

Borderline Personality Disorder: Disorder of Trauma or Personality, a Review of the Empirical Literature

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This goal of this review was to explore empirical research examining the question of whether borderline personality disorder (BPD) is a disorder of “personality” or a disorder arising out of experiences of childhood trauma. The review highlighted the complexities in the relationship between childhood disorder (CT) and BPD and identified important implications for research and practice. Although relationships between specific trauma types and outcomes in adulthood are inconsistent, overall associations between CT and the development of BPD are strong and consistently identified. Research exploring the specific mechanisms through which CT may be related to the development of BPD in adulthood is beginning to untangle the complex web of interrelated factors such as heritable personality traits, affect regulation and dissociation, and trauma symptoms as mediators in the relationship between CT and BPD. Our strongest recommendation is for future researchers to further explore transdiagnostic factors such as the self capacity of affect regulation to further disentangle the complex pathways between CT, inherited personality traits, and the development of all forms of traumatogenic psychopathologies in adulthood.

Keywords: childhood trauma, childhood sexual abuse, borderline personality disorder, complex posttraumatic stress disorder, personality disorders

Over the past 30 years, the deleterious impact of childhood trauma (CT; i.e., child sexual abuse, child psychological or physical neglect, parental violence, witnessing intimate partner violence, and other types of child maltreatment) has been well established in the literature. CT has been associated with a wide range of pervasive difficulties in all areas of adult functioning including psychological (e.g., Briere, Hodges, & Godbout, 2010; Godbout & Briere, 2012) and relational difficulties (e.g., Godbout, Dutton, Lussier, & Sabourin, 2009; Godbout, Lussier, & Sabourin, 2006; Godbout, Runtz, MacIntosh, & Briere, 2013). In one particular line of inquiry, researchers began to explore the question of whether a history of childhood sexual abuse (CSA) or other forms of CT, especially when committed by an attachment figure (i.e., “high-betrayal trauma”; Freyd, 1996; Kaehler & Freyd, 2009), played a causal role in the development of borderline personality disorder (BPD; Herman, Perry, & van der Kolk, 1989; Landecker, 1992). As this discussion emerged in the literature, so too did the question of whether BPD was the most appropriate diagnosis to

better reflect the symptomatology of CSA survivors, whose clinical presentation was more complex than other adult trauma survivors diagnosed with posttraumatic stress disorder (PTSD; Herman, 1992; Pearlman, 2001; Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997; van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005).

BPD continues to be a controversial diagnosis with this controversy being based on the significant symptom overlap with other disorders including PTSD and other axis I disorders (Pagura et al., 2010; Zanarini et al., 1998), the diversity of patients receiving the diagnosis (Maffei, 2005), and the lack of support in the literature regarding the reliability and validity of BPD as a diagnostic entity (Becker, 2000; Gunderson, 2009). Over time the diagnosis of BPD came to have significant negative connotations in the minds of many clinicians, whereby individuals diagnosed with PTSD were more likely to be viewed as victims of traumatic events rather than possessing intractable personality problems (Quadrio, 2005). However, the *symptom complexity* (Hodges et al., 2013) or simultaneous presence of multiple, different types of symptomatology in the same trauma survivor was not well represented by the diagnostic category of PTSD (e.g., Briere et al., 2010; van der Kolk et al., 2005). One of the main preoccupations of clinicians and researchers has been to better understand the etiological origins of BPD to formulate well-tailored approaches to prevention and intervention.

In the initial literature considering these questions, authors consistently reported associations with CT, CSA in particular (Herman et al., 1989; Zanarini et al., 1997). These researchers posited that

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survivors of CT, especially severe long-lasting CT, were suffering from a complex form of PTSD, Complex PTSD, or Disorders of Extreme Stress Not Otherwise Specified (DESNOS) that shared many similarities with BPD including affect dysregulation, impulsivity, suicidality, feelings of emptiness and other identity impairments, chronic interpersonal difficulties, abandonment issues, anger, and dissociation (Pearlman, 2001; Roth et al., 1997; van der Kolk et al., 2005). This debate has continued to date with researchers assuming positions encompassing a wide expanse of conceptualizations of BPD. These range from the position that BPD is “little more than a sophisticated insult” (Herman, 1992, p. 123) and that the symptoms identified in most trauma survivors (mis)diagnosed with BPD reflect Complex PTSD (Briere et al., 2010; Ford, Courtois, Coutois, & Ford, 2009; Herman, 1992; Herman et al., 1989), all the way to positions that suggest a strong genetic and neurobiological foundation for the disorder regardless of trauma history.

At the heart of this controversy is a diagnostic conceptual confusion. PTSD is an axis I disorder that suggests a disorder of short duration that is amendable to treatment, whereas BPD is an axis II disorder suggesting a disorder of long duration that, based on clinical evidence, has a poor prognosis. These diagnostic differences reflect the theory that these disorders are conceptually distinct (Zlotnick et al., 2003). However, despite this conceptual separation, the durations of both disorders can be longstanding and persistent (Gunderson & Zanarini, 1987; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Zlotnick et al., 1999). Empirical studies have identified rates of comorbidity between PTSD and BPD in clinical samples ranging from 56% to 76% (Heffernan & Cloitre, 2000; Shea, Zlotnick, & Weisberg, 1999; Zlotnick et al., 2003) and up to 30% in community samples (Swartz et al., 1990). Additionally, there is significant overlap of core features including affective instability and interpersonal dysfunction (Rowan, Foy, Rodriguez, & Ryan, 1994; van der Kolk et al., 1996; Zanarini et al., 1997). It is no surprise, then that a number of authors have proposed that BPD consists of a complex or chronic form of PTSD. While the pejorative connotation of BPD may have played a role in this movement (e.g., Gunderson & Sabo, 1993; Herman, 1992; Herman & van der Kolk, 1987), this proposal arose out of clinical observations and research that identified significant associations between BPD and PTSD, and CSA (Lewis & Grenyer, 2009). Herman and van der Kolk (1987) further observed that both disorders (BPD and PTSD) shared similar disturbances in five core domains: affect regulation, impulse control, reality testing, interpersonal relationships, and self-integration. Supporters of this proposal argued that these associations might offer a plausible explanation for the higher rates of BPD diagnosed in women: 75% according to *Diagnostic and Statistical Manual for Mental Disorders-Fifth Edition (DSM-V)*; American Psychological Association, 2013; Landecker, 1992) because of the higher prevalence of abuse, particularly CSA in girls (Herman & van der Kolk, 1987; Skodol & Bender, 2003). More recently, however, it has been observed that rates of BPD are similar between men and women (Grant et al., 2008) but that women may present with higher levels of disability and comorbidities such that the clinical presentation differs enough to result in sampling biases in research studies (Grant et al., 2008; Sansone, Hanh, Dittoe, & Wiederman, 2011). Many researchers have come to the conclusion that the long-term impacts of CT are not readily encompassed within any

