

# The Protective Role of Attachment Security for Adolescent Borderline Personality Disorder Features via Enhanced Positive Emotion Regulation Strategies

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While studies have documented significant associations between insecure attachment, emotion dysregulation, and borderline personality disorder (BPD) features, no research to date has empirically delineated the specific mechanisms by which these constructs are related. The present study brings together 2 lines of research that have hitherto separately examined attachment disturbance and emotion dysregulation as they respectively manifest in the pathogenesis of BPD, and explores the complex relations between the 2 well-established correlates of borderline traits in a clinical sample of adolescents ( $N = 228$ ). We examined the adolescents' use of positive and negative emotion regulation strategies, along with their maternal and paternal attachment security. Results indicated that positive and negative emotion regulation strategies were differentially implicated in the link between attachment insecurity and BPD features. Attachment security functioned as a buffer against adolescent BPD by enhancing positive emotion regulation strategies, while negative emotion regulation strategies served to dilute the protective effect of attachment and positive regulation strategies, culminating in clinically significant levels of borderline traits. Findings are discussed with regard to interventions in the developmental trajectory of BPD as it unfolds during adolescence.

*Keywords:* borderline personality disorder, attachment, emotion dysregulation, adolescence, mediated moderation

Emotional and interpersonal dysregulation are understood to constitute the central features of borderline personality disorder (BPD). Since the birth of the diagnosis, the field has documented severe disturbances in interpersonal relatedness in BPD in addition to pervasive deficits in emotion regulation (Grinker, Werble, & Drye, 1968; Gunderson & Singer, 1975; King-Casas et al., 2008; Knight & Friedman, 1954; Stern, 1938). These observations have given rise to theories that underscore the role of attachment disturbance in the pathogenesis of BPD (Fonagy, Target, Gergely, Allen, & Bateman, 2003; Kernberg, 1967), along with those that locate borderline phenomenology on the continuum of biologically

based disorders of the affective system (Akiskal et al., 1985; Linehan, 1993; Stone, 1980).

These related but separate lines of conceptual work were formalized in the constellation of symptoms that came to comprise current diagnostic criteria, with factor analytic studies of the *DSM-III-R* and *IV* diagnostic criteria (Clarkin, Hull, & Hurt, 1993; Sanislow et al., 2002) suggesting that interpersonal, affective, and behavioral clusters underlie BPD. A growing literature holds that the complex bidirectional and interactive pathways between the affective and interpersonal dimensions account for an array of phenomenological observations and etiological hypotheses that have thus far been proposed (Fonagy & Luyten, 2009; Linehan, 1993).

Empirical literature to date has explored the role of attachment disturbance and emotion dysregulation separately as they relate to BPD. Concomitant manifestations of BPD and insecure attachment have been well represented in both interview-based (Fonagy et al., 1996) and self-report data (Dutton, Saunders, & Starzomski, 1994). Data converge to point to BPD's strong association with unresolved/fearful attachment, followed by preoccupied attachment, as well as its robust inverse relationship with secure attachment (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004; Levy, Beeney, & Temes, 2011). High prevalence of what Lyons-Ruth, Melnick, Patrick, and Hobson (2007) termed hostile-helpless attachment in BPD further corroborates these patients' contradictory and malevolent internal representations of caregivers, a finding

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This article was published Online First December 23, 2013.

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This research was supported by the Child and Family Program of the Menninger Clinic.

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consistent with patients' retrospective self-reports (Zanarini et al., 2000). Prospective longitudinal studies have also complemented the cross-sectional evidence: early caregiving variables (e.g., maternal inconsistency, hostility) emerged as potent predictors of adolescent and adult BPD; attachment disturbance in infancy and adolescence was also shown to predict BPD symptoms in adulthood (Bezirgianian, Cohen, & Brook, 1993; Carlson, Egeland, & Sroufe, 2009). The role of attachment in the ontogeny of BPD has been particularly well articulated in the mentalization model of BPD (Fonagy & Luyten, 2009; Sharp & Fonagy, 2008), which links failures in attachment to deficits in mentalization (i.e., the capacity for affectively enriched understanding of self, others, and the social world) that are seen to characterize BPD.

Studies of emotion dysregulation in BPD have followed from Linehan's (1993) conceptualization and highlighted BPD patients' lack of emotional awareness (Levine, Marziali, & Hood, 1997), heightened negative emotional reactivity (Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007), dampened positive emotional reactivity (Sadikaj, Russell, Moskowitz, & Paris, 2010), affect intensity (Yen, Zlotnick, & Costello, 2002), and delayed recovery to baseline (Reisch, Ebner-Priemer, Tschacher, Bohus, & Linehan, 2008). Fronto-limbic abnormalities (Schmahl & Bremner, 2006) have been implicated in the observed profile, as well as altered functions of serotonergic (Ni, Chan, Chan, McMain, & Kennedy, 2009) and dopaminergic (Friedel, 2004) neurotransmitter systems. The pool of cognitive emotion regulation strategies routinely employed by BPD patients is seen to further perpetuate and propel their disturbed emotional profile (Conklin, Bradley, & Westen, 2006; Linehan, 1993). Specifically, BPD has been associated with low levels of acceptance (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006; Schramm, Venta, & Sharp, 2013) and cognitive reappraisal (Koenigsberg et al., 2009), while linked to high levels of rumination (Smith, Grandin, Alloy, & Abramson, 2006), catastrophization (Selby, Anestis, Bender, & Joiner, 2009), suppression (Rosenthal, Cheavens, Lejuez, & Lynch, 2005), avoidance (Chapman, Dixon-Gordon, & Walters, 2011), and internalization/externalization of blame (Conklin et al., 2006). Noteworthy in this regard is the emotional cascade model (Selby et al., 2009), which located rumination at the heart of the BPD syndrome. The model proposes that rumination (and catastrophization as future-oriented rumination) potentiates the magnitude of negative affect which, in sequence, amplifies the level of rumination, initiating a vicious, self-perpetuating cascade of negative emotions from which a full syndrome of BPD emerges.

