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Prospective Associations Between Features of Borderline Personality Disorder, Emotion Dysregulation, and Aggression

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Abstract

Difficulties with emotion regulation and behavioral instability, including impulsive aggression, are seen as core dimensions underlying borderline personality disorder (BPD). Although both BPD and antisocial personality disorder (ASPD) are associated with impulsivity and aggressive behavior, difficulties regulating emotions may be associated uniquely with BPD and may explain distinctive associations between BPD and aggression. This study was designed to examine the unique prospective associations between BPD symptoms at baseline, difficulties with emotion regulation and trait impulsivity, and psychological and physical aggression (both perpetration and victimization) over the course of a year after controlling for ASPD symptoms in a mixed clinical and community sample of adults (N = 150). Results of a multivariate path analysis demonstrated that associations between BPD symptoms at baseline and later psychological and physical aggression were fully mediated by difficulties with emotion regulation. Although BPD symptoms also predicted trait impulsivity, impulsivity did not predict aggression after controlling for emotion dysregulation. ASPD symptoms were directly associated with physical assault perpetration and victimization but were not associated with emotion dysregulation, impulsivity, or psychological aggression. These findings suggest that although both BPD and ASPD are associated with aggressive behaviors, associations between BPD symptoms and aggression are mediated uniquely by difficulties regulating emotions.

Keywords

borderline personality disorder; antisocial personality disorder; aggressive behavior; victimization; emotion dysregulation

Borderline personality disorder (BPD) is one of the most complex, functionally debilitating, and costly psychiatric conditions currently facing the mental health and criminal justice systems. Both theory and empirical studies suggest that emotion dysregulation is a central area of dysfunction in BPD (Kernberg, 1984; Linehan, 1993; McCloskey et al., 2009; Siever, 2008), which often leads to behavioral dysregulation as manifested in self-destructive, impulsive, and/or aggressive behavior. Although the link between BPD and self-directed aggression (e.g., suicide attempts and self-injury) is well-established, evidence also

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suggests that BPD is associated with aggressive and violent behavior directed toward others (Newhill, Eack, & Mulvey, 2009, 2012; Sansone & Sansone, 2012), as well as with heightened risk for experiencing victimization (Stepp, Smith, Morse, Hallquist, & Pilkonis, 2012; Zanarini et al., 1999). In addition, previous research suggests that aggression perpetrators are at higher risk for victimization and vice versa (e.g., Stith, Smith, Penn, Ward, & Tritt, 2004; Stepp et al., 2012), and the overlap between perpetration and victimization is especially high among those with BPD (Cloitre, Tardiff, Marzuk, Leon, & Portera, 2001). However, most research on BPD has focused on internalized affective processes and self-harm, with relatively few studies examining externalized aggression or victimization. Hence, the specific mechanisms by which BPD features increase risk for aggression and victimization are understudied and poorly understood.

In the relatively few studies focused on externalized aggression and BPD, associations between BPD and aggressive behavior have been demonstrated using self-report instruments (e.g., Burnette & Reppucci, 2009; Fossati et al., 2004; Ostrov & Houston, 2008), collateral sources of information (i.e., informants' reports, official police and hospital records; e.g., Walsh et al., 2010; Newhill et al., 2009), and laboratory-based behavioral measures (Dougherty, Bjork, Huckabee, Moeller, & Swann, 1999; McCloskey et al., 2009; New et al., 2009). When individuals with BPD engage in aggressive behavior, research suggests it is often in situations of conflict with intimate partners or in other close relationships (Newhill et al., 2009; Sansone & Sansone, 2012). Among both men and women, BPD has been associated with heightened risk for both perpetrating and being a victim of intimate partner violence, nonintimate assaults, child maltreatment, and more subtle forms of aggression such as psychological and relational aggression or property damage (McGowan, King, Frankenburg, Fitzmaurice, & Zanarini, 2012; Ostrov, Hart, Kamper, & Godleski, 2011; Perepletchikova, Ansell, & Axelrod, 2012; Sansone & Sansone, 2012).

BPD features tend to covary with other known risk factors for aggressive behavior such as antisocial personality disorder (ASPD), low socioeconomic status, and young age (Capaldi, Knoble, Shortt, & Kim, 2012; Látalová & Prasko, 2010; Monahan et al., 2001). Thus, for purposes of risk assessment and treatment development, it is important to determine the degree of risk that BPD confers for aggressive behavior above and beyond other associated risk factors. Some have argued that aggressive behavior in those with BPD is attributable to comorbid ASPD features (Allen & Links, 2012), but recent evidence suggests that a substantial proportion (i.e., 66%; Newhill et al., 2009) of those with BPD and without comorbid ASPD engage in aggressive behavior directed toward others. In another study, BPD was uniquely associated with violent behavior after controlling for antisocial features (Newhill et al., 2012). However, BPD may be associated with different types of aggression and in different contexts than is ASPD. A recent study (Weinstein, Gleason, & Oltmanns, 2012) showed that BPD symptoms, but not ASPD symptoms, were associated with aggression against romantic partners among a sample of late middle-age adults (ages 55–64), suggesting distinctions in the long-term course of BPD and ASPD and their associations with aggressive behavior in close relational contexts. Evidence also suggests that when associations between BPD and violence diminish after controlling for ASPD, this may be due to features shared by these disorders and that increase risk for reactive aggression, such as proneness to impulsivity, irritability, and anger (Newhill et al., 2009).

