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RESPIRATORY SINUS ARRHYTHMIA MEDIATES LINKS BETWEEN BORDERLINE PERSONALITY DISORDER SYMPTOMS AND BOTH AGGRESSIVE AND VIOLENT BEHAVIOR

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Although emotion dysregulation (ED) is a core symptom of borderline personality disorder (BPD), tests of associations between ED and aggression and violence—which are common to BPD—are sparse. The authors evaluated mediating effects of an autonomic vulnerability to ED on links between BPD symptoms and (a) reactive aggression, (b) proactive aggression, and (c) histories of interpersonal violence in a sample of young adults (N = 104), ages 18–22 years. Low baseline respiratory sinus arrhythmia (RSA) mediated the association between BPD symptoms and reactive aggression. In contrast, although BPD symptoms were correlated with proactive aggression, no mediational effect was found. In addition, low RSA mediated the association between BPD symptoms and histories of interpersonal violence. Collectively, these findings add evidence that neurobiological vulnerability to ED contributes to aggressive and violent behavior among those with BPD.

Keywords: borderline personality disorder, reactive aggression, proactive aggression, violence, emotion dysregulation, respiratory sinus arrhythmia

Borderline personality disorder (BPD) is a complex and often severe psychiatric condition characterized by interpersonal problems, cognitive dysfunction, and both behavior and emotion dysregulation (American Psychiatric Association, 2013). Theory and research identify emotion dysregulation (ED) as an especially central feature of the disorder (Crowell, Beauchaine, & Linehan, 2009; Kuo & Linehan, 2009; Linehan, 1993). To manage challenges of extreme emotional states, people with BPD often engage in impulsive and maladaptive behaviors, such as involvement in abusive relationships,

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risk-taking, intentional self-injury, and suicidal acts (e.g., Scott, Stepp, & Pilkonis, 2014). Furthermore, aggression is considered a core feature of BPD (Látalová & Praško, 2010). Research indicates that individuals with BPD are more likely to commit serious violent and aggressive behavior than individuals without BPD (Newhill, Eack, & Mulvey, 2009). However, when accounting for antisocial personality disorder (ASPD), the association is diminished. Newhill and colleagues (2009) conclude, based on further analyses, that the diminished association between BPD and violence is not because the construct is irrelevant, but rather due to strong overlap of BPD and ASPD (p. 550). This is unsurprising given shared etiology and therefore high rates of comorbidity between BPD and ASPD (see Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009). In community and outpatient samples, BPD symptoms increase risk of aggressive behavior, even controlling for ASPD (Scott et al., 2014). Moreover, greater risk of aggressive and risky behavior and high rates of comorbidity with ASPD may explain the high prevalence of BPD in forensic settings (25%–50%; Black et al., 2007; Sansone & Sansone, 2009).

Although the prevalence and severity of BPD in the general public is lower (1%–5.9%; Grant et al., 2008; Torgersen, Kringlen, & Cramer, 2001), many young people endorse significant symptoms (Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002). Depending on the sample, prevalence rates in university settings range from 0.5% to 32.1%, with a lifetime prevalence of 7.8% (Meaney, Hasking, & Reupert, 2016). Consistent with these data, young adults report more interpersonal tension than older adults (Birditt, Fingerman, & Almeida, 2005) and use less effective emotion regulation strategies to deal with such tensions (Blanchard-Fields, Mienaltowski, & Seay, 2007). This may place young adults at particular risk for interpersonal aggression.

Emerging research has begun to explore associations between BPD symptoms and aggression among adolescents and young adults. Similar to older adult patients (see Mancke, Herpertz, Kleindienst, & Bertsch, 2017), self-reported ED among young adults and adolescents appears to mediate associations between BPD symptoms and aggression (Gardner, Archer, & Jackson, 2012). However, these studies have relied exclusively on self-report measures of ED, which may be problematic for several reasons, such as inflation of statistical associations given common method variance (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). In addition, people with BPD symptoms have difficulties with accurately identifying and reporting emotions (Cole, Llera, & Pemberton, 2009; Putnam & Silk, 2005). Furthermore, contemporary models of BPD specify biological vulnerabilities to impulsivity and ED (Crowell et al., 2009; Linehan, 1993). However, few studies have evaluated biological vulnerabilities as mediators of associations between BPD symptoms and aggressive behavior (Kuo & Linehan, 2009). Thus, mechanisms through which BPD symptoms increase risk for aggression and violence remain poorly understood (Scott et al., 2014, p. 2). To our knowledge, this is the first study to evaluate the mediating role of a biological vulnerability to ED in associations between BPD symptoms and both aggression and interpersonal violence.