particular *DSM* diagnostic category (Ford, 1999; Ford, Stockton, Kaltman, & Green, 2006; van der Kolk & Courtois, 2005; van der Kolk et al., 2005).

However, the proposal that BPD should be reconceptualized as a form of Complex PTSD has been criticized as too simplistic with authors such as Goodman and Yehuda (2002) pointing to the lack of empirical support for the causal relationship between BPD and CT. Critics have argued that the main problem with the Complex PTSD proposal is that it conflates risk with causation. The presence of a risk factor, CT, is not necessary or sufficient to explain the emergence of the BPD, and to explain why some survivors will develop BPD symptoms whereas others do not, although no clear data is available on the exact percentage of CT survivors who will grow up developing BPD (Lewis & Grenyer, 2009). These critics further suggest that the conceptualization of BPD as Complex PTSD places too much emphasis on CT as the primary cause of BPD, and fails to integrate the systemic multidimensional etiologic factors of BPD (Lewis & Grenyer, 2009). Offering a more sophisticated interactionist position based on their review of the scientific documentation, authors such as Goodman and Yehuda (2002) have proposed that for some individuals, the emergence of a constellation of BPD related symptoms is a result of CT interacting with pre-existing temperament and biological vulnerabilities.

A limited body of research has explored the interrelations between a diagnosis of BPD and the specific conceptualization of Complex PTSD. In one study with a small clinical sample of 65 women diagnosed with BPD, Complex PTSD was present in all cases (McLean & Gallop, 2003) whereas another larger study of 472 consecutively admitted patients into inpatient psychotherapy treatment centers demonstrated that Complex PTSD, in patients reporting having experienced severe trauma, could be differentiated from BPD and found only 40% comorbidity (van Dijke, Ford, van Son, Frank, & van der Hart, 2013). In a recent statistical exploration of this question, Cloitre, Garvert, Weiss, Carlson, and Bryant (2014) used a latent class analysis of archival data to determine whether the patterns of symptoms endorsed by 280 women seeking treatment for CT form classes that are consistent with diagnostic criteria for PTSD, Complex PTSD, or BPD, and identified four distinct groups: (a) a low symptom group, (b) a high levels of PTSD symptoms and low levels of CPTSD symptoms group, (c) a Complex PTSD group with high levels of PTSD and self-organization symptoms characteristic of CPTSD and low on symptoms of BPD, and (d) a group with symptoms of BPD alone. They identified four symptoms that strongly predicted class membership in the BPD group: desperate attempts to avoid abandonment, an unstable sense of self, unstable and intense interpersonal relationships, and impulsiveness. These findings support the argument that BPD and Complex PTSD, as currently conceptualized, may be conceptually distinct and that research is needed to further understand the links between CT and BPD.

In response to these important questions, conceptualizations and critiques, this article presents a review of the empirical literature specifically exploring the relationship between CT and BPD in adulthood, organized according to the three main domains of research that will be presented in the next sections: (a) studies on bivariate links between CT and BPD, (b) studies on multivariate links between CT and BPD, and (c) studies on the mechanisms linking CT to BPD. All of the studies reviewed utilized diagnostic criteria for BPD and PTSD from *DSM-IV* or earlier. Research

published in 2014 and earlier did not utilize new diagnostic criteria for BPD or PTSD.

Bivariate Links Between CT and BPD

A number of researchers explored bivariate relationships between CT and BPD using univariate models assessing correlations between, and prevalence of a history of CT, mainly in patients diagnosed with BPD. A large body of empirical research has demonstrated that patients diagnosed with personality disorders report a high number of CT experiences (Paris, 1994, 1998). Much of this first generation of research focused on the relationship between a history of CSA and the development of BPD in adulthood (Gunderson & Sabo, 1993; Ruegg & Frances, 1995).

Studies have identified that between 30% and 90% of BPD patients reported a history of CT including sexual, physical and emotional abuse, compared with 17–45% in the control groups (Ball & Links, 2009; Battle et al., 2004; Bornovalova, Gratz, Delany-Brumsey, Paulson, & Lejuez, 2006; Bouchard, Godbout, & Sabourin, 2009; Carlson, Yates, & Sroufe, 2009; Gladstone, Parker, Wilhelm, Mitchell, & Austin, 1999; Golier et al., 2003; Laporte & Guttman, 1996; Zanarini, 2000). These associations have also been noted in a variety of samples including psychiatric inpatients (Bradley, Jené, & Westen, 2005), outpatients (Golier et al., 2003), urban drug users (Bornovalova et al., 2006), and community adolescents (Rogosch & Cicchetti, 2005). These univariate analyses consistently pinpointed CT, mainly CSA, as a significant risk factor in the development of BPD.

Although much of the research arising from this literature has focused on the relationship between a history of CSA and the development of BPD, researchers also started to document the pervasive impacts of exposure to other forms of CT on BPD related symptoms. For example, in a study of 467 psychiatric inpatients diagnosed with BPD, up to 91% of the sample reported a history of CT and 92% reported experiences of neglect. Moreover, reports of CT were significantly higher in individuals with BPD than in a comparison group. This data highlighted that those CSA survivors diagnosed with BPD reported more chaotic childhood environments with co-occurring emotional, physical, and verbal abuse and neglect (Zanarini et al., 1997).