Despite the temperamental characteristics shared by BPD and ASPD, difficulties regulating emotions may be a unique feature of BPD that underlies reactive aggression perpetration and victimization in this population. Emotion dysregulation (also referred to as affective instability) is a central characteristic of BPD (Conklin, Bradley, & Westen, 2006; Linehan, 1993), but this characteristic is not consistently observed in those with ASPD (Paris, Chenard-Poirier, & Biskin, 2012). Studies indicate that BPD is associated with deficits in a broad range of affect regulation skills (e.g., Glenn & Klonsky, 2009; Salsman & Linehan, 2012). Empirical work also suggests that those with BPD demonstrate intensely negative and rapidly shifting emotions (Santangelo, Bohus, & Ebner-Priemer, 2012), especially within interpersonal contexts (Berenson, Downey, Rafiaeli, Coifman, & Paquin, 2011; Chapman, Walters, & Dixon Gordon, 2012; Sadikaj, Moskowitz, Russell, Zuroff, & Paris, 2012). Dysregulated emotions during interpersonal conflict may contribute to impulsive and frantic attempts to regain control, manifesting in psychological or physical aggression directed toward others. In addition, those who have difficulty regulating intense emotional experiences are more prone to risky behaviors and involvement in antagonistic or even abusive relationships, increasing their vulnerability to interpersonal violence and victimization (Finkelhor, Ormrod, Turner, & Holt, 2009).

Researchers have previously posited that difficulties regulating emotions are key mechanisms in the link between BPD and aggression (e.g., Newhill & Mulvey, 2002; Newhill et al., 2009). This idea has only recently begun to be tested empirically. A recent cross-sectional study showed that the association between BPD symptoms and reactive aggression was mediated by maladaptive emotional coping strategies (Gardner, Archer, & Jackson, 2012). In addition, difficulties with emotion regulation have been shown to fully mediate associations between BPD symptom severity and interpersonal problems (Herr, Rosenthal, Geiger, & Erikson, 2012). A recent longitudinal study with adults recruited from inpatient units demonstrated that slower decreases in emotion dysregulation over the course of a year fully mediated the association between BPD and future violence (Newhill et al., 2012). Thus, although longitudinal evidence is scarce, emerging evidence supports the role of emotion dysregulation as an intermediate pathway in the association between BPD and aggression.

However, the specificity of emotion dysregulation as a mechanism for explaining both aggression and victimization in those with BPD after controlling for ASPD features is unclear. Supporting a specific link between BPD and emotionally provoked aggression, studies suggest that BPD is more closely associated with reactive aggression (i.e., hostile or impulsive aggression in response to provocation) in the context of perceived insult or injury and intense negative affective arousal, whereas ASPD is more closely associated with proactive aggression (i.e., premeditated, goal-oriented, or instrumental aggression), which may or may not be associated with aversive affect (Gilbert & Daffern, 2011; Ostrov & Houston, 2008; Ross & Babcock, 2009; Siever, 2008). Nonetheless, in the longitudinal study cited above (Newhill et al., 2012), emotion dysregulation not only mediated the association between BPD and violence, but also partially mediated the association between ASPD and violence after controlling for BPD. This finding may have been due in part to the nature of the sample (i.e., patients who were hospitalized by civil commitment, many of whom were diagnosed with severe mental illness such as bipolar or psychotic disorders). In addition, the

measure of emotion dysregulation used in this study was derived from an instrument designed to assess anger, a construct common to both BPD and ASPD. Follow-up studies are necessary to determine if more generalized difficulties with emotion regulation uniquely mediate prospective associations between BPD symptoms and aggression within community and outpatient psychiatric samples.

We posit that emotion dysregulation may uniquely explain aggressive behavior and victimization among those with BPD features, even after controlling for ASPD symptoms and temperamental characteristics that are common to BPD and ASPD (e.g., impulsivity). In the current study, our primary goal was to examine prospective associations between BPD symptoms, difficulties with emotion regulation, and subsequent aggressive behaviors and experiences of victimization after controlling for ASPD symptoms and trait impulsivity. Our hypotheses were as follows: (a) both BPD and ASPD symptoms would predict greater impulsivity, aggression, and victimization; and (b) BPD would uniquely predict difficulties with emotion regulation, which in turn, would predict greater aggression and victimization. Thus, we hypothesized that emotion dysregulation would be a specific mediator of associations between BPD symptoms and aggression.

