for the Biological Sensitivity to Context model of Boyce and Ellis (2005) and suggest that incorporating the moderating influence of biological reactivity may improve the precision of models of the effects of aggression on adult adjustment.

Keywords

Aggression; couples; cardiovascular reactivity; context sensitivity

Why is it that some people in noxious intimate relationships appear exceptionally “hard hit” while others seem relatively “unfazed?” It is well known that being the recipient of a partner’s psychological and physical aggression is particularly toxic for adult functioning. For example, there are links between being the victim of partner aggression and alcohol use, poor physical health, depression, post-traumatic stress, and poor parenting (e.g., Campbell, 2002; Cascardi & K.D. O’Leary, 1993; Cascardi, K. D. O’Leary, & Schlee, 1999; Coker et al., 2002; Vaeth, Ramisetty-Mikler, & Caetano, 2010). Despite compelling main effects, however, there is great variability in how people are affected by aggressive relationships. Behind the prototypical correlation of .30 between aggression and outcome lie many observations that fall substantially off the best fit regression line. Some members of highly

aggressive couples show greatly compromised functioning, whereas others do not. Advances in developmental theory offer as of yet untested hypotheses to explain why.

It has long been recognized that not all individuals respond the same way to environmental factors. Recent years have seen progress toward discerning what factors make some individuals more readily influenced by their environments, with factors such as negative emotionality (e.g. Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007), genotype (e.g., Rutter, Moffitt, & Caspi, 2006), and biological stress reactivity (Boyce & Ellis, 2005) at the forefront of this wave of research (Belsky & Pluess, 2009). In some models – referred to here as *vulnerability models* – people are viewed as having specific inborn risks for pathological behavior that are expressed in certain environmental conditions (e.g., Rutter, 2006). Such models bear resemblance to diathesis-stress models (e.g., Monroe & Simons, 1991).

From another group of theoretical perspectives – previously referred to as either sensitivity or susceptibility models and more recently conceptualized as the comprehensive *differential susceptibility* model – some people are thought to have qualities that make them more susceptible to environmental influences (e.g., Belsky & Pluess, 2009; Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2011). Risky environments are more destructive for these people than for other individuals. Likewise, supportive environments are more positive forces for them. Although the focal moderating characteristics have been conceptualized as phenotypic and of genetic origin (e.g., temperament; Belsky & Pluess, 2009) or endophenotypic and of dual genetic-environmental origin (e.g., biological reactivity; Boyce & Ellis, 2005), the rapprochement of Ellis et al. (2011) suggests that “whatever the level of analysis employed in a given study, *neurobiological susceptibility to the environment* is the fundamental construct of interest” (p. 13). The hypotheses offered herein are influenced by this differential susceptibility to the environment perspective.

Biological Reactivity As a Moderator of Environmental Influence

Boyce and Ellis (2005) theorized that high levels of biological stress reactivity mark “biological sensitivity to context” (BSC). BSC, reflected primarily in increased activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, is thought to strengthen the impact of environmental factors on a range of behavioral and physical health outcomes. Environments, be they positive or negative, matter more for individuals higher in biological reactivity. In contrast to “main effect” models of biological reactivity and psychopathology (e.g., Beauchaine, 2001; Lorber, 2004), biological reactivity is not itself considered to be inherently pathogenetic. In adverse circumstances, individuals with high biological reactivity are expected to show especially compromised functioning. However highly biologically reactive/context sensitive individuals are also theorized to disproportionately benefit from supportive environments. Emerging evidence from research on children supports BSC theory. For example, Boyce, Chesney, Alkon, and Tschann (1995) found that children with greater cardiovascular reactivity to laboratory stressors had the lowest incidences of respiratory illness in favorable environments, but more respiratory illness in high stress environments. Boyce, Essex, Alkon, Goldsmith, Kraemer, and Kupfer

(2006) further found that high cortisol reactivity strengthened the association of father involvement and child behavior problems. In a third report by the same group, Obradovi , Bush, Stamperdahl, Adler, and Boyce (2010) found that those children with greater cortisol reactivity to stress, relative to their lower cortisol reactive peers, showed the greatest effects of family adversity. In highly adverse environments, high cortisol reactivity was associated with suboptimal adaptation; in less adverse environments, children with high cortisol reactivity exhibited superior adaptation.

The findings of El Sheikh and colleagues are also partially consistent with BSC theory (El Sheikh, 2005; El-Sheikh, Keller, & Erath, 2007). She and her colleagues found stronger associations between marital conflict and behavior problems among girls with greater electrodermal reactivity to an inter-adult argument; findings were mixed for boys. However, whereas biological reactivity is not viewed by BSC theory to be a vulnerability factor for psychopathology, El Sheikh's findings suggest that elevated electrodermal reactivity is associated with child behavior problems. Moreover, high levels of electrodermal reactivity did not confer adaptive advantage to children from low conflict homes, as BSC theory would predict.