These early studies identified a dose response relationship between CT and BPD, with more severe CT being associated with higher levels of BPD symptoms and overall impairment (Bierer et al., 2003; Sansone, Songer, & Miller, 2005; Silk et al., 1995; Zanarini et al., 2002). This is consistent with the literature examining the impact of CT on adult functioning, which has repeatedly identified that the impacts appeared to be dependent upon severity, chronicity, frequency, and co-occurrence with multiple forms of CT (Browne & Finkelhor, 1986; Malinosky-Rummell & Hansen, 1993; McLean & Gallop, 2003; Silk et al., 1995; Yen et al., 2002; Zanarini et al., 2002). Increasing complexity of CT experiences have been shown to be significantly associated with corresponding increases in the extent, severity and range of clinically significant symptoms in adulthood (Briere, Kaltman, & Green, 2008; Chapman et al., 2004; Cloitre et al., 2009; Edwards, Holden, Felitti, & Anda, 2003; Finkelhor, Ormrod, & Turner, 2007; Ford, 2010; Ford, Connor, & Hawke, 2009; Holt, Finkelhor, & Kantor, 2007). However, other studies have not found a relationship be-

tween severity, duration of CSA, and severity of BPD (Soloff, Lynch, & Kelly, 2002).

Prospective longitudinal studies examining the associations between CT and BPD have consistently been identified, strengthening the argument that CT might act as an etiological risk factor for the development BPD (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Johnson, Cohen, Chen, Kasen, & Brook, 2006; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Soloff et al., 2002). In their meta-analytic study, Fossati, Madeddu, and Maffei (1999) reviewed 21 studies over 15 years of univariate research examining the associations between CSA and BPD in 2,479 (primarily female) participants. All but one of these studies found significant associations between a history of CSA and the development of BPD. However, the overall pooled effect size was considered moderate (pooled $r = .28$). These authors concluded that these findings did not support the hypothesis that CSA is a necessary risk factor or that it plays a causal role in the development of BPD. Of the studies reviewed, the only study that did not find a significant association between a history of CSA and BPD was the one study that obtained corroboration for self-reported CSA, highlighting a possibly central role of interpretation, beliefs, perception, and meaning-making as mediators of the link between CT and borderline symptoms (Goldman, D'Angelo, DeMasoc, & Mezzacappa, 1992).

Multivariate Relationships Between Types of CT and BPD

As researchers began responding to the limitations of these early studies, more complex multivariate approaches to statistical analyses began to be added to investigations. For example, Carr and Francis (2009) assessed the differential role of diverse trauma types with a sample of 178 students and found that emotional abuse was the only significant predictor of a diagnosis of BPD when controlling for other forms of CT and family functioning factors. Using a prospective cohort design, Widom, Czaja, and Paris (2009) followed a group of children into adulthood, matching those who had experienced CT with nonabused participants. They found a direct effect of CT on the development of BPD (especially physical abuse and neglect). However, this association did not maintain significance when other family variables were entered into the multivariate analysis. Utilizing a path analytic approach, Hengartner and colleagues (2013) explored the relationships between CT and BPD in a community sample of 512 women. Bivariate analyses indicated that all types of CT (sexual, physical, emotional, and neglect) explained a large proportion of the variance in BPD (27.8% of the variance explained, $f^2 = .39$, where $f^2 = .35$ is considered a large effect), but multivariate path analysis showed that CSA demonstrated no specific significance over and above other types of CT (Hengartner et al., 2013). Conversely, Sansone and colleagues (2011) examined associations between five forms of CT and BPD symptoms in a sample of 250 cardiac patients. Although all forms of CT were associated with BPD symptoms, multiple regression showed that only CSA remained a significant predictor of BPD ($\beta = .22$) while controlling for the other types of CT. Utilizing a large scale representative sample of 34,563 community participants, Afifi et al. (2011) examined the relationship between a wide range of CT types and axis II disorders, while adjusting for axis I disorders. Consistent with previous

research, all CT types were significantly associated with increased likelihood of a diagnosis of cluster B personality disorders, including BPD. Those associations remained after the inclusion of covariates (i.e., socioeconomic status, marital status, education, and select axis I disorders) in the analyses.

Fewer studies have assessed the specific role of physical abuse (Golier et al., 2003; Herman et al., 1989; Paris et al., 1994; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989) or neglect (Zanarini et al., 2002) in the development of BPD. Although many types of CT were related to BPD, Battle et al. (2004) found CSA to be particularly predictive (OR [Odds ratio] = 3.08 for CSA by a caretaker, OR = 2.61 for noncaretaker CSA, OR = 1.90 for emotional abuse; Battle et al., 2004), whereas Carr and Francis (2009) found CSA to be the only CT related to BPD. In two studies, CSA (OR = 2.6 to 3) and emotional abuse (OR = 1.9) were found to be particularly predictive of the development of BPD (Battle et al., 2004; Carr & Francis, 2009). In four studies, diverse forms of CT including emotional and physical abuse and neglect were significantly associated with BPD, in addition to sexual abuse (Afifi et al., 2011; Grover et al., 2007; Johnson et al., 1999; Soloff et al., 2002). Put together, these studies confirm a link between CT, other types of family dysfunction and BPD, especially CSA. However, more information is needed to further understand the specific contributions of different types of CT and other etiological factors in the development of BPD.

Overall, results from these bi- and multivariate studies revealed high rates of CT in BPD patients. However, these findings are inconsistent with some studies not finding associations between CT and BPD, and one meta-analysis identifying only a moderate association (Fossati et al., 1999). Additionally, the differential impacts of diverse types of CT, their characteristics (e.g., perpetration, abuse by mother vs. father, etc.) or their cumulative effects on BPD remain to be documented (Dong, Anda, Dube, Giles, & Felitti, 2003; Faust, Runyon, & Kenny, 1995; Zanarini et al., 1999). These findings add further questions to the quest for understanding the etiological role of CT in the development of BPD.

