Emotional Dysregulation and Childhood Adversity in Borderline Personality Disorder

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Long-standing theories of borderline personality disorder (BPD) suggest that symptoms develop at least in part from childhood adversity. Emotion dysregulation may meaningfully mediate these effects. The current study examined three factors related to emotion dysregulation—alexithymia, affective lability, and impulsivity—as potential mediators of the relation between childhood adversity and BPD diagnosis in 101 individuals with BPD and 95 healthy controls. Path analysis compared three distinct models informed by the literature. Results supported a complex mediation model wherein (a) alexithymia partially mediated the relation of childhood adversity to affective lability and impulsivity; (b) affective lability mediated the relation of childhood adversity to BPD diagnosis; and (c) affective lability and impulsivity mediated the relation of alexithymia to BPD diagnosis. Findings suggest that affective lability and alexithymia are key to understanding the relationship between childhood adversity and BPD. Interventions specifically targeting affective lability, impulsivity, and alexithymia may be particularly useful for this population.

Keywords: borderline personality disorder, abuse, alexithymia, impulsivity, affective lability

Borderline personality disorder (BPD) is a complex, often debilitating illness characterized by severe dysregulation in mood, behavior, cognition,

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and relationships (American Psychiatric Association, 2013). At least 75% of individuals with BPD attempt suicide, including approximately 10% who ultimately die by suicide (Black, Blum, Pfohl, & Hale, 2004). Enormous emotional distress and functional impairments associated with the disorder contribute to reduced productivity, severe impairments in social functioning, high utilization of mental health services, and disproportionate involvement with the criminal justice system (Comtois & Carmel, 2016; Conn et al., 2010; Gunderson et al., 2011; Samuels, 2011).

Reducing the burden of BPD on both society and the individual requires an accurate understanding of factors underlying BPD. Etiological theories suggest that borderline symptoms develop at least in part from experiences of childhood adversity (e.g., abuse, neglect, or disruptions in attachment, often occurring within the context of a relationship with a parent or caregiver). Linehan's biosocial theory, for example, proposes that borderline symptoms develop through a transaction between biological emotional vulnerabilities (i.e., high emotional sensitivity, high emotional reactivity, and slow return to emotional baseline) and an emotionally invalidating social environment (i.e., an environment that characterizes the person's internal experience as wrong, inappropriate, and/or unacceptable; Crowell, Beauchaine, & Linehan, 2009; Linehan, 1993). Similarly, the mentalization model of BPD suggests that early experiences of abuse and/or neglect by caregivers contribute to the development of maladaptive biological, behavioral, and emotional adaptations to avoid threat and abandonment (Fonagy & Luyten, 2009).[1]

Widespread empirical evidence has accumulated in support of these theories. A recent meta-analysis of nearly 100 studies suggests that individuals with BPD are 13.91 times more likely to report experiences of childhood adversity (e.g., abuse, neglect) than healthy controls and 3.15 times more likely to report such experiences than individuals with other psychiatric disorders (Porter et al., 2020). Similarly, higher severity of reported abuse is associated with greater severity of symptoms among individuals with BPD (Kaplan et al., 2016; Soloff, Lynch, & Kelly, 2002). Prospective analyses similarly suggest that experiences of child abuse and neglect increase the risk of subsequently developing borderline traits in adolescence and adulthood (Goodman & Yehuda, 2002; Johnson et al., 2001; Widom, Czaja, & Paris, 2009, but see Infurna et al., 2016). Despite this widespread evidence, however, comparatively little research has explored potential mediators to explain the relationship between trauma and BPD diagnosis.

Growing evidence suggests that emotion dysregulation may meaningfully mediate this relationship. Emotion dysregulation is a multifaceted construct, consisting of impairments in modulation, awareness, understanding, acceptance, and behavior subsequent to emotional arousal (Bridges, Denham, & Ganiban, 2004; Gratz & Roemer, 2004). It is commonly considered a core feature of BPD (Glenn & Klonsky, 2009; Linehan, 1993), and its development is closely associated with experiences of childhood adversity, particularly

1. Notably, although experiences of childhood adversity are commonly referenced in etiological theories of borderline personality development, research suggests that various factors contribute to such development. Genetic and biological effects, for example, play a key role in the development of various features associated with BPD (Amad, Ramoz, Thomas, Jardri, & Gorwood, 2014).