Compared to the rapidly developing literature on BSC in children, much less is known about how the model may apply to adults. Two studies, however, are relevant. In the first, Gannon, Banks, Shelton, and Luchetta (1989) measured a broad range of physiological measures in college students who were exposed to a laboratory stressor. Daily hassles and depressive symptoms were more strongly associated among students who exhibited greater increases in blood pressure and respiration, compared to their lower-reactivity counterparts. Cardiovascular and respiratory reactivity similarly moderated the association of daily hassles and physical symptoms. Important to BSC theory, the interactions took the predicted "cross-over" shape, indicating that high biological reactors had the highest levels of depressive and physical symptoms in the most adverse environments but the best adjustment given less stressful circumstances. In the second BSC-relevant study of adults, Clements and Turpin (2000) found that life events were more strongly associated with global psychological symptoms among college students who had greater heart rate reactions to laboratory stimuli. However, it is unclear to what extent the stimuli were stressful (repeated tones participants were instructed to ignore and a challenging cognitive task from an intelligence test). Accordingly, the relevance of these results to BSC theory is somewhat ambiguous. Taken together, however, these two results suggest the possibility that BSC theory is a useful model to explain variation in adult functioning.

Biological Reactivity as a Consequence of Environment

In BSC theory, biological reactivity is thought not only to moderate environmental effects but also to be subject to them, as the result of an evolutionary process. Context sensitivity – hence biological reactivity – is viewed as an adaption favored by natural selection that allows a given genotype to repeatedly adjust the phenotype to prevailing environmental conditions that influence reproductive fitness over the course of its evolutionary history. Context sensitivity in a given individual allows her/him to adjust to the present environment by enhancing cognitive and behavioral processes that are adaptive in helping the person

respond effectively to both supports and threats in the environment (Flinn, 2006). In adverse environments, high biological reactivity may support adaptive increased threat vigilance and fight/flight behavior. High biological reactivity may be similarly adaptive in people's full engagement of promotive environments. More moderate environments, neither particularly stressful nor supportive, are thought, on average, to offer no adaptive advantage to biological reactivity. Accordingly, the association between environmental stress/support and biological reactivity is thought to be U-shaped, with high levels of biological reactivity found in both highly stressful *and* supportive environments, with intermediate levels of reactivity found in intermediate environments.

Evidence in support of the U-shaped biological reactivity-environment association has been slower to accumulate than evidence on the moderating impact of biological reactivity. Ellis, Essex, and Boyce (2005) found that the hypothesized U-shaped biological reactivity-environment association was evident in analyses of a sample of children with a wide range of family stress. Children living in families marked by both high and low levels of family stress showed greater heart rate reactivity to social, cognitive, physical, and emotional challenges in the laboratory. Intermediate family stress was associated with low levels of heart rate reactivity. Boyce is unaware of any further attempts to evaluate such a curvilinear association (personal communication, June 19, 2010). Clearly, the U-shaped environment-biological reactivity association specified in the BSC model is in need of further evaluation.

The Present Investigation

We aimed to evaluate the usefulness of the BSC model in explaining variation in how people are affected by their intimate partners' aggressive behavior. We theorized that adults vary in their susceptibility to the effects of their intimate partners' behavior and that this susceptibility is marked by cardiovascular reactivity. We additionally postulated that cardiovascular reactivity is likely enhanced in both the least or most aggressive couple environments. Flinn (2006) argues that the family is a primary source of stressful (and supportive) events in a child's world, helping calibrate biological reactivity. We argue that it remains so for adult members of couples. Couple environments provide a major context for adult development and couple functioning is a powerful predictor of adult adaptation.

Based on BSC theory, it was hypothesized that associations between victimization by one's partner and multiple aspects of adult adjustment (affective functioning, alcohol problems, and parenting) would be strengthened with higher levels of cardiovascular reactivity to laboratory stressors (Hypothesis 1). A U-shaped relation between aggression victimization and cardiovascular reactivity, with higher levels of reactivity found in recipients of both the most and least partner aggression, was also hypothesized (Hypothesis 2).

Method

Participants

Four hundred fifty-three couples in the vicinity of Stony Brook University, in the New York City suburbs, were recruited through a random digit dialing procedure over a three year period (1999-2002). Eligible participants needed to be living as a couple for at least one

year, parent a three to seven year-old who was the biological child of at least one member of the couple, and be able to complete questionnaires in English. Comparison to the 2000 U.S. Census indicated the random digit dialing produced a sample fairly representative of the county's population. Please see Table 1.

Procedure

The study couples completed two, 3-hour sessions or one 6-hour session, for which they were paid \$250. The couples first gave informed consent for an IRB-approved protocol and were then separated to first fill out questionnaires and then complete additional tasks. Only questionnaires and tasks relevant to the present investigation are described herein. Interested readers are referred to O'Leary, Slep, and O'Leary (2007) for additional details.

Questionnaires—Partners completed questionnaires on several aspects of affective functioning (e.g., depressive symptoms), family dynamics (e.g., partner aggression), and personal characteristics (e.g., alcohol problems) in separate rooms. All data were collected in an anonymous manner (i.e., randomly coded subject identifier not linked to respondent identity; questionnaires completed in different rooms).

Physiological baseline—To establish resting measures of the cardiovascular system, participants were individually seated in a private room where they rested for a period of eight minutes. The first four minutes were an adaptation period; physiological recording occurred in the last four minutes.

Mental arithmetic—Upon completion of the baseline physiological measurement period, participants were individually asked to perform a 3-min mental arithmetic task consisting of vocal serial subtractions by steps of three from a four-digit number (2,357) as quickly and accurately as possible. Participant's performance was monitored via intercom by the experimenter in the adjacent room. Mental arithmetic is a standard stressful task, known to produce increases in cardiovascular measures, and commonly used in psychophysiological research (e.g., Tomaka, Blascovich, Kelsey, & Leitten, 1993).

