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The Case for Borderline Personality Disorder as an Emotional Disorder: Implications for Treatment

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The goal of this article is to advance understanding of borderline personality disorder (BPD) as an emotional disorder and to use this information as a heuristic for reconceptualizing targeted treatment approaches. The first section reviews evidence that BPD is characterized by the hallmark of emotional disorders, frequent intense negative emotions, and adverse reactions to them. Next, overlap between BPD and other emotional disorders is described, followed by a section delineating how these similarities can be largely accounted for by a shared underlying vulnerability, namely, high levels of neuroticism. Finally, we discuss the treatment implications of this conception of BPD in the context of recent transdiagnostic approaches to emotional disorders.

Key words: **borderline personality disorder, comorbidity, emotion, neuroticism.** ***[Clin Psychol Sci Prac 21: 118–138, 2014]***

"Emotional disorder" is a term that has been used to group psychopathology that is characterized by (a) frequent and intense negative emotions, (b) strong aversive reactions to negative emotions, and (c) efforts to escape or avoid these emotional experiences (Barlow, 1991). Grouping disorders in this way is very much in keeping with a more dimensional and functional basis for classification, as exemplified by the National Institute of Mental Health's Research Domain Criteria (RDoC; Insel et al., 2010). A major priority of the RDoC initiative is the translation of evidence from basic science to elucidate core fundamental mechanisms that cut across existing disorder categories. There is support from genetic, neurobiological, cognitive, and behavioral sources to suggest that the personality trait of neuroticism, all but isomorphic with the defining features of emotional disorders outlined above, is a core mechanism in the development and maintenance of this functional category of disorders (Andrews, 1990; Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, in press; Krueger, 1999). As such, disorders falling on the traditional "neurotic spectrum," sharing the characteristics specified above, could be classified as emotional disorders. These include classic internalizing disorder diagnoses such as *DSM-5* (American Psychiatric Association [APA], 2013) depressive disorders, anxiety disorders, obsessive–compulsive disorders, trauma-related disorders, somatic symptom disorders, dissociative disorders, and some eating disorders. In contrast, substance-related disorders,[1] impulse control/conduct disorders, psychotic disorders, and other cognitive disorders would not meet the definitional characteristics of emotional disorders. It is important to note that the umbrella label of "emotional disorders" does not represent a return to a nonempirical system of classification, but rather is a way to emphasize the functional similarities and underlying mechanisms that may be more appropriate targets for intervention across conditions.

Empirical support for this premise is most evident among anxiety and depressive disorders (Barlow et al., in press). Individuals with these disorders endorse high levels of neuroticism, and nearly all of the temporal

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covariance among comorbid anxiety and depressive disorders can be accounted for by temperamental factors, particularly neuroticism (Brown, 2007; Griffith et al., 2010; Kessler et al., 2011). Phenotypic differences among disorders (somatic, social, or cognitively focused anxiety) can be regarded as of secondary importance. The purpose of the current article is to forward a case for borderline personality disorder (BPD) as an emotional disorder by first focusing on its functional similarities with mood, anxiety, and related disorders, followed by a discussion of possible shared underlying vulnerabilities among these disorders. This characterization of BPD may provide a useful, theoretically informed heuristic to reconceptualize current treatments and guide intervention strategies that directly target the definitional properties of emotional disorders: the experience of strong emotions and subsequent negative responses to them.

Borderline personality disorder is a costly and debilitating disorder (McGlashan et al., 2000; Sansone, 2004; Skodal et al., 2002) characterized by impairment across several areas of functioning. In *DSM-5*, symptoms associated with BPD include emotional difficulties (labile affect and intense anger), interpersonal problems (efforts to avoid abandonment and unstable relationships), behavioral dysregulation (chronic suicidality, self-injury, and other impulsive actions), identity disturbance (unstable self-image and chronic emptiness), and cognitive symptoms (transient dissociation and paranoia in response to stress; APA, 2013).

At first glance, BPD may not appear to share obvious definitional similarities or treatment priorities with the emotional disorders described above. It is important to note, however, that two patients with BPD may look as disparate as an individual with BPD compared to an individual with panic disorder; due to an interesting artifact of the current classification system, it is possible that two individuals meeting criteria for BPD could share only one common diagnostic criterion (e.g., chronic feelings of emptiness). Regardless of phenotypic differences, a large literature (reviewed below) supports the notion that individuals with BPD, as with other emotional disorders, are prone to strong emotions and tend to evaluate these experiences negatively. Factor-analytic work by Sanislow et al. (2002) has suggested that BPD is comprised of three highly related factors; the authors suggest that one factor, affect dysregulation, may moderate the other two factors (behavioral dysregulation and disturbed relatedness). Further, emerging evidence suggests that BPD appears to share substantial underlying temperamental variance with anxiety and mood disorders (Eaton et al., 2011; James & Taylor, 2008) represented largely by a neuroticism factor. In fact, much of the clinical presentation of BPD can be accounted for by these emotional factors (Linehan, 1993).

The conceptualization of BPD as an emotional disorder is not entirely inconsistent with other views of BPD. For example, Linehan (1987), perhaps the leading clinical investigator in this area, described emotion dysregulation as the core characteristic of BPD from which the other diagnostic features arise; emotion dysregulation is further described as the product of a biologically inherited vulnerability to experience intense, easily elicited emotions, coupled with an inability to modulate emotions adaptively. Widiger, Trull, Clarkin, Sanderson, and Costa (2002) view BPD as corresponding closely to the neuroticism facets identified by the Big Five theory of personality (Costa & McCrae, 1992). Specifically, they purport that the anxiety, depression, and angry hostility facets account for BPD emotional difficulties and that interpersonal problems and behavioral dysregulation are encompassed by neurotic impulsivity. Finally, identity disturbance and cognitive symptoms are accounted for by neuroticism facets of vulnerability to stress and self-consciousness. Additionally, Siever and Davis (1991) have described personality pathology as existing on a psychobiological continuum with Axis I disorders; specifically, they describe similarities between BPD and mood disorders in terms of the intensity and regulation of negative emotions. Finally, other authors have proposed shifting the BPD diagnosis to Axis I (New, Triebwasser, & Charney, 2008) as a mood disorder (Schultz & Goldberg, 1984).