While attachment and emotion regulation have established their individual robust links to BPD, data is sparse and inconclusive concerning the nature of the interplay between the two constructs in relation to the disorder. Accordingly, the field has long recognized attachment disturbance and emotion dysregulation as precursors of BPD without demonstrating the capacity to explicate the relationship between the two factors. This paucity of research is striking given the importance that this bears on the development of effective prevention and intervention strategies. Only a few recent studies have started to tackle the issue: Scott, Levy, and Pincus (2009) reported that traits of negative affect (defined as depression, angry hostility, and anxiety) and impulsivity mediated the link between attachment and BPD in a nonclinical sample of adults. Fossati et al. (2005) found a similar effect of mediation for impulsive and aggressive traits using a sample of adult outpatients.

Morse et al. (2009), in an exploratory analysis of adult outpatients, documented that a subtype of BPD was characterized by an interaction between temperamental anger and preoccupied attachment. Despite marking the first attempts to elucidate the interrelationships between interpersonal and affective dimensions of BPD, these studies focused on trait/dispositional variations (e.g., depression, anger, impulsivity) and hence on the temperamental variability underlying emotion dysregulation, not on emotion dysregulation per se (see John and Gross (2004) for a clearer demarcation between temperament, emotion, and emotion regulation). No study has yet delineated how specific aspects of regulatory phenomena, processes, or strategies work in concert with or in opposition to attachment variables to culminate in BPD. Studies that directly capture the manner(s) in which one monitors, modulates, and manages emotions (Gross & Thompson, 2007) are called for to unravel such mechanisms of interplay. To our knowledge, the only work to date in this area is that of Cheavens et al. (2005), in which thought suppression (as a regulatory strategy) partially mediated the link between perceived parental criticism and BPD.

The present cross-sectional study explored the mechanisms of interplay between attachment disturbance and emotion dysregulation as they relate to BPD traits in a clinical sample of adolescents. We specifically adopted a trait-based approach, in line with recent dimensional conceptualizations of BPD. The current study sought to extend the small body of extant research cited above, but moved beyond the previous work in three important ways. First, we examined both maternal and paternal attachment security in relation to BPD. The role of fathers in the ontogeny of BPD is mostly unknown. Maternal attachment has received more attention from classic psychoanalytic theories (e.g., Masterson & Rinsley, 1975), centering on Mahler's theory of separation-individuation (Mahler & Kaplan, 1977). Recent studies have started to include a focus on the unique role of paternal attachment in the child's developmental outcomes (Grossmann, Grossmann, Kindler, & Zimmermann, 2008). We examined paternal attachment alongside maternal attachment in efforts to add to this growing body of literature.

Second, to elucidate the differential profiles of adaptive and maladaptive emotion regulation in the pathways between attachment and BPD, we examined two classes of cognitive emotion regulation strategies, the routine use of which has been differentially linked to psychopathology. Several emotion regulation strategies have received attention for their consistent associations with good health outcomes, while others have been recognized for their links to psychopathology. Included under the former are cognitive reappraisal (John & Gross, 2004) and acceptance (Hayes, Strosahl, & Wilson, 1999), as well as other strategies that involve adaptive shifts of attention (e.g., positive refocusing, refocus on planning, putting into perspective; Garnefski, Kraaij, & Spinhoven, 2002). Conversely, rumination, catastrophization, and internalization/externalization of blame (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Garnefski et al., 2002) have come under the latter heading. We created and examined two composite variables, positive emotion regulation strategies and negative emotion regulation strategies, to contrast the differential profiles of the two classes of emotion regulation strategies as they relate to BPD.

Third, the present research utilized a clinical sample of adolescents and extended the investigation to a younger age group wherein initial vulnerability to BPD may appear. Despite the controversies surrounding the diagnosis of personality disorders in

adolescence (Miller, Muehlenkamp, & Jacobson, 2008), distinct features of BPD have been reported to emerge during adolescence (Sharp & Bleiberg, 2007). With growing evidence lending support to the reliability and validity of adolescent-onset BPD (Miller et al., 2008) and the development of valid tools for the assessment of BPD in adolescents (Michonski, Sharp, Steinberg, & Zanarini, 2013; Sharp, Ha, Michonski, Venta, & Carbone, 2012), this age group is considered apropos for the study of early trajectories of BPD. Some strides (e.g., Gratz et al., 2009; Sharp et al., 2011) have recently been made in adding to the scant extant literature on the developmental precursors of BPD; we attempted to further this endeavor.

To inform our hypotheses, we rely on the theoretical accounts of BPD discussed above, all of which emphasize the links between attachment, emotion dysregulation, and BPD features (Fonagy & Luyten, 2009; Linehan, 1993; Selby et al., 2009). To recapitulate, attachment theory (Bowlby, 1982/1969) provides one (of several) conceptual approaches to understanding the capacity to regulate emotions. It suggests that proximity to and responsiveness of attachment figures undergird the child's emotional equanimity, while suboptimal dyadic experiences between infant and caregiver disrupt the optimal development of the child's regulatory strategies (Contreras, Kerns, Weimer, Gentzler, & Tomich, 2000; Sroufe, 2005). In cases of severe disruption, it is thought that BPD features may develop as suggested by both Fonagy and Luyten's (2009) mentalization model and Linehan's (1993) biosocial model. Building on these theoretical models, our first aim was to test the hypothesis that the link between attachment insecurity and BPD features is mediated by elevated levels of negative emotion regulation strategies as well as decreased levels of positive regulation strategies. While emotion dysregulation is seen as a mediator in Fonagy's and Linehan's models, it is best described as a moderator in Selby and colleagues' (2009) model, where the effects of attachment insecurity are amplified in the presence of disturbances in emotion regulation. To test this possibility, we further aimed to test the moderational role of emotion regulation variables in the relation between attachment insecurity and BPD features. Finally, because emotion dysregulation may capture both mediational and moderational functions, we expected that post hoc mediated moderation analyses would be applied to simultaneously examine the mediating and moderating relations respectively proposed by the theoretical accounts described above. Taken together, our aim was to provide a coherent picture elucidating complex relations between attachment, adaptive and maladaptive emotion regulation, and BPD features.