Family conflict video tasks—Upon completion of the mental arithmetic task, each participant, sitting in a private room, viewed several professionally acted scenes depicting family conflicts on a large color monitor.. The scenes included four couple conflict vignettes, and a similar block of four parent-child conflicts. The video order (i.e., parent-child or partner scenes first) was counterbalanced. Participants were asked to "put yourself in the position of the (wife/husband/mother/father) while you watch the scene. Even if you wouldn't handle things exactly the way (she/he) is, I want you to imagine that you are anyway."

The four couple conflict videos were each approximately 90 seconds in length and shown in counterbalanced order. Two scenes were of an African-American couple, and two were of a Caucasian couple. These interactions began with a disagreement that escalated over time, with both partners becoming obviously upset. The scenes ended with the conflicts unresolved. All scenes contained verbal, but not physical, aggression. Two of the

disagreements were initiated by the female partner and two by the male partner, counterbalanced by the race of the actors.

The four parent-child conflict videos were also each approximately 90 s in length and shown in counterbalanced order. Participants were shown two scenes of parent-son (age 4.5 years) and two scenes of parent-daughter (age 7 years) conflict. Parent-son scenes involved African-American actors; parent-daughter scenes involved Caucasian actors. Males viewed scenes of fathers and children, and females viewed identically scripted scenes of mothers and children. Each scene began with a request by the parent to the child, which was then ignored or disobeyed by the child. The conflicts escalated over time, with the parent raising his/her voice and repeating himself/herself, ending with no resolution. The parents threatened, but did not carry out, punishment and were clearly angry.

Measures

Affective Functioning—Affective functioning was estimated as a latent composite of self-reported anger expression, depressive symptoms, and perceived stress. Descriptions of each of the indicators follow.

Anger: The Anger Expression Index from the widely used State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988) was computed by adding the means of the Anger Expression-In and Anger Expression-Out subscales and subtracting the mean of the Anger Control items; 24 items rated on a four point scale (1 = *almost never* to 4 = *almost always*). The Cronbach's α for anger was .85 for males and .80 for females.

Depressive symptomatology. (Beck Depression Inventory-II; BDI-II; Beck, Steer, & Brown, 1996): The BDI-II is a widely used and validated measure of depressive symptoms. It contains 21 items that are measured on a four point Likert-scale and summed to obtain the level of symptoms ($\alpha = .87$ males; $\alpha = .91$ for females).

Perceived stress: The Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) is made up of 14 items rated on a five-point scale (from 1 = *never* to 5 = *very often*), which were summed ($\alpha = .85$ for both males and females). The PSS is a widely-used measure of stress (Parks et al., 2000). The PSS has been found to strongly correlate with the Perceived Stress Questionnaire (Levenstein et al., 1993), a similar measure (Cohen et al., 1983).

Latent composite: Women's and men's respective standardized loadings from a measurement model with all constructs simultaneously modeled (reported below) were .51 and .57 for the BDI, .58 and .63 for the PSS, and .70 and .76 for the STAXI, $ps < .001$. The residuals for the BDI and STAXI scores were allowed to covary, $\beta_s = .54$ and .36, $ps < .001$. For men, the standardized loadings were .57, .63, and .76

Overreactive discipline—Parents gave self-reports of their discipline over the past two months on the Parenting Scale (PS; Arnold, O'Leary, Wolff, & Acker, 1993). The PS is a 30-item self-report scale of dysfunctional discipline with acceptable reliability. It is also associated with home observations of discipline and toddlers' behavior problems, and with parent reported child problem behavior (Arnold et al., 1993). Scores based on the item

average of the 10-item Overreactivity (e.g., “I get so frustrated or angry that my child can see that I’m upset”) was used for analysis ($\alpha = .68$ for both males and females).

Alcohol problems—Participants answered questions about their alcohol use and abuse on the 10-item Alcohol Use Disorders Identification Test (AUDIT; Allen, Litten, Fertig, & Babor, 1997), a measure assessing alcohol dependence. The AUDIT is a 10-item self-report measure that was created by the World Health Organization (WHO) through the collaborative efforts of six countries. It has well established sensitivity and specificity (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). The AUDIT has demonstrated moderate validity when correlated with other self-report measures and distal predictors of significant alcohol use (Allen et al., 1997).

Partner aggression—Partner aggression was estimated as a latent composite of partner-to-self psychological and physical aggression, as reported by both the perpetrator and victim. Descriptions of each of the four indicators follow.

Couple aggression was measured with the Conflict Tactics Scale-II (Straus, Hamby, Boney-McCoy, & Sugarman, 1996), the most widely adopted measure of intimate partner violence and with good reliability and construct validity. This is a 78-item questionnaire assessing partner conflict during the last year through frequencies (scales ranging from 0 = never to 6 = *more than 20 times*). Aggression frequencies on the 8 psychological aggression items (e.g., “called partner fat or ugly”) and the 12 physical aggression items (e.g., “pushed or shoved”) were, respectively, averaged to calculate psychological and physical aggression scores. Both psychological and physical aggression scores were calculated separately for perpetration (reports of aggressing against one’s partner) and victimization (reports of one’s partner’s aggression).

Women’s and men’s respective standardized loadings from a measurement model with all constructs simultaneously modeled (reported below) were .68 and .58 for the partner’s reports of psychological aggression perpetration, .65 and .37 for the partner’s reports of physical aggression perpetration, .91 and .94 for self-reports of psychological aggression victimization, and .57 and .62 for self-reports of physical aggression victimization, $ps < .001$. For women, the residuals for self-reported psychological aggression victimization and partner-reported physical aggression perpetration were allowed to covary, $\beta = -.75$, $p < .001$. For men, the residuals for partner-reported psychological and physical aggression perpetration ($\beta = .40$, $p < .001$), and for self-reported victimization and partner-reported perpetration of physical aggression ($\beta = .30$, $p < .001$), were allowed to covary.