BORDERLINE PERSONALITY DISORDER AS AN EMOTIONAL DISORDER

"Emotional disorder" is a term that has been used to group anxiety, unipolar mood, and related disorders, such as somatoform and dissociative disorders (Barlow, 1991; Barlow et al., 2011; Brown & Barlow, 2009). Individuals with emotional disorders report experiencing

more frequent and intense negative emotions than healthy individuals (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Mennin, Heimberg, Turk, & Fresco, 2005). Further, they describe the experience of negative emotions as more aversive than healthy individuals (Roemer, Salters, Raffa, & Orsillo, 2005), and as such, they engage in a range of cognitive and behavioral strategies aimed at escaping or avoiding negative emotions (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Baker et al., 2004; Moore, Zoellner, & Mollenholt, 2008; Tull & Roemer, 2007; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). A wide range of research suggests that these ways of interpreting and responding to negative emotions paradoxically serve to increase and maintain such emotions and emotional disorder symptomatology. The following section discusses in greater detail the literature supporting the case for BPD as characterized by these response tendencies and their sequelae.

Borderline Personality Disorder Is Characterized by Strong Negative Emotions

An important characteristic of emotional disorders is the propensity to experience negative emotions. A substantial literature supports the notion that individuals with BPD indeed experience frequent, strong negative emotions. Linehan (1993) described individuals with BPD as emotionally vulnerable or, in other words, characterized by high baseline levels of negative affect (intensity), coupled with substantial changes in the intensity of this affect when faced with an emotionally evocative cue (reactivity). Several studies have employed self-report methods to demonstrate greater levels of negative emotions in BPD compared to nonclinical controls and other personality disorders (Henry et al., 2001; Koenigsberg et al., 2002; Levine, Marziali, & Hood, 1997) and that this emotional intensity is significantly associated with severity of BPD symptoms (Cheavens et al., 2005; Rosenthal, Cheavens, Lejuez, & Lynch, 2005; Yen, Zlotnick, & Costello, 2002).

Additional studies have utilized physiological measures to demonstrate heightened emotional intensity and reactivity in BPD. For example, Ebner-Priemer et al. (2005, 2007) found greater startle responses and larger changes in heart rate following a stressor for individuals with BPD compared to healthy control subjects. Further, BPD has been associated with decreases in respiratory sinus arrhythmia and heart period following a stressor, which has been described as reflecting heightened emotional reactivity (Austin, Riniolo, & Porges, 2007). It is important to note, however, that the specificity of psychophysiological measures has been questioned (Beauchaine, 2001; Berntson, Cacioppo, & Quigley, 1993; Berntson, Cacioppo, Quigley, & Fabro, 1994), and several studies have shown no differences between BPD participants and healthy controls on various physiological indicators of emotional reactivity (Herpertz, Kunert, Schwenger, & Sass, 1999; Herpertz et al., 2000, 2001; Kuo & Linehan, 2009). More recently, neuroimaging studies have demonstrated heightened emotional responding in individuals with BPD (for a review, see Rosenthal et al., 2008); across studies, results indicate that reduced hippocampal, orbitofrontal, and amygdala volumes are observed in BPD and that the amygdala displays increased activation in response to emotional cues. Overall, self-report and physiological data support the notion that BPD is characterized by strong emotional experiences.

Borderline Personality Disorder Is Characterized by Negative Reactions to Strong Emotions

Another important feature of emotional disorders is the tendency to find emotional experiences aversive and, as such, engage in attempts to avoid them. Self-report studies have demonstrated that individuals with BPD are high in experiential avoidance (Chapman, Specht, & Cellucci, 2005; Rusch et al., 2006), defined as the unwillingness to remain in contact with uncomfortable internal experience (e.g., thoughts, emotions, sensations, memories, urges) through escape or avoidance (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Experiential avoidance accounts for significant incremental variance in predicting BPD symptom severity beyond frequency of negative emotions (Gratz, Tull, & Gunderson, 2008; Iverson, Follette, Pistorello, & Fruzzetti, 2012) and mediates the relationship between early life stressors and BPD symptoms (Sturrock, Francis, & Carr, 2009). Underscoring their similarities with BPD, experiential avoidance has also been linked to the development of other emotional disorders, including anxiety (Begotka, Woods, & Wetterneck, 2004; Hayes,

Luoma, Bond, Masuda, & Lillis, 2006; Kashdan, Breen, Afram, & Terhar, 2010) and depressive disorders (Berking, Neacsiu, Comtois, & Linehan, 2009; Cribb, Moulds, & Carter, 2006; Hayes et al., 2006; Shahar & Herr, 2011; Tull, Gratz, Salters, & Roemer, 2004). In fact, Cheavens and Heiy (2011) recently found that avoidant coping partially mediates the relationship between negative emotions and both BPD and major depressive symptoms. Taken together, these findings suggest that BPD and other emotional disorders symptoms are not simply a product of high levels of negative affect; instead, the combination of strong negative emotions and how one relates to them when they occur appears to be important for the development of these disorders.