disrupted attachment (Calkins & Hill, 2007; Steele, Steele, & Croft, 2008). Recent research suggests that emotion dysregulation at least partially accounts for associations between childhood adversities and outcomes closely associated with BPD, including eating disorders, depression, and nonsuicidal self-injury (Burns, Fischer, Jackson, & Harding, 2012; Hopfinger, Berking, Bockting, & Ebert, 2016; Huh, Kim, Lee, & Chae, 2017; Jennissen, Holl, Mai, Wolff, & Barnow, 2016; Peh et al., 2017). Preliminary evidence from nonclinical samples (e.g., samples recruited from college or community settings rather than treatment settings) also suggests that emotion dysregulation may mediate associations between childhood adversity and borderline traits (Fossati et al., 2015; Kuo, Khoury, Meltcalfe, Fitzpatrick, & Goodwill, 2015). To date, however, these mediational relationships have not been investigated in samples diagnosed with BPD.

ALEXITHYMIA, AFFECTIVE LABILITY, AND IMPULSIVITY AS POTENTIAL MEDIATORS

Given the particularly salient role of emotion regulation within the relation between childhood adversity and psychopathology, it stands to reason that alexithymia, affective lability, and impulsivity—each of which conceptually overlaps with emotion regulation and is strongly associated with BPD—may similarly mediate the relation between childhood adversity and BPD. Alexithymia is a pervasive deficit in emotion processing and understanding characterized by difficulties in identifying and communicating emotional experiences and information (Bagby, Parker, & Taylor, 1994). Prior work, including work from our group, indicates that individuals with BPD have trouble accurately describing their emotional reactions and have more severe alexithymic trait severity compared with healthy controls (e.g., Hazlett et al., 2007; New et al., 2012). A more recent meta-analysis confirms a strong association between BPD and emotional awareness in studies comparing BPD with healthy controls (Derks, Westerhof, & Bohlmeijer, 2017). In addition, studies report associations between severity of alexithymia and BPD symptoms, including behavioral impulsivity, suicidality, and interpersonal dysfunction (Edwards & Wupperman, 2017; Kealy, Ogrodniczuk, Rice, & Oliffe, 2018; Spitzer, Siebel-Jurges, Barnow, Grabe, & Freyberger, 2005). Like BPD, disrupted child–caregiver relationships, interpersonal trauma, childhood adversity, and socialization experiences surrounding emotional expression, experience, and regulation are commonly theorized to contribute to alexithymic traits (Edwards, Micek, Mottarella, & Wupperman, 2017; Le, Berenbaum, & Raghavan, 2002; Thorberg, Young, Sullivan, & Lyvers, 2011). Some evidence also suggests that alexithymia is critical to the understanding of emotion dysregulation because it mediates the effects of trauma on other factors related to emotion dysregulation (e.g., distress intolerance; Fang & Chung, 2019; Gaher, Arens, & Shishido, 2015; Gaher, Hofman, Simons, & Hunsaker, 2013). Preliminary evidence suggests that this mediation effect may have especially notable implications for understanding borderline personality disorder. In a nonclinical sample of

young adults, alexithymia, negative urgency, and distress tolerance mediated the association between trauma and borderline symptoms, with alexithymia mediating effects of trauma on negative urgency and distress tolerance (Gaher et al., 2013).

Individuals with BPD and/or significant emotion dysregulation also often experience their emotions as intense, unpredictable, and in need of avoidance (Holm & Severinsson, 2011; Kreisman & Straus, 2010; Linehan, 1993; Spodenkiewicz et al., 2013). Correspondingly, affective lability is common among persons with BPD (Reich, Zanarini, & Fitzmaurice, 2012; Silvers et al., 2016; Trull et al., 2008) and positively associated with BPD symptom severity (Links et al., 2007; Wedig et al., 2012). Like emotion dysregulation, childhood adversity likely contributes to the development of affective lability (Kim-Spoon, Cicchetti, & Rogosch, 2013; Shields & Cicchetti, 1998).

Lastly, individuals with BPD often display disrupted, impulsive cognitive and behavioral reactions, particularly in response to emotional experiences (American Psychiatric Association, 2013). Impulsivity is a core feature of BPD (Barker et al., 2015; Henry et al., 2001; Links, Heslegrave, & van Reekum, 1999) and manifests as poor self-control, nonplanning, behavioral reactivity, and/or inattention (Patton, Stanford, & Barratt, 1995). Like alexithymia and affective lability, impulsivity is closely associated with experiences of childhood adversity (Roy, 2005; Shin, Lee, Jeon, & Wills, 2015; Sujan, Humphreys, Ray, & Lee, 2014), although genetics also appears to be a strong contributor (Arce & Santisteban, 2006).