## Method

### Participants

Data were collected from 275 consecutive voluntary admissions to the Adolescent Treatment Program of a private tertiary care inpatient treatment facility specializing in adolescents who had failed to respond to previous interventions. Thirty-nine adolescents were excluded from the analyses for reasons including declined or revoked consent, discharge prior to completion of research assessments, or other exclusion criteria (active psychosis, IQ <70, diagnosis of autism spectrum disorder, and primary language other than English). Our final sample consisted of 228 adolescents

ranging in ages from 12 to 17 ( $M = 15.43$ ;  $SD = 1.42$ ), including 132 females (57.9%) and 96 males (42.1%). Ethnic breakdown was as follows: 91.2% Caucasian, 2.6% Hispanic, 1.8% Asian, 1.8% mixed, 0.9% African American, 0.4% Native Hawaiian/Pacific Islander and 1.3% unreported. One or more suicide attempts were reported by 19% of the sample within the last year, while 23% had a lifetime history of one or more suicide attempts. In addition, 35% of the sample reported cutting during the last year, and 40% reported cutting over their lifetimes. Fifty-eight adolescents in the sample (25.4%) scored above the clinical cut-off for BPD (70T) on the Personality Assessment Inventory-Adolescent (Morey, 2007). When assessed using the Diagnostic Interview Schedule for Children (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), 44% of the sample carried a diagnosis of a mood disorder (dysthymia, major depressive disorder, bipolar disorder), while 50% had an anxiety disorder diagnosis (PTSD, GAD, OCD, social phobia, other phobias); 41% were diagnosed with a disruptive behavior disorder (ADHD, conduct disorder, oppositional defiant disorder).

### Measures

**Security Scale (SS).** The SS (Kerns, Klepac, & Cole, 1996) is a 15-item self-report measure designed to assess an adolescent's perceptions of security in parent-child relationships. The SS measures the adolescent's belief that a particular attachment figure is responsive and available, tendency to rely on the attachment figure in times of stress, and reported ease and interest in communicating with the attachment figure. Adolescents were instructed to choose statements that are characteristic of them and to rate on a 4-point scale the degree to which they perceive the statement to be true (1 = *really true*, 4 = *sort of true*). Scores were averaged across items to yield a dimensional score of security, with higher scores representing a more secure attachment. The SS has demonstrated good test-retest reliability across a 2-week period ( $r = .75$ ), as well as good construct and predictive validity in children and adolescents (Kerns et al., 1996; Van Ryzin & Leve, 2012). Specifically, the SS has been found to be associated with observed parent-adolescent relationship quality and parent- and teacher-reported social competence, and to be predictive of adolescents' self-reported attachment security 3 years later (see Van Ryzin & Leve, 2012). In the present study, Cronbach's alpha was .91 for mother and .90 for father.

**Cognitive Emotion Regulation Questionnaire (CERQ).** The CERQ (Garnefski et al., 2002) is a 36-item self-report measure of cognitive regulation strategies used in responding to negative events. Participants were instructed to indicate on a 5-point Likert scale the extent to which they make use of the nine theoretically and empirically derived cognitive emotion regulation strategies, ranging from 1 (*almost never*) to 5 (*almost always*), which in turn were summed and converted to scores ranging from 25T to 80T for each of the nine subscales. The CERQ was validated in a large community sample of adolescents (Garnefski, Kraaij, & Spinhoven, 2001) and Cronbach's alpha was .85 in the present sample. In the present study, scores on the Acceptance, Positive Refocusing, Refocus on Planning, Positive Reappraisal, and Putting into Perspective subscales were averaged to form a measure of positive emotion regulation strategies (CERQ Pos) and scores on the Self-blame, Rumination, Catastrophizing, and

Other-blame subscales were likewise combined to form a measure of negative emotion regulation strategies (CERQ Neg; see d'Acremont & Van der Linden, 2007; Garnefski et al., 2001).

**Personality Assessment Inventory–Adolescent, Borderline Features scale (PAI-A BOR).** The present study utilized the Borderline Features scale of the PAI-A (Morey, 2007). The PAI-A-BOR is a 24-item self-report questionnaire of features associated with BPD and taps into the four conceptually and empirically derived subdomains: affective instability, identity problems, negative relationships, and self-harm. Items rated on a 4-point Likert scale (0 = *false*, 1 = *slightly true*, 2 = *mainly true*, 3 = *very true*) were summed for each scale/subscale, and converted into *T* scores for comparison with the standardization sample. Scores that exceeded 70*T*, two standard deviations above the mean, represented a clinically significant deviation from the normative sample in the respective domain. Studies have supported the validity and reliability of the PAI-BOR in adolescents (Morey, 2007).

## Procedures

The study was approved by the appropriate institutional review board. All adolescents admitted to the inpatient psychiatric unit were approached on the day of admission about participation in the study. Informed consent from the parents was collected first and, if granted, assent from the adolescent was obtained in person. Adolescents were then consecutively assessed by doctoral-level clinical psychology students, licensed clinicians, and/or trained clinical research assistants following the instructions specified for each assessment tool. All assessments were conducted independently and in private with the adolescents within the first 2 weeks following admission. The average length of stay in this program is 5 to 7 weeks.