Individuals with BPD also show deficits in mindfulness (Cheavens et al., 2005; Hayes et al., 1996), a related construct that refers to attention and awareness toward the present moment in an accepting manner regardless of the nature of the experience (Kabat-Zinn, 1982). Mindfulness is negatively correlated with several difficulties encountered by individuals with BPD, including interpersonal problems, impulsivity, and emotion regulation deficits characterized by the use of avoidant strategies. Consistent with data on experiential avoidance, studies suggest that the degree to which one responds to negative emotions in a mindful manner predicts psychological symptoms over and above the contributions of a trait-like tendency to experience negative emotions (Sauer & Baer, 2009; Segal, Williams, & Teasdale, 2002). Relatedly, Wupperman, Neumann, and Axelrod (2008) and Wupperman, Neumann, Whitman, and Axelrod (2009) found that difficulties with mindful attention appear to explain borderline symptom severity, even when controlling for constructs that have been described as central to the disorder's pathology by various authors—heightened negative affectivity (Widiger, Costa, & McCrae, 2002), impulsivity (Trull, 2001), emotion regulation difficulties (Linehan, 1993), and interpersonal problems (Benjamin, 1996; Meyer & Pilkonis, 2005). Low levels of mindfulness are also common in other emotional disorders (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006; Brown & Ryan, 2003; Dekeyser, Raes, Leijssen, Leysen, & Dewulf, 2008; Rasmussen & Pidgeon, 2011), supporting the case for similar underlying constructs in these disorders. Overall, these data suggest that deficits in mindfulness (e.g., responding to negative emotions in a judgmental, avoidant manner) appear to predict BPD symptoms beyond the contributions of the frequency and intensity of negative emotions alone.

Another transdiagnostic construct that has been implicated in BPD and other emotional disorders is anxiety sensitivity, defined as a propensity for developing beliefs that anxiety-related symptoms will have negative somatic, cognitive, and social consequences (Reiss, 1991). Like experiential avoidance and mindfulness, anxiety sensitivity represents an individual's characteristic way of evaluating and responding to emotional experiences (specifically anxiety) when it occurs, distinct from the frequency or intensity of anxiety itself (Cox, Borger, & Enns, 1999; Lilienfeld, 1999). There is a large literature that implicates anxiety sensitivity in the development of anxiety disorders beyond the contributions of the tendency to experience anxiety (for a review, see Naragon-Gainey, 2010). More recently, researchers have found elevated levels of anxiety sensitivity in individuals with BPD features compared to normal control participants (Lilienfeld & Penna, 2001). Further, Gratz et al. (2008) found that outpatients with BPD reported significantly higher anxiety sensitivity than outpatients with other personality disorders and that anxiety sensitivity levels in BPD were comparable to patients with generalized anxiety disorder, obsessive–compulsive disorder, and social phobia. Additionally, they found that anxiety sensitivity predicts BPD symptoms above the contributions of trait negative affect and that the relationship between anxiety sensitivity and BPD was mediated by the use of avoidant coping. Again, these results support the notion that how one relates to negative emotions is just as important in the development of BPD and other emotional disorders as the frequency and intensity of emotional experience.

Borderline Personality Disorder Difficulties Are a Product of Strong Emotions Coupled With Negative Reactions to Them

The difficulties in regulating emotional experiences that characterize BPD may result from the product of intense negative affect coupled with negative reactions (e.g., anxiety sensitivity, described above) to this experience. Emotion regulation has been defined as

"monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features" (Thompson, 1994, p. 27). Putnam and Silk (2005) noted that, given this definition, individuals with BPD are actually quite effective at noticing their distress and engaging in strategies to reduce it. The strategies chosen, however, are informed by negative evaluations of their emotional experience, motivated by the desire to avoid it altogether, and can lead to increased future negative emotions. First, there is evidence to suggest that the behavioral difficulties associated with BPD function as avoidance of intense negative emotion. For example, self-harm has long been considered an intentional effort to escape from emotional pain (Carr, 1977; Chapman, Gratz, & Brown, 2006; Gratz, 2003). Empirical studies utilizing self-report methods provide support for the notion that engaging in self-harm most often leads to quick reductions in anxiety, anger, depression, and shame (Bentley, Nock, & Barlow, in press; Gratz, 2000; Haines, Williams, Brain, & Wilson, 1995; Nock & Prinstein, 2004). Further, experiential avoidance mediates the relationship between BPD and self-harm, suggesting that unwillingness to experience emotions is one mechanism by which individuals with BPD engage in self-harm (Bentley et al., in press; Chapman et al., 2005).

Other behaviors commonly observed in BPD may also be accounted for by experiential avoidance, as they may function to distract from negative emotions (see Bijttebier & Vertommen, 1999). Specifically, substance abusers with BPD, compared to substance abusers without BPD, reported greater instances of drug and alcohol use as a means to escape negative emotions (Kruedelbach, McCormick, Schulz, & Grueneich, 1993). Further, dissociation and binge eating have also been associated with experiential avoidance (Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003; Paxton & Diggens, 1997; Wagner & Linehan, 1995). Avoidant behavioral coping is also quite common in other emotional disorders. For example, interoceptive and situational avoidance are hallmarks of panic disorder; compulsions serve to escape from the distress associated with obsessions in obsessive–compulsive disorder; and persistent avoidance is one of the diagnostic criteria for posttraumatic stress disorder (American Psychiatric Association, 2000).

In addition to engaging in problematic avoidant behaviors, individuals with BPD and other emotional disorders also engage in cognitive coping motivated by avoidance. Such processes include thought suppression and rumination. Thought suppression is a strategy in which individuals deliberately attempt to push unpleasant, emotion-inducing cognitions out of awareness; paradoxically, this strategy has been shown to produce rebound effects in which the suppressed thoughts return with greater frequency and intensity (Rassin, Murkelbach, & Muris, 2000; Wegner, Schneider, Carter, & White, 1987). Thought suppression has been shown to mediate the relationship between emotional vulnerability (emotional intensity and reactivity) and BPD symptoms (Cheavens et al., 2005; Rosenthal et al., 2005). Emotional vulnerability may lead individuals with BPD to fear their emotional experience, leading to thought suppression as a way to minimize consequences associated with strong reactions (Sauer & Baer, 2009). Like most avoidant coping, there is evidence that thought suppression leads to short-term reductions in negative affect associated with BPD (Chapman, Rosenthal, & Leung, 2009), but longer-term consequences include higher rates of self-injury (Chapman et al., 2005), presumably due to rebound effects. High levels of thought suppression have also been demonstrated in many emotional disorders. This strategy has been associated with depression, generalized anxiety disorder, obsessive–compulsive disorder, and posttraumatic stress disorder (Purdon, 1999) and has been shown to exacerbate symptoms across these disorders (Abramowitz, Tolin, & Street, 2001).