CURRENT RESEARCH

Previous research suggests that the relation between early interpersonal trauma and BPD may be at least partially explained by emotion dysregulation. However, reliance on nonclinical samples raises questions of clinical implications, and it remains unclear how alexithymia, affective lability, and impulsivity may also play a role in these mediational relationships. To address this gap in the literature, the current research examined alexithymia, affective lability, and impulsivity as potential mediators of the relation between childhood adversity and BPD diagnosis. Competing models, each informed by the literature, were compared using path analysis to best understand relations between these constructs. Consistent with previous research, we hypothesized:

1. Greater severities of alexithymia, affective lability, impulsivity, and childhood adversity would be associated with BPD diagnosis.
2. Alexithymia, affective lability, and impulsivity would each at least partially mediate relations between childhood adversity and BPD diagnosis.
3. Alexithymia would at least partially mediate effects of childhood adversity on affective lability and impulsivity.

METHOD

Participants

The sample included 196 individuals, including 101 with BPD and 95 healthy controls (HC) recruited as part of a larger program of research. Significant heterogeneity in age (M = 35.19, SD = 11.28, range = 18–65), race (45% White, 32% Black, 13% Asian, 6% Mixed Race), and years of education (M = 14.93, SD = 2.76, range = 3–24) were noted across the sample. Alexithymia data from a subset of these participants have been published elsewhere (e.g., New et al., 2012); nevertheless, the current study represents a novel analysis of such data by including different variables than those examined in previous work. See Table 1 for a summary of participant demographic information.

Participants were recruited through advertisements in local newspapers and Internet postings (e.g., Craigslist). To support recruitment of both individuals with BPD and HCs, multiple advertisements were used (e.g., advertisements noting difficulties with intense emotion targeted recruitment of individuals with BPD, whereas more generalized advertisements targeted HCs). Less than 10% of the individuals with BPD were recruited by referrals from the outpatient mental health clinics at Mount Sinai Hospital. All assessments were conducted at the Mood and Personality Research Program offices at Mount Sinai Hospital. All patients were free of any psychotropic medication for at least 2 weeks (6 weeks for fluoxetine) before the assessment, and most were never previously medicated. Exclusion criteria included history of schizophrenia or schizoaffective disorder, bipolar disorder type I, head trauma with loss of consciousness, neurological disease, organic mental syndrome, intellectual disability disorder, current substance use disorder (occurring within the past 3 months), or current major depressive episode (occurring within the past 3 months). Additional exclusion criteria for HC participants included personal history of any Axis I or personality disorder and first-degree family history of psychotic disorders.

MEASURES AND PROCEDURE

Diagnosis of Borderline Personality Disorder. All participants received a structured diagnostic interview administered by a clinical psychologist with expertise in evaluation of personality disorders using the Structured Clinical Interview for *DSM-IV* (SCID-IV; First, Spitzer, Gibbon, & Williams, 2002) and the Structured Interview for *DSM-IV* Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997). Our group has achieved an interrater reliability of k = 0.80–0.81 for the diagnosis of BPD (e.g., Goodman et al., 2014), as was true for the current sample.

Following the diagnostic interview, the participants completed the Childhood Trauma Questionnaire, the Toronto Alexithymia Scale-20, the Affective Lability Scale, and the Barratt Impulsiveness Scale as part of a larger study. Participants were provided monetary compensation for their time and travel. All procedures were approved by the Institutional Review Board at Icahn School of Medicine at Mount Sinai.

TABLE 1. Demographic and Clinical Measures

	Subjects With Borderline Personality Disorder (BPD) (*n* = 101)		Healthy Controls (*n* = 95)		
	M* or *n	***SD* or %**	***M* or *N***	***SD* or %**	**Statistical Comparison**
Age	34.35	11.31	34.25	11.32	$t(194) = .06$, $p = .95$, $d = 0.01$
Gender (female)	65	64.36%	55	57.89%	χ^2 (1) = 0.86, $p = .35$
Race (minority)	54	53.47%	53	55.79%	χ^2 (1) = 0.11, $p = .74$
Years of Education	14.13	2.68	15.83	2.58	t (189) = −4.46, $p < .01$, $d = 0.65$
CTQ	60.79	16.58	43.64	8.79	t (194) = 8.97, $p < .01$, $d = 1.29$
TAS-20	52.18	13.20	35.72	9.57	t (194) = 9.94, $p < .01$, $d = 1.43$
ALS	84.42	31.79	22.43	20.74	t (183) = 15.53, $p < .01$, $d = 2.31$
BIS	73.44	11.83	54.65	10.51	t (194) = 11.73, $p < .01$, $d = 1.68$

Note. CTQ = Childhood Trauma Questionnaire; TAS-20 = Toronto Alexithymia Scale-20; ALS = Affective Lability Scale; BIS = Barratt Impulsivity Scale.