Research on the Mechanisms of CT in the Development of BPD

In response to these bi- and multivariate studies, researchers began to consider the mechanisms through which CT might play a role in the development of BPD and consider mediators and moderators in these relationships. The two variables that have received the most study in this line of research are deficits in affect regulation and the interplay between personality traits that are hypothesized to be heritable and create a genetic predisposition to the development of BPD in the presence of exposure to trauma. In terms of the former, authors have suggested that chronic, unresolved trauma might decrease the development of self-capacities such as affect regulation capacities. In terms of the latter, models have been proposed including that the development of BPD represents an interaction between genetic and environmental factors including CT (Silk et al., 1995; Zanarini et al., 1997). In other words, researchers have begun to examine the complexity of relationships between CT variables and adult functioning, including PTSD and BPD, in an effort to better understand the mechanisms through which CT may contribute to the development of BPD symptoms. While other variables such as attachment history

have been discussed in the literature that have not been explored empirically to the same degree as these two models. As such, we will focus our review on these two variables.

CT, BPD, and Affect Regulation Deficits

Affect regulation difficulties have been proposed as key mediators in the relationship between CT and BPD (van Dijke et al., 2013). It has been argued that BPD may be one of a number of potential outcomes that arise from compromised affect regulation as a result of CT (van Dijke et al., 2011). CT has been associated with well-documented, pervasive, and significant deficits in affect regulation including underregulation and overregulation, particularly when abuse is perpetrated by a caregiver (Bermond, Moormann, Albach, & van Dijke, 2008; Freyd, DePrince, & Gleaves, 2007; Paivio & Laurent, 2001; Roth et al., 1997). In CT both overregulation and underregulation are related to psychological distress (van Dijke et al., 2013). Based on clinical and empirical findings, it has also been suggested that individuals who respond to CT experiences with the inhibition of emotional regulation, will be less likely to overcome their negative experiences and more likely to develop BPD (Fonagy et al., 1995; Stalker & Davies, 1995). Some research has suggested that CT by attachment figures causes alterations in the development of the autonomic and central nervous systems leading to disruptions in the development of the capacity to regulate affect (Fosha, Siegel, & Solomon, 2009; Schore, 2001, 2002; Siegel, 1999, 2001).

Empirical research has identified significant affect regulation difficulties in children who experienced diverse forms of CT. These children were less able to understand and regulate emotions, received less emotional support, encountered more interpersonal conflict, had difficulty discriminating emotional expressions and identifying discrete emotions, and responded with dysregulation to angry faces in experimental conditions (Maughan & Cicchetti, 2002; Shipman, Edwards, Brown, Swisher, & Jennings, 2005; Shipman, Zeman, Penza, & Champion, 2000). Studies assessing the role of CT in the development of affect dysregulation in adolescents have also found consistent associations between CT and affect dysregulation (Burns, Jackson, & Harding, 2010; Messman-Moore, Walsh, & DiLillo, 2010; Tull, Barrett, McMillan, & Roemer, 2007).

Affect dysregulation is a core symptom of BPD (Linehan, 1993a; Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002; Tragemmer, Solhan, Schwartz-Mette, & Trull, 2007; Trull, 2001; van Dijke, 2008; van Dijke et al., 2010; Watson, Chilton, Fairchild, & Whewell, 2006; Zanarini et al., 2002) and is also frequently present in CT survivors (Briere et al., 2010; van Dijke et al., 2010). For instance, in a recent study exploring the impact of childhood trauma on emotion regulation and interpersonal functioning in a sample of 139 women, 44% endorsed moderate to severe levels of childhood trauma (Stevens et al., 2013). CT had a direct effect on emotion regulation and the relationship between current trauma symptoms and CT was significantly mediated by affect dysregulation. Briere et al. (2010) observed a moderated relationship between cumulative CT and affect dysregulation ($r = .35$) and the link remained stable in an integrative structural model of the relations between CT, affect dysregulation, posttraumatic symptoms, and dysfunctional avoidance (i.e., acting out, anger, and dissociation). Further, investigating emotion regulation in

BPD patients, van Dijke et al., (2010) emphasized the importance of looking at both underregulation, a core symptom found in the majority of BPD patients (Ford, 2010; van Dijke, 2008; van Dijke et al., 2010; Zanarini et al., 2002), as well as overregulation such as numbing and dissociation, reported by 1 in 5 BPD patients (Lanius et al., 2010; van Dijke et al., 2010). Self-injury, addictions, and difficulties with memory and interpersonal relationships, often seen in trauma survivors and BPD patients, are largely seen as efforts at regulating unbearable emotional states (Briere et al., 2010; Conklin et al., 2006; De Zulueta, 1999; Ebner-Priemer & Sawitzki, 2007; Linehan, 1993a).

Recent research has attempted to delineate the complex interrelationships between CT, BPD, PTSD, and affect regulation. Examining the contributions of diverse experiences of CT, affect dysregulation and BPD symptoms in a group of 472 adult survivors, van Dijke et al. (2013) found that underregulation of affect was associated with the severity of BPD symptoms while overregulation was not. Underregulation of affect partially mediated the relationship between CT and BPD (indirect effect $B = 1.09$), such that when underregulation was added to the mediational model, the direct effect of CT on BPD symptoms ($B = 7.54$ that corresponds to a medium effect size according to Cohen, 1988) was reduced, but still statistically significant ($B = 6.45$). These results suggest that addressing underregulation of affect as a sequelae of CT rather than only as a feature of BPD may enhance treatment for BPD patients. Utilizing a structural equation model, Trull (2001) examined the relations between BPD features such as affect regulation and hypothesized etiological factors, including CT, in a large sample of nonclinical participants. Results supported the proposed model, in which parental disinhibition, mood disorder, CT, and affect dysregulation represent etiological factors leading to the development of borderline features.

Investigating these relationships more fully, Santangelo et al. (2014) hypothesized that retrospective self-report measurements of affect dysregulation utilized in previous studies may have limited the capacity of researchers to identify real differences in affect dysregulation between diverse diagnostic and trauma types. As a result, they used high frequency diaries that measured both valence of emotion regulation difficulties and emotional distress at the time of prompting and found that BPD patients evidenced affect instability, both in terms of valence and distress. However, this affect dysregulation was not more heightened than in PTSD or eating disorders Santangelo et al. (2014). Based on their findings they suggested that affect dysregulation may be a core feature of many *DSM* disorders not just PTSD and BPD.