## Results

### Descriptive Statistics and Preliminary Analyses

Means, standard deviations, and ranges of the study variables are presented in Table 1 and show that significant effects of gender were found for several variables. Females displayed higher levels of BPD features in addition to greater signs of paternal attachment insecurity. Females also employed positive regulation strategies to a lesser degree than males. Bivariate correlations of the main study variables are shown in Table 2. As seen in Table 2, age did not

correlate with parental attachment insecurity, emotion regulation strategies, or BPD features. However, in building the blocks for the mediational analyses, BPD features were positively and significantly associated with negative regulation strategies ( $r = .38, p < .001$ ). BPD features were also negatively associated with positive regulation strategies ( $r = -.34, p < .001$ ), paternal attachment security ( $r = -.30, p < .001$ ) and maternal attachment security ( $r = -.18, p = .016$ ).

### Tests of Mediation

A series of hierarchical regression analyses (Holmbeck, 2002) tested the model in which the two classes of emotion regulation strategies (i.e., positive and negative) mediated the link between parental attachment security and BPD features. The effect of gender was controlled for and partialled out in the estimation of direct, indirect, and total effects.

Results supported the model in which the decreased use of positive emotion regulation strategies (CERQ Pos) mediated the relationship between attachment insecurity and BPD features (see Table 3). Specifically, analyses revealed that: (a) attachment security was positively associated with CERQ Pos ( $\beta = .22, p = .002$  for father;  $\beta = .19, p = .006$  for mother); (b) attachment security was negatively associated with BPD features ( $\beta = -.27, p < .001$  for father;  $\beta = -.15, p = .043$  for mother); (c) CERQ Pos predicted BPD features when controlling for attachment security ( $\beta = -.24, p = .001$  for father;  $\beta = -.28, p < .001$  for mother); and (d) the predictive strength of attachment security to BPD features decreased when CERQ Pos was controlled for ( $\beta = -.21, p = .004$  for father;  $\beta = -.09, p = .218$  for mother). The Sobel's test revealed that the mediation effect was statistically reliable ( $z = -2.24, p = .025$  for father;  $z = -2.27, p = .023$  for mother). CERQ Pos accounted for roughly 19% of the path from paternal attachment insecurity to BPD features and 41% of the path from maternal attachment insecurity to BPD features. Results, on the other hand, indicated a nonsignificant mediating effect of negative regulation strategies (CERQ Neg). Regression analyses did not reveal significant relationship between attachment security and CERQ Neg ( $\beta = -.01, p = .924$  for father,  $\beta = .01, p = .896$  for mother), suggesting a nonsignificant indirect path between attachment and BPD through CERQ Neg.

As gender remained a significant predictor in all analyses (see Table 3), the above analyses were conducted separately for males and females. A nonmediating effect of CERQ Neg was confirmed

Table 1  
Descriptive Data for Main Study Variables (N = 228)

	Range	M (SD)			Gender difference ( <i>t</i> ) <sup>a</sup>
		Full sample	Male (n = 96)	Female (n = 132)	
Age (in years)	12–17	15.43 (1.42)	15.48 (1.44)	15.39 (1.41)	–0.49
Paternal attachment (SS Father)	1–4	2.61 (.74)	2.80 (.75)	2.48 (.72)	–2.96**
Maternal attachment (SS Mother)	1–4	2.88 (.75)	2.98 (.66)	2.80 (.80)	–1.89 <sup>†b</sup>
Positive regulation strategies (CERQ Pos)	0–72	51.70 (10.18)	53.98 (9.32)	50.05 (10.48)	–2.93**
Negative regulation strategies (CERQ Neg)	0–74	57.92 (9.77)	58.34 (8.86)	57.61 (10.40)	–0.56
BPD Features (PAI-A BOR)	30–90	61.01 (12.90)	57.05 (12.61)	63.61 (12.48)	3.52**

<sup>a</sup> *t*-statistic from independent samples *t*-test comparing the means of males and females. <sup>b</sup> Levene's test for equality of variances was significant and adjusted *t*-values are reported. The results remain unchanged if homogeneous variances are assumed.

<sup>†</sup> < .10. \*  $p < .05$ . \*\*  $p < .01$ .



Table 2  
Bivariate Correlations Among Study Variables (N = 228)

	1	2	3	4	5	6
1. Age	1.00					
2. Paternal attachment (SS Father)	-.04	1.00				
3. Maternal attachment (SS Mother)	-.11	.20**	1.00			
4. BPD features (PAI-A BOR)	-.01	-.30***	-.18*	1.00		
5. Positive regulation strategies (CERQ Pos)	-.07	.26***	.21**	-.34***	1.00	
6. Negative regulation strategies (CERQ Neg)	-.08	.01	.01	.38***	.36***	1.00

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

for both males and females. The mediating role of CERQ Pos, however, could not be confirmed for either of the gender groups, likely due to a lack of statistical power.

**Tests of Moderation**

The moderating role of emotion regulation strategies was examined in two sets of hierarchical regression analyses using centered predictors. Each analysis controlled for gender in step 1 and included the first-order effects of attachment (paternal or maternal) and emotion regulation (CERQ Pos or CERQ Neg) in step 2 before adding their respective interaction terms in step 3. Results did not support the model in which CERQ Pos served as a moderator between parental attachment security and BPD. A nonsignificant interaction effect emerged when BPD was regressed on attachment security and CERQ Pos ( $\beta = -.02, R^2_{\text{change}} = .00, F_{\text{change}}(1, 175) = .06, p = .804$  for father;  $\beta = -.07, R^2_{\text{change}} = .01, F_{\text{change}}(1, 182) = 1.02, p = .313$  for mother). However, a significant moderation effect of CERQ Neg was found on the relation between paternal attachment insecurity and BPD. As seen in Table 4, the first-order effects of attachment and CERQ Neg were qualified by the significant interaction for father ( $\beta = .29, R^2_{\text{change}} = .07, F_{\text{change}}(1,$

$175) = 18.11, p < .001$ ), but not for mother ( $\beta = .11, R^2_{\text{change}} = .01, F_{\text{change}}(1, 182) = 2.34, p = .128$ ). Post hoc probing of the moderation (Holmbeck, 2002) revealed that the simple slope of paternal attachment security was significant at low level ( $z = -1$ ) of negative regulation strategy use ( $b = -10.06, t(175) = -6.01, p < .001$ ), but nonsignificant at high level ( $z = 1$ ) of negative regulation strategy use ( $b = 0.44, t(175) = .28, p = .782$ ; Figure 1). This indicated that the protective effect of paternal attachment security on BPD diminished as the deleterious effect of negative regulation strategy use increased. As seen in Figure 1(b), paternal attachment security buffered the increased risk of BPD associated with negative regulation strategies when the negative strategy use was roughly below  $z = 1.0$  (i.e., CERQ Neg = 67T; Very High range). However, when the use of negative regulation strategy reached this threshold, the effect of paternal attachment dropped to nonsignificance and predicted BPD scores reached clinically significant level ( $>70T$ ), as can be seen in Figure 1(a).