Rumination refers to repetitively and passively focusing on negative mood and its possible causes, meanings, and consequences (Nolen-Hoeksema, 1991). Like thought suppression, rumination can also be conceptualized as an avoidant strategy, as passive focus on surface matters may serve to protect individuals from more distressing concerns (see CaR-F-A-X model: Williams & Moulds, 2007). Rumination has been shown to intensify negative affect (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), leading to more rumination, in what Selby, Anestis, and Joiner (2008) described as an emotional cascade; this continues until a maladaptive behavior (e.g., self-injury, substance use, binge eating) interrupts the cycle. Several authors have

confirmed that rumination, particularly in response to anger, is prominent in people with BPD (Abela, Payne, & Moussaly, 2003), is correlated with symptom severity (Baer & Sauer, 2011; Smith, Grandin, Alloy, & Abramson, 2006), and indeed predicts dysregulated behavior (Sauer & Baer, 2012; Selby, Anestis, Bender, & Joiner, 2009). Like thought suppression, rumination is commonly used across emotional disorders and has been demonstrated to exacerbate symptoms (Nolen-Hoeksema et al., 2008). Thus, it seems clear that negative reactions to strong emotions lead to similar forms of avoidant cognitive coping (e.g., thought suppression and rumination) common to all emotional disorders. These strategies that intensify already strong negative emotions appear to lead to greater use of disorder-specific behavioral coping.

Overall, the literature suggests that individuals with emotional disorders experience strong negative emotions with frequency and evaluate these experiences as aversive. Due to these negative reactions to their emotions, they are more likely to engage in avoidant coping strategies to manage emotional experiences, and these strategies, in turn, paradoxically increase the frequency/intensity of negative emotions, which maintains symptoms.

BORDERLINE PERSONALITY DISORDER AND COMORBIDITY WITH OTHER EMOTIONAL DISORDERS

Given the overlap in emotional disorder features described above, it is no surprise that BPD is characterized by a high degree of comorbidity with other emotional disorders (Zanarini et al., 1998; Zimmerman & Mattia, 1999). Grant et al. (2008), utilizing a large, representative sample of adults from the United States, reported a high degree of lifetime comorbidity among BPD and emotional disorders. Specifically, they found that 75% of individuals with a lifetime BPD diagnosis will meet criteria for a lifetime mood disorder and 74.2% of individuals will meet criteria for a lifetime anxiety disorder. The mood disorders most commonly found co-occurring with BPD are major depressive disorder (32.1%) and bipolar I disorder (31.8%), while the most commonly co-occurring anxiety disorders are posttraumatic stress disorder (39.2%), generalized anxiety disorder (35.1%), social anxiety disorder (29.3%), and panic disorder (28.5%). The overall rates of comorbidity for any mood or anxiety disorder with BPD are similar to comorbidity rates among mood and anxiety disorders alone; estimates suggest that 55% of patients with a principal anxiety disorder had at least one additional anxiety or depressive disorder at the time of assessment, and this rate increases to 76% when lifetime diagnoses are considered (Brown, DiNardo, Lehman, & Campbell, 2001).

Impact of BPD Comorbidity on Treatment

Comorbid BPD is thought to complicate severity and response to treatment for Axis I conditions. For example, patients with panic disorder and comorbid BPD display more severe panic symptomology, greater depressive and agoraphobia symptoms, earlier onset, and complicating factors such as separation anxiety and history of trauma (Weiler et al., 1988), and that these factors limit treatment effectiveness (Ozkan & Altindag, 2005). Similarly, individuals with comorbid BPD and obsessive–compulsive disorder also exhibit increased severity such that they are far more likely to engage in self-injurious behavior (Mckay, Kulchych, & Danyko, 2000). The impact of comorbid BPD and posttraumatic stress disorder has received the most empirical attention, and the literature suggests that comorbidity between these disorders also negatively impacts severity and treatment response (van Dijke et al., 2012; Vignarajah & Links, 2009). Finally, depressed patients with comorbid BPD demonstrate earlier age of depressive onset, more chronic depressive symptoms, greater likelihood of alcohol and cannabis use disorders, greater likelihood of suicide attempts, and decreased treatment response (Joyce et al., 2003).

Despite the fact that comorbidity among BPD and other emotional disorders is extremely common and clearly affects the course of treatment, few interventions have provided guidance for dealing with comorbid conditions. For example, dialectical behavior therapy (DBT; Linehan, 1993), a well-designed treatment for BPD with strong empirical support, does not provide strategies to manage comorbid anxiety conditions; in fact, the use of concurrent exposure-based procedures to address posttraumatic stress disorder symptoms is explicitly discouraged until patients have completed a course of DBT. Relatedly, commonly used cognitive-behavioral treatments for anxiety

disorders, such as mastery of your anxiety and panic (for panic disorder; Barlow & Craske, 2007), managing social anxiety (for social anxiety disorder; Hope, Heinberg, & Turk, 2010), and mastery of your anxiety and worry (for generalized anxiety disorder; Craske & Barlow, 2006) also do not directly address comorbid anxiety and mood disorders, let alone personality disorders.