BPD and Dissociation

Dissociation has long been considered a form of dysfunctional affect dysregulation and CT has been consistently associated with long-lasting dissociation in adulthood. The relationship between CT and dissociation has been confirmed in a number of clinical studies (Chu & Dill, 1990; De Zulueta, 1999; Herman et al., 1989; Korzekwa, Dell, Links, Thabane, & Fougere, 2009; Meehl, 1995; Ross, Joshi, & Currie, 1991; Sar et al., 2003; Shearer, 1994; Van IJzendoorn & Schuengel, 1996), and nonclinical studies (Irwin, 1999; Sar, Akyüz, Kugu, Öztürk, & Ertem-Vehid, 2006), although contradicted by two others (Paris et al., 1994; Zweig-Frank, Paris, & Guzder, 1994).

Regarding the relationship between dissociation and BPD, four risk factors for dissociation in BPD patients have been identified including inconsistent treatment, sexual abuse by caretaker, witnessing sexual violence as child, and rape in adulthood (Zanarini, Ruser, Frankenburg, Hennen, & Gunderson, 2000). Severe dissociation has been argued to be a distinguishing component of BPD (Skodol et al., 2002; Zweig-Frank et al., 1994). Patients with BPD report extensive dissociative symptoms, especially identity confusion, unexplained mood changes, and depersonalization (Brodsky, Cloitre, & Dulit, 1995; Kemperman, Russ, & Shearin, 1997; Korzekwa et al., 2009; Shearer, 1994; Watson et al., 2006; Zanarini et al., 2000). These symptoms have been associated with other core BPD symptoms such as self-injury and impulsive aggression.

Research assessing the role of dissociation has identified that emotional abuse or neglect has the strongest relationship between CT and dissociation in BPD patients (Draijer & Langeland, 1999; Sar et al., 2006; Timmerman & Emmelkamp, 2001). Loffler-Stastka, Szerencsics, and Bluml (2009) identified that derealization was significantly correlated with emotional, $r = .38, p = .006$, and sexual abuse, $r = .38, p = .007$ during childhood in BPD patients, whereas overall dissociation was associated with emotional abuse, $r = .28, p = .045$. However, Watson et al. (2006) found a significant relationship between CT and dissociation for emotional abuse ($r = .33$), physical abuse ($r = .19$), and emotional neglect ($r = .17$), but not for sexual abuse or physical neglect in BPD patients.

Some studies have not found significant associations between dissociation and BPD (Simeon, Nelson, Elias, Greenberg, & Hollander, 2003; Timmerman & Emmelkamp, 2001; Zweig-Frank et al., 1994). However, other studies, assessing the role of CT in the dissociative experiences of BPD patients, have found associations between some types of CT (emotional abuse, physical abuse, and neglect) and dissociation in BPD patients, and not other CT types (CSA, physical neglect; Watson et al., 2006). As in other domains of study, relationships between different trauma types and dissociation remain unclear and inconsistent, suggesting that dissociation may share a common etiological factor with CT and BPD rather than acting as a core symptom of both (Watson et al., 2006).

Toward an Integrative Model of PTSD, Affect Regulation, and Dissociation

The results of the integrative model of Briere et al. (2010) provided additional evidence that some affective phenomena associated with a diagnosis of BPD (i.e., affect dysregulation, tension reduction behaviours, suicidality, substance abuse, and dissociation) may represent dysfunctional avoidance, a form of compromised affect regulation, related to a history of multiple types of interpersonal trauma. They suggest that this can be and explained through multiple pathways involving posttraumatic stress and affect regulation difficulties. The authors suggested that some survivors may respond to trauma with BPD symptoms, others may engage in dysfunctional avoidance and demonstrate BPD symptoms primarily because of insufficient affect regulation capacities, and some may present with BPD symptoms in response to the additive combination of posttraumatic distress and regulation difficulties.

Genetic or Personality Trait Variables, CT and BPD

Research exploring the role of genetics and the heritability of BPD might help to further understand and contextualize the links between CT and BPD. In a comprehensive review of the literature, Paris (1998) noted that the parents of individuals with BPD are frequently diagnosed with “impulsive spectrum disorders” or “depressive spectrum disorders” and, based on this, suggested that the mechanism through which CT has its impact on the developing person may be through shared, heritable personality traits. Researchers interested in gene environment interactions have also conducted a number of studies to explore their hypothesis that the role of CT in the development of BPD is best understood as a complex interplay between genes and environment (Paris, 1998). For example, Kraemer et al. (1997) suggested that impulsive or depressed parents may be more likely to abuse their children or be unable to maintain a stable family environment and relationships.

Examining the role of heritability, studies of first-degree relatives of patients with BPD have demonstrated a high prevalence of disorders marked by impulsive and affective symptoms as well as BPD itself (Baron, Gruen, Asnis, & Lord, 1985; Silverman et al., 1991; White, Gunderson, Zanarini, & Hudson, 2003; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1988). Additionally, patients with BPD are five times more likely to have a first-degree relative with BPD (Distel et al., 2008). These studies compared BPD patients to patients diagnosed with schizotypal personality disorder or nonclinical controls (Baron et al., 1985), schizophrenia and bipolar disorders (Loranger, Oldham, & Tulis, 1982), schizophrenia and “other” personality disorders (Silverman et al., 1991), schizophrenia and depression (Soloff & Millward, 1983), and antisocial personality disorder and dysthymic disorder (Zanarini et al., 1998). These researchers generally found significantly higher levels of personality disorders or traits in first degree relatives, as compared with the rates of these disorders or traits in the first degree relatives of other psychiatric patients and comparisons groups without diagnosis, suggesting heredity.