BPD AND EMOTION DYSREGULATION

Chaotic interpersonal relationships and difficulties regulating emotions are associated strongly with aggressive behavior among those with BPD (e.g., Newhill et al., 2009; Newhill, Eack, & Mulvey, 2012). Furthermore, developmental models view ED as the single most important feature of BPD in eliciting symptoms, including mood lability, fear of abandonment, and instabilities in self-image and interpersonal relationships (Beauchaine et al., 2009; Linehan, 1993). Biosocial models specify longitudinal, transactional associations between biological and social factors that contribute to development of ED among those with BPD (Crowell, Derbidge, & Beauchaine, 2014). According to these models, ED develops specifically among neurobiologically vulnerable children and adolescents when they are reared in punitive, invalidating, and emotionally labile family environments. Such environments, which have been verified in recent empirical studies (e.g., Crowell et al., 2017), are proposed to canalize the high negative emotional intensity and physiological reactivity that characterize BPD (Kuo & Linehan, 2009).

To date, findings regarding emotional and physiological reactivity among those who are vulnerable to or suffer from BPD are mixed. Although we have found considerable emotional and physiological reactivity among adolescents who meet criteria for BPD (e.g., Crowell et al., 2014, 2017), negative findings have appeared in the adult literature (see Kuo & Linehan, 2009). In contrast, relations between BPD symptoms and biomarkers of ED collected at rest appear to be more consistent across age groups (e.g., Crowell et al., 2005; Kuo, Fitzpatrick, Metcalfe, & McMain, 2016). Resting parasympathetic tone in particular—assessed by measuring respiratory sinus arrhythmia (RSA)—marks individual differences in emotion regulation capabilities (see Beauchaine, 2001; Shader et al., 2018) and is compromised among those who regulate their emotions ineffectively (Beauchaine & Thayer, 2015; Porges, 2007; Thayer & Lane, 2009).

When respiration is appropriately controlled, RSA marks parasympathetic efference to the heart via the vagus (10th cranial) nerve (Beauchaine, 2001; Porges, 1995). Given inverse associations with a wide range of psychiatric disorders characterized by ED, RSA has become a valuable biomarker of vulnerability to emotion regulation difficulties (see Beauchaine, 2015). High resting RSA is associated with prosocial outcomes and behaviors (Oveis et al., 2009), whereas low resting RSA is associated with problems regulating emotions, as manifested in anxiety and anxiety disorders (e.g., Thayer, Friedman, & Borkovec, 1996), anger (e.g., Ellis, Shumake, & Beevers, 2016), trait hostility (e.g., Sloan et al., 1994), depression (e.g., Kemp, Quintana, Felmingham, Matthews, & Jelinek, 2012), suicidality (e.g., Rottenberg, Wilhelm, Gross, & Gotlib, 2002), longer periods of psychiatric hospitalization (Lin et al., 2015), reactive aggression (e.g., Zhang & Gao, 2015), and BPD (e.g., Koenig, Kemp, Feeling, Thayer, & Kaess, 2016), among other adverse outcomes across the life span (see Beauchaine & Thayer, 2015; Shader et al., 2018; Thomson & Centifanti, 2018). Despite overwhelming evidence for low RSA marking vulnerability to ED, no research conducted to date

has evaluated its associations with BPD symptoms, aggression, and violent behavior.