There are several possible explanations for high rates of comorbidity among emotional disorders, which have been reviewed extensively elsewhere (Brown & Barlow, 2002, 2009). One explanation with increasing support is that this pattern of comorbidity may be best accounted for by what has been called a "general neurotic syndrome" (Andrews, 1990, 1996; Tyrer, 1989). Under this conceptualization, heterogeneity in the expression of emotional disorder symptoms (e.g., individual differences in the prominence of interpersonal conflicts, panic attacks, anhedonia) is regarded as relatively trivial variation in the manifestation of a broader syndrome (Barlow et al., in press). This theory has focused for the most part on mood and anxiety disorders, but emerging evidence suggests that an underlying neurotic syndrome may also have implications for BPD.

BORDERLINE PERSONALITY DISORDER AND NEUROTICISM

As noted above, a substantial literature underscores the role of neuroticism in accounting for the onset, overlap, and maintenance of many emotional disorders, largely anxiety and depressive disorders (Brown, 2007; Brown & Barlow, 2009; Brown, Chorpita, & Barlow, 1998; Gershuny & Sher, 1998; Griffith et al., 2010; Kasch, Rottenburg, Arnow, & Gotlib, 2002; Kessler et al., 2011; Krueger, 1999; Watson, Clark, & Carey, 1988). Exploration of the latent factor structure of emotional disorders has revealed that neuroticism represents a higher-order factor with significant pathways to depression, social anxiety disorder, generalized anxiety disorder, posttraumatic stress disorder, obsessive–compulsive disorder, and panic disorder (Brown et al., 1998; Griffith et al., 2010; Kessler et al., 2011; Krueger, 1999). In fact, virtually all the considerable temporal covariance among latent variables corresponding to the *DSM* constructs of these disorders can be explained by the higher-order dimensions of neuroticism (Brown, 2007). Again, these findings suggest that heterogeneity in the presentation of emotional disorders (e.g., presence of panic attacks vs. social anxiety) may represent trivial variations of a broader underlying vulnerability, mostly accounted for by neuroticism. Given that it is not often classified in terms of its similarities to mood and anxiety disorders, BPD was not included in these investigations, despite obvious theoretical overlap. The following section summarizes work that has been carried out to characterize the latent temperamental structure of BPD and presents evidence suggesting that individuals with BPD display high levels of neuroticism that may underscore comorbidity among BPD and other emotional disorders.

The Role of Neuroticism in BPD

Research has consistently shown that BPD is strongly associated with high levels of neuroticism (Clarkin, Hull, Cantor, & Sanderson, 1993; Henry et al., 2001; Koenigsberg et al., 2001; Larstone, Jang, Livesley, Vernon, & Heike, 2002; Morey et al., 2002; Putnam & Silk, 2005; Samuel & Widiger, 2008; Saulsman & Page, 2005). In fact, levels of neuroticism in patients with BPD were elevated relative to patients with other personality disorder diagnoses (Morey & Zanarini, 2000), forwarding the notion that BPD may operate more similarly to emotional disorders. It is important to note that other personality disorders are also characterized by high levels of neuroticism (Widiger, Trull, et al., 2002); however, although we consider neuroticism (frequent and strong negative emotions) to be important for the development of emotional disorders, responding to these negative emotions with distress and avoidant regulatory strategies (as seen in BPD) is perhaps more important. Additionally, although BPD appears to be a disorder with a varied presentation and impairment in several areas of functioning, the four important features of BPD (affective instability, cognitive distortion, identity problems, and insecure attachment) are best explained by a common latent factor that is highly correlated with trait neuroticism (Kendler, Myers, & Reichborn-Kjennerud, 2011).

Some studies have included BPD in multivariate analyses designed to uncover underlying structures that may explain comorbidity among disorders that commonly occur together. For example, James and

Taylor (2008) examined associations between BPD and internalizing, a latent factor synonymous with neuroticism representing a propensity for emotional disorders, and externalizing, a latent factor representing the propensity to experience disinhibitory disorders such as substance use. They found that BPD is best described as an internalizing *and* externalizing disorder, although it is significantly more strongly associated with internalizing (factor loadings = .60 vs. .23 for externalizing). Eaton et al. (2011) conducted similar analyses in a large sample of 34,653 individuals who participated in the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. They replicated previous findings in which BPD was associated with both latent internalizing and externalizing factors, although again more strongly with internalizing. Thus, these findings suggest that comorbidity among BPD and other emotional disorders may be largely due to the shared underlying vulnerability of neuroticism.

ETIOLOGY OF BPD AS AN EMOTIONAL DISORDER

A useful framework in which to understand the contributions of trait neuroticism in the development of BPD is within the context of the triple vulnerabilities model that has been forwarded to describe the etiology of other emotional disorders (Barlow, 1988, 2000, 2002). As the name suggests, the model integrates three factors thought to be important in the development of emotional disorders: a biological (heritable) vulnerability common across disorders, a shared psychological vulnerability, and a more specific psychological vulnerability to account for why a particular emotional disorder may emerge instead of another.

Generalized Biological Vulnerability

In terms of biological vulnerability, there is evidence to suggest that neuroticism, underlying emotional disorders, is heritable with genetic contributions to the expression of this trait estimated between 30% and 50% of the variance (Barlow, 2000; Clark, Watson, & Mineka, 1994; Kendler, Prescott, Myers, & Neale, 2003). Genetically mediated trait neuroticism has also been positively linked to BPD symptoms. For example, Distel et al. (2009) conducted multivariate genetic analysis to explore relationships between five-factor model (FFM) personality traits and BPD symptoms; using data on 10,489 twins and siblings, covariance between these constructs was broken into genetic and environmental influences. They found that level of neuroticism significantly predicts BPD symptoms and that 50% of this phenotypic association can be accounted for by shared genetic factors. Further, they found that all genetic variation in BPD is shared with personality traits. Kendler et al. (2011) found similar results in regard to their latent BPD factor (analogous to neuroticism); specifically, they found that this factor was highly heritable (60%), with the remaining variance in BPD symptoms explained by specific psychological factors. Interestingly, they found affective instability (one of the four areas of BPD dysfunction measured in their study) had the highest genetic contribution (67%), consistent with the notion that BPD is primarily a disorder of emotion dysregulation.