Exploring the role of heritable personality traits as a vehicle for the genetic transmission of risk to develop BPD in response to environmental stressors, researchers such as Bandelow et al. (2005) highlighted the challenges of disentangling genetic from environmental factors. They hypothesized that personality traits might be transmitted genetically or via learning models as a result of being parented by someone with difficulties in emotion regulation and impulsivity. A number of studies have identified that BPD is associated with personality traits that have been found to demonstrate a higher levels of heritability including impulsivity, neuroticism, affective lability, negative affect, low effortful control, agreeableness, and conscientiousness (Joyce et al., 2003; Paris, 2004; Saulsman & Page, 2004; Skodol et al., 2002), which may serve as core biological vulnerabilities (Distel et al., 2008; Skodol et al., 2002) to develop BPD. Behavioral genetics studies have demonstrated that approximately half of the variance in the development of personality traits can be accounted for by genetic factors (Torgersen et al., 2000). For example, Trull (2001) developed a structural model looking at parental psychopathology and temperament in a sample of college students and found that the impact of CT remained a significant predictor of BPD in a multivariate model that included parental psychopathology and personality factors (i.e., disinhibition and negative affectivity). It has

been suggested that impulsivity and affective instability might be heritable as a result of intergenerational alterations in the serotonin and norepinephrine systems (Siever & Davis, 1991). In their prospective, longitudinally designed study assessing the role of temperament and CT in the development of BPD, Jovev et al. (2013) followed 245 children over 3 years and identified that childhood abuse ($b = .27$) and neglect ($b = .26$) was a significant predictor of increases in BPD symptoms. However, abuse and neglect acted as moderators in interaction with dimensions of temperament (i.e., low affiliation and effortful control) to predict increases in BPD symptoms, leading to the conclusion that both temperament and CT contributed to the development of BPD symptoms.

A number of researchers have examined the potential genetic associations in the relationship between CT and BPD and other personality disorders through twin studies. In their study of 221 twin pairs, Torgersen et al. (2000) identified a concordance rate for BPD of 35% in monozygotic twins and 7% in dizygotic twins; thus, suggesting a genetic component. In a large sample of 2,780 drawn from the Norwegian Twin Registry, Berenz et al. (2013) identified significant but small associations between CT and personality disorder criteria. After common family factors were considered in the discordant twin sample, CT continued to be significantly related to BPD and ASPD but not other personality disorders although the effects were moderate. In a longitudinal discordant twin design, Bornovalova et al. (2013) explored a diathesis stress model to explain aspects of the interaction between heritable traits that serve as vulnerability factors in the development of BPD and environmental factors, such as childhood trauma, that may play a role in triggering the development of BPD traits. They argued that internalizing and externalizing traits serve as genetic vulnerabilities that interact with the environmental stressor of abuse and results in the development of BPD traits. Although there was a direct relationship between CT and BPD traits, such that as BPD traits increased, so did the probability of a history of CT. This relationship was only significant when internalizing and externalizing factors were considered suggesting that this association between CT and BPD can be explained by common genetic influences. These authors argued that internalizing and externalizing traits serve as pre-existing vulnerabilities whereas CT acts as an environmental risk factor. Although these authors cited research that provides some indirect support for this hypothesis, no direct tests of this model have yet been conducted (Bornovalova et al., 2013).

Utilizing a design that integrated biological sisters, Laporte and colleagues (2011) examined 56 women with BPD and their sisters on measures of psychopathology, personality traits, and CT experiences. Participants were administered two standardized measures of personality disorders based on the *DSM-III*, a self-report measure of personality pathology, a measure of overall psychiatric symptom severity and two self-report trait measures of personality, one focused on affect lability and the other on impulsivity and a retrospective interview about histories of childhood abuse and neglect (CT). Only three sisters were concordant for BPD and most sisters demonstrated little evidence of psychopathology. Although all participants rated high on levels of CT and dysfunctional parent-child relationships, the BPD participants scored significantly higher on scales of compulsivity and affect lability. Given these findings, Laporte et al. (2011) argued that the existence of

higher rates of these personality traits (affect lability and impulsivity) in the BPD patients suggests heritability and that these create a sensitivity to CT that helps to explain the strong relationship between CT and BPD. These researchers did not utilize a measure that assessed complex trauma.

Discussion

Critiques of Research

The literature examining the relationships between a history of CT and adult psychopathology has been criticized for overreliance on clinical populations, retrospective self-report, and inconsistent measurement of constructs. The most significant of these criticisms is the almost complete reliance upon cross sectional, retrospective designs (Afifi et al., 2011; Cohen et al., 2014; Johnson et al., 2003; Paris, 1998). Critics suggest that these findings may be spurious because of recall and reporting bias, over or underreporting, or misunderstanding of questions (Afifi et al., 2011; Barone, 2003). Given that most individuals who experience CT do not develop personality disorders and very few studies have assessed the impact of CT on development longitudinally on in community samples, this is an important criticism as researchers attempt to better understand the complex interactions between CT and later psychopathology (Bornovalova et al., 2013). However, critical analysis of retrospective reports suggest that such presumed biases do not systematically affect the association between CT and later outcomes (Brewin, Andrews, & Gotlib, 1993), the veracity of retrospective reports on CT (Brewin et al., 1993; Hardt & Rutter, 2004; Henry, Moffitt, Caspi, Langley, & Silva, 1994), and prospective longitudinal studies following documented cases of CT and adult psychopathology have been found to support findings of retrospective studies (e.g., Boney-McCoy & Finkelhor, 1996).

A number of studies could also be criticized for failing to assess a full range of deleterious childhood experiences, mainly focusing on CSA or only one trauma type (Afifi et al., 2009, 2011; Carr & Francis, 2009; Paris, 1998), and not examining the effects of moderators or confound variable such as frequency, severity, and chronicity. In particular, the role of multiple trauma or cumulative trauma has not been adequately considered with statistical models, primarily focusing on identifying the associations between discreet trauma types and the emergence of BPD. Some of these studies did make reference to the frequency with which participants experienced multiple forms of trauma (Bandelow et al., 2005; Battle et al., 2004; Cohen et al., 2014; Widom et al., 2009; Yen et al., 2002), but only Bandelow et al. (2005) examined the impact of what they identified to be trauma severity and appeared to represent the impact of cumulative traumas as well as the severity of specific ones. They found that the BPD group had a higher overall trauma severity score than nonclinical controls.