Methods

Participants and Recruitment Procedures

The study sample consisted of 150 adult participants recruited for a longitudinal study of interpersonal and emotional functioning among individuals with a range of BPD features (*M* age = 44.85, *SD* = 10.42, range = 22 to 61 years old; 65% female). The sample included 75 patients receiving treatment at a general outpatient psychiatric clinic and 75 community residents who were not receiving mental health care. The recruitment procedures for the current study sample have been described in detail elsewhere (Scott et al., 2013). Briefly, our recruitment criteria were designed to sample the full spectrum of BPD features within both clinical and community (i.e., nontreatment-seeking) populations. Thus, the community sample was not intended to be a healthy comparison group, but rather, was selected to represent a range of psychopathology in a nontreatment-seeking population. Sample demographics and clinical characteristics of the sample are displayed in Table 1. Although only 26 and nine individuals met full diagnostic criteria for BPD and ASPD, respectively, 64 participants (43%) met three or more criteria for BPD, and 44 participants (29%) met two or more criteria for ASPD, suggesting that a sizable proportion of the sample had at least subthreshold symptoms of these disorders. The most commonly met (above threshold) symptoms of BPD were excessive anger (20%), affective instability (16%), and impulsivity (12%). The most commonly met symptoms of ASPD were consistent irresponsibility (16%) and failure to conform to social norms with respect to lawful behaviors (13%).

Assessment Procedures

All procedures of this study were approved by the University Institutional Review Board. At the initial assessment meeting, clinicians described the study in detail and obtained written, informed consent. Participants completed a battery of self-report questionnaires and clinical interviews at intake, and then completed selected self-report questionnaires at 3-month

follow-up intervals over the course of the year. Interviewers were trained clinicians who had a Master's or doctoral degree and at least 5 years of assessment/clinical experience. Clinical interviewers were blind to participants' community or patient status and initial screening responses. At the conclusion of each participant's interviews, a consensus diagnostic case conference was conducted by a research team comprised of at least three individuals. At the case conferences, interviewers presented all historical and concurrent information collected during the intake process. Consensus-rated diagnostic measures were completed in the case conference sessions. A complete description of the consensus rating process used in this research program has been provided in previous reports (Pilkonis et al., 1995; Scott et al., 2013; Stepp, Hallquist, Morse, & Pilkonis, 2011). Assessment time points for the measures used in this analysis (described below) were selected to allow for prospective examination of constructs (without temporal overlap).

Measures

BPD and ASPD symptoms—Clinician-rated personality disorder (PD) symptoms were assessed at baseline using a Diagnostic and Statistical Manual of Mental Disorders (*DSM*) checklist that was rated by the consensus team using all available information from intake, including responses from administration of the Structured Interview for *DSM-IV* Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997). The individual diagnostic criteria for each PD were rated on a 0–2 scale (0 = absent, 1 = present, 2 = strongly present). The clinician-rated PD dimensional scores were calculated by summing these scores for the corresponding PD criteria. Because these clinician ratings of PD criteria were based largely on structured interviews, they were highly correlated with dimensional scores from the SIDP-IV (r = .89 for BPD and r = .84 for ASPD dimensional scores, ps < .001). A randomly selected sample of 19 participants' SIDP-IV interviews were videotaped and rated by independent clinically trained judges for calculation of interrater reliability. Three participants' interviews were rated by six judges, 12 were rated by five judges, and four were rated by four judges. Intraclass correlation coefficients (ICC's) were calculated based on one-way random effect models for the unequal number of raters per case, and demonstrated adequate interdiagnostician agreement for BPD (ICC = .81) and ASPD (ICC = .82) dimensional scores.

Emotion dysregulation—Emotion dysregulation was assessed using participants' self reports on the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) at 3-month follow-up. The DERS contains 36 items and produces a total score and six subscales, including lack of emotional clarity, limited access to emotion regulation strategies, lack of emotional awareness, impulse control difficulties, difficulties engaging in goal-directed behavior, and nonacceptance of emotional responses. Participants were asked to indicate how often each item applied to themselves in the last 3 months on a scale from 1 (*almost never*) to 5 (*almost always*). In this analysis, we used the sum of all 36 DERS items to summarize emotion dyresgulation from intake through the 3-month follow-up assessment (α = .96).

Impulsivity—Participants completed the Revised NEO Personality Inventory (NEO-PI–R; Costa & McCrae, 1992) at the 3-month follow-up assessment. The NEO-PI–R is a reliable

and valid measure of the five personality traits that comprise the five factor model of personality, each of which is further broken down into six trait facets, with eight items corresponding to each facet. Participants were asked to rate the degree to which each item described them using a 5-point scale ranging from 0 (*strongly disagree*) to 4 (*strongly agree*). For this analysis, we calculated the average of items corresponding to the Impulsiveness, Self-Discipline (reversed), and Deliberation (reversed) facet scales to comprise a single measure of trait impulsivity (24 items, $\alpha = .87$). These facets have been used in several previous studies as indicators of trait impulsivity (e.g., Bagge et al., 2004; Trull, 2001).

