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Association of Childhood-Trauma-by-Primary Caregiver and Affect Dysregulation With Borderline Personality Disorder Symptoms in Adulthood

Annemiek van Dijke
Delta Psychiatric Hospital, Rotterdam, The Netherlands

Julian D. Ford
University of Connecticut School of Medicine

Maarten van Son, Laurence Frank, and Onno van der Hart
Utrecht University

Potentially traumatizing experiences by a primary caregiver during childhood (TPC) may lead to affect dysregulation, which is a hallmark of borderline personality disorder (BPD) in adulthood. Path analyses with bootstrap confidence intervals were used to explore the relationships of TPC and under- and overregulation of affect to BPD symptoms. Almost 63% of a sample of 472 patients diagnosed with either BPD alone, comorbid BPD + somatoform disorder, or other mental disorder, reported childhood TPC. TPC and underregulation of affect were associated with severity of BPD symptoms, but overregulation was not. Underregulation of affect partially mediated the relationship between TPC and BPD symptoms. Study findings suggest that also addressing underregulation of affect as a sequela of TPC rather than as only on feature of BPD, may enhance treatment of BPD symptoms. Research is needed to determine if targeting overregulation of affect, next to TPC and underregulation of affect, enhances the efficacy of treatment for severe BPD symptoms with childhood TPC and with comorbid other mental disorders, especially somatoform disorder.

Keywords: borderline personality disorder, childhood trauma, primary caregiver, affect dysregulation

Psychological trauma is known to be associated with mental disorders such as borderline personality disorder (BPD; Yen et al., 2002; Zanarini, Yonge, & Frankenburg, 2002). Although the association between childhood trauma history and mental disorders is complex (D'Andrea, Ford, Stolbach, Spinazzola, & Van der Kolk, in press), sexual and physical abuse appear to be important etiological contributors to BPD symptoms (Bradley, Jenei, & Westen, 2005; Weaver & Clum, 1993). However, little is known about how childhood psychological trauma contributes to the etiology or severity of BPD symptoms later in life (Verdurmen et al., 2007). In light of the well documented association between early childhood trauma and affect dysregulation (D'Andrea et al., in press; Ford, 1999; Paivio & Laurent, 2001; Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997), one possible mediator of the relationship between childhood psychological trauma and BPD symptoms is affect dysregulation.

Affect dysregulation is considered a core symptom of BPD (Van Dijke, 2008; Van Dijke, Ford, et al., 2010; Zanarini et al., 2002). However, descriptions of affect dysregulation in BPD primarily have been based on only one of the two domains of affect dysregulation, "underregulation": A deficiency in the capacity to modulate affect such that emotions become uncontrolled, expressed in intense and unmodified forms, and overwhelm reasoning (Koenigsberg et al., 2002; Zittel Conklin, Bradley & Westen, 2006; Zittel Conklin & Westen, 2005). More recent clinical research has suggested that another distinct domain of affect dysregulation may play a role in mental disorders, "overregulation": the numbing of, and an inability to recognize and articulate emotions (Van Dijke, Ford et al., 2010). Marx and Sloan (2002) found that among survivors of child sexual abuse, both overregulation and underregulation of affect were significantly related to psychological distress. Only overregulation mediated the relationship between child sexual abuse status and distress, indicating that overregulation is influential in the development of psychological symptoms in childhood sexual abuse survivors. However, underregulation nevertheless may play a role in BPD pathology. Although adult psychiatric inpatients with severe BPD symptoms were particularly likely to report underregulation of affect, one in five also reported clinically significant overregulation of affect (Van Dijke, Ford et al., 2010). Moreover, Lanius and colleagues (e.g., Frewen et al., 2006, 2008, Lanius, Hopper, & Menon, 2003, Lanius et al., 2010, 2011) consequently demonstrated in their neuroimaging studies support for the presence of qualitatively different forms of posttraumatic self-dysregulation characterized by underregulation (hyperarousal) or overregulation (emotional numbing).

Developmentally adverse interpersonal trauma occurring during early life (Ford, 2005), including but not limited to abuse and neglect, has been found to be associated with complex forms of

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Annemiek van Dijke, Delta Psychiatric Hospital, Rotterdam, The Netherlands; Julian D. Ford, Department of Psychiatry, University of Connecticut School of Medicine; Maarten van Son, Laurence Frank, and Onno van der Hart, Utrecht University.