BPD, AGGRESSION, AND VIOLENCE

Research on aggressive behavior has focused largely on etiological functional differences between aggression subtypes—particularly reactive and proactive aggression. Although highly correlated, these subtypes are distinct theoretically (see Raine et al., 2006). Reactive aggression is thought to be driven by frustration (Berkowitz, 1993) and is characterized by hostility to real or perceived provocations and threats. Empirically, it is associated with poor behavioral control, anxiety, emotionality, and attributional biases toward social threats (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Dodge et al., 2015). In contrast, proactive aggression is goal-directed and predatory, and may be learned through both observation and reinforcement mechanisms because it yields personal gain (Dodge & Coie, 1987; Huesmann, 1998). These topographical and functional differences have led to theories linking reactive aggression in particular to BPD symptoms—a perspective with considerable empirical support (Berenson, Downey, Rafaeli, Coifman, & Paquin, 2011; Dougherty, Bjork, Huckabee, Moeller, & Swann, 1999; Gardner et al., 2012; McCloskey et al., 2009; New et al., 2009; Ostrov & Houston, 2008; Ross & Babcock, 2009). Evidence for an association between BPD and proactive aggression is more mixed (e.g., Gardner et al., 2012; Ostrov & Houston, 2008; Ross & Babcock, 2009).

To date, few studies have explored interrelations among ED, BPD symptoms, and aggression (Scott et al., 2014). One cross-sectional study found that the association between BPD symptoms and reactive aggression was mediated by self-reported maladaptive emotion coping strategies (Gardner et al., 2012). In addition, the same study found that BPD symptoms were related to proactive aggression only through self-reported avoidant coping (withdrawing from emotional contact with stressful situations via denial, avoidance, or disengagement; Gardner et al., 2012). Among adults, self-reported ED is a longitudinal mediator of the association between BPD and interpersonal violence (Newhill et al., 2012), and between BPD and both psychological and physical aggression (Scott et al., 2014). However, these studies have not included autonomic measures of vulnerability to ED to corroborate self-reports.

THE PRESENT STUDY

Following from this discussion, we expected BPD symptoms to be associated with lower RSA. We also expected BPD symptoms to be correlated with reactive aggression, proactive aggression, and histories of interpersonal violence. However, our primary aims were to test mediating effects of RSA on links between BPD symptoms and both aggression and interpersonal violence. Given its empirical associations with poor emotion regulation (Beauchaine, 2015), we expected low RSA to mediate links between BPD and reactive aggression

and histories of violence. However, we did not expect mediation effects when predicting proactive aggression.

METHOD

PARTICIPANTS

Participants were recruited from a convenience sample of undergraduate students (N = 104; 83% female) in the North East of England. Participants were ages 18–22 years (M = 19.85, SD = 1.17) and self-identified as White British (84%), Asian (9%), White European (4%), and Other (3%).

PROCEDURE

Participants were recruited from online and poster advertisements. Participants were apprised of the study by an information letter and enrolled by e-mail. Once they arrived at the laboratory and signed consent forms, electrodes and a respiration belt were fitted to participants. Self-report questionnaires were completed prior to the baseline to accommodate a stabilization for the physiological assessments. Next, participants underwent a 3-min rest period during which they sat still and were asked to relax.

MEASURES

Borderline Personality Disorder Symptoms. The Minnesota Borderline Personality Disorder scale (MBPD; Bornovalova, Hicks, Patrick, Iacono, & McGue, 2011) consists of 19 true/false items drawn from the Multidimensional Personality Questionnaire-Brief Form (Patrick, Curtin, & Tellegen, 2002). The MBPD has robust associations with BPD symptoms and diagnosis, scores on established self-report measures (e.g., Personality Assessment Inventory-Borderline scale; Morey, 1991), and clinical correlates of BPD (e.g., suicidality, internalizing distress, substance abuse). Cronbach's alpha coefficient was good (α = 0.70).

Reactive and Proactive Aggression. Participants completed the Reactive–Proactive Aggression Questionnaire (RPQ; Raine et al., 2006). The 23-item scale captures physical and verbal aggression of each aggression subscale. The reactive and proactive aggression subscales consist of 11 items (e.g., "Gotten angry or mad or hit others when teased") and 12 items (e.g., "Hurt others to win a game"), respectively. Each item is reported on a 3-point scale ranging from 0 (*never*) to 2 (*often*). The RPQ is considered a cross-culturally valid measure of aggression (Fossati et al., 2009) and is often used in psychophysiological studies (see Raine, Venables, & Mednick, 1997; Xu, Raine, Yu, & Krieg, 2014). Cronbach's alphas for the total score (α = 0.86) and reactive (α = 0.84) and proactive (α = 0.71) scales were good and consistent with prior research (see Raine et al., 2006).