Self-Report Measures. The Childhood Trauma Questionnaire-Short Form (CTQ-SF; Bernstein et al., 2003) was used to assess experiences of childhood adversity. The CTQ is a 28-item retrospective self-report assessment of physical abuse (e.g., "I was punished with a belt, a board, a cord, or some other hard object"), emotional abuse (e.g., "People in my family said hurtful or insulting things to me"), sexual abuse (e.g., "Someone tried to make me do sexual things or watch sexual things"), emotional neglect (e.g., "I felt loved"), and physical neglect (e.g., "I didn't have enough to eat") occurring during childhood. It has demonstrated strong test–retest reliability, internal consistency, convergent validity, and discriminant validity across samples (Bernstein et al., 1994; Scher, Stein, Asmundson, McCreary, & Forde, 2001). Internal reliability in the current sample was α = 0.87.

The Toronto Alexithymia Scale-20 (TAS-20; Bagby et al., 1994) was employed to assess alexithymia. Commonly considered the gold standard in alexithymia assessment (Kooiman, Spinhoven, & Trijsburg, 2002), this 20-item self-report questionnaire assesses the difficulties identifying feelings (e.g., "I am often confused about what emotion I am feeling"), difficulties describing feelings (e.g., "It is difficult for me to find the right words for my feelings"), and externally oriented thinking (e.g., "I prefer to analyze problems rather than just describe them") aspects of alexithymia. The TAS-20 has demonstrated strong concurrent and discriminant validity, test–retest reliability, and internal reliability across populations and contexts (Kooiman et al., 2002; Taylor, Bagby, & Parker, 2003). In the current sample, α = 0.89.

The Affective Lability Scale (ALS; Harvey, Greenberg, & Serper, 1989) was used to assess affective lability. The ALS is a 54-item self-report measure of rapidly shifting mood, focusing specifically on depression, elation, anxiety, and anger (e.g., "One minute I can be feeling OK and then I feel tense, jittery, and nervous"). It has demonstrated strong test–retest reliability, internal reliability, and construct validity in both clinical and nonclinical samples (Aas et al., 2015; Harvey et al., 1989). In the current sample, α = 0.98.

The Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995) was used to examine impulsivity. This self-report questionnaire includes 30 items assessing attention, motor impulsiveness, self-control, cognitive complexity, perseverance, and cognitive instability (e.g., "I do things without thinking"). The BIS-11 is the most widely used self-report measure of impulsivity (Stanford et al., 2009). Across contexts and populations, it has demonstrated strong test–retest reliability, internal consistency, and convergent validity with other self-report measures (but not behavioral measures; Stanford et al., 2009). Internal reliability for the current sample was $\alpha = 0.91$.

Statistical Analysis

Path analysis using the lavaan package for R (v 3.4.4; R Core Team, 2013) was then used to explore the potential mediating roles of alexithymia, affective lability, and impulsivity on the association between childhood adversity and BPD (to preserve statistical power, only scale total scores were used in analyses). Post hoc power analyses using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) suggested that the sample size provided adequate statistical power for regression analyses to detect medium-sized effects ($1\text{-}\beta > 0.99$, $\alpha = .05$, effect size $f^2 = 0.15$). To best understand these potential relations, three models, each informed by the previous literature, were compared:

A. Consistent with correlational studies (e.g., Barker et al., 2015; Kaplan et al., 2016; New et al., 2012, Reich et al., 2012), Model A assessed childhood adversity, alexithymia, affective lability, and impulsivity independently predicting BPD with no mediating relationships.
B. Consistent with mediational analyses suggesting emotion dysregulation to mediate the relation of childhood adversity to psychopathology (e.g., Huh et al., 2017; Peh et al., 2017), Model B assessed alexithymia, affective lability, and impulsivity fully mediating the association between childhood adversity and BPD.
C. Consistent with research suggesting alexithymia to mediate the relation of childhood adversity to other factors associated with emotion dysregulation (e.g., Fang & Chung, 2018), Model C assessed affective lability and impulsivity fully mediating relations of childhood adversity on BPD, with alexithymia partially mediating relations of adversity to affective lability and impulsivity.

For each model, global and local fit statistics were analyzed to determine goodness of fit of the model to the data. Estimated indirect effects were also calculated to better understand potential mediating relationships. Lastly, chi-square difference tests were used to compare models and determine which model was a best fit to the data.