As with the mediation analyses, gender remained significant in all models (see Table 4). All above results were replicated when males and females were examined separately, except that the moderating effect of CERQ Neg on the relation between paternal

Table 3  
Positive Emotion Regulation Strategies (CERQ Pos) as a Mediator Between Parental Attachment Security and BPD Features

Steps/Predictors	$\beta$	$b (SE)$	$R^2$ (Adj.)	$\Delta R^2$	$F$
Step 1					
Gender	-.24**	-6.38 (1.91)	.06 (.05)	.06**	11.19**
Step 2					
Gender	-.19**	-5.03 (1.88)	.13 (.12)	.07***	12.98***
Paternal attachment security	-.27***	-4.64 (1.24)			
Step 3					
Gender	-.14*	-3.74 (1.87)	.18 (.16)	.12***	12.66***
Paternal attachment security	-.21**	-3.63 (1.25)			
Positive regulation strategies	-.24**	-0.34 (0.10)			
Step 1					
Gender	-.26***	-6.76 (1.87)	.07 (.06)	.07***	13.07***
Step 2					
Gender	-.24**	-6.27 (1.87)	.09 (.08)	.02*	8.72***
Maternal attachment security	-.15*	-2.49 (1.22)			
Step 3					
Gender	-.17*	-4.52 (1.86)	.16 (.14)	.09***	11.32***
Maternal attachment security	-.09	-1.49 (1.21)			
Positive regulation strategies	-.28***	-0.40 (0.10)			

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

Table 4  
*Negative Emotion Regulation Strategies (CERQ Neg) as a Moderator Between Paternal Attachment Security and BPD Features*

Steps/Predictors	$\beta$	$b$ (SE)	$R^2$ (Adj.)	$\Delta R^2$	$F$
Step 1			.06 (.05)	.06**	11.19**
Gender	-.24**	-6.38 (1.91)			
Step 2			.28 (.27)	.22***	23.23***
Gender	-.21**	-5.54 (1.71)			
Paternal attachment security	-.27***	-4.61 (1.13)			
Negative regulation strategies	.40***	0.62 (0.10)			
Step 3			.35 (.34)	.07***	23.64***
Gender	-.21**	-5.45 (1.63)			
Paternal attachment security	-.28***	-4.81 (1.08)			
Negative regulation strategies	.52***	0.82 (0.11)			
Pat att $\times$ Negative reg strategies	.29***	0.54 (0.13)			

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

attachment and BPD was significant only in females ( $\beta = .48$ ,  $R^2_{\text{change}} = .15$ ,  $F_{\text{change}}(1, 104) = 22.08$ ,  $p < .001$ ), but not in males ( $\beta = .04$ ,  $R^2_{\text{change}} = .002$ ,  $F_{\text{change}}(1, 68) = .180$ ,  $p = .673$ ).

### Test of Mediated Moderation

The mediation and moderation analyses reported above were followed by a post hoc test of mediated moderation, with CERQ Pos specified as a mediator and CERQ Neg as a moderator. The analysis controlled for the effect of gender. Mediated moderation probes underlying mediation process(es) by which an observed moderation effect is produced. The presence of mediated moderation is established by the following: (a) there must be a significant moderation effect on the relation between the predictor and the outcome variable; (b) the predictor must be directly associated with the mediator; (c) the relation between the mediator and the outcome variable must be significantly moderated by the moderator specified in condition (a); and (d) the moderation effect described in condition (a) must be reduced in magnitude when the mediator is entered in the model (Muller, Judd, & Yzerbyt, 2005).

Significant mediated moderation emerged as follows (see Table 5): (a) there was a moderation of CERQ Neg on the relation between paternal attachment and BPD ( $b = 0.54$ ,  $\beta = .29$ ,  $t = 4.26$ ,  $p < .001$ ); (b) there was a direct effect of paternal attachment on the mediator, CERQ Pos ( $b = 2.70$ ,  $\beta = .22$ ,  $t = 3.25$ ,  $p = .001$ ); (c) the effect of the mediator, CERQ Pos, on BPD was moderated by CERQ Neg ( $b = 0.01$ ,  $\beta = .22$ ,  $t = 2.65$ ,  $p = .009$ ); and (d) the moderation effect of CERQ Neg on the relation between paternal attachment and BPD reduced in magnitude and no longer remained significant when the mediator, CERQ Pos, was accounted for in the model ( $b = 0.24$ ,  $\beta = .13$ ,  $t = 1.68$ ,  $p = .095$ ). This suggested that CERQ Neg interacted with paternal attachment security, in part, through its interaction with CERQ Pos in predicting BPD. That is, paternal attachment security served as a buffer against BPD by enhancing positive emotion regulation strategies, but only among adolescents whose use of negative emotion regulation strategies was low. As adolescents' use of negative regulation strategies increased, the protective effect that paternal attachment security exerted via positive regulation strategies lessened.