History of Interpersonal Violence. Fourteen items were used to capture histories of interpersonal violence. Participants scored each item as either 1 (*yes*) or 0 (*no*) to being asked if they had in the past year committed violence against another person. Consistent with prior research, items were summed to form a total score (see Vitacco, Neumann, & Pardini, 2014). Items were based on the violence subscale of the Self-Reported Delinquency scale (SRD; Elliott & Ageton, 1980; Huizinga & Elliott, 1984). These items cover violence directed at parent(s) ("Hit one of your parents?"), friends ("Hit one of your friends?"), romantic partner ("Hit your girlfriend/boyfriend or ex-partner?"), people other than family or friends ("Hit or threatened to hit other people, not friends, family, or partner?"), nonvictim-specific violence ("Beaten someone up?" "Choked someone?" "Attacked someone with the idea of seriously hurting or killing them?"). Cronbach's alpha for the total score was moderate ($\alpha = 0.63$).

Respiratory Sinus Arrhythmia. Two Ag-AgCl electrocardiogram (ECG) electrodes were placed in a modified Lead II configuration. Respiration was recorded using an RSPEC-R amplifier with a wireless respiration belt transducer. To ensure that the belt was placed at maximum point of sensitivity, participants were asked to exhale, and at full exhalation the respiration belt was fastened around the abdomen. Data were recorded using a Biopac MP150 system with a BioNomadix module transmitter (MP150-BIOPAC Systems Inc., Goleta, CA). The sampling rate was 1 kHz. Data were reduced and analyzed offline using the Biopac's Acknowledge 4.3 software. Data were visually inspected for motion artifacts and outliers. The ECG was reduced offline using computer-aided event detection, but modified by visual inspection so that midbeats were created if missing (< .001%) and errors in R-wave detection were adjusted. To compensate for fluctuations due to movement, the electrocardiogram was reduced at 250 Hz and respiration was passed through a 0.5 Hz digital band filter. RSA was computed using AcqKnowledge automated function for RSA analysis, which applies the peak–valley method (Grossman, van Beek, & Wientjes, 1990). RSA values reflect the millisecond difference between the minimum and maximum R-R intervals during each respiration cycle. RSA values were averaged across 30-s epochs.

DATA ANALYSES

Bivariate correlations were first computed to assess zero-order associations among study variables. To test the hypothesis that ED (RSA) mediates relations between BPD symptoms and reactive aggression, proactive aggression, and histories of interpersonal violence, regression analyses with 95% bootstrapped confidence intervals (CIs) of indirect effect were computed using PROCESS (Hayes, 2013) in SPSS with 5,000 replication samples. The proactive aggression scale was positively skewed (2.23) and kurtotic (6.23). To resolve this, we used a square root transformation to improve normality. This yielded skew of 0.68 and kurtosis of –0.57. When reactive aggression was tested, proactive aggression was included as a covariate, and vice versa,

TABLE 1. Correlations, Means, and Standard Deviations for the Main Study Variables

Measure	1	2	3	4	5	6	7
1. Sex[a]	—						
2. Age	–.26**	—					
3. BPD Traits	.06	–.09	—				
4. Reactive aggression	–.00	.04	.39***	—			
5. Proactive aggression	–.02	.05	.21*	.57***	—		
6. Interpersonal violence	–.05	-.03	.34***	.57***	.31**	—	
7. RSA	.11	–.06	–.35***	.-42***	–.10	–.62***	—
M		19.85	6.41	8.19	1.20	1.74	4.23
SD		1.17	4.11	4.52	1.78	2.40	0.77

RSA = respiratory sinus arrhythmia. [a]Based on Spearman's correlation. *$p < .05$. **$p < .01$. ***$p < .001$.

to test for unique effects of each subtype. Age and sex were also included as covariates.