Correspondence concerning this article should be addressed to Annemiek van Dijke, Delta Psychiatric Hospital, Albrandswaardsedijk 74, 3172 AA, Poortugaal, the Netherlands. E-mail: a.van.dijke@deltapsy.nl

posttraumatic self-dysregulation, of which affect dysregulation is a core feature (Cook et al., 2005; D'Andrea et al., in press). Affective disruptions, including underregulation and overregulation of affect, are at the heart of disturbances stemming from childhood abuse, especially when the agent of abuse is a caregiver (Bermond, Moormann, Albach, & Van Dijke, 2008; Freyd, DePrince, & Gleaves, 2007; Paivio & Laurent, 2001). Early life abuse by caretakers has been hypothesized to alter the developing autonomic and central nervous systems (Fosha, Siegel, & Solomon, 2009; Schore, 2001, 2002; Siegel, 1999, 2001). Enduring structural changes in these areas secondary to abuse and attachment disruption could produce the inefficient stress coping mechanisms that lie at the core of the posttraumatic symptoms associated with severe adult mental disorders (Mueser & Taub, 2008). Moreover, the long-term sequelae of childhood trauma do not seem to be encompassed by any single *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [*DSM–IV–TR*] American Psychiatric Association [APA], 2004) disorder (e.g., Ford, 1999; Van der Kolk, 2005; Van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). Studies have reported an elevated risk of childhood trauma exposure or complex posttraumatic stress disorder (PTSD) symptoms in persons reporting BPD symptoms (e.g., Ford, 1999; McLean & Gallop, 2003).

However, a recent study showed that complex PTSD can be distinguished from BPD and/or somatoform disorder (SoD; Van Dijke, et al., 2012). Almost a quarter of all participating patients in the study met criteria for complex PTSD. Complex PTSD symptoms were significantly and differentially distributed across diagnostic groups. Patients diagnosed with comorbid BPD + SoD most frequently (almost 40%) met criteria for complex PTSD, while also reporting the most extensive trauma histories. BPD and BPD + SoD patients reported similar complex PTSD symptoms, except for subscale alterations in one's system of meaning, in which BPD patients report the highest mean score, and subscale total somatic complaints, in which BPD + SoD patients report the highest mean score. Thus, although a prior report with a smaller clinical sample of women diagnosed with BPD reported that complex PTSD was present in all cases (McLean & Gallop, 2003), by including patients of both genders and other diagnoses as well as BPD patients, this study demonstrated that complex PTSD is not exclusively or always found with (and therefore not synonymous with) BPD. Caretaker-caused traumatic stressors are likely to occur in and contribute to troubled family environments (Weaver & Clum, 1993). Trauma-by-primary caregiver (TPC) has been found to be associated with neglect (Lieberman & Amaya-Jackson, 2005), insufficient affective responsivity, and stimulation (Tronick & Weinburg, 1997), and a failure to protect the child from posttraumatic states of enduring negative affect (Fonagy, Gergely, Jurist, & Target, 2002; Lieberman & Amaya-Jackson, 2005). In addition, the abusive caretaker might provide insufficient protection against other potential abusers of the child. The abusive caretaker also often is inaccessible and unattuned and/or rejecting of the infant's expressions of emotions and stress (Fosha, Siegel, et al., 2009; Schore, 2001). Therefore, the abusive caretaker is likely to show minimal or unpredictable participation in helping the child to develop the capacity to regulate arousal and recognize and utilize emotions (Lyons-Ruth, Dutra, Schuder, & Bianchi, 2006). Instead of modulating extreme levels of stimulation and arousal, the infant and caretaker tend to experience extreme levels of arousal episodically when abuse, affective unattunement, neglect, or other stressors occur. If responsive and well-attuned repair from a caretaker is not available to the infant, intense negative states can persist and be experienced by the infant as unmanageable. Until these states subside, the infant must devote most or all of her or his available biological and affective resources to withstand this state of distress and dysregulation (Schore, 2001; Tronick & Weinberg, 1997). As a result of these complex relational adversities that occur in a context of TPC, the biological, cognitive, behavioral, and relational foundations of emotion that are built during infancy may be profoundly altered. The infant may instead feel a pervasive sense of fear, or anxiety, and powerlessness when she or he experiences even mild degrees of negative affect. The infant is likely to develop biological and affective compensatory processes to withstand these dysregulated states of affect, which can become embedded into the core of the evolving body and personality (Fosha, Paivio, et al., 2009; Schore, 2001, 2002; Siegel, 1999, 2001).