Cardiovascular Reactivity—Mean reactivity scores across tasks were separately calculated for systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart period (HP). These mean reactivity scores were then used as the three indicators of a latent cardiovascular reactivity construct (see Figure 1). Descriptions of each indicator follow. Electrocardiogram (ECG; sampling rate = 1000Hz) was continuously recorded during baseline, mental arithmetic, and each family conflict video task with Coulbourn LabLinc V series equipment (V75-11 isolated ECG amplifier). Blood pressure was measured with a cuff of appropriate size placed around the nonpreferred arm using an

automated oscillometric device (Dinamap model, Johnson & Johnson). Systolic and diastolic blood pressure (SBP and DBP, respectively) was recorded once per minute during baseline and task periods. All signals were recorded by an IBM-compatible computer.

Heart period reactivity: Mean heart interbeat interval or heart period (HP), the time between R-waves in the ECG (and the inverse of heart rate), was calculated separately for mental arithmetic, partner video, and parent-child video tasks. HP reactivity scores for mental arithmetic were calculated by subtracting the 4-min baseline mean from the mean across the entire 3-min task. HP reactivity scores were calculated for each of the video vignettes by subtracting the baseline mean from the mean across the last 60 s of each vignette. These individual reactivity scores were subsequently averaged to yield partner video and parent child video HP reactivity measures. The last 60 s of each vignette was chosen for the computation of reactivity scores because conflict was the most intense during these sections.

Blood pressure reactivity: SBP and DBP reactivity scores were also formed by subtracting baseline means from task means. Mental arithmetic blood pressure reactivity scores were calculated by subtracting the mean across the 4 baseline measurements from the mean of the 3 measurements made during mental arithmetic. Partner video blood pressure reactivity scores were the average of the 4 measurements of blood pressure made during the partner videos (one assessment per video) minus the baseline average. Parent-child video blood pressure reactivity scores were the average of the 4 measurements of blood pressure made during the parent-child videos (one assessment per video) minus the baseline average.

Latent composite: To form a latent cardiovascular reactivity variable that jointly took into account SBP, DBP, and HP, each of the above reactivity scores was first standardized and then averaged across tasks within measure. To illustrate, SBP reactivity scores for mental arithmetic, partner videos, and parent-child videos were each standardized, then averaged to form a summary SBP reactivity score. The same procedure was used to produce summary DBP and HP reactivity scores. Women's and men's respective standardized loadings from a measurement model with all constructs simultaneously modeled (reported below) were .89 and .99 for SBP, .72 and .60 for DBP, and $-.50$ and $-.35$ for HP, $ps < .001$.

Measurement model fit—Measurement models were separately estimated for women and men. All latent variables (affective functioning, aggression, cardiovascular reactivity) were simultaneously estimated with the indicators described in detail above by construct and allowed to freely covary amongst themselves and with the manifest outcome variables (overreactive discipline, alcohol problems). Modification indices suggested the need to allow a few correlated residuals within construct. The fit of women's and men's respective measurement models was acceptable, χ^2 (44 and 43) = 57.49 and 58.82 ($ps = .08$ and $.05$), CFIs = .99, TLIs = .98, RMSEAs = .03.

Results

Descriptive data for each study measure are presented in Table 2. Correlations among the variables evaluated in the latent regression models are reported in Table 3.

Missing Data and Nonnormality

The rate of missing data was 1.82%. Cases with missing data were included in the estimated models using Maximum Likelihood (ML) estimation, using all available cases for each analysis. This was done in order to avoid the known biases of listwise deletion (Schafer & Graham, 2002). The robust maximum likelihood (MLR) estimator was selected as several variables were positively skewed (e.g., couple aggression, depressive symptoms).

Analytic Strategy

Latent variable interactions, using the Latent Moderated Structural Equations (LMS) approach of Klein and Moosbrugger (2000), were estimated to test interaction using Mplus software (Muthén & Muthén, 2004). Although tested in a structural equation modeling framework, latent variable interactions follow the familiar format of testing interactions in ordinary multiple regression. The criterion is regressed on two or more predictors (main effects) and a product term representing their interaction. A significantly nonzero product term indicates the presence of an interaction. Results from all LMS models are reported in Table 4.

The most commonly used fit indices (e.g., chi-square) are not available. Thus, overall model fit could not be evaluated in the customary manner and individual path coefficients were interpreted in a manner usually associated with ordinary correlation and regression. Further, standardized path coefficients are not available for LMS models.

Significant interactions were followed-up with additional graphical and statistical analysis, following Preacher, Curran, and Bower (2006) and using the computational utilities at the following Internet site: <http://www.people.ku.edu/~preacher/interact/mlr2.htm>. Outcomes were plotted at +1, 0, and -1 SDs on the moderators (cardiovascular reactivity) and aggression variables. Additionally, simple slopes representing aggression victimization-outcome associations at each level of the cardiovascular reactivity moderators were calculated.