RESULTS

Descriptive statistics and zero-order correlations appear in Table 1. Reactive and proactive aggression were moderately correlated ($r = .57$, $p < .001$). Histories of violence correlated with both reactive aggression ($r = .57$, $p < .001$) and proactive aggression ($r = .31$, $p = .001$). Borderline symptoms were positively correlated with reactive aggression ($r = .39$, $p < .001$), proactive aggression ($r = .21$, $p = .031$), and histories of violence ($r = .34$, $p < .001$), and were negatively correlated with RSA ($r = –.35$, $p < .001$).

MEDIATION MODELS

Reactive Aggression. Model 1 included BPD symptoms and reactive aggression, and mediation by RSA, while controlling for sex, age, and proactive aggression. The overall model predicting reactive aggression was significant, $F(4, 99) = 22.53$, $p < .001$, $R^2 = .54$. Furthermore, the total effect was significant, $b = .33$, $SE = .08$, $p < .001$, and both the direct effect of BPD symptoms on reactive aggression, $b = .22$, $SE = .08$, $p = .009$, and the indirect effect of BPD symptoms on reactive aggression through RSA were significant, $b = .11$, $SE = .04$, 95% CI [.04, .21] (see Figure 1). Thus, BPD was predictive of reactive aggression, and RSA mediated the association between BPD symptoms and reactive aggression.

Proactive Aggression. Model 2 tested the mediating role of RSA on the relation between BPD symptoms and proactive aggression. The overall model predicting proactive aggression, which included age, sex, and reactive aggression as covariates, was significant, $F(4, 99) = 13.81$, $p < .001$, $R^2 = .41$. However, the total effect was not significant, $b = –.02$, $SE = .02$, $p = .373$.

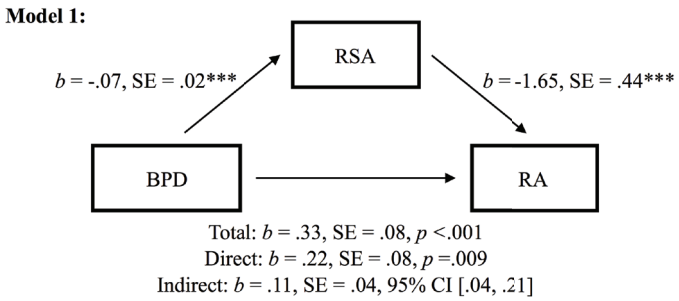

FIGURE 1. Mediation of BPD symptoms and reactive aggression by RSA. Note. Sex, age, and proactive aggression included as covariates. ***$p < .001$.

Furthermore, the direct effect of BPD symptoms on proactive aggression was not significant, $b = -.01$, $SE = .02$, $p = .637$, and the indirect effect of BPD symptoms on proactive aggression through RSA was also not significant, $b = -.01$, $SE = .01$, 95% CI [–.02, 00] (see Figure 2). Thus, BPD symptoms did not predict proactive aggression, and RSA did not mediate an association between BPD symptoms and proactive aggression.

Histories of Interpersonal Violence. Finally, Model 3 tested the mediating role of RSA on the relation between borderline symptoms and histories of interpersonal violence. Model 3 controlled for sex and age. The overall model, including all predictor variables and covariates, was significant, $F(3, 100) = 16.69$, $p < .001$, $R^2 = .40$. In addition, the total effect was significant, $b = .20$, $SE = .06$, $p < .001$, but the direct effect of BPD symptoms on interpersonal violence was not significant, $b = .08$, $SE = .05$, $p = .095$. However, the indirect effect of BPD symptoms on interpersonal violence through RSA was significant, $b = .12$, $SE = .03$, 95% CI [.06, .19] (see Figure 3). Thus, RSA mediated the association between BPD symptoms and histories of interpersonal violence.

DISCUSSION

When attempting to manage extreme emotional states, people with BPD sometimes engage in aggression (Newhill et al., 2009) and interpersonal violence (Sansone & Sansone, 2009; Soloff et al., 2003). In this cohort of nonclinical young adults, our findings support associations between BPD symptoms and risk for aggression (particularly reactive aggression) and interpersonal violence. Both theory and empirical evidence suggest that ED is a principal mechanism through which borderline symptoms emerge (Crowell et al., 2009; Kuo & Linehan, 2009; Linehan, 1993; Putnam & Silk, 2005), as described in developmental models of BPD (e.g., Crowell et al., 2017, 2014). According to such models, biological vulnerabilities transact with social risk factors to shape and maintain ED (Linehan, 1993). To date, however, few