Thus, BPD symptoms might be among the numerous final common pathways that occur when affect regulation is compromised as a result of early life interpersonal psychological trauma and disruption in (or unavailability of) caregiving relationships. Van Dijke and colleagues (2011a) found that underregulation and exposure to psychological TPC in childhood were associated with BPD symptoms in a large sample of adult psychiatric inpatients. However, they did not examine the specific role of TPC nor whether affect regulation mediated the relationship between childhood trauma exposure and BPD symptoms. This study therefore examined the relative contributions of self-reported exposure to potentially traumatizing emotional, physical, and sexual events involving a primary caregiver (TPC), and two forms of affect dysregulation, to the severity of BPD symptoms in adulthood in that adult inpatient psychiatric sample.

Method

Participants and Procedure

Participants in the original sample were 472 consecutive admissions to two adult inpatient psychotherapy treatment centers, Altrecht Centre for Psychosomatic Medicine, Altrecht Mental Health, Utrecht ($N = 117$) and De Waard, Centre for Personality Disorders, Delta Psychiatric Hospital, Rotterdam ($N = 355$). During intake diagnoses of BPD and SoD were made according to the *DSM–IV–TR* criteria by trained and officially registered clinicians (psychiatrists, psychotherapists). Where possible, general practice and former hospital records were obtained (with patient's consent) and studied. All participants had a well-documented history of somatic and/or psychiatric symptoms. All had received previous inpatient or outpatient treatment at psychiatric or somatic hospitals and were referred for specialized psychotherapy.

After admission, all patients participated in the multicenter project Clinical Assessment of Trauma-Related Self and Affect Dysregulation (Van Dijke, 2008). This is a diagnostic and orientation period integrated in the first phase (1 to 6 weeks) of psychotherapeutic inpatient group treatment. Assessment was started after 2 weeks of acclimatizing to the therapeutic milieu, and was finished before the end of the fifth week. Evaluation of all assessments, multidisciplinary observations, and participants'

findings (including the findings reported by the participants significant other) and evaluation of the prospective treatment possibilities and treatment goals, took place in the sixth week after admission and before definite admission to the full program.

For all participants, history of potential brain damage (e.g., head injury, electroshock therapy) and the use of psychotropic medications that may impair executive function (e.g., antipsychotics, neuroleptics, lithium) were exclusion criteria. Moreover, for the psychiatric comparison (PC) group, severe mental illness (i.e., schizophrenia, other psychotic disorders, bipolar disorder), eating disorders with severe underweight, or imminent suicidality that would interfere with cognitive functioning, and developmental disorders (i.e., autism spectrum disorders or attention deficit hyperactivity disorder), were exclusion criteria. All psychiatric control patients met *DSM–IV–DR* criteria for generalized anxiety disorder and/or unipolar depression with sufficient psychosocial impairment that admission for inpatient psychiatric care was indicated.

This study was approved by the local ethics committee (Toetsingscommissie Patientgebonden Wetenschappelijk Onderzoek, Arnhem, The Netherlands). After complete description of the study and procedure, participants provided written informed consent to participate, according to the Declaration of Helsinki (World Medical Association, 2009).

Table 1 presents the demographic characteristics of the total sample. No significant effects were found for age, gender, and level of education on the dependent variables. Final decision for inclusion in the study was after multidisciplinary consensus on diagnoses and clinical presentation in relation to self-report findings (Van Dijke, 2008).

Measures

After being admitted and for inclusion in the study, BPD and SoD (i.e., somatization disorder, undifferentiated somatoform disorder, severe conversion, and pain disorder) and other diagnoses were confirmed by trained clinicians using two clinical interviews: BPDSI (Borderline Personality Disorder Severity Index; Weaver & Clum, 1993; Dutch version IV, Arntz, 1999) and CIDI (Composite International Diagnostic Interview; World Health Organization [WHO], 1997; Dutch version Ter Smitten, Smeets, & Van den Brink, 1998). Trained clinicians performed all assessments and were supervised by A. Van Dijke.

The BPDSI (Weaver & Clum, 1993; Dutch version IV, Arntz, 1999) is a semistructured interview that contains nine sections (abandonment, relationships, self-image, impulsivity, parasuicide, affect, emptiness, anger, and dissociation and paranoia) corresponding to the symptom clusters of BPD. Each section contains items asking about events, for example, "Did you, during the last three months, ever become desperate when you thought that someone you cared for was going to leave you?" The BPDSI has been shown to have good validity and reliability (subscales .70 to .93; total score .96; Arntz et al., 2003). Positive reports for six of the nine sections with BPD symptom severity scores over 20 were considered meeting *DSM–IV–TR* criteria.