Neuroticism has been linked to a functional polymorphism in the promoter region of the serotonin transporter gene (5HTTPR; Lesch et al., 1996). This functional polymorphism, specifically the presence of the s/s genotype (presence of two short alleles), has been associated with a greater magnitude of amygdala responses to emotional stimuli (Caspi et al., 2003; Hariri & Weinberger, 2003) and reductions in positive connectivity between the amygdala and ventromedial prefrontal cortex (Pezawas et al., 2005). The presence of two short alleles of the 5-HTTLPR polymorphism has also been associated with BPD symptoms (Pascual et al., 2007). For example, Maurex, Zaboli, Ohman, Asberg, and Leopardi (2010) found that this genetic variation is associated with not only BPD-specific symptoms, but also symptoms of depression and anxiety. The 5-HTTPR-S allele has also been implicated in obsessive–compulsive disorder (Lin, 2007), major depressive disorder (Contreras et al., 2009), general anxiety disorder (Narasimhan et al., 2011), and posttraumatic stress disorder (Wang et al., 2011), perhaps underscoring comorbidity among these disorders. Overall, the literature suggests that there is a strong biological component in the development of BPD and other emotional disorders; there is evidence supporting the notion that neuroticism is inherited and positively linked to overactive amygdala functioning, likely increasing an individual's vulnerability to such disorders.

Generalized Psychological Vulnerability

It is important to note that, in the context of the triple vulnerability model, a person with biological vulnerability in the form of the genetic predisposition described above would be unlikely to develop an emotional disorder in the absence of concurrent psychological vulnerabilities; rather, he or she would experience exaggerated responses to stress within the realm of normal functioning. Global psychological vulnerability in the triple vulnerability model refers to a sense of unpredictability or uncontrollability of life events and the perception that one is ill-equipped to manage such events as they occur (Barlow, 1988). Among other sources, such as generally disruptive early life experiences, this sense of uncontrollability is thought to arise from two early learning experiences with parents (for a review, see Chorpita & Barlow, 1998). First, parents who are more contingently responsive afford their children greater opportunity to exert control by influencing parents' behavior in a predictable manner, and, second, parents who are less protective provide their children with more opportunity to develop independent skills to cope with unexpected environmental events. The notion that there are environmental factors that lead to characteristic psychological responding is consistent with the results found in the studies (described above) identifying the genetic contribution of neuroticism in BPD and other emotional disorders. Although both Distel et al. (2009) and Kendler et al. (2011) found that a large proportion of the variance in BPD was genetically mediated, the authors also note that significant variance was accounted for by environmental influences. Lyons-Ruth (2008) reviewed the literature on early parent–child interactions and their impact on subsequent development of BPD symptoms. Results suggest that maternal withdrawal in response to attachment overtures from their 18-month-old children significantly predicted BPD features at age 19, even when controlling for genetic contributions and later traumatic experiences. In other words, infants with mothers who fail to respond to them or respond in a contradictory manner may have difficulty developing a sense of control over their environment, putting them at risk of emotional disorders, including BPD.

Specific Psychological Vulnerability

The third component in the triple vulnerability model is a specific psychological vulnerability that may account for why one particular *DSM* emotional disorder emerges instead of another. For example, an individual may develop social anxiety disorder, in particular, by observing caregivers modeling behavior which suggests that being evaluated negatively in social situations because of clothing styles or behavior is catastrophic (Suarez, Bennett, Goldstein, & Barlow, 2009). Linehan (1993) described a parenting style, deemed "childhood invalidating environment," that may go beyond the effects of lack of perceived control in explaining the emergence of BPD symptoms, specifically. Invalidating environment refers to a childhood home life characterized by repeated criticism, minimization, and punishment of emotional expressions. Linehan suggests that these parental attitudes toward emotions cultivate the belief that strong emotions are truly damaging and should be quickly terminated, leading to the use of extreme avoidant behaviors (i.e., self-injury, substance use) as a way to accomplish this goal. Several self-report studies have demonstrated a significant relationship between Linehan's invalidating environment and the development of BPD symptoms (Cheavens et al., 2005; Rosenthal et al., 2005) and that fear of emotions mediates the relationship between invalidating environment and avoidant coping (Sauer & Baer, 2009).

Overall, the literature suggests that a genetically mediated diathesis and acquired deficits in perceived control over environmental events represent global vulnerabilities for the development of neuroticism and emotional disorders, including BPD. But, as noted above, research also suggests that certain life experiences, such as childhood parental invalidation (Linehan, 1993), may lead to the expression of BPD, in particular.

Malleability of Neuroticism in BPD

Despite overlap between personality traits and personality disorder diagnoses/symptoms, it is believed that they are separate constructs that are each important in the prediction of functioning (Morey et al., 2007; Zanarini et al., 2007). A five-factor model representation of BPD, chiefly featuring high levels of

neuroticism, is a stronger (stable) predictor of 2-year and 4-year outcomes (BPD symptoms and functional impairment) than *DSM-IV* criteria severity (APA, 2000; Morey & Zanarini, 2000). A similar pattern of results regarding stability of temperamental and *DSM* symptom variables has been demonstrated for other emotional disorders (Brown, 2007). Further, changes in FFM representations of BPD significantly predict future change in BPD symptoms, whereas changes in BPD symptoms do not reflect changes in temperamental functioning (Warner et al., 2004). These findings are consistent with the notion that personality functioning, particularly neuroticism, is at the core of BPD, whereas symptoms may represent more transient manifestations. Zanarini and Frankenberg (2007) made a similar observation, suggesting that temperamental features (high levels of neuroticism) represent the core feature of BPD and that ineffective strategies for modulating strong emotions (impulsive actions) lead to symptomatic presentations (unstable relationships). Overall, this pattern of results suggests that neuroticism is a relatively stable temperamental vulnerability for BPD and other emotional disorders, whereas disorder symptoms may vary widely over time in response to life events.