Findings for Women

Affective functioning interaction tests—Women's affective functioning (latent composite of depression, anger, and perceived stress scores) was regressed on male aggression (latent composite of male and female reports of his physical and psychological aggression; $p < .001$), women's cardiovascular reactivity (latent composite of SBP, DBP, and HP reactivity over mental arithmetic, partner conflict, and parent-child conflict videos; $p = .294$), and a latent Male Aggression $\times$ Cardiovascular Reactivity interaction term ($p = .004$). Consistent with Hypothesis 1, follow-up analyses indicated that male aggression became a stronger predictor of women's affective functioning as women's cardiovascular reactivity increased (Figure 1, Panel A). Simple slopes for the aggression-affective functioning association were significant at -1 SD (slope = 3.35, $t = 3.29$, $p = .001$), average (slope = 5.37, $t = 5.03$, $p < .001$), and +1 SD (slope = 7.39, $t = 4.97$, $p < .001$) on cardiovascular reactivity.

Further follow-up analyses were conducted, which suggested that at average and ± 1 SD on female cardiovascular reactivity and ± 1 SD on male aggression, the confidence intervals (CIs) for the expected values of women's affective functioning overlapped. In other words, mean affective functioning did not differ among women of high ($+1$ SD), medium (*mean*), and low (-1 SD) cardiovascular reactivity, at high ($+1$ SD), medium, and low (-1 SD) levels of male aggression. Replotting the interaction at the mean and ± 1.5 SD on female cardiovascular reactivity and male aggression however revealed clearly nonoverlapping CIs among high, medium, and low levels female cardiovascular reactivity at high, medium, and low levels of male aggression. In other words, greater female cardiovascular reactors had both disproportionately poor affective functioning in the context of high male aggression and disproportionately good affective functioning in the context of low male aggression, when slightly more extreme values of each were plotted.

Alcohol problems interaction tests—Women's alcohol problems (manifest AUDIT scores) were regressed on the latent male aggression ($p = .006$), female cardiovascular reactivity ($p = .625$), and male aggression $\times$ female cardiovascular reactivity ($p = .09$) factors described above. Consistent with Hypothesis 1, the association of male aggression and women's alcohol problems was stronger among women with higher levels of cardiovascular reactivity (Figure 1, Panel B). The simple slope was nonsignificant at -1 SD (slope = 0.24, $t = 0.96$, $p = .339$) cardiovascular reactivity and significant at average (slope = 0.60, $t = 2.72$, $p = .007$) and $+1$ SD (slope = .96, $t = 2.74$, $p = .006$) on cardiovascular reactivity.

However, this interaction was only marginally significant. We subsequently explored the possibility that the interaction could depend on which cardiovascular reactivity measure was modeled. The same analytic methods as those above were employed, with the exception that each cardiovascular reactivity measure was modeled separately with indicators from each task. To illustrate, SBP reactivity was modeled as a latent variable with manifest SBP reactivity scores from mental arithmetic, partner, and parent-child videos. Latent variables for DBP and HP were estimated by the same method. Interaction tests were repeated for each cardiovascular reactivity measure. A significant interactions was found for SBP ($p = .049$), with marginal interaction for DBP ($p = .061$) and a nonsignificant interaction for HP ($p = .146$). Each interaction followed the same general pattern of a strengthening male aggression-female alcohol problems relation at higher levels of female cardiovascular reactivity.

Further follow-up analyses were conducted, which suggested that at average and ± 1 SD on female cardiovascular reactivity and ± 1 SD on male aggression, the confidence intervals (CIs) for the expected values of women's alcohol problems overlapped. In other words, mean alcohol problems did not differ among women of high ($+1$ SD), medium (*M*), and low (-1 SD) cardiovascular reactivity, at high ($+1$ SD), medium (*M*), and low (-1 SD) levels of male aggression. Replotting the interaction at the mean and ± 1.75 SD on female cardiovascular reactivity and male aggression however revealed clearly nonoverlapping CIs among high, medium, and low levels female cardiovascular reactivity at high, medium, and low levels of male aggression. In other words, greater female cardiovascular reactors had both disproportionately high levels of problem drinking in the context of high male

aggression and disproportionately low levels of problem drinking in the context of low male aggression, when more extreme values of each were plotted.

Overreactive discipline interaction tests—Women's overreactive discipline practices (manifest variable) were regressed on the latent male aggression ($p = .013$), female cardiovascular reactivity ($p = .712$), and male aggression $\times$ female cardiovascular reactivity ($p = .536$) factors described above. In contrast to Hypothesis 1, cardiovascular reactivity did not significantly moderate the male aggression-female discipline association.

Controlling for possible confounds—The above moderation analyses were reevaluated controlling for mother and child age, child sex, and family income, as preliminary analyses suggested these were correlated with at least one of the outcome variables. The inclusion of these covariates did not significantly affect the results of the moderation tests reported above (analytic detail available from the authors).

Associations between male aggression and female cardiovascular reactivity—Women's cardiovascular reactivity was regressed on male aggression (Table 5, Step 1). Male aggression was associated with reduced female reactivity ($p < .001$). To determine the possibility of curvilinear associations of male aggression with female autonomic reactivity (Hypothesis 2), cardiovascular reactivity was next simultaneously regressed on male aggression and male aggression squared (quadratic effect; estimated via a generalization of the LMS method; Step 2). The quadratic term was nonsignificant ($p = .712$), indicating that association of male aggression and female cardiovascular reactivity was linear.

Findings for Men

All analyses involving men are directly parallel to those for women, and will therefore be described in less detail.