Aggression perpetration and victimization—The frequency of aggressive behaviors and experiences of victimization was assessed via participants' retrospective reports at the 6-, 9-, and 12-month follow-ups using the revised Conflict Tactics Scale (CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). Participants completed 40 items from the CTS2 comprising the Psychological Aggression (e.g., yelling, verbal insults) and Physical Assault (e.g., shoving, slapping) perpetration and victimization subscales to indicate the frequency of their experiences with each item in the last 3 months. Items are scored on a 7-point scale ranging from 0 (*0 times*) to 6 (*21 or more times*). Although the original CTS2 items focus solely on romantic relationships, items were reworded for the current study to reflect perpetration against anyone and victimization by anyone in the past three months, not just romantic partners. The CTS2 was scored by summing the ratings regarding the frequency of behaviors reported on each subscale, differentiating between perpetration and victimization, resulting in four constructs: Psychological Aggression Perpetration (8 items; average alpha across time points $\alpha = .72$), Psychological Aggression Victimization (8 items; average $\alpha = .74$), Physical Assault Perpetration (12 items; average $\alpha = .74$), and Physical Assault Victimization (12 items; average $\alpha = .82$). For each of these four constructs, we calculated the sum of the 6-, 9-, and 12-month follow-up scores to summarize aggressive behaviors and experiences in the final nine months of the follow-up period (i.e., covering months 3–12). In this sample, 89% of participants reported perpetrating psychological aggression, 89% reported being a victim of psychological aggression, 33% reporting perpetrating physical assault, and 36% reported being a victim of physical assault at least once in the last nine months. As expected with relatively low base-rate behaviors such as aggression, these constructs were positively skewed. Therefore, we conducted square root transformations for these variables prior to statistical analyses, which reduced nonnormality to nonextreme levels, that is, absolute values of skew <3 and kurtosis <10 per guidelines provided by Kline (2011), which can be adequately handled with robust estimation procedures as described in further detail below.

Statistical Procedures

Preliminary data analyses were conducted using SPSS 21.0. Hypotheses were tested in Mplus Version 7 for Windows (Muthén & Muthén, 2012) using full-information maximum likelihood estimation with robust standard errors (MLR estimator). MLR estimation can include missing data and produces unbiased parameter estimates and standard errors that are robust to moderate nonnormality. We estimated a multivariate regression model using a path analysis framework to simultaneously predict four types of aggressive behaviors and

experiences (i.e., psychological aggression perpetration, experiences of psychological aggression victimization, physical assault perpetration, and experiences of physical assault victimization). We controlled for the influence of age, clinical group status (i.e., recruitment source; 0 = community, 1 = clinic), gender (0 = male, 1 = female), minority race (0 = Caucasian, 1 = minority race), and education level (coded on a 7-point scale ranging from 1 [*less than 7 years of school*] to 7 [*graduate or professional training*]) on impulsivity, emotion dysregulation, and aggression by entering these variables as covariates. Indirect effects were tested using the "model indirect" command in Mplus, which calculates the product of component path coefficients and uses the delta method to calculate standard errors (MacKinnon, 2008).

Because we were primarily interested in the strength of regression coefficients in this multivariate context rather than model fit, we first tested a fully saturated model with all possible pathways estimated, which has 0 degrees of freedom and therefore is a perfectly fitting model. Fit of more restricted models was evaluated using commonly accepted criteria, including a nonsignificant ($p > .05$) adjusted chi-square (χ^2) test; standardized root mean square residual [SRMR] <.08; CFI and TLI > .95; and root mean square error of approximation (RMSEA) < .06 (Hu & Bentler, 1999; Kline, 2011). Hierarchical (nested) models were compared using the adjusted chi-square difference ($\chi^2\Delta$) test.

Results

Preliminary Analyses

Descriptive statistics and bivariate correlations for all study variables are presented in Table 2. Means and standard deviations of untransformed aggression variables are presented in the table for ease of interpretation. Attrition over the course of the study was low, with only nine participants (6%) failing to complete the 12-month follow-up assessment. Because the MLR estimation method can use cases with missing data, the full sample was used to estimate the reported model. Bivariate correlations demonstrated that both BPD and ASPD symptoms were positively correlated with emotion dysregulation, impulsivity, aggression, and victimization constructs. To test for multicollinearity, we ran a series of multiple regressions in which each predictor was regressed on all other predictors, and examined variance inflation factors (VIFs). The highest VIF value was 2.38, which fell below the most conservative VIF cutoff value of 2.5 (O'Brien, 2007), indicating that multicollinearity was not an issue.

Primary Analyses

Emotion dysregulation was tested as a potential mediator of the effects of BPD symptoms on aggression perpetration and victimization after controlling for sociodemographic characteristics, ASPD symptoms, and impulsivity. We estimated a multivariate path analysis model simultaneously predicting aggressive behaviors and victimization experiences from sociodemographic covariates, BPD and ASPD symptoms, and emotion dysregulation and impulsivity. Specifically, emotion dysregulation and trait impulsivity at 3-month follow-up were regressed on clinical group status, age, gender, minority race, education, and baseline BPD and ASPD symptoms. In addition, the four aggression constructs were regressed on

clinical group status, age, gender, minority race, education, baseline BPD and ASPD symptoms, and emotion dysregulation and trait impulsivity at 3-month follow-up. Given the significant bivariate correlation observed between emotion dysregulation and impulsivity constructs, the residuals of these variables were allowed to correlate in the model.