The relations between paternal attachment security, positive regulation strategies, negative regulation strategies, and BPD fea-

tures are plotted in Figure 2, adapting methods illustrated by Fritz and MacKinnon (2008). Note that the magnitudes of the overall effect of paternal attachment security on BPD (depicted as  $\hat{c}$ ) as well as the portion of the effect mediated by positive regulation strategies (depicted as  $\hat{a}\hat{b}$ ) decreased as negative regulation strategies increased. Also visible is increased negative regulation strategies shifting the plot upward, pulling up BPD scores. At high level of negative strategy use, BPD features are shown to remain stably high while  $\hat{c}$  and  $\hat{a}\hat{b}$  are observed to be of negligible magnitude, and the slope (quantified by  $\hat{b}$ ) representing the relation of positive strategies and BPD is rendered flat.

### Discussion

Consistent with attachment theory, the present results supported the model in which attachment insecurity was associated with BPD features through its relation with emotion dysregulation. Specifically, parental attachment security (in particular, secure attachment to father) functioned as a buffer against adolescent BPD via enhanced positive emotion regulation strategies, while negative emotion regulation strategies served as a potent correlate of clinically significant levels of BPD, weakening the protective effect of attachment and positive regulation strategies.

Some notable implications follow the differential profiles of positive and negative strategies revealed in our mediational data. In our study, the mediating effect of emotion dysregulation was characterized by low use of positive emotion regulation strategies, while high use of negative emotion regulation strategies did not constitute a link between attachment insecurity and BPD features. This finding extends emerging psychobiological data on the effects of attachment on regulatory functioning (e.g., Gilissen, Bakermans-Kranenburg, van Ijzendoorn, & Linting, 2008), and converges with the view of secure attachment as a basic foundation upon which the human adaptation system is built (Sapientza & Masten, 2011). Attachment relationships may be understood as a context in which adolescents develop and mobilize positive emotion regulation strategies that sustain them in the face of difficulties. While it follows that the lack of attachment security may limit one's opportunities to learn positive regulation strategies, the present results did not support the generally held supposition that attachment insecurity may lead one to adopt negative regulation strategies. A growing body of literature has portrayed

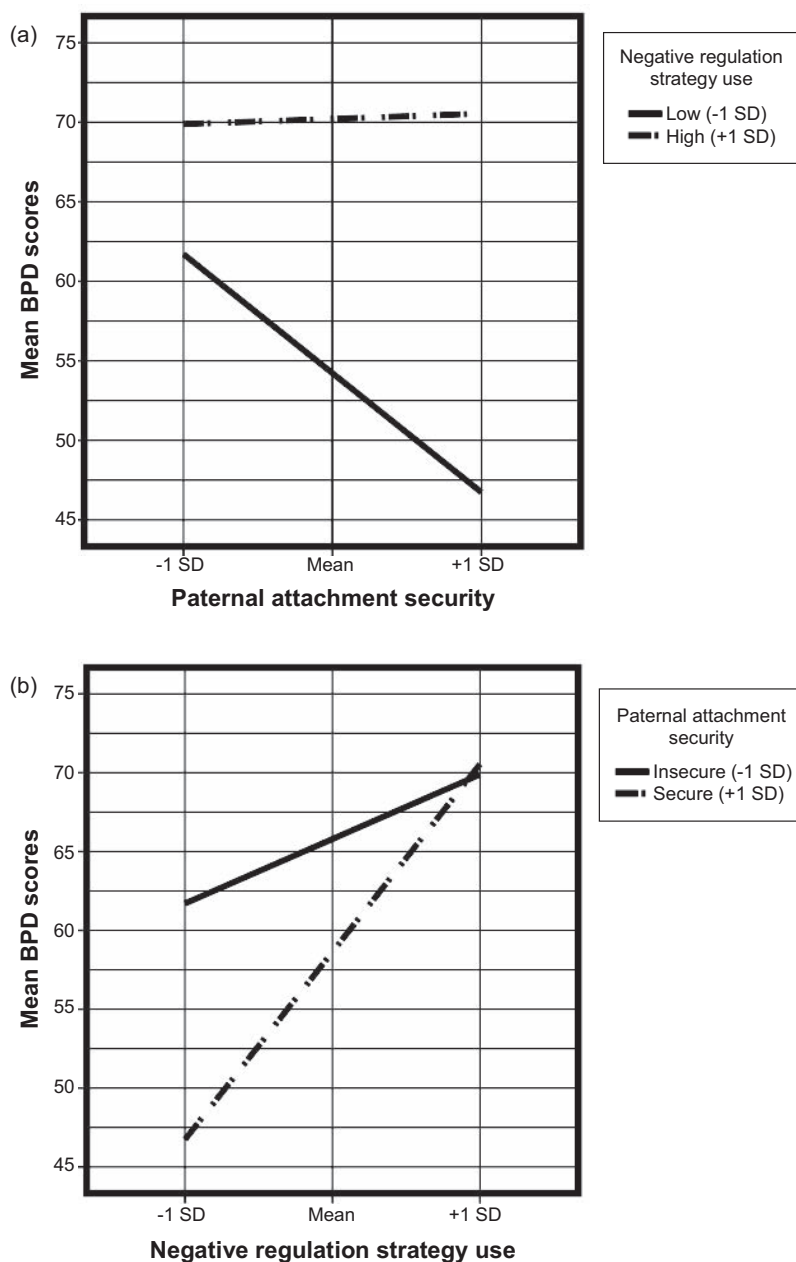


Figure 1. (a) Simple slopes of paternal attachment security predicting BPD features at high and low levels of negative regulation strategy use; (b) Simple slopes of negative regulation strategy use predicting BPD features at high and low levels of paternal attachment security.

attachment security as a key ingredient of resilience (Sapienza & Masten, 2011). Our findings augment this picture by providing an empirical delineation of a process by which attachment interacts with the complex pathways toward resilience—namely, attachment promotes resilience by undergirding the ability to adaptively regulate emotion. Adaptive emotion regulation, though long proposed as a putative protective factor (Curtis & Cicchetti, 2003), had rarely been studied in conjunction with other factors that shield against maladaptive functioning.