Although temperamental variables appear to be more stable than disorder symptoms, these traits do show malleability over time (Paris, 1993; Zanarini, Frankenburg, Henen, Reich, & Silk, 2005). Investigations examining longitudinal changes in neuroticism in the normal population show gradual age-related decreases that continue into old age (Roberts & Mroczek, 2008; Roberts, Walton, & Viechtbauer, 2006). Further, in a recent review of the literature, Clark (2009) noted that traits and behaviors making up the definition of most personality disorders also change slowly over time, with the greatest change occurring in the behavioral manifestations of these traits. Studies examining the stability of temperamental functioning in BPD specifically have also yielded interesting results. For example, Hopwood et al. (2009) found that individuals with BPD exhibit greater decreases in neuroticism over time than individuals with other personality disorders; BPD patients also demonstrate increased variability in the amount of change relative to patients with other personality disorders, with some people remaining at stable levels and others changing a great deal. In general, neuroticism in individuals with BPD appears to decrease and become more stable as individuals age. Given that trait conceptualizations of BPD, particularly featuring high neuroticism, strongly predict symptoms and functioning (Warner et al., 2004), increasingly stable decreases in neuroticism may call into question the notion that BPD is largely unchangeable. Interestingly, cross-lagged path analyses revealed that changes in temperamental features of BPD led to change in acute symptoms (e.g., impulsive behaviors, stormy relationships; Hopwood & Zanarini, 2010). Hopwood et al. (2009) postulated that these decreases in neuroticism may reflect the effects of treatment and that future research should address the direct treatment of underlying temperamental vulnerabilities.

IMPLICATIONS FOR THE TREATMENT OF BPD

Extant Treatments for BPD

Currently, the treatment for BPD with by far the most empirical support is dialectical behavior therapy (DBT; Linehan, 1993; Neacsiu & Linehan, in press). Over 20 years ago, when little was known about the nature of emotional disorders and even less about personality disorders, Marsha Linehan developed a heterogeneous treatment approach for BPD influenced by the tenets of behavior therapy, Eastern religious traditions, and her own clinical and personal experiences. This seminal effort resulted in an efficacious treatment, the first for a personality disorder, that has relieved unremitting suffering in thousands of individuals. In its standard outpatient form, DBT involves weekly individual sessions and weekly group skills sessions lasting approximately 1 year or more. The content of the individual sessions is organized around a target hierarchy aimed at reducing behavioral symptoms of BPD and increasing skillful behavior; reduction in suicidal and self-injurious behaviors is paramount, followed by "therapy-interrupting" behavior, followed by "quality-of-life-interrupting" behavior. Weekly skills group covers broad topics including interpersonal skills, distress tolerance (coping skills), skills for understanding and tolerating emotions, and mindfulness. Additionally, DBT also includes a therapist consultation group for providers of the treatment to obtain support while working with this difficult population.

In a qualitative review summarizing the early years of DBT outcome research, including Linehan, Armstrong, Suarez, Allmon, and Heard (1991), Linehan, Armstrong, and Heard (1993), Linehan, Tutek, Heard, and Armstrong (1994), and two inpatient adaptations (Barley et al., 1993; Springer, Lohr, Buchtel, & Silk, 1996), Scheel (2000) indicated that DBT is associated with reduced engagement in nonsuicidal self-injury, psychiatric hospitalization, anger, and psychotropic medication use and with increased client retention, overall level of functioning, and overall social adjustment. The author notes, however, that DBT does not appear to be associated with improved depression, hopelessness, suicidal ideation, and anxiety symptoms. More recently, Kliem, Kroger, and Kosfelder (2010) conducted a meta-analysis of the efficacy and long-term effectiveness of DBT. The authors, summarizing multiple outcome measures within and across studies, determined that there is a moderate global effect for DBT, which holds true when DBT is compared to treatment as usual. The authors, however, urge caution in the interpretation of this effect; due to the fact that a variety of outcomes from each study were included, the effect size likely represents a combination of some outcomes that changed quite a bit (e.g., nonsuicidal self-injury) and some outcomes that changed very little (e.g., depression symptoms), consistent with Scheel's (2000) review. Emerging research (Neacsiu, Rizvi, & Linehan, 2010) examining mediators of response in DBT suggests that acquisition of DBT skills, broadly defined, mediated reductions in symptoms; the study did not attempt to examine specific effects of the multiple components included in DBT.

Several additional interventions have garnered some empirical support for the treatment of BPD. Psychodynamic approaches currently receiving attention include transference-focused therapy (TFT; Clarkin et al., 2001; Kernberg, 1975, 1984) and mentalization-based treatment (MBT; Batemen & Fonagy, 2004). The goal of TFT is the resolution of intrapsychic conflict by therapist employment of interpretation, maintenance of technical neutrality, and transference analysis. MBT entails increasing mentalization or increasing the patient's ability to understand the actions of the self and others on the basis of intentional mental states such as desire, feelings, and beliefs; specific techniques are not delineated, as therapists are encouraged to use any strategy that best restores mentalization (Bateman & Fonagy, 2010). Additionally, schema-focused therapy (SFT; Young, Klosko, & Weishaar, 2003), a cognitive-behavioral approach, has also demonstrated efficacy in the treatment of BPD. SFT includes techniques developed to challenge early schemas by identifying dysfunctional schema modes that influence the individual's thoughts, emotions, and behaviors (i.e., detached protector, punitive parent, abandoned/abused child, angry/impulsive child). While DBT has accumulated considerable empirical support and TFT, MBT, and SFT somewhat less (for a review, see Neacsiu & Linehan, in press), these approaches are all intensive, long-term, technically sophisticated treatments requiring extensive training and support for therapists, as befitting treatment for a severe personality disorder.