Standardized regression coefficients for the fully saturated model are presented in Table 3. Only results that directly pertain to our study hypotheses are described here. In partial support of our first hypothesis, baseline BPD symptoms significantly predicted greater emotion dysregulation and trait impulsivity at 3-month follow-up. However, ASPD symptoms did not significantly predict either emotion dysregulation or impulsivity after controlling for the effects of BPD and sociodemographic covariates. Also partially supporting our first hypothesis, emotion dysregulation, but not impulsivity, significantly predicted greater psychological aggression perpetration, psychological aggression victimization, physical assault perpetration, and physical assault perpetration over the remainder of the year. In addition, ASPD symptoms directly predicted greater physical assault perpetration and victimization over the course of the year. However, BPD symptoms did not directly predict aggressive behaviors and experiences after controlling for emotion dysregulation, but rather indirectly predicted these constructs via their effects on difficulties with emotion dysregulation, consistent with our second hypothesis. As depicted in Figure 1, there was a significant indirect effect of BPD symptoms through emotion dysregulation on perpetration of psychological aggression ($\beta = .20$, $SE = .07$, $z = 2.95$, $p = .003$), being a victim of psychological aggression ($\beta = .18$, $SE = .07$, $z = 2.58$, $p = .01$), perpetration of physical assaults ($\beta = .15$, $SE = .07$, $z = 2.09$, $p = .04$), and being a victim of physical assaults ($\beta = .19$, $SE = .07$, $z = 2.75$, $p = .006$). There were no significant indirect effects of BPD symptoms on aggression outcomes through impulsivity, nor were there any significant indirect effects of ASPD symptoms on aggression outcomes through either emotion dysregulation or impulsivity (all ps > .05).

We also tested a more restrictive model in which the direct paths from BPD symptoms to each of the aggression outcomes were not estimated (i.e., fixed to 0), allowing us to test whether these direct pathways are necessary to the model. A nonsignificant decrement in model fit of this restricted model in comparison to the fully saturated model would suggest that the associations between BPD symptoms and aggressive behavior outcomes are fully mediated by emotion dysregulation. Results suggested that this more restricted model without direct pathways from BPD symptoms to aggression outcomes fit the data well (RMSEA < .001; CFI/TLI = 1.0; SRMR < .01) and did not result in a significant decrement in model fit as compared to the saturated model ($\chi^2\Delta(4) = 3.35$, $p = .50$). Hence, emotion dysregulation fully mediated the effects of BPD symptoms on the aggression outcomes.

To verify that the above results were not due to item content overlap between our measures of distinct constructs, we conducted a supplemental analysis in which symptoms directly referring to anger or aggression were removed from calculation of BPD and ASPD dimensional scores, and items referring to impulse control were removed from calculation of DERS scores. Specifically, the BPD criterion, “inappropriate, intense anger or difficulty controlling anger” and ASPD criterion, “irritability and aggressiveness” were excluded from calculation of BPD and ASPD scores, respectively. In addition, to better differentiate

between emotion dysregulation and impulsivity, the items corresponding to the "impulse control difficulties" subscale of the DERS were excluded from calculation of DERS scores. The same fully saturated multivariate model reported above was then rerun with these modified variables as predictors. Results of this supplemental analysis (details available upon request from the first author) were virtually identical to those reported above; that is, all regression coefficients, direct effects, and indirect effects were similar in magnitude and significance.

Discussion

We sought to prospectively examine the mediating role of emotion dysregulation in the association between BPD symptoms and aggression in a mixed community and outpatient psychiatric sample of adults. Results supported our primary hypothesis in demonstrating that BPD symptoms were uniquely associated with emotion dysregulation at 3-month follow-up after controlling for ASPD symptoms. In turn, emotion dysregulation predicted both psychological and physical aggression perpetration and victimization in the following 9 months. Although bivariate correlations showed that BPD symptoms were associated with all four aggression constructs, BPD no longer directly predicted aggression after controlling for emotion dysregulation in our multivariate model. In addition, these direct pathways from BPD symptoms to aggression outcomes were not necessary to produce a well-fitting model after accounting for the indirect relationship between BPD symptoms and aggressive behaviors through emotion dysregulation. Hence, we found that difficulties with emotion regulation fully mediated the relationships between baseline BPD symptoms and subsequent aggressive behaviors and experiences of victimization. This finding is consistent with recent evidence of emotion dysregulation as an intermediate pathway in the association between BPD and aggression (Gardner et al., 2012; Newhill et al., 2012).

Moreover, this mediational pathway to all four types of aggressive behavior was unique to BPD symptoms, as ASPD symptoms were not associated with difficulties regulating emotions in our multivariate model. Rather, ASPD features were directly associated with physical assault perpetration and victimization, but not psychological aggression. This is the first study to demonstrate that emotion dysregulation has specificity to BPD as a mediator of aggressive behavior and victimization. This result is consistent with Paris and colleagues' (2012) assertion that BPD and ASPD can be differentiated from each other on the basis of emotion dysregulation, which appears to be specific to BPD. It is possible that other emotion-related constructs not measured in this study, such as callousness, may further differentiate between BPD and ASPD and may serve as specific intermediate pathways between ASPD and aggression after controlling for BPD. This could be further explored in future studies.