While delineating protective factors, our moderational findings draw further attention to the adverse effect of negative

regulation strategies, thereby extending support to the emotional cascade model (Selby et al., 2009). Though not intended to provide a direct test of the model in its entirety, our mediated moderation data serve to substantiate the model’s central proposal: elevated BPD features, intensified by negative regulation strategies, are made impervious to the effect of protective factors at play, including positive regulation strategies. Major conceptualizations of BPD have spoken to the immobilization of adaptive capacities in similar contexts: mentalization theory points to the inhibition or decoupling of reflective capacities in the grip of intense emotional arousal (Allen, 2003; Fonagy &

Table 5  
*A Mediated Moderation Model of Paternal Attachment Security, Positive Regulation Strategies, Negative Regulation Strategies, and BPD Features*

Predictors	Equation 1 DV: BPD		Equation 2 DV: Positive regulation strategies		Equation 3 DV: BPD	
	<i>b</i>	<i>t</i>	<i>b</i>	<i>t</i>	<i>b</i>	<i>t</i>
IV: Paternal attachment	-4.81	-4.45***	2.70	3.25**	-3.34	-3.21**
MO: Negative regulation strategies	0.82	7.68***	0.12	1.49	0.96	9.15***
IV × MO	0.54	4.26***	-0.19	-1.92	0.24	1.68
ME: Positive regulation strategies					-0.36	-3.91***
ME × MO					0.01	2.65**

*Note.* The regression analyses controlled for gender. MO = Moderator variable; ME = Mediator variable; DV = Criterion variable.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

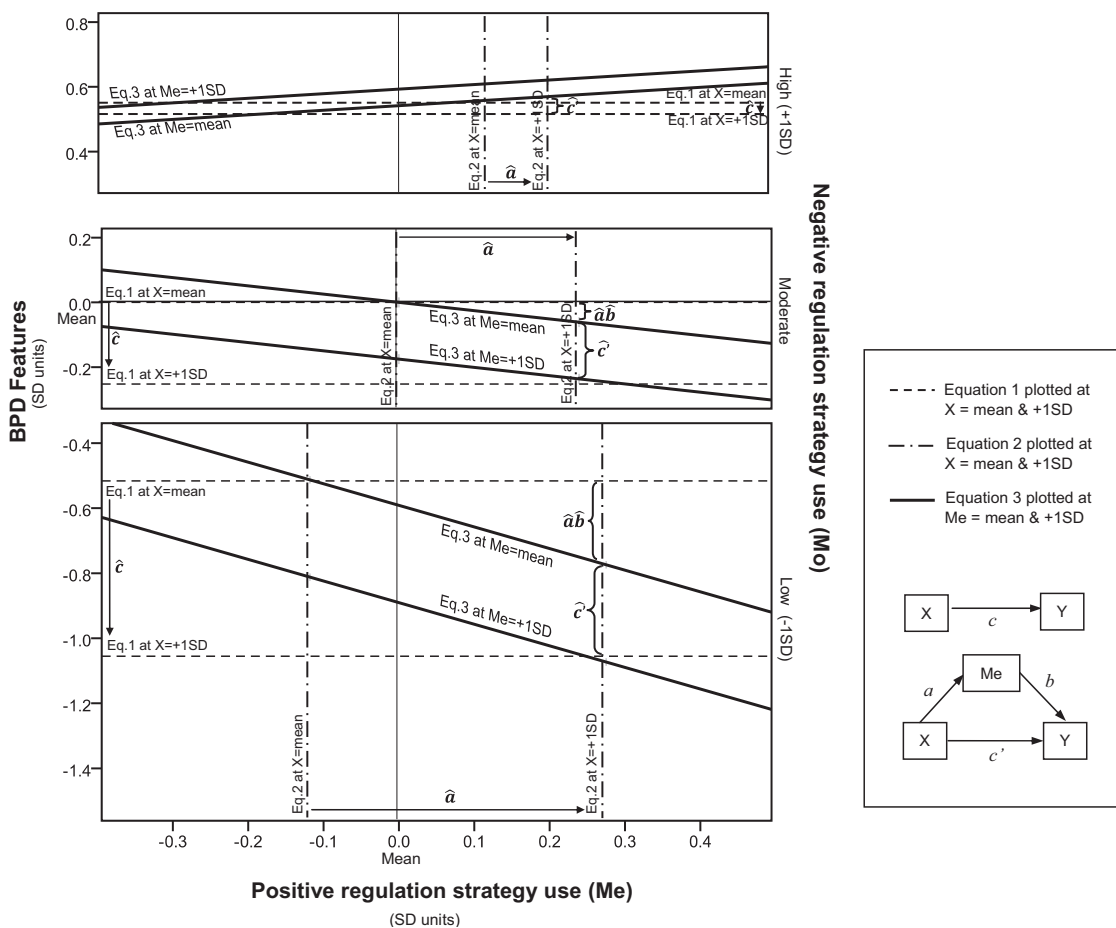
Luyten, 2009); dialectical-behavior theory highlights the disrupted activation of the mindful stance or “wise mind” when emotions are maladaptively handled (Linehan, 1993). Our findings provide preliminary empirical corroboration of this line of phenomena that has been construed as central to BPD impairment, while shedding light on the likely process targeted in both mentalization-based therapy (MBT) and dialectical behavior therapy (DBT).

In our data, adolescents’ paternal attachment produced larger and more consistent associations with our variables of interest than maternal attachment. We did not expect this difference, and this result is therefore considered preliminary. In comparison to maternal attachment, which has often been examined during the infant’s separation and reunion with the mother, paternal attachment is understood to grow in importance following the child’s early years of life and to develop in the context of the child’s joint play with the father (Grossmann et al., 2002). Paternal attachment security in childhood and adolescence has received attention for its roles in the child’s competent exploration and mastery of the environment, as well as the child’s management of frustration in the service of goal-directed behaviors (Grossmann et al., 2008). While still at its nascent stage, there is growing interest in this line of research. Despite their preliminary nature, our findings add to this small but expanding body of research by pointing to the significant role of paternal attachment in adolescent adjustment. Another potentially important gender difference concerns that of adolescent gender. Adolescent gender remained a significant predictor of BPD in all our analyses. Our post hoc analyses of gender effects indicated that the results reported here may be more relevant for girls than boys (also see Gratz et al., 2009). Specifically, the moderating role of negative regulation strategies on the link between paternal attachment and BPD was found to be specific to girls. This may underscore a particularly deleterious role that negative regulation strategies play in immobilizing adaptive capacities that adolescent girls develop in the context of their relationship with their father. A lack of adequate statistical power prohibits drawing further conclusions pertaining to boys and girls separately. Further research is needed to bring greater clarity to the role of adolescent gender.