The CIDI is a comprehensive, standardized instrument for assessing mental disorders according to the definitions and diagnostic criteria of *DSM–IV–TR* and ICD–10 (WHO, 1997). The CIDI has been shown to have good reliability and validity (Andrews, & Peters, 1998). After completion of the interviews, for inclusion in the study the diagnosis of SoD was confirmed—and somatic disease was ruled out—by a specialist in internal medicine.

To assess underregulation of affect, each participant completed the Dutch self-report version of the Structured Interview for Disorders of Extreme Stress Not Otherwise Specified, Revised (SIDES–R; Ford & Kidd, 1998), an adaptation of the interview that provides a subscale for dysregulated affect (Ford & Kidd, 1998; Dutch translation Van Dijke & Van der Hart, 2002; Cronbach's $\alpha = .75$). The items include: (1) often getting "quite upset" over daily matters, (2) being unable to get over the upset for hours or not being able to stop thinking about it, and (3) having to "stop everything to calm down and it took all your energy" or "getting drunk, using drugs or harming yourself" to cope with emotional distress. Thus, the measure addresses the core components of underregulation of affect, that is, frequent/intense distress, inability to modulate or recover from distress, and use of self-defeating coping to deal with distress.

To assess overregulation of affect, each subject completed the Bermond Vorst Alexithymia Questionnaire (BVAQ; Vorst & Ber-

Table 1
Demographic Characteristics of the Original Total Sample

Variable	BPD	SoD	BPD + SoD	PC	Total sample
N	120	159	129	64	472
Male	40	47	30	28	145
Female	80	112	99	36	327
Age *M* (*SD*)	29.9 (8.8)	38.3 (10.5)	33.6 (9.1)	36.8 (9.9)	34.7 (10.1)
Social					
None	30.8%	45.3%	40.3%	28.1%	37.9%
T	60.8%	41.5%	47.3%	56.3%	50.0%
S	8.3%	13.2%	12.4%	15.6%	12.1%
Education					
L	24.2%	22.6%	27.1%	23.4%	24.4%
M	35.8%	45.9%	37.2%	46.9%	41.1%
H	40.0%	31.4%	35.7%	29.7%	34.5%

Note. BPD = borderline personality disorder; SoD = somatoform disorder; PC = psychiatric comparison group; Social = primary relationship status; T = living together; S = separated by death or divorce; L = primary and low-level secondary education; M = middle level secondary education; H = high-level secondary education.

mond, 2001), which is a Dutch 40-item questionnaire using Likert-type scales. The cognitive dimensions of the BVAQ are highly correlated with the Toronto Alexithymia Scale (TAS–20; Bagby, Parker, & Taylor, 1994; $r = .80$). Of the BVAQ, the cognitive dimensions were used as the measure for overregulation of affect (diminished ability to verbalize, diminished ability to identify, and diminished ability to analyze emotions). High scores represent stronger alexithymic tendencies. A reliability analysis was performed for the whole sample (Cronbach's $\alpha = .88$). The internal consistency for the total scale and its subscales in other samples is good and varies between .75 and .85 (Vorst & Bermond, 2001).

Reports of potentially traumatizing events were collected using the Traumatic Experiences Checklist (TEC; Dutch version, Nijenhuis, Van der Hart, & Kruger, 2002), a retrospective self-report questionnaire concerning adverse experiences and potential traumatic events. Reports of traumatic experiences by primary caregiver were confirmed by close relatives in a subsample of Delta Psychiatric Centre participants, with 100% agreement. A TPC (i.e., potentially traumatizing-event-by-primary caregiver) variable was computed by categorizing participants dichotomously as potentially traumatizing events by the caregiver were present (in any form) and regardless of the developmental epoch the event or absent and by summarizing positive variables. The TEC has been shown to have good reliability and validity among psychiatric outpatients (Nijenhuis, Van der Hart, & Kruger, 2002).

Statistical Analysis

The Statistical Package for Social Sciences (SPSS, version 17.0) was used for computing descriptive statistics and correlations. Mediation analysis was carried out on continuous scores with path analysis and the indirect effects were tested with bootstrap confidence intervals using software package Mplus, version 4.2 (Muthén & Muthén, 2007) and following the guidelines on mediation analysis given by Baron and Kenny (1986); Bollen and Stine (1990); MacKinnon (2008); MacKinnon, Lockwood, Hoffman, West, and Sheets (2002); Muthén & Muthén (2007).