RESULTS

Consistent with common comorbidities of BPD, many participants with BPD also met criteria for a range of other psychiatric diagnoses. The most common

comorbid diagnoses included intermittent explosive disorder (n = 64), major depressive disorder (past; most recent episode >3 months prior to study participation; n = 39), alcohol use disorder (n = 37), substance use disorder (past; no substance use diagnosis during the past 3 months; n = 29), and posttraumatic stress disorder (n = 26). Exploratory t tests and chi-square analyses suggested that participants with BPD and HCs were comparable in terms of age, gender, and race (all p values ≥ .35). Participants with BPD had slightly less education than HCs, $t(189) = -4.46$, $p < .01$, $d = 0.65$. See Table 1 for a summary of these demographic comparisons. A multivariate ANOVA also suggested significant differences between participants with BPD versus HCs in study measures, Wilks's $\Lambda = 0.45$, $F(4, 180) = 66.08$, $p < .01$. Follow-up univariate analyses suggested that participants with BPD reported significantly more severe alexithymia, affective lability, impulsivity, and histories of early-life interpersonal trauma than HCs (all ps < .01, ds = 1.29–2.31; Table 1). Diagnostic tests suggested no notable violations of regression assumptions (i.e., linearity, normality of residuals, homoscedasticity, independence of residuals) and no significant instances of multicollinearity (all variance inflation factors (VIFs) < 2.75). Data missingness was minimal (1.15%); therefore, listwise deletion was used throughout analyses to account for missing data.

Path Analysis

Correlational analyses suggested strong associations between assessed aspects of emotion dysregulation (i.e., alexithymia, affective lability, and impulsivity; see Table 2). Path analysis using maximum likelihood estimation further explored these associations in accordance with the proposed models. Given group differences in education level, education level was included as a covariate in all analyses. Model A tested whether childhood adversity, alexithymia, affective lability, and impulsivity each independently predicted BPD diagnosis with no mediating relationships between variables. Consistent with hypotheses, global fit statistics suggested that this model was not a good fit to the data, $\chi^2(6, N = 196) = 367.45$, $p < .01$, CFI = 0.38, RMSEA = 0.55, 90% CI [0.51, 0.60], SRMR = 0.35. Local fit statistics similarly suggested that this model underestimated all associations between variables (correlation residuals = 0.09–0.69).

Model B tested a simple mediation model wherein alexithymia, affective lability, and impulsivity fully mediated the relation of childhood adversity to BPD diagnosis. Global fit statistics reflected only marginal improvement in comparison to Model A and suggested that Model B was also not a good fit to the data, $\chi^2(4, N = 196) = 203.32$, $p < .01$, CFI = 0.66, RMSEA = 0.50, 90% CI [0.45, 0.56], SRMR = 0.19. Local fit statistics suggested that Model B showed good fit with regard to childhood adversity (correlation residuals = 0.00–0.03), but underestimated associations between other variables (correlation residuals = 0.04–0.45). Estimated indirect effects suggested that both affective lability and impulsivity significantly mediated the relation of adversity to BPD diagnosis.

Lastly, Model C tested whether affective lability and impulsivity fully mediated the relation of childhood adversity to BPD diagnosis, with

TABLE 2. Correlation Statistics

	Childhood Adversity	Alexithymia	Affective Lability	Impulsivity
Childhood Adversity	—			
Alexithymia	.51 (< .01)	—		
Affective Lability	.58 (< .01)	.67 (< .01)	—	
Impulsivity	.44 (< .01)	.69 (< .01)	.74 (< .01)	—
n	196	196	185	196

alexithymia partially mediating the relations of adversity to affective lability and impulsivity. Global fit statistics reflected a notable improvement in model fit in comparison to Models A and B and suggested that Model C was a good fit to the data, $\chi^2(2, N = 196) = 2.51$, $p = .29$, CFI = 1.00, RMSEA = 0.04, 90% CI [0.00, 0.15], SRMR = 0.01. Local fit statistics also showed good fit at the local level (correlation residuals = 0.00–0.05). All paths were positive and statistically significant at $\alpha = .05$. The model accounted for 60% of the variance in BPD diagnosis, 53% in affective lability, 51% in impulsivity, and 25% in alexithymia. Estimated indirect effects suggested that alexithymia significantly mediated the relationship between childhood adversity and affective lability and between adversity and impulsivity; affective lability significantly mediated the relationship between adversity and BPD diagnosis and between alexithymia and BPD diagnosis; and impulsivity significantly mediated the relationship between alexithymia and BPD diagnosis. Impulsivity did not, however, significantly mediate the relationship between adversity and BPD diagnosis. See Figure 1 for a graphical representation of Model C and Table 3 for further information about model parameters and mediation analyses.