The failure to find this specificity of emotion dysregulation as a mediator of aggression in BPD within a prior longitudinal investigation (Newhill et al., 2012) could potentially be attributable to sampling or measurement differences between studies. Newhill and colleagues' sample was recruited from inpatient units and included those with severe mental illnesses that were not included in our study sample (e.g., bipolar and psychotic disorders). In addition, whereas the emotion dysregulation measure used in this previous study was

comprised of items culled from a measure of anger (with a relatively smaller number of items referring to other negative emotions), we used a more generalized measure of emotion dysregulation that does not ask about specific emotions, but rather focuses on general difficulties with emotion regulation. It is possible that BPD and ASPD may both relate to experiences of intense affects, especially anger, but individuals with BPD may perceive themselves as less able to control and regulate their emotions relative to those with ASPD.

Contrary to our hypotheses, although bivariate correlations demonstrated that ASPD symptoms were related to impulsivity, ASPD symptoms were not associated with impulsivity after controlling for BPD symptoms in the multivariate model. This is surprising given that impulsivity is a feature that is common to both BPD and ASPD according to *DSM-5* criteria (APA, 2013; Fossati et al., 2004). Interestingly, impulsivity was also not significantly associated with aggression after controlling for emotion dysregulation. This finding held even when we recalculated the emotion dysregulation construct after excluding items referring to difficulties controlling impulses. These results suggest that emotion dysregulation and trait impulsivity may be more relevant to BPD than ASPD symptoms, and further, that emotion dysregulation may differentially predict aggression and victimization above and beyond trait impulsivity. Nonetheless, these results should be interpreted with caution in the context of the nature of our sample and our measure of impulsivity. Specifically, our sample was representative of patients seeking treatment in an outpatient psychiatric facility and nontreatment-seeking individuals in the community with a range of PD, depressive, and anxiety-related symptoms. In addition, only nine individuals in our sample met criteria for ASPD, and four of them also had a diagnosis of BPD. Given these characteristics, it is possible that the types of impulsivity seen in our sample may have occurred more in the context of BPD symptoms, including emotion dysregulation, and this is consistent with the strong correlation observed between our measures of emotion dysregulation and impulsivity. In addition, our measure of impulsivity was drawn from an instrument designed to assess normative personality traits, and therefore, may not tap into aspects of impulsiveness (e.g., urgency; Tragesser & Robinson, 2009) that could be more strongly related to ASPD and aggression.

Previous studies (e.g., Fossati et al., 2004; Ostrov & Houston, 2008) suggest that ASPD is more strongly related to direct (e.g., physical) forms of aggression, whereas BPD appears to be more strongly related to indirect (e.g., psychological or relational) aggression. Partially consistent with these findings, we found that ASPD symptoms were only associated with physical forms of aggression after controlling for features of BPD. However, BPD symptoms were indirectly associated with both physical and psychological forms of aggression via emotion dysregulation, even after controlling for features of ASPD. These results suggest that emotion dysregulation confers heightened risk for various types of interpersonal conflict, including physical assault, among those with BPD symptoms, which cannot be accounted for by comorbid ASPD features. Nonetheless, our findings are consistent with others demonstrating strong associations between distinct forms of aggression, such that physical and psychological forms of aggression are highly correlated with each other, and aggression perpetration is associated with heightened risk for being victimized (e.g., Fossati et al., 2007; Stith et al., 2004).

This study is one of few longitudinal studies of associations between BPD and aggression, and represents an extension of previous studies in several respects. The prospective design of this study satisfies the condition of temporal precedence of BPD symptoms, followed by emotion dysregulation, followed by aggression, thus providing more compelling evidence of causality than can be derived from cross-sectional designs. In addition, we controlled for several well-known risk factors for aggression, allowing us to establish more confidence in the specificity of emotion dysregulation as a mediator of aggression in those with BPD symptoms. Our assessment of aggression and victimization was also more comprehensive than most previous studies given that both psychological and physical forms of aggression were assessed within all types of relationships (not just with romantic partners). Our use of dimensional measures of PD symptoms, which were rated by well-trained clinicians based on thorough semistructured interviews, are also a benefit over studies that have used self-report measures of PDs (which are susceptible to bias and a high false-positive rate) or categorical diagnostic entities (which result in loss of important information regarding severity).

Despite the prospective design, we were unable to control for baseline levels of emotion dysregulation and aggression, which prohibits us from making inferences about within-individual changes over time. The constructs we repeatedly assessed in this study were generally stable over time, leaving little within-person variance to be explained, and power for examining such individual-level processes is limited in this relatively small sample. Another limitation of this study is the exclusive use of self-report measures of constructs other than BPD/ASPD symptoms, and the noted limitations of our measure of impulsivity. We also lack comprehensive measures of trait anger, which may be another important mediator of aggression in those with BPD. Our study is also limited in its contribution to understanding the interpersonal and contextual determinants of aggression and victimization, and the potential within-individual and reciprocal relations between constructs. A particularly useful direction for future research is to use methods such as ecological momentary assessment to study the dynamic mechanisms underlying emotion, aggression, and victimization with heightened ecological validity, specificity, and temporal resolution. Moreover, as previously mentioned, our study sample is limited in ASPD relative to BPD. Thus, results may not generalize to more severely impaired inpatient samples or violent criminal populations with a wider range of ASPD symptoms.