Another significant criticism of the research examining the impact of CT on psychopathology in adulthood is the overreliance on self-report, which may result in negative response biases, reverse causation, or even under reporting of traumatic experiences (Barone, 2003; Carr & Francis, 2009; Goodman et al., 2003). In terms of measuring trauma, the majority of the studies that we reviewed utilized a retrospective self-report measure. The most frequently utilized measure was the Childhood Trauma Questionnaire, which is a 28 item self-report questionnaire measuring a

history of five types of CT. Although this measure has received extensive psychometric validation, it simply measures the presence or absence of a five forms of childhood trauma. It does not provide any information about trauma symptoms, symptom severity, or traumatogenic diagnoses. Therefore, the majority of the studies used categorical approaches to indicate the presence or absence different types of CT rather than a dimensional scale of trauma severity or cumulative trauma, with few notable exceptions (Briere et al., 2010; Zlotnick et al., 2003). Another critique is the limiting of the measurement of crucial constructs to one variable that may or may not be representative of the construct of interest. For instance, Afifi et al. (2011) reported that although they used a number of measures to measure CT the assessment of family environment was limited to one question about violence against the maternal caregiver, which is indeed considered as a form of CT in itself. The overlap between family environment and CT is typical and might be difficult to distinguish. These measurement issues limit the capacity of researchers to untangle the complex relationships between CT and adult psychological distress. The limited information that is currently available due, to these measurement issues, on the role of cumulative trauma from childhood has also limited how much we can take away from studies that have focused, primarily, on drawing associations between the occurrence of discreet CT types and a later diagnosis of BPD. However, although most studies did rely upon retrospective, self-report, categorical measures of childhood trauma exposure, these measures were psychometrically validated and there was significant overlap between studies in the use of the CTQ. This allows for greater ease in comparing studies.

Inconsistency in the measurement of constructs has also been criticized in the CT and BPD literature. The majority of studies used a categorical approach to the measurement of BPD (e.g., Carr & Francis, 2009; Hernandez, Arntz, Gaviria, Labad, & Gutierrez-Zotes, 2012). The most frequently utilized measures, in this literature, were the Structured Clinical Interview for *DSM* and the Structured Interview for *DSM-IV* Personality Disorders (SIDP-IV, Pfohl, Blum, & Zimmerman, 1995). These two measures are well validated and, as interviews, they may be more rigorous than self-report measures of psychopathology. However, they are categorical measures that provide thresholds for diagnosis versus no diagnosis. There is substantial evidence to argue for the dimensional measurement of personality disorders, with clinical populations representing an extreme end of a continuum of distress and functionality (e.g., Shedler & Westen, 1999). More recently, van Dijke et al. (2010, 2012, 2013) has utilized the borderline personality disorder Severity Index (Weaver & Clum, 1993), which is a semistructured interview assessing the frequency and severity of BPD symptoms. Although this is only the work of one researcher, this represents a shift in the literature to an awareness of the importance of assessing symptom severity as well as diagnosis.

Overreliance on predominantly female clinical samples is also an issue. The use of clinical populations may lead to inflations in estimates of the impact of CT on adult distress as well as difficulties in generalizing this research to other populations (Cohen et al., 2014; Johnson et al., 2003; Sansone et al., 2011). Using predominantly female clinical samples may also fail to capture the outcomes of a large number of traumatized children who grow up to be well-functioning adults (Paris, 1998). This is, essentially, a "base rate problem" given the high prevalence of CT present in the

general population (Rutter & Maughan, 1997). These resilient survivors can tell us much about the pathways and mechanisms to thriving after CT and, comparisons with survivors who are struggling with significant deficits in functioning may facilitate the disentanglement of the complex mechanisms in the pathways from CT to adult psychopathology.

Finally, further research is needed to provide data on the links between CT, BPD, and PTSD based on the new *DSM-5* criteria, which are similar for BPD but changed for PTSD. These include the addition of Criterion D “negative alterations in cognitions and mood” (e.g., negative beliefs and expectations, negative emotions such as anger, self-blame, feeling alienated from others, and inability to experience positive emotions). Whether the new PTSD criteria will accurately represent the complex symptom profiles of CT survivors, in particular, will need to be assessed in future studies.

Implications for Research

The literature examining the relationship between a history of CT and later PTSD, CPTSD, and/or BPD consistently find significant associations between CT and later diagnosis of BPD. However, although early studies found strong associations suggestive of a causal relationship between CT and later BPD, later multivariate studies identified more complex indirect relationships between specific trauma types and later diagnosis of BPD. In particular, this second wave of research has failed to confirm that a history of CSA is a specific or unique risk factor for the diagnosis of BPD. Later studies have also highlighted the complexities in the relationships between CT and later pathologies including BPD and PTSD. Owing to the complexity and variability of documented symptoms in BPD, integrative theoretical or clinical models are highly advocated to understand how and why CT might lead to a diagnosis of BPD in some survivors, and the many factors that might explain this association.

Given the significant conceptual criticisms of the diagnosis of BPD, including challenges to the validity and reliability of the diagnostic category itself, the concerns about the failure of the diagnostic construct to integrate etiological factors, and the high levels of inconsistency in research regarding the relationships and comorbidities between a history of CT and adult psychopathology, a radical shift in the focus of research in this area appears important. One consistent finding in the literature is that many of the core consequences of CT, including affect dysregulation and interpersonal distress are shared core features of PTSD, CPTSD, and BPD and, in fact, may be transdiagnostic throughout the *DSM*. This suggests that research needs to shift away from categorical, diagnostic foci toward exploring the underlying core deficits that are shared by distressed adult survivors of CT regardless of their diagnostic status.

One approach to this shifting lens would be to move toward research that examines, in prospective longitudinal designs, personality traits, the development of self capacities related to adult psychopathology such as affect regulation and interpersonal functioning, experiences of CT utilizing a cumulative and continuous measure of trauma types and severity, and then compare these to aspects of adult psychological and interpersonal functioning. This would represent a biopsychosocial approach to untangling these complex questions that begins to

consider the interrelated roles of genetics and CT in the development of adult psychopathology.