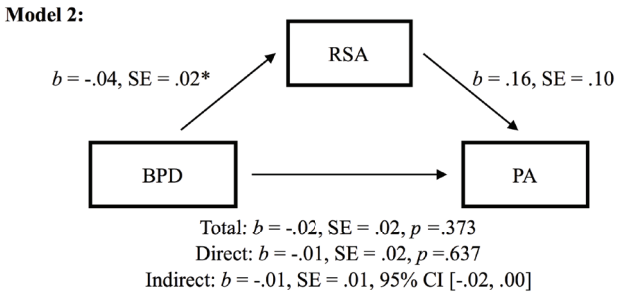

FIGURE 2. Mediation of BPD symptoms and proactive aggression by RSA. Note. Sex, age, and reactive aggression included as covariates. *$p < .05$.

studies have tested the role of ED or associated neurobiological vulnerabilities such as RSA in mediating links between BPD symptoms and risky behaviors—including aggression and interpersonal violence (Scott et al., 2014).

We expected that BPD symptoms would be associated with reactive aggression, and that this association would be mediated by low resting RSA. This expectation was supported. Although such findings are consistent with biosocial perspectives, longitudinal data are needed to verify mechanisms. Indeed, a primary limitation of our models is their cross-sectional nature. An alternative possibility is that BPD symptoms moderate links between RSA and aggression. Although this is less consistent with developmental theory, it cannot be ruled out with cross-sectional data.

We expected similar results for interpersonal violence because BPD-associated violence is often precipitated by strong emotional states (e.g., fear of abandonment). As expected, RSA mediated the association between borderline symptoms and histories of interpersonal violence. It is important to note, however, that when RSA was included, the association between BPD and violence was no longer significant. This indicates that within this sample of young adults, the link between BPD symptoms and violence is accounted for by low resting RSA. This is consistent with community samples in which ED mediates associations between BPD symptoms and interpersonal problems (Herr, Rosenthal, Geiger, & Erikson, 2013). Unfortunately, our measure of interpersonal violence did not assess whether such violence was reactive or proactive. However, based on several findings (low prevalence of proactive aggression, high correlation between the reactive aggression scale and the interpersonal violence scale, and consistent mediating effects of RSA), it seems likely that interpersonal violence perpetrated by our sample may be more reactive than proactive. Our findings may have implications for treatment of young adults with high levels of BPD symptoms; improving emotion regulation will likely reduce violence.

We did not find a mediating effect for proactive aggression, nor a direct effect between BPD and proactive aggression when accounting for reactive aggression. This is consistent with prior research (Gardner et al., 2012; Ross & Babcock, 2009) and is potentially significant because prior research shows

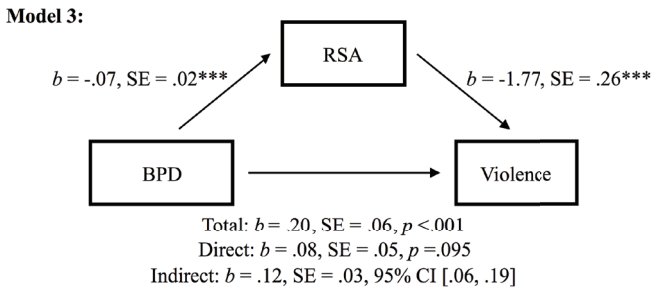

FIGURE 3. Mediation of BPD symptoms and history of interpersonal violence by RSA. Note. Sex and age included as covariates.
$^{***}p < .001$.

that BPD symptoms are related to manipulative behaviors to achieve desired goals (Zanarini et al., 2007). This may suggest, albeit indirectly, that BPD symptoms should be associated with proactive aggression. However, our findings were specific to reactive aggression.