Treating BPD as an Emotional Disorder

If BPD is an emotional disorder, as we are suggesting, then transdiagnostic interventions targeting emotional disorders generally may provide a parsimonious, cost-effective, and focused approach for BPD and associated comorbid conditions. Of course, interventions can be "transdiagnostic" in a number of ways. For example, cognitive therapy is broadly transdiagnostic in that efficacy has been shown to extend across the full range of psychopathology (Chambless & Ollendick, 2001). Similarly, mindful awareness approaches and acceptance and commitment therapy (ACT; Hayes et al., 2006) have been utilized across a number of disorders with some evidence for efficacy (Smout et al., 2012). But these general treatment approaches, much like pharmacological interventions such as selective serotonin reuptake inhibitors, are not necessarily theoretically linked to a specific class of disorders but rather postulate a broad, nondiagnosis-specific therapeutic goal such as altering attributions and appraisals, increasing mindful awareness of experience, or reducing experiential avoidance. Although these treatment approaches have proven heuristic, each addressing deficits that exist across disorders and would likely benefit patients with BPD in a similar manner, recent evaluations have demonstrated rough equivalence in the context of specific disorders, suggesting possible common, overarching mechanisms of action. For example,

Hayes-Skelton, Roemer, and Orsillio (2013) recently demonstrated no advantage of acceptance-based behavior therapy over applied relaxation in the treatment of generalized anxiety disorder. Similarly, there appear to be no differences in the ability of ACT versus traditional cognitive-behavioral interventions to address a range of anxiety disorder symptoms (Arch et al., 2012; Wolitzky-Taylor, Arch, Rosenfield, & Craske, 2012). An intriguing question is whether these treatments are each successful by targeting totally different, yet equally important mechanisms, or whether there is a common mechanism of action that is similarly addressed across these treatments.

More recently, transdiagnostic treatment approaches have emerged that address putative core mechanisms and temperamental factors underlying a class of psychopathology. The first example of this strategy was Christopher Fairburn's transdiagnostic approach to eating disorders where the core causal factor of distorted perceptions of body shape, common to all eating disorders, was directly addressed (e.g., Fairburn et al., 2009). In our own setting, we have recently developed a unified transdiagnostic approach specific to emotional disorders focusing on the temperament of neuroticism itself as the common underlying element across disorders of emotion (Barlow et al., 2011). This treatment involves careful assessment of psychopathology and the functional establishment of an "emotional disorder" diagnosis, characterized by the frequent experience of negative affect and aversive reactions to it; however, considerations of broad patters of comorbidity with other emotional disorders are less necessary, as the putative core psychopathology in all emotional disorders is being addressed directly. Preliminary data suggest promising outcomes using this approach across the spectrum of anxiety and mood disorders (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010; Ellard et al., 2010; Farchione et al., 2012). Treating five individuals with BPD and comorbid mood and anxiety disorders with an unmodified version of this treatment (unified protocol for the transdiagnostic treatment of emotional disorders) produced significant improvements in BPD, anxiety and depressive symptoms, and emotion regulation skills in an open case series (Sauer-Zavala, Bentley, Wilner, & Barlow, in preparation). Overall, these data suggest that transdiagnostic treatments focusing on core mechanisms of emotional disorders represent a theoretically plausible approach to treating BPD, but additional confirmation is, of course, necessary.

SUMMARY AND CONCLUSIONS

The goal of this article is to review the literature supporting the notion that BPD is an emotional disorder and to use this information as a heuristic to reconceptualize existing treatments and develop more streamlined, targeted interventions for emotional disorders that would be applicable to BPD. Thus, we review literature providing evidence that BPD is characterized by the hallmarks of emotional disorders: the experience of intense emotions coupled with negative reactions to these experiences. Such negative evaluations of internal experience may lead to maladaptive avoidant coping, both cognitive (thought suppression and rumination) and behavioral (self-injury, binge eating, ritual development, etc.), that paradoxically exacerbates symptoms. Second, overlap between BPD and other emotional disorders was described, highlighting the high rates of comorbidity among BPD, anxiety disorders, and mood disorders. It is clear that comorbidity impacts the presentation and treatment response of these disorders, and few interventions have been designed with such high rates of overlap in mind. Next, temperamental variables including neuroticism were forwarded as shared underlying vulnerabilities that may account for the similarities among emotional disorders, including BPD. Levels of self-reported neuroticism are high in individuals with BPD and mood, anxiety, and related disorders; additionally, BPD, panic disorder, social anxiety disorder, major depressive disorder, generalized anxiety disorder, and posttraumatic stress disorder all load highly on a latent neuroticism factor that explains a high degree of variance in their presentation. These findings suggest that comorbidity among BPD and other emotional disorders may be largely accounted for by a shared underlying vulnerability. Further, although temperamental variables such as neuroticism are relatively stable, there is evidence that they demonstrate malleability over time, suggesting that they may be a reasonable target for the treatment.

Finally, one implication of BPD as an emotional disorder is that emerging transdiagnostic treatments for emotional disorders that are more parsimonious,

scalable, and cost-effective than extant approaches to personality disorders come into play. This could include reconceptualizing and streamlining existing longer-term approaches to BPD by identifying and focusing on active mechanisms. BPD is a costly and debilitating disorder (McGlashan et al., 2000; Sansone, 2004; Skodal et al., 2002) that warrants continued treatment development. A deeper understanding of the nature of BPD as an emotional disorder may facilitate that process.

NOTE

1. There is evidence to suggest that emotional dysregulation (anxiety and depression) often precedes substance use, which may function to escape or avoid these negative emotions (Fischer, Smith, Annus, & Hendricks, 2007); however, once physiological addiction sets in, the substance use can become functionally independent from the negative emotion experienced.

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