Model comparisons using chi-square difference tests suggested that Model C best fit the data of the current sample: comparing Model A to Model B, $\chi^2(2) = 164.13$, $p < .01$; comparing Model A to Model C, $\chi^2(4) = 364.94$, $p < .01$; comparing Model B to Model C, $\chi^2(2) = 200.81$, $p < .01$. See Table 3 for a summary of statistics for each model.

DISCUSSION

The main findings of this study are that (a) affective lability mediated the relation of childhood adversity to BPD diagnosis, and (b) alexithymia partially mediated the relation of childhood adversity to other aspects of emotion dysregulation. This is consistent with growing research suggesting that emotion dysregulation mediates the relation of childhood adversity to outcomes closely related to BPD and that alexithymia may play a notable role in these mediational relationships (e.g., Burns et al., 2012; Fossati et al., 2015; Gaher et al., 2013; Hopfinger et al., 2016; Huh et al., 2017; Jennissen et al., 2016; Kuo et al., 2015; Peh et al., 2017). Although childhood adversity is not the only factor contributing to the development of BPD (genetics also appears

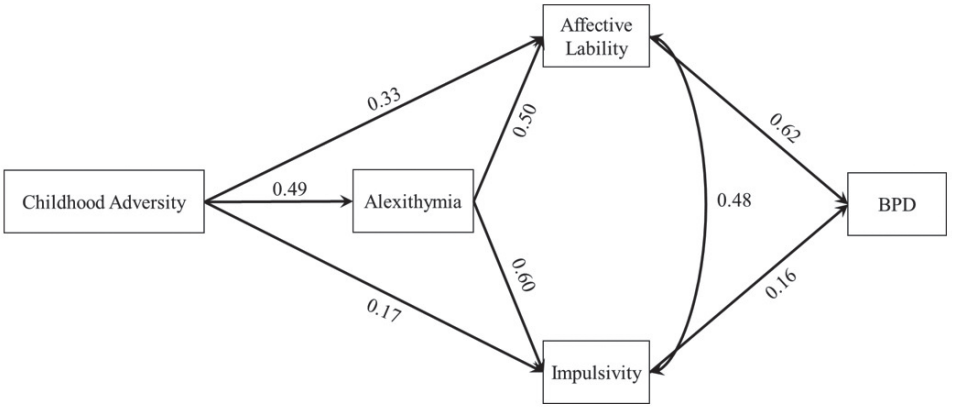

FIGURE 1. Model C. Due to the cross-sectional nature, results cannot be used to inform causal inference. Directionality of arrows in the figure is therefore purely theoretical and informed by previous research rather than by the current study design.

to play a key role; Amad et al., 2014), clarifying the nature of associations between childhood adversity and BPD could have direct implications for treatment. The current study is cross-sectional and therefore cannot inform causal models of this association; however, observed mediational relationships suggest that maintaining and/or developing healthy emotion regulation after childhood adversity may be key to minimizing the effects of adversity on the risk of developing BPD. Early interventions to develop and strengthen the emotion regulation of maltreated children may be helpful in this regard (e.g., Mazza, Dexter-Mazza, Miller, Rathus, & Murphy, 2016; Zenner, Herrnleben-Kurz, & Walach, 2014).

Results also suggest that affective lability may play a notable role in explaining these associations. Although all assessed factors (i.e., alexithymia, affective lability, and impulsivity) were significantly related to both a BPD diagnosis and severity of childhood adversity, affective lability showed notably stronger conditional associations with BPD and adversity in path analyses (see Table 3). By comparison, conditional effects of alexithymia and impulsivity were less pronounced. These findings suggest that affective lability may be more central to distinguishing individuals with BPD from healthy controls than disruptions in processing and/or responding to emotional experiences (i.e., alexithymia and impulsivity, respectively). This central function echoes characterizations of BPD as tending toward intense, labile emotional reactions (Holm & Severinsson, 2011; Kreisman & Straus, 2010; Linehan, 1993; Spodenkiewicz et al., 2013; Trull & Carpenter, 2014) and the emphasis of BPD-focused treatment programs on reducing emotional instability (e.g., Bateman & Fonagy, 2006; Linehan, 1993). Further research is needed to replicate and clarify this role of affective lability within the broader context of emotion dysregulation and BPD.