Overall, further research is clearly needed to continue to explore this debate and inform practitioners on the etiology or risk factors associated with BPD, and to inspire efficient intervention strategies well-tailored to the specific needs of different patients. Clarifying these relationships and pathways will help establish comprehensive models of intervention based on the most relevant factors contributing to BPD symptoms in survivors of CT. Finally, future study should separately study the links between CT and BPD, in clinical and community samples, in male and female survivors, as well as document the role of CT committed by fathers versus mothers and explore resiliency factors in survivors who do not go on to develop psychopathology.

Clinical Implications

The focus in this literature, to date, has been on assessing the role of CT in the development of BPD and in questioning the diagnostic construct of BPD as to whether it would be better labelled a disorder of complex trauma. However, by engaging in research that focuses on the diagnoses rather than the underlying etiological and resulting pathological features, we may be missing important pieces to the puzzle of the development of severe distress in the intrapsychic and interpersonal lives of childhood trauma survivors.

This literature review has highlighted implications for treatment, including the importance of assessing trauma exposure in patients presenting with BPD symptoms, and emphasizes the possibility that trauma-relevant interventions may be helpful in resolving some of the symptoms typically associated with BPD, and vice versa. Given the current results, trauma-focused interventions might include therapeutic exposure to reduce posttraumatic stress (Briere et al., 2010) and various cognitive—behavioral and relational treatments shown to increase affect regulation capacity and/or reduce experiential avoidance (Cloitre, Koenen, Cohen, & Han, 2002; Linehan, 1993b). The relationship between CT, PTSD, and reduced affect regulation also reinforces concerns that therapeutic exposure paradigms, at least when applied to patients who are survivors of CT, may need to be carefully titrated to match existing affect regulation capacities in case such procedures overwhelm patients with emotional states that cannot be easily regulated (e.g., Briere & Scott, 2014). Other treatment approaches to both individuals with BPD and trauma-survivors that have received some empirical support include interventions developed based on mentalization, attachment theory, transference, and object relations theory (e.g., Allen, Fonagy, & Bateman, 2008; Clarkin, Yeomans, & Kernberg, 2006; Yeomans, Clarkin, & Kernberg, 2002; Masterson, 1976). Furthermore, careful assessment of CT could reveal that behind BPD symptoms might lie basic posttraumatic suffering that practitioners must address. As such, these findings support the importance of assessing and attending to unresolved childhood trauma concurrently, or before, addressing BPD symptoms. However, comprehensive treatment reviews along with empirical treatment outcome studies are needed to compare the efficacy of trauma-related and BPD treatments to help patients struggling with symptoms of diagnoses of BPD, CT, PTSD, and complex-PTSD. Meanwhile, the findings of our review suggest that interventions should adopt a more trans-

diagnostic approach based on the observed clinical complexity common to trauma-survivors and individuals diagnosed with BPD. These transdiagnostic interventions would focus on deficits in self capacities that underlie the development of psychopathology such as focusing on increasing affect regulation capacities, reducing experiential avoidance, promoting relational adjustment, as well as identity integration.

Conclusions

This review has highlighted the complexities in the relationship between CT and BPD and identified important implications for research and practice. While relationships between specific trauma types and outcomes in adulthood are inconsistent, overall associations between CT and the development of BPD are strong and consistently identified. Research exploring the specific mechanisms through which CT may be related to the development of BPD in adulthood is beginning to untangle the complex web of interrelated factors such as heritable personality traits, affect regulation and dissociation, trauma symptoms as mediators in the relationship between CT and BPD. The question of whether BPD is a disorder of personality or a disorder arising out of a history of trauma continues to be explored. However, significant questions remain to be answered through more complex research that will consider the conceptual questions related to conceptual distinctness of the BPD diagnosis versus complex posttraumatic responses and the importance of assessing core features of both such as affect regulation and dissociation rather than focusing on diagnoses in research studies.

In short, there is evidence that CT is present in the histories of a majority of individuals who go on to be diagnosed with BPD. There is also evidence that genetics may play a role in the etiology of BPD via heritable personality traits that may interact with CT in the genesis of BPD. Additionally, recent research suggests that BPD and PTSD or Complex PTSD may be conceptually distinct categories. Therefore, it is our conclusion that BPD is, likely, both a disorder of personality and a disorder of trauma. However, our strongest recommendation is for future researchers to further explore transdiagnostic factors such as the self capacity of affect regulation to further disentangle the complex pathways between CT, inherited personality traits and the development of all forms of traumatogenic psychopathologies in adulthood.

Résumé

Cette revue a pour but d'explorer la recherche empirique qui vise à déterminer si le trouble de la personnalité limite (TPL) est un trouble associé à la « personnalité » ou plutôt un trouble découlant de traumatismes subis pendant l'enfance. La revue met en relief la complexité de la relation entre un trouble de l'enfance (TE) et le TPL et établit d'importantes répercussions pour la recherche et la pratique. Bien que les liens entre des types de traumatismes précis et leurs conséquences à l'âge adulte sont inconsistants, les associations globales entre les TE et le développement du TPL sont fortes et clairement cernées. La recherche explorant les mécanismes précis par lesquels les TE pourraient être reliés au TPL à l'âge adulte commence à démêler la toile de facteurs interreliés, tels les traits de personnalité héréditaires, la régulation et la dis-

sociation des affects, et les symptômes de traumatismes en tant que médiateurs dans la relation entre le TE et le TPL. Il est recommandé que les recherches futures explorent davantage les facteurs transdiagnostiques, tels que la capacité en matière de régulation des affects chez les personnes touchées, afin de continuer à démêler les voies complexes entre les TE, les traits de personnalité héréditaires et le développement de toutes les formes de psychopathologies traumatogènes à l'âge adulte.

Mots-clés : traumatisme pendant l'enfance, abus sexuels d'enfants, trouble de la personnalité limite, trouble de stress post-traumatique complexe, troubles de la personnalité.

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