

Research Report

Post-Traumatic Stress Disorder Mediates the Relationship Between Major Depression and Borderline Personality Disorder

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Previous research has examined the relationship between Borderline Personality Disorder (BPD) and other forms of psychopathology. Although the association between Major Depressive Disorder (MDD) and BPD has received empirical (e.g., Pinto, Grapentine, Francis, & Picariello, 1996) and conceptual (e.g., *DSM-IV-TR*, APA, 2000) support this association falls short of fully depicting the complex etiology of BPD. Previous research suggests that Post-traumatic Stress Disorder (PTSD) may be a cognitive-affective mediator between MDD and BPD. Participants ($n = 324$) were administered the Coolidge Axis II Inventory (CAII), a measure of Axis I and II psychopathology. Multiple regression analysis confirmed the original mediating hypothesis. These findings provide impetus for the development of a more informative model concerning the relationship between MDD and BPD.

Keywords: Borderline Personality Disorder, Depression, PTSD, mediator

The Diagnostic and Statistical Manual, 4th Edition, Text Revision (DSM-IV-TR) defines a personality disorder as “an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual’s culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment” (p. 685; APA, 2000). Borderline Personality Disorder (BPD) is one of 10 personality disorders classified in the *DSM-IV-TR*. BPD is frequently diagnosed in clinical settings and continues to be one of the most researched personality disorders in terms of phenomenology, biological markers, treatment response, family history, and outcome (Kavoussi, Coccaro, Klar, Bernstein, & Siever, 1990; Linehan, Cochran, & Kehrer, 2001). Individuals that suffer from BPD display great affective and interpersonal instability, including major instability in mood (e.g., major depression, shame, rage), an unstable self-image, and poor regulation of behavior (e.g., impulse control, suicidality). These symptoms combine to create unstable relationships (*DSM-IV-TR*, 2000).

Due to its complex and problematic symptomatology, BPD is considered one of the most demanding clinical disorders that therapists encounter (Linehan, Cochran, & Kehrer, 2001). Traits associated with BPD that create treatment challenges include emotional instability, unstable relationships, and impulsivity. These behaviors can lead to reckless and unhealthy behaviors, a high rate of

parasuicidal behavior, as well as completed suicide (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2005). Additionally, individuals with BPD commonly meet criteria for other Axis I disorders, including substance abuse, major depression, and Post-Traumatic Stress Disorder (PTSD). The comorbidity of BPD with Axis I disorders, which is characteristic of BPD, often makes an accurate diagnosis and treatment planning difficult. Therefore, clarifying the complex etiological nature of BPD may facilitate diagnosis, assessment, and treatment of this disorder.

In an effort to better understand BPD’s complex pathology, investigators have focused on elucidating the association between BPD and Major Depressive Disorder. A wide variety of literature clearly specifies the etiological relationship between Major Depressive Disorder (MDD) and BPD, where MDD is represented as a significant causal factor in the development and maintenance of BPD in both adolescents (Pinto, Grapentine, Francis, & Picariello, 1996) and adults (Russ, Clark, Cross, Kemperman, Kakuma, & Harrison, 1996). The *DSM-IV-TR* (2000) clearly delineates emotional variability to be a major diagnostic indicator of BPD, yet the diagnostic criteria for BPD show little, if any, overlap with depressive symptomatology.

Although empirically established, the relationship between MDD and BPD is not well understood at a conceptual and clinical level. Previous research suggests that PTSD may help explain this relationship, given the high prevalence of PTSD and trauma history in persons with BPD (McGlashan et al., 2000; Sar et al., 2003; Zlotnick, Franklin, & Zimmerman, 2002). For example, a replication of the National Comorbidity Study found a moderate

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correlation ($r = .50, p < .05$) between MDD and PTSD (Kessler, Chiu, Demler, & Walters, 2005), however correlation does not indicate causation and so these results should be interpreted with caution.

Some research indicates that these factors also potentially contribute to the development of BPD. Golier et al. (2003) found that individuals with BPD reported significantly higher rates of childhood/adolescent physical abuse and were twice as likely to develop PTSD. Additionally, trauma associated with BPD has been linked to pervasive physiological and neuropsychological changes (Goldman, D'Angelo, DeMaso, & Mezzacappa, 1992; Guzder, Paris, Zerkowitz, & Feldman, 1999; Guzder, Paris, Zerkowitz, & Marchessault, 1996; Yen, et al., 2002). In summary, empirical evidence suggests that PTSD may be critically related in elucidating the complex relationship between MDD and BPD.