Affective functioning interaction tests (Table 3)—Men's affective functioning was regressed on female aggression ($p < .001$), men's cardiovascular reactivity ($p = .088$), and female aggression $\times$ cardiovascular reactivity ($p = .652$). The interaction was nonsignificant, contrary to Hypothesis 1.

Alcohol problems interaction tests—Men's affective functioning was regressed on female aggression ($p = .001$), men's cardiovascular reactivity ($p = .803$), and female aggression $\times$ cardiovascular reactivity ($p < .419$). Contrary to Hypothesis 1, the interaction was nonsignificant.

Overreactive discipline interaction tests—Men's overreactive discipline practices were regressed on female aggression ($p < .001$), female cardiovascular reactivity ($p = .764$), and female aggression $\times$ female cardiovascular reactivity ($p = .785$). Contrary to Hypothesis 1, the interaction was nonsignificant.

Controlling for possible confounds—The above moderation analyses were reevaluated controlling for father and child age, child sex, male occupational prestige and family income, as preliminary analyses suggested these were correlated with at least one of

the outcome variables. The inclusion of these covariates did not significantly affect the results of the moderation tests reported above (analytic detail available from the authors).

Associations between female aggression and male cardiovascular reactivity—

Female aggression was associated with reduced male reactivity ($p < .001$; Table 5, Step 1). In contrast to Hypothesis 2, there was no evidence of curvilinearity in this relation ($p = .125$; Table 5, Step 2).

Discussion

Cardiovascular Reactivity as a Moderator of the Effects of Partner Aggression

Partial support was found for the hypothesis based on BSC theory (Boyce & Ellis, 2005), and differential susceptibility to the environment theory more broadly (Ellis et al., 2011), that higher levels of cardiovascular reactivity to stress strengthen the associations of aggressive couple environments with multiple aspects of adults' functioning (Hypothesis 1) – but only among women. Women whose partners were more aggressive toward them experienced more depression, stress, and anger, and reported more problem drinking. These associations were strengthened among women who showed greater increases in cardiovascular reactivity, as jointly indicated by increases in blood pressure and heart rate in response to laboratory stressors. However, where problem drinking was concerned, this moderation was weaker and was statistically significant only for systolic blood pressure.

The above interactions showed evidence of a crossover pattern. At lower levels of male aggression, women who were greater biological reactors enjoyed an adaptive advantage; they drank less and were less depressed, stressed, and angry. The opposite was true at higher levels of male aggression, at which women with higher levels of cardiovascular reactivity fared progressively worse. Thus, women's cardiovascular reactivity behaved in the present investigation less like a vulnerability factor that enhanced the harmful effect of a hostile couple environment and more like an environmentally conditional, bivalent risk/promotive factor, consistent with BSC theory.

Although women's cardiovascular reactivity could be either a promotive or risk factor, conditional on their partners' aggressive behavior, it is interesting to note that *overall* associations indicated that women with lower levels of cardiovascular reactivity tended to experience worse affective functioning and greater alcohol problems. BSC theory is agnostic to whether biological reactivity absent information about the environment is a risk or promotive factor. However, the associations of lower cardiovascular reactivity with alcohol problems and poor affective functioning are consistent with findings of physiological underreactivity in depression and substance dependence (e.g., Salomon, Clift, Karlsdóttir, & Rottenberg, 2009; Taylor, 2004).

Cardiovascular reactivity was not found to moderate the association of women's overreactive discipline practices with victimization by their partner's aggression. Men's aggression was positively associated with women's angry, harsh parenting of their children irrespective of women's cardiovascular reactivity. The moderating role of cardiovascular reactivity may be limited to more "individualistic" aspects of women's adjustment (e.g.,

depression, drinking). Some more biologically reactive women victimized by a highly aggressive partner may make efforts to limit the impact of their partner's behavior on their own parenting, thus protecting their children and counteracting the interaction effect noted for affective functioning and problem drinking.

Turning to men, no support for the predicted moderational effects of cardiovascular reactivity was found. The authors are unaware of a theoretical rationale for this pattern. If such findings prove to be replicable, gender specificity (at least among adults) may need to be incorporated into BSC theory.

Association of Cardiovascular Reactivity with the Partner's Aggression

BSC theory stipulates not only that biological reactivity moderates environmental effects, but that biological reactivity is itself affected by the environment, and in a curvilinear fashion. Biological reactivity is thought to be highest in very supportive and very adverse environments, with intermediate levels of reactivity in the middle ranges of environment – a U-shaped function (Hypothesis 2). Findings were not consistent with this hypothesis. Instead men and women whose partners were more aggressive tended to exhibit lesser cardiovascular reactivity, with this association taking on a linear form. It is possible that being the recipient of aggressive behavior by the partner down-regulates one's cardiovascular reactivity. Considered in the context of high reactivity women's especially compromised functioning when aggressed against by their partners, the down regulation of a woman's cardiovascular reactivity in aggressive environments may actually serve a protective function. Women who are low cardiovascular reactors may not be as strongly influenced by an aggressive mate. Note that this is in contrast to BSC theory which states that high biological reactivity is adaptive in hostile environments. Alternatively, it may also be that a U-shaped partner aggression-cardiovascular reactivity function would have been obtained if our sample had included a greater range of environment, with more extremely aggressive men and women. The random digit dialing sampling method we used produced a wide range of aggression, but cases of extreme aggression are rare in community samples and couples with extremely aggressive people may not have agreed to participate in the study.