Our findings carry important implications for the treatment of BPD. Given their role in maintaining high levels of BPD, negative regulation strategies may be an important target at the outset of treatment, particularly in the service of mobilizing patients’ adaptive capacities. Also critical may be the activation of an attachment relationship and its use as a vehicle in which positive regulation strategies are taught. What seems particularly important given our pattern of results is the striking of a balance between focusing on emotions with the aim of enhancing adaptive regulation strategies and simultaneously refraining from provoking emotional overarousal that diminishes adaptive regulation and leads to immobilization. These principles can in fact be understood as the common grounds of MBT and DBT; here we point to these principles to join in the ongoing endeavor to empirically elucidate specific mechanisms of change that underpin MBT and DBT (e.g., Fonagy & Bateman, 2006; Lynch, Chapman, Rosenthal, Kuo, & Linehan, 2006).

Several limitations of the study should be recognized. First, the cross-sectional nature of the present study precludes inferences concerning temporal sequence and causality, while providing an impetus for future prospective and longitudinal studies. Second, adolescent and parent temperament was not measured in this study. Temperament and attachment are distinct but mutually influencing constructs (Levy, 2005; Vaughn, Bost, & Van Ijzendoorn, 2008), and interact closely in the development of BPD (Fonagy & Luyten, 2009; Linehan, 1993). With the aim of elucidating the links between attachment, emotion dysregulation, and BPD, we largely ignored how temperament intersects with different components of these paths. It should, however, be acknowledged that temperamental factors may also provide avenues for understanding the links described here. Third, the use of an inpatient sample may limit the generalizability of our results. The dominant nature of negative regulatory strategies and their deleterious effects may not be as prominent in outpatient or subclinical populations. At the same time, focused study of our high-risk sample points us toward a strategy for intervening in this population in adolescence, a juncture at which BPD diagnosis is found to be held only moderately stable (Chanen et al., 2004). Fourth, comorbid internalizing and externalizing symptoms were not controlled for in this study, as were some familial variables (e.g., family





**Figure 2.** The relationship between paternal attachment security, positive regulation strategies, negative regulation strategies, and BPD features. *Note.* Equation 1:  $Y = \beta_{10} + \beta_{11}X + \beta_{12}Mo + \beta_{13}XMo$ ; Equation 2:  $Me = \beta_{20} + \beta_{21}X + \beta_{22}Mo + \beta_{23}XMo$ ; Equation 3:  $Y = \beta_{30} + \beta_{31}X + \beta_{32}Mo + \beta_{33}XMo + \beta_{34}Me + \beta_{35}MeMo$ ;  $X$  = paternal attachment security;  $Me$  (Mediator) = positive regulation strategies;  $Mo$  (Moderator) = negative regulation strategies;  $Y$  = BPD features;  $\hat{c}$  is decomposed into  $\hat{a}\hat{b} + \hat{c}'$ , and each component of the model represents the following at a given level of negative regulation strategies ( $Mo$ ):  $\hat{c}$  = total effect of paternal attachment security on BPD (equals  $\beta_{11} + \beta_{13}Mo$ );  $\hat{a}$  = effect of paternal attachment security on positive regulation strategies (equals  $\beta_{21} + \beta_{23}Mo$ );  $\hat{b}$  = effect of positive regulation strategies on BPD adjusted for paternal attachment security (equals  $\beta_{34} + \beta_{35}Mo$ );  $\hat{a}\hat{b}$  = indirect effect of paternal attachment security on BPD mediated via positive regulation strategies;  $\hat{c}'$  = residual direct effect of paternal attachment security on BPD adjusted for positive regulation strategies (equals  $\beta_{31} + \beta_{33}Mo$ ); All scales are presented in standard deviation units.

structure, living arrangement) that may have been important. Future studies should determine whether the mechanisms disentangled here are specific to BPD or are also implicated in other disorders. The findings should also be explored in the context of familial covariates. Fifth, the use of *T* scores for the CERQ may have restricted the range at the low end of the distribution. Subsequent studies may alternatively consider using raw scores. A final caveat concerns the use of self-report measures. The present research should be followed by further investigations utilizing interview and/or experimental measures, though some reports have been made concerning relatively high convergence of these measures and self-report instruments (Fossati et al., 2005; Mikulincer & Shaver, 2007).

Despite these limitations, the present study marks the first attempt to spell out mechanisms of interplay between attach-

ment and emotion regulation as they relate to BPD. We have shown that attachment is differentially linked to BPD on the basis of the nature of emotion regulation strategies one adopts. Our findings serve as an illustration of how the interpersonal context intersects with one's choice of behaviors and strategies in influencing one's developmental trajectory. Emotion regulation strategies are relatively easy to target and modify in treatment; results reported here hence bear important implications for treatment at its critical juncture of adolescence.

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### Correction to Clark (2013)

In the article, “A Critique of Gunderson’s Views of *DSM-5*: Commentary on ‘Seeking Clarity for Future Revisions of the Personality Disorders in *DSM-5*’” by Lee Anna Clark (*Personality Disorders: Theory, Research, and Treatment*, Vol. 4, No. 4, pp. 379–380. doi: 10.1037/per0000050), the appendices were omitted. The online version has been corrected.

DOI: 10.1037/per0000067