TABLE 3. Model Comparisons

	Model A (Independent Direct Effects)	Model B (Simple Mediation)	Model C (Complex Mediation)
Parameters			
CTQ → Diagnosis	$b = 0.10$, $p = .07$	—	—
TAS20 → Diagnosis	$b = 0.02$, $p = .67$	$b = 0.04$, $p = .47$	—
ALS → Diagnosis	$b = 0.64$, $p < .01$	$b = 0.64$, $p < .01$	$b = 0.62$, $p < .01$
BIS → Diagnosis	$b = 0.15$, $p < .01$	$b = 0.15$, $p < .01$	$b = 0.16$, $p = .02$
CTQ → TAS20	—	$b = 0.49$, $p < .01$	$b = 0.49$, $p < .01$
CTQ → ALS	—	$b = 0.58$, $p < .01$	$b = 0.33$, $p < .01$
CTQ → BIS	—	$b = 0.47$, $p < .01$	$b = 0.17$, $p < .01$
TAS20 → ALS	—	—	$b = 0.50$, $p < .01$
TAS20 → BIS	—	—	$b = 0.60$, $p < .01$
ALS → BIS	—	—	$b = 0.48$, p < .01
Variance Explained			
Diagnosis	$R^2 = 0.52$, $p < .01$	$R^2 = 0.56$, $p < .01$	$R^2 = 0.60$, $p < .01$
CTQ	$R^2 = 0.11$, $p < .01$	$R^2 = 0.11$, $p < .01$	$R^2 = 0.11$, $p < .01$
TAS20	$R^2 = 0.04$, $p < .01$	$R^2 = 0.25$, $p < .01$	$R^2 = 0.25$, $p < .01$
ALS	$R^2 = 0.15$, $p < .01$	$R^2 = 0.34$, $p < .01$	$R^2 = 0.53$, $p < .01$
BIS	$R^2 = 0.05$, $p < .01$	$R^2 = 0.24$, $p < .01$	$R^2 = 0.51$, $p < .01$
Mediation Effects			
TAS20 on CTQ → Diagnosis	—	$b = .02$, $p = .47$	—
ALS on CTQ → Diagnosis	—	$b = .37$, $p < .01$	$b = .20$, $p < .01$
BIS on CTQ → Diagnosis	—	$b = .07$, $p < .01$	$b = .03$, $p = .07$
TAS on CTQ → ALS	—	—	$b = .25$, $p < .01$
TAS on CTQ → BIS	—	—	$b = .29$, $p < .01$
ALS on TAS → Diagnosis	—	—	$b = .31$, $p < .01$
BIS on TAS → Diagnosis	—	—	$b = .09$, $p = .02$
Fit Statistics			
χ^2	367.45, $p < .01$	203.32, $p < .01$	2.51, $p = .29$
CFI	0.38	0.66	1.00
RMSEA	0.55	0.50	0.04
SRMR	0.35	0.19	0.01

Note. CTQ = Childhood Trauma Questionnaire; TAS20 = Toronto Alexithymia Scale-20; ALS = Affective Lability Scale; BIS = Barratt Impulsiveness Scale; CFI = comparative fit index; RMSEA = root-mean square error of approximation; SRMR = standardized root mean square residual.

The role of alexithymia in observed mediational relationships appears unique. Consistent with hypotheses and previous research (e.g., Fang & Chung, 2018; Gaher et al., 2013, 2015), alexithymia partially mediated the relation of childhood adversity to other factors related to emotion dysregulation. Fundamentally, alexithymia is an impairment in processing internal emotion cues (Lane, 2020). Because these cues provide necessary information to guide, control, and regulate behavior according to situational demands (Baumeister, Vohs, DeWall, & Zhang, 2007), restricted access to such information—as

occurs in alexithymia—results in various forms of emotion dysregulation as observed in the current study. Findings suggest that histories of childhood adversity in BPD are associated with affective lability and impulsivity because of this restriction to emotional information. This may at least partially explain why treatments that increase attention to and processing of internal emotional cues (e.g., those based in mindfulness) are helpful for individuals with BPD. These mediation relationships are consistent with previous research, including models of subclinical borderline symptoms (e.g., Fang & Chung, 2018; Gaher et al., 2013, 2015). Such similarity in results across studies and populations suggests that the mediational role of alexithymia may extend across diagnoses and to both clinical and nonclinical populations. Findings are also consistent with evidence highlighting alexithymia as a necessary target in the treatment of trauma-related disorders and trauma-exposed persons (Berke et al., 2017; Hyer, Woods, & Boudewyns, 1991; O'Brien, Gaher, Pope, & Smiley, 2008; Zorzella, Muller, Cribbie, Bambrah, & Classen, 2020). Although treatment of alexithymia is often difficult for clinicians (O'Brien et al., 2008; Ogrodniczuk, Piper, & Joyce, 2011), recent findings suggest that psychoeducational and emotion-focused interventions may be effective in reducing alexithymic trait severity (Cameron, Ogrodniczuk, & Hadjipavlou, 2014; Edwards, Shivaji, & Wupperman, 2018; McMurran & Jinks, 2012). Given the role of alexithymia in mediating the relation of childhood adversity to other factors related to emotion dysregulation and, by extension, BPD, future research may examine the utility of alexithymia-focused interventions in the treatment and prevention of BPD.