The general purpose of the present study was to investigate the role played by PTSD in the relationship between MDD and BPD. Specifically that PTSD may be a critical variable in elucidating the complex relationship between MDD and BPD. The present study was part of a larger study examining the relationship between depression, emotional variability, and trauma in a college student population.

Method

Participants and Procedure

Participants were undergraduate college students enrolled in Introduction to Psychology classes at two public universities. Of the 315 total participants, 208 (63.4%) were female and 107 (36.6%) were male. The mean age of the sample was 22.07 years ($SD = 6.07$; range = 18 to 81 years). When asked to self-identify their ethnic background, 256 (70.1%) students in the sample identified themselves as Caucasian, nine (2.8%) as Native American, seven (2%) as Hispanic, 14 (4.3%) identified themselves as Other, and 38 (12%) did not complete this question. No participants had received a diagnosis of PTSD, MDD, or BPD.

Participants were administered the Coolidge Axis II Inventory (CATI; Coolidge, 1993). Students either received extra credit as compensation for their participation or their participation fulfilled a course requirement. Participants completed all study measures in one sitting.

Materials

The CATI is a well-validated 225-item self-report measure designed to assess Axis I and II disorders and neuropsychological functioning in individuals over 15 years of age (Coolidge, 1993). The measure contains four

validity scales, with the BPD scale demonstrating acceptable score reliability ($\alpha = .80$; Coolidge, 1993) for research purposes as outlined by Nunnally and Bernstein (1994). The measure takes 35 to 40 minutes to complete and assesses five Axis I disorders, 13 Axis II personality disorders, and contains nine normative scales. For the purpose of this study, the CATI was used to capture MDD, BPD, and PTSD as separate psychological constructs, consistent with the criteria outlined in the *DSM-IV-TR* (APA, 2000).

Results

Multiple regression analysis confirmed that PTSD mediated the relationship between MDD and BPD. Results showed that MDD significantly predicted BPD (see Figure 1, Path *c*; $B = .741, t(323) = 18.63, p < .0001, Adj R^2 = .517$). Additionally, MDD significantly predicted the mediator variable, PTSD (see Figure 2, Path *a*; $B = .664, t(323) = 29.11, p < .0001, Adj R^2 = .724$). While statistically controlling for the effect of MDD, the findings showed that PTSD significantly predicted BPD above and beyond the influence of MDD (see Figure 2, Path *b*; $B = 1.04, t(323) = 22.98, p < .0001, Adj R^2 = .620$). Lastly, results revealed that MDD significantly predicted BPD while controlling for the effect of PTSD (see Figure 2, Path *c*; $B = .184, t(323) = 2.76, p = .006, Adj R^2 = .628$).

According to Baron and Kenny (1986), the strongest evidence of mediation is found when the *beta* value from

Figure 1. Major Depressive Disorder (MDD) significantly predicted Borderline Personality Disorder (BPD).

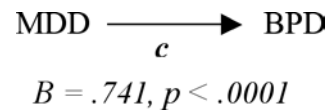
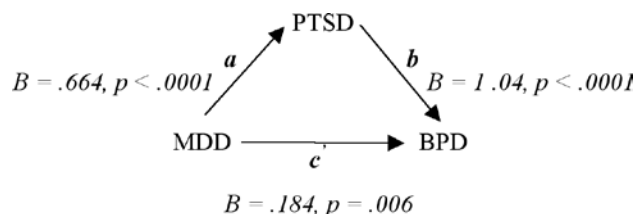


Figure 2. Post-Traumatic Stress Disorder (PTSD) significantly mediates the relationship between Major Depressive Disorder (MDD) and Borderline Personality Disorder (BPD).



the initial association (e.g., between MDD and BPD) is reduced to zero after controlling for the effect of a mediating variable. In the current analysis, the *beta* value decreased from .741 to .184. Given the resulting *beta* value was not zero, the Sobel test (Kenny, 2006; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Sobel, 1982) was used to determine whether the indirect effect of MDD on BPD via PTSD was significantly different from zero. A *z* score was calculated using the equation $\sqrt{b^2s_a^2 + a^2s_b^2 + s_a^2s_b^2}$. Using a standard confidence interval of $\alpha = .05$, PTSD was found to be a statistically significant mediator in the relationship between MDD and BPD ($z = 18.03, p < .0001$).