Limitations

The present results provide insights into why some people may be more or less strongly affected by partner aggression. Nonetheless, there are a few important caveats. First, causal interpretations of these associations are complicated by the cross-sectional design; temporal ordering of the hypothesized cause (partner aggression) and consequences (adult functioning and cardiovascular reactivity) could not be established. Prospective and/or experimental designs will be required in future tests of the causal relations implied by the present findings. Second, aggression in couples co-occurs at a high rate (Slep & O'Leary, 2005), and associations between cardiovascular reactivity and partner aggression may reflect perpetration effects as well as victimization effects. Put another way, couple aggression is a highly dyadic phenomenon and many people are active contributors to the environments that they are then victimized by. Thus it would be inaccurate to construe the present findings as pure victimization effects. Third, as previously described, the strength of the sampling

procedure may have inadvertently truncated the range of aggression, with extremely aggressive couples not well represented. This may have had the effect of dampening statistical interactions, as they are heavily influenced by the tails of the predictors' distributions (McClelland & Judd, 1993). Moreover, it may have limited our ability to detect the hypothesized U-shaped associations of cardiovascular reactivity and partner aggression. Much larger samples or oversampling of the extremes may be required to determine more conclusively the shape of such associations. Fourth, the cardiovascular reactivity $\times$ partner aggression interaction in predicting women's problem drinking did not reach conventional levels of statistical significance. Only one of the cardiovascular measures – systolic blood pressure – reliably moderated the partner aggression-drinking problems association. Thus, this finding must be interpreted with some caution. Yet this caution is balanced against the well-known difficulties in detecting interactions (McClelland & Judd, 1993).

Implications for Research on the Effects of Aggression

The present results suggest that the precision of models of aggression's effects – particularly on women – would be enhanced by accounting for individual differences in biological reactivity to stress that may moderate such effects. What remains unknown, however, is the extendability of these findings to different moderators and outcomes. Does greater reactivity in other measures of biological reactivity suggested by BSC theory (e.g., cortisol, salivary alpha amylase) yield moderation of aggression-outcome associations in the same way cardiovascular reactivity does? These associations could not be tested here. Also, what other outcomes might be moderated by biological reactivity, whether cardiovascular or noncardiovascular? As previously noted, BSC theory, as a member of the “sensitivity to context” class of models, does not explicitly stipulate limits to the range of outcomes that may potentially be differentially influenced by environmental factors, depending on one's biological reactivity. We did not find biological moderation of aggression-parenting associations, suggesting that biological moderation may not apply equally to all outcomes. However, the limits are presently unknown.

The questions of the previous paragraph are more than academic. More precisely specified models for compromised adult functioning in the face of partner aggression can lead to improved intervention efforts. Key moderators identified in the present and related research could be assessed to inform the selection of interventions specifically tailored to individual moderator profiles. To illustrate, high level cardiovascular reactivity in a female client experiencing partner aggression may be taken as an indication for more or different interventions to mitigate anger, stress, depressive symptoms, and drinking, and more extensive monitoring of these potential outcomes. Less reactive women reporting victimization by the partner may not require the same interventions and assessments. Such tailoring must clearly await future replications of these initial findings.

Implications for the BSC Model

In addition to being topically relevant to the effects of couple aggression, the present investigation contributes to the empirical validation of the BSC model (Boyce & Ellis, 2005) by (a) providing a test of its relevance to explaining adults' reactions to their environments, with a substantially larger sample than either Gannon et al. (1989) or Clements and Turpin

(2000), and (b) contributing a much needed empirical evaluation of the curvilinear environment-biological reactivity hypothesis of the BSC model. The stronger partner aggression-outcome associations found among women with greater cardiovascular reactivity are clearly consistent with BSC theory and with the results several investigations, primarily with children. The relative lack of such patterns in men is not. Thus, the present findings suggest the applicability of the BSC model to adult behavior, but unevenly so.

Evaluations of the curvilinear environment-biological reactivity association theorized by Boyce and Ellis (2005) are rare. Thus, it is of great interest that the lack of curvilinear partner aggression-cardiovascular reactivity associations was inconsistent with BSC theory. Future, more conclusive evaluations of the curvilinearity hypothesis will require greater representation in the upper ranges of environmental adversity.

Conclusion

Greater cardiovascular reactivity in women enhanced the associations of male-to-female aggression with women's affective functioning and alcohol problems. High reactive women in less aggressive couple environments fared better than their peers with lower cardiovascular reactivity. They fared worse however, in more aggressive environments. Men's cardiovascular reactivity was generally not a moderating factor in their reactions to female-to-male aggression. Like women, however, men's cardiovascular reactivity was lower given more aggressive partners. The present results provide partial support for BSC theory and suggest that incorporating the moderating influence of biological reactivity may improve the precision of models of the effects of aggression on adult adjustment. These novel findings await replication and extension before their potential application in intervention settings may be capitalized on.

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Table 1

Participant Characteristics

Variable	%		<i>M</i>		<i>SD</i>		Range	
	M	F	M	F	M	F	M	F
Age (Years)			37	35.1	6.0	5.0	21-57	21-48
Education (Years)			14.2	14.3	2.3	2.2	10-20	10-20
Minority	20.8	18.1						
Employed full time	93.2	30.0						
Employed part-time	2.4	37.7						
Family income (\$)			81,498		43,099		4,700-500,000	
Married	94.5							
Family size			4.6		1.2		1-10	

Note. M = male; F = female.