Our findings should be interpreted with several limitations in mind. First, our aim was to explore BPD symptoms in a nonclinical population. However, findings from undergraduates may not generalize to the broader community. This is particularly true for our proactive aggression findings. Scores on proactive aggression were low, with limited variance. This is consistent with prior research, including university and community samples (see van Dongen, van Schaik, van Fessem, & van Marle, 2018; van Dongen et al., 2017; White & Turner, 2014). Furthermore, we did not have enough male participants to examine sex differences. This is important because research indicates that links between BPD and aggression sometimes differ by sex (Archer & John, 2004; Rausch et al., 2015). In addition, we did not account for symptoms of common comorbid disorders, such as ASPD. However, statistically controlling for shared symptoms of BPD may remove important construct variance and symptoms from the BPD scale (Gardner et al., 2012; Scott et al., 2014). Finally, despite mounting evidence that low RSA marks vulnerability to ED (Beauchaine, 2001, 2015; Beauchaine & Thayer, 2015), this is the first study to investigate RSA as a mediator of links between BPD symptoms and both aggression and interpersonal violence. Thus, additional studies are needed to explore these links further, including studies in both clinical and longitudinal samples.

There are benefits to assessing mechanisms of BPD-related aggression and violence. The aim of aggression research is to help inform and direct interventions in the hope of reducing aggressive and violent behavior. Prior research has provided support for the understanding that emotion dysregulation is a central component driving BPD-related aggression. Our study expands this research, demonstrating that RSA mediates associations between BPD and reactive aggression and violence. This important finding has several key implications for how we understand BPD-related aggression and for interventions targeting aggressive and violent behavior. First, it implies that

BPD-related aggression is motivated reactively (i.e., by perceived provocation) and is not a result of predatory and goal-directed intent. Furthermore, BPD-related aggression and violence appear to be driven at least in part by biological vulnerability to emotion dysregulation. Importantly, Kuo and Linehan (2009) found that BPD was related to lower RSA and higher levels of emotional intensity at baseline (compared to controls), but was not related to greater increases in physiological or emotional reactivity during emotion induction tasks. This may indicate that individuals with BPD symptoms suffer from a trait vulnerability to emotion dysregulation before provocation, making challenging social situations more acute to manage, resulting in rapid physiological escalation and more frequent displays of reactive aggression and violence.

Thus, interventions designed to address emotion dysregulation may be more effective for reducing BPD-related aggression and violence. Indeed, dialectical behavioral therapy (DBT), a treatment developed for individuals with BPD, targets cognitive dysregulation, interpersonal chaos, affective lability, and impulsivity and mood-dependent behaviors. DBT is effective in reducing violence among adolescent (Shelton, Kesten, Zhang, & Trestman, 2011) and adult male offenders (Shelton, Sampl, Kesten, Zhang, & Trestman, 2009) and reduces criminogenic risk among female offenders (Nee & Farman, 2005). However, it remains unknown if DBT affects neurobiological vulnerabilities to emotion dysregulation, such as those reflected in RSA, resulting in a reduction of aggressive and violent behavior. Nevertheless, other treatments that effectively improve emotion regulation (e.g., meditation, mindfulness, cognitive-behavioral therapies), increase resting RSA for individuals with anxiety (Middleton & Ashby 1995), depression (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014), substance use disorders (Carroll & Lustyk, 2018), and other externalizing problems (Bell, Shader, Webster-Stratton, Reid, & Beauchaine, 2018). Thus, RSA is not a fixed/static vulnerability. In addition, RSA may be used as an early indicator of vulnerability to psychopathology. For instance, low resting RSA in infancy predicts later externalizing behaviors (e.g., oppositional defiant disorder, callous unemotional behaviors; Wagner et al., 2017). Thus, low RSA may hold promise in identifying vulnerable populatons for preventative approaches. For example, children with low resting RSA whose parents participated in early parental intervention (e.g., Parent–Child Interaction Therapy) were found to have greater improvements in disruptive behaviors than children with low RSA from a waitlist control comparison group (see also Beauchaine et al., 2013). Thus, RSA seems responsive to early intervention in childhood and to treatment in adulthood.

In sum, this study extends research indicating that RSA is an important biomarker of vulnerability to emotion dysregulation, which characterizes many forms of psychopathology. However, an important line of inquiry will be to test if treatments designed to target emotion dysregulation affect RSA specifically among adults with BPD and BPD symptoms, and if such changes mediate reductions in reactive aggression and violent behavior. We look forward to finding such studies in the literature in the years ahead.

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