Nevertheless, these results advance our knowledge regarding understudied phenomena of high clinical and societal importance. The current results suggest that emotion regulation strategies, which are the primary focus of several empirically supported treatments for BPD (Weinberg, Ronningstam, Goldblatt, Schechter, & Maltsberger, 2011), may be useful in reducing the risk for aggression and victimization, particularly in those with elevated BPD features. Specifically, interventions aimed at improving the ability to understand, tolerate, and modulate negative emotions could be applied to reduce aggressive behavior and victimization among those with BPD symptoms who present in clinical as well as in forensic settings. These findings also imply that emotion regulation capacities may be more relevant for reducing aggression among those with BPD symptoms than among those with a more antisocial presentation. This research adds to an emerging line of evidence suggesting that aggression and victimization in those with BPD might be best understood in the context of

emotion dysregulation and not as merely an epiphenomenon of general impulsivity or antisocial features.

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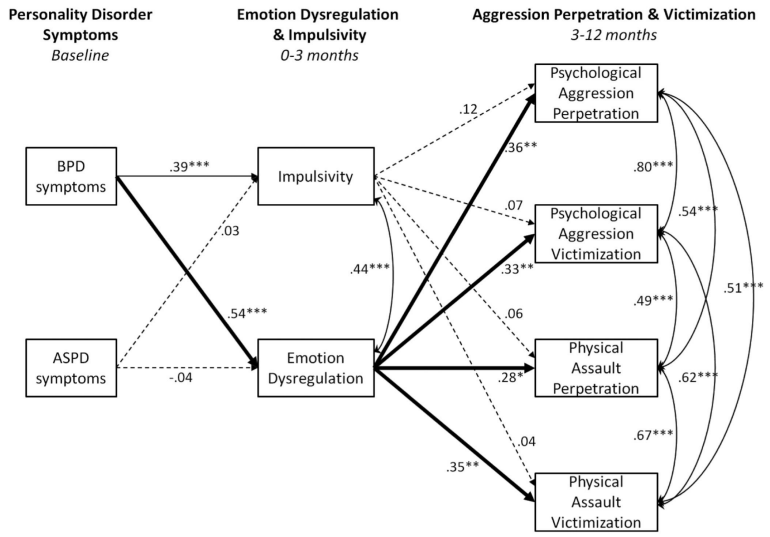

Figure 1.

Emotion dysregulation as a mediator of the relationship between borderline personality disorder (BPD) symptoms and aggression perpetration and victimization. Numbers presented are completed standardized robust maximum likelihood regression coefficients after controlling for clinical group (i.e., community vs. clinic), age, gender, minority race, and education. Only significant indirect pathways are shown. Bold lines represent significant mediational pathways. ASPD = antisocial personality disorder. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 1

Sample Demographics and Axis I and II Diagnostic Information

	Full sample (*n* = 150)		Clinic (*n* = 75)		Community (*n* = 75)	
Demographic	***n***	**%**	***n***	**%**	***n***	**%**
Race						
Caucasian	86	57.3				
African American	57	38.0				
Asian	1	0.7				
More than one race	6	4.0				
Ethnicity						
Hispanic	4	2.7				
Marital status						
Never married	68	45.3				
Married	47	31.3				
Separated or divorced	33	22.0				
Widowed	2	1.3				
Highest completed education						
Junior high school	3	2.0				
Some high school	10	6.7				
High school graduate	28	18.7				
Some college or vocational	63	42.0				
Four-year college degree	28	18.7				
Graduate or professional school	18	12.0				
Current Axis I disorders						
Mood	70	46.7	57	76.0	13	17.3
Anxiety	47	31.3	31	41.3	16	21.3
Substance-related	30	20.0	21	28.0	9	12.0
Eating	1	0.7	1	1.3	0	0
Somatoform	5	3.3	4	5.3	1	1.3
Other Axis I	4	2.7	2	2.7	2	2.7
Any Axis I diagnosis (1 or more)	95	63.3	65	86.7	30	40.0
Current Axis II disorders						
Paranoid	5	3.3	1	1.3	4	5.3
Schizoid	3	2.0	3	4.0	0	0
Schizotypal	2	1.3	2	2.7	0	0
Histrionic	4	2.7	2	2.7	2	2.7
Narcissistic	4	2.7	3	4.0	1	1.3
Antisocial	9	6.0	9	12.0	0	0
Borderline	26	17.3	22	29.3	4	5.3
Avoidant	18	12.0	15	20.0	3	4.0
Dependent	1	0.7	1	1.3	0	0
Obsessive-compulsive	8	5.3	6	8.0	2	2.7

	Full sample (*n* = 150)		Clinic (*n* = 75)		Community (*n* = 75)	
Demographic	***n***	**%**	***n***	**%**	***n***	**%**
PD-NOS	25	16.7	17	22.7	8	10.7
Any Axis II diagnosis (1 or more)	85	56.7	62	82.7	23	30.7

Note. Axis I and II diagnoses were rated by the consensus team based on all available intake information, including the Structured Clinical Interview for *DSM-IV* Axis I disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997) and the Structured Interview for *DSM-IV* Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997). “Mood” disorders include depressive and dysthymic disorders. No participants in this sample met criteria for bipolar or psychotic disorders. “Other Axis I” disorders include adjustment, dissociative, and sexual disorders. Several participants met criteria for more than one disorder, particularly within the clinic sample. The mean numbers of Axis I and II diagnoses in the clinic sample were 1.55 (*SD* = 0.90) and 1.08 (*SD* = 0.82), respectively; 40 (53.3%) clinic participants met criteria for more than one Axis I disorder, and 15 (6.6%) met criteria for more than one Axis II disorder. The mean numbers of Axis I and II diagnoses in the community sample were 0.55 (*SD* = 0.81) and 0.32 (*SD* = 0.66), respectively; eight (10.7%) community participants met criteria for more than one Axis I disorder, and 3 (4.0%) met criteria for more than one Axis II disorder.