Our results invite discussion about potential commonalities and differences between BPD and other types of psychopathology. For example, similar to persons with BPD, individuals with psychotic, depressive, eating, and trauma disorders display substantial degrees of alexithymia (Edwards, 2019; Kimhy et al., 2012; Kimhy et al., 2016; Li, Zhang, Guo, & Zhang, 2015; De Panfilis, Rabbaglio, Rossi, Zita, & Maggini, 2003) along with increased prevalence of childhood trauma and adversity (Brewin, Andrews, & Valentine, 2000; Hund & Espelage, 2006; Krabbendam, 2008; Mandelli, Petrelli, & Serretti, 2015; Morgan & Fisher, 2007; Thompson et al., 2009). Although preliminary, some evidence also suggests that alexithymia may play a similar mediating role for these disorders (Berenbaum, Valera, & Kerns, 2003; Güleç et al., 2013; Hund & Espelage, 2006; O'Brien et al., 2008). Future research should therefore investigate the extent to which results of the current study may also apply to other forms of psychopathology. It would be useful for future work to compare individuals with BPD to a clinical control group. Such comparisons could clarify the extent to which observed relationships are specific to BPD or reflective of broader patterns in psychopathology.

The current study has many strengths, most notably inclusion of a large sample of carefully diagnosed individuals with BPD. Nevertheless, results should be considered in light of a few methodological limitations. First, although the sample was prudently vetted to exclude potential confounding diagnoses, including active depression and a history of substance use, this vetting may also limit generalizability of findings to treatment settings. Typically, treatment-seeking individuals who meet criteria for BPD also meet

criteria for at least three comorbid psychiatric conditions (Zimmerman & Mattia, 1999). Future research may therefore benefit from including a more diagnostically heterogeneous sample. Second, although etiological and developmental theories imply temporal order of effects, the current study used a cross-sectional design. Thus, findings cannot be used to imply or inform theories of temporal order or causal direction of observed effects. Relatedly, some evidence suggests that mediation analyses risk overestimating effects when using cross-sectional data (Maxwell & Cole, 2007). However, the present findings inform our understanding of the associations between childhood adversity, emotion regulation, and a BPD diagnosis. Longitudinal research is needed to investigate whether these models also reflect development of BPD in individuals over time. Third, strong reliance on use of self-report measures may have introduced issues of biased responding and/or method variance. Some research suggests that the limitations of self-report may be particularly salient for measures of trauma (Wilson & Keane, 2004) and alexithymia (Derks et al., 2017). Future research should therefore consider integrating laboratory, behavioral, and/or clinician-administered measures to provide a more complete perspective on mediating factors. Fourth, the study's sample size, while large for a clinical sample, limited statistical power to test more complex models, such as those controlling for potential confounds (e.g., comorbid conditions) or identifying latent variables that may be driving observed effects (e.g., different types of childhood trauma, subfactors of alexithymia, etc.). Future research may investigate these more complex models by employing larger samples. Lastly, the study focused on a narrow subset of constructs related to emotion dysregulation. It is likely that other factors, such as experiential avoidance, mindfulness, and use of specific regulation strategies, also play important roles in mediating the relation between trauma and BPD. Further research is needed to clarify these roles.

CONCLUSIONS

Theory and research have long suggested that experiences of childhood adversity are key to the development of BPD (e.g., Fonagy & Luyten, 2009; Linehan, 1993). However, only limited research has investigated potential mediating factors underlying the relation of childhood adversity to BPD. The current study replicates previous findings by offering further evidence to suggest a strong association between childhood adversity and BPD diagnosis. Extending previous findings, results also support a complex mediation model in which alexithymia, affective lability, and impulsivity mediate the association between childhood adversity and BPD, and alexithymia partially mediates the association of adversity to other aspects of emotion dysregulation.

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