Table 2

Descriptive Statistics on Study Measures

Measure	Women				Men			
	<i>M</i>	<i>SD</i>	Min	Max	<i>M</i>	<i>SD</i>	Min	Max
HP reactivity (mental arithmetic)	-113.39	101.91	-933.57	531.87	-123.53	117.51	-763.30	617.48
HP reactivity (partner video)	-20.54	44.22	-188.56	139.39	-20.06	46.37	-250.85	89.61
HP reactivity (parent video)	-19.58	41.65	-146.81	128.33	-18.86	46.08	-272.91	191.23
SBP Reactivity (mental arithmetic)	12.60	10.23	-10.42	45.92	15.58	10.85	-8.00	59.00
SBP reactivity (partner video)	5.08	6.04	-13.75	28.50	5.76	6.03	-20.75	29.00
SBP reactivity (parent video)	4.59	5.74	-5.75	34.25	4.71	6.32	-13.00	28.50
DBP reactivity (mental arithmetic)	6.82	5.48	-12.17	37.50	8.16	5.72	-12.00	28.67
DBP reactivity (partner video)	1.96	3.95	-10.25	16.25	2.73	4.07	-11.25	15.75
DBP reactivity (parent video)	1.89	3.81	-16.50	16.50	1.69	4.22	-14.75	17.00
Psychological aggression (self-report)	1.25	0.81	0.00	4.38	1.05	0.79	0.00	4.63
Psychological aggression (partner report)	1.21	0.91	0.00	5.25	1.14	0.86	0.00	4.50
Physical aggression (self-report)	0.13	0.30	0.00	2.42	0.09	0.25	0.00	2.58
Physical aggression (partner report)	0.11	0.36	0.00	4.33	0.15	0.36	0.00	2.83
Depressive symptoms	8.92	7.85	0.00	46.00	6.71	5.90	0.00	35.00
Perceived stress	26.27	7.41	5.00	50.00	24.38	7.14	4.00	47.00
Anger	22.34	8.64	3.00	51.00	22.40	8.67	2.00	54.00
Alcohol problems	1.87	1.79	0.00	12.00	3.03	2.79	0.00	20.00
Overreactive discipline	2.73	0.72	1.10	5.10	2.61	0.73	1.00	5.40

Note. HP = heart period; SBP = systolic blood pressure; DBP = diastolic blood pressure.

Table 3

Associations Among Modeled Constructs

Variable	1	2	3	4	5
1. Autonomic Reactivity	-	-.23**	-.20**	-.06	-.08
2. Partner's Aggression	-.17**	-	.42**	.15**	.15**
3. Affective Functioning	-.21**	.60**	-	.71**	.09
4. Overreactive Discipline	-.02	.27**	.64**	-	.03
5. Alcohol Problems	-.05	.21**	.29**	.09	-

Note. Figures are standardized covariances with robust *SEs* computed in the context of measurement models; women above diagonal; men below.

**
p < .01.

Table 4
Moderation of Partner Aggression-Outcome Associations by Cardiovascular Reactivity

Dependent variable	Men					Women				
	B	SE	t	CI low	CI high	B	SE	t	CI low	CI high
<i>Affective Functioning</i>										
Aggression victimization	4.88	0.88	5.53**	3.15	6.61	5.37	1.07	5.04**	3.28	7.46
Cardiovascular reactivity	-0.65	0.38	-1.71 [†]	-1.40	0.10	-0.55	0.52	-1.05	-1.57	0.47
Aggression Victimization × Cardiovascular Reactivity	-0.48	1.07	-0.45	-2.57	1.61	2.92	1.01	2.88**	0.93	4.90
<i>Alcohol Problems</i>										
Aggression victimization	1.21	0.38	3.22**	0.47	1.94	0.60	0.22	2.72**	0.17	1.04
Cardiovascular reactivity	-0.05	0.18	-0.25	-0.40	0.31	-0.07	0.15	-0.49	-0.36	0.22
Aggression Victimization × Cardiovascular Reactivity	-0.39	0.48	-0.81	-1.34	0.56	0.52	0.31	1.69 [†]	-0.08	1.12
<i>Overreactive Discipline</i>										
Aggression victimization	0.43	0.10	4.30**	0.23	0.63	0.21	0.09	2.48*	0.04	0.38
Cardiovascular reactivity	0.02	0.06	0.30	-0.11	0.14	-0.02	0.06	-0.37	-0.14	0.09
Aggression Victimization × Cardiovascular Reactivity	0.04	0.16	0.27	-0.27	0.36	0.06	0.10	0.62	-0.14	0.27

Note. CI = 95% confidence interval; DV = dependent variable.

[†] $p < .10$.

* $p < .05$.

** $p < .01$.

Table 5

Linear and Curvilinear Associations of Partner Aggression and Cardiovascular Reactivity

Variable	Men					Women				
	<i>B</i>	<i>SE</i>	<i>t</i>	<i>CI low</i>	<i>CI high</i>	<i>B</i>	<i>SE</i>	<i>t</i>	<i>CI low</i>	<i>CI high</i>
Step 1: Partner aggression	-0.28	0.08	-3.76**	-0.43	-0.14	-1.40	0.35	-3.95**	-2.09	-0.70
Step 2: Partner aggression	-0.39	0.11	-3.46**	-0.61	-0.17	-1.28	0.49	-2.60**	-2.24	-0.32
Partner aggression squared	0.20	0.13	1.53	-0.06	0.46	-0.12	0.33	-0.37	-0.78	0.53

Note. CI = 95% confidence interval; partner aggression refers to aggression of opposite sex partner.

* $p < .05$.

** $p < .01$.