Table 2

Bivariate Correlations and Descriptive Statistics for All Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
Baseline covariates													
1. Clinical group	—												
2. Age	−.16*	—											
3. Female gender	.04	−.11	—										
4. Minority race	−.01	−.02	<.01	—									
5. Education	−.21*	.05	.01	−.14	—								
6. BPD symptoms	.40***	−.17*	.14	.07	−.20*	—							
7. ASPD symptoms	.33***	−.14	−.03	.19*	−.27**	.44***	—						
Emotion dysregulation & impulsivity, 0–3 months													
8. Emotion dysregulation	.48***	−.07	<.01	−.16*	−.16	.60***	.24**	—					
9. Impulsivity	.37***	−.19*	−.01	−.11	−.13	.48***	.23**	.63***	—				
Aggression & victimization, 3–12 months													
10. Psychological aggression perpetration	.20*	−.21*	<.01	.07	−.25**	.34**	.30***	.42***	.38***	—			
11. Psychological aggression victimization	.20*	−.12	−.02	<.01	−.28**	.30***	.24**	.40***	.32***	.84***	—		
12. Physical assault perpetration	.12	−.27**	.07	.15	−.17*	.24**	.43***	.26**	.24**	.64***	.55***	—	
13. Physical assault victimization	.16	−.22**	−.11	.12	−.28**	.23**	.38***	.32***	.26**	.63***	.68***	.74***	—
N	150	150	150	150	150	150	150	149	145	141	141	141	141
M	0.50	44.85	0.65	0.43	5.05	2.62	1.39	82.92	2.37	31.96	33.41	3.40	4.48
SD	0.50	10.42	0.48	0.50	1.14	3.30	2.36	26.32	0.53	42.55	44.93	11.21	14.08

Note. Clinical group coded 0 = community, 1 = clinic patient. Gender coded 0 = male, 1 = female. Race coded 0 = Caucasian, 1 = Minority race. Education coded 1 = less than 7 years of school, 2 = junior high school (7–9 years), 3 = some high school (10–11 years), 4 = high school graduate, 5 = some college or vocational training, 6 = 4-year college degree, 7 = graduate or professional training. BPD = borderline personality disorder; ASPD = antisocial personality disorder. The means and standard deviations reported are for variables prior to square root transformation.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

Standardized Multiple Regression Coefficients From the Fully Saturated Path Model

	Emotion dysregulation, 0–3 months		Impulsivity, 0–3 months		Psychological aggression perpetration, 3–12 months		Psychological aggression victimization, 3–12 months		Physical assault perpetration, 3–12 months		Physical assault victimization, 3–12 months	
	β	*SE*	β	*SE*	β	*SE*	β	*SE*	β	*SE*	β	*SE*
Baseline covariates												
Clinical group	.29***	.07	.17*	.08	−.09	.08	−.05	.08	−.14*	.07	−.11	.08
Age	.04	.06	−.11	.07	−.17*	.07	−.09	.08	−.22**	.07	−.20**	.07
Female Gender	−.08	.06	−.08	.07	.01	.07	−.02	.08	.09	.07	−.09	.08
Minority race	−.20***	.05	−.17*	.07	.10	.07	.03	.07	.13	.07	.12	.06
Education	−.02	.05	−.04	.07	−.15*	.08	−.21 *	.08	−.05	.07	−.18*	.07
BPD symptoms	.54***	.07	.39***	.08	−.03	.08	−.01	.08	−.12	.12	−.14	.11
ASPD symptoms	−.04	.08	.03	.09	.14	.09	.09	.09	.37***	.11	.27*	.11
Emotion dysregulation & impulsivity, 0–3 months												
Emotion dysregulation					.36**	.11	.33**	.12	.28*	.13	.35**	.13
Impulsivity					.12	.09	.07	.10	.06	.09	.04	.09

Note. Results are from the fully saturated path model with all parameters estimated (*df* = 0). Deletion of direct paths from BPD symptoms to each of the four aggression outcomes did not result in significant decrement in model fit ($\chi^2\Delta(4) = 3.35$, $p = .50$). Clinical group coded 0 = community, 1 = clinic. Gender coded 0 = male, 1 = female. Race coded 0 = Caucasian, 1 = Minority race. Education coded 1 = less than 7 years of school, 2 = junior high school (7–9 years), 3 = some high school (10–11 years), 4 = high school graduate, 5 = some college or vocational training, 6 = 4-year college degree, 7 = graduate or professional training. BPD = borderline personality disorder; ASPD = antisocial personality disorder.

* $p < .05$.

** $p < .01$.

*** $p < .001$.