Discussion

The purpose of the present investigation was to determine whether PTSD mediated the relationship between MDD and BPD. The original mediational hypothesis was supported. Based on these findings, PTSD appears to be a prominent mechanism through which MDD affects BPD. This relationship was also supported conceptually, as this finding appears to fit well into the existing literature based on this topic. To our knowledge, this study provides the first empirical support for the mediating effect of PTSD on the relationship between MDD and BPD.

These results have important research and clinical implications. From a research perspective, comorbid MDD and BPD is a well-documented clinical phenomenon (Pinto, Grapentine, Francis, & Picariello, 1996; Russ, et al. 1996). However, at the present, little empirical evidence exists which demonstrates that individuals with MDD develop more severe presentations of BPD due to history of trauma (e.g., PTSD). From a clinical perspective, individuals with MDD who are exposed to significant and repeated trauma may be at increased risk for developing BPD, which is consistent with Briere's (2002) self-trauma model. This model suggests that severe childhood trauma interrupts normal development, conditions negative affect to stimuli related to abuse, and interferes with the typical acquisition of affect regulation skills. Perhaps when some individuals with MDD experience a significant trauma, the negative affect and cognitive distortions related to MDD are exacerbated and become even more complex, thus triggering symptoms related to BPD.

The findings also have important implications for the diagnosis and assessment of BPD. Although the *DSM-IV-TR* (2000) explicitly states that emotional valence is a major diagnostic indicator of BPD, the diagnostic criteria for BPD notably do not include depressive symptoms. Should our proposed model withstand replication with a large and diverse clinical sample, future editions of the

DSM may include MDD symptomatology as an important criteria for BPD.

Another implication of the current findings involves assessment considerations with persons with BPD. Our findings suggest that the prevalence rates of BPD in individuals with Major Depressive Disorder increase when there is a history of trauma. In other words, individuals with Major Depressive Disorder and PTSD may be more likely to develop BPD compared with individuals diagnosed only with Major Depressive Disorder. Therefore, including PTSD measures when assessing for BPD may provide clinicians with a greater understanding of the origins and development of their patient's BPD.

These results have important implications for the development of psychological interventions aimed at reducing the symptoms of BPD. Given that PTSD was a significant mediator in the relationship between MDD and BPD, PTSD should be considered a prominent risk factor for the development of BPD in those individuals with comorbid MDD and PTSD. Incorporating treatments geared toward addressing trauma-related symptoms may improve therapy outcomes in patients with BPD. For example, one effective intervention for persons with BPD (Feigenbaum, 2007) that considers the impact of trauma is Dialectical Behavior Therapy (DBT; Linehan et al., 2001).

The generalization of the current results are limited due to fact that the sample consisted of non-clinical participants. Although the inclusion of a sample of BPD patients would greatly extend the external validity of the study and allow researchers to speak more specifically to the diagnostic and treatment aspects of BPD, the present sample does little to limit the primary findings of this study. In an effort to examine the mediational effects of PTSD on MDD and BPD, a sample with clinical levels of psychopathology is not statistically or conceptually required. An additional limitation is the homogeneous nature of the sample. Although a power analysis suggested that the sample size was adequate to detect medium effect sizes, the sample included primarily Caucasian (non-Hispanic) college students. Future studies in this area may wish to gain more cultural and life-span diversity in their samples in an effort to generalize results to populations of various ages and ethnic and cultural backgrounds.

Future research should explore alternative or additional variables to fully explain the strong association between MDD and BPD, and additionally, future research may attempt to examine which latent constructs of PTSD best explain the relationship between MDD and BPD. Future empirical endeavors should also use a pure longitudinal design to explore whether the duration of PTSD-related symptoms in participants with MDD influence the observed incidence of BPD.

Results from this study provide a more comprehensive depiction of the multifarious nature of BPD. In addition to providing meaningful scientific evidence of the robust mediating nature of PTSD in the relationship between MDD and BPD, findings suggest that individuals with MDD and PTSD may be more likely to develop BPD than individuals with MDD alone. Replication of similar findings with clinical samples may aid more accurate diagnostic procedures, facilitate more efficient assessment procedures, and guide clinicians in effective treatment planning with patients with BPD.

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