Results

From the original sample of size ($N = 472$), 450 participants were used in the statistical analyses due to missing values. From the 450 participants, 283 participants (63%) experienced TPC. Table 2 presents means, standard deviations, and intercorrelations for all variables involved in the path analysis for the tests of mediation. No anomalies were detected in the shape of the distributions of these variables. None of the assumptions of the statistical analyses were violated.

Table 2
Descriptive Statistics and Pearson Correlations Coefficients of the Variables Used in the Mediation Analyses

Variables	*M*	*SD*	1	2	3
1. BPD symptoms	22.90	12.46	—		
2. TPC	0.63	0.48	.29**	—	
3. Underregulation	7.62	2.12	.48**	.09*	—
4. Overregulation	75.46	18.24	.17**	.01*	.12*

Note. $N = 450$.
* $p < .05$. ** $p < .01$.

BPD symptoms were positively related to reports of childhood TPC, higher levels of underregulation and higher levels of overregulation. In addition, reports of childhood TPC were positively related to underregulation, but not to overregulation. Therefore, the mediation analyses reported next do not include overregulation of affect.

Figure 1 shows a path analysis model that represents the relation between childhood TPC and the presence of BPD symptoms, mediated by underregulation of affect. The direct relation between childhood TPC and BPD symptoms, without the presence of underregulation of affect in the model, was statistically significant ($B = 7.54$, 95% CI [5.25, 9.83]) and corresponds to a medium effect size according to Cohen (1988). The presence of childhood TPC was associated with higher levels of BPD symptoms.

When underregulation of affect was added to the model, the direct effect between TPC and BPD symptoms became smaller, but remained statistically significant ($B = 6.45$, bootstrapped 95% CI [4.37, 8.24]). The total indirect effect of childhood TPC via underregulation of affect was statistically significant ($B = 1.09$, bootstrapped 95% CI [0.03, 2.64]). The fact that the direct effect remains statistically significant after taking into account underregulation of affect shows that there is partial mediation.

Discussion

The principal findings of this study were that childhood TPC had both a direct association with BPD symptoms (although not necessarily to meeting *DSM–IV–TR* BPD diagnostic criteria) and an indirect relationship that was partially mediated by underregulation of affect. Although the results imply that both TPC and underregulation of affect warrant attention in assessment, treatment, and research on BPD, they do not rule out the possibility that other factors not assessed in this study may contribute to BPD (e.g., parental psychopathology, other forms of childhood psychological trauma, genetic factors), or rather than being independent factors, some of these factors might be highly related to TPC (e.g., parental psychopathology) and explain some of the associations observed. Research is needed to determine whether those other potential contributing factors to BPD are also associated with either or both TPC and underregulation of affect.

TPC was not associated with overregulation of affect, and therefore overregulation could not serve as a mediator of the TPC–BPD relationship. Overregulation was previously found to be associated with BPD in this inpatient psychiatric sample (Van Dijke, Ford et al., 2010), but not as strongly as underregulation of affect. In those analyses, overregulation of affect also was found to be most strongly associated with somatoform disorders, which were not associated with trauma history (Van Dijke et al., 2011a). Taken together, the findings suggest that although some patients diagnosed with BPD may experience overregulation of affect, the primary focus for assessment and treatment in BPD should be on underregulation of affect.

The direct and indirect relationship of TPC to BPD symptoms suggests that treatment of BPD may be enhanced for patients with histories of TPC in childhood by addressing underregulation of affect as a sequela of TPC. Therefore, evidence-based and evidence-informed treatment models that focus on repair of disor-

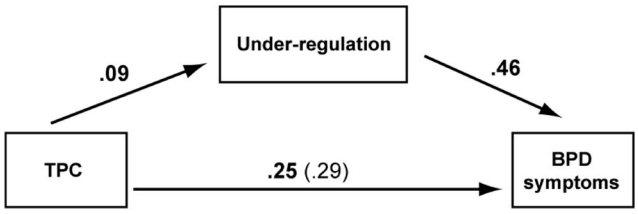

Figure 1. Standardized regression coefficients for the relation between presence of TPC and the development of BPD symptoms mediated by underregulation of affect. The direct relation between TPC and BPD symptoms without taking into account underregulation yields a standardized regression coefficient of .29. TPC = trauma-by-primary caregiver; BPD = borderline personality disorder.

ganized attachment working models (e.g., Courtois, 2010; Courtois & Ford, 2009; Fosha, Siegel, & Solomon, 2009), affect regulation (Cloitre et al., 2010; Ford, Steinberg, & Zhang, in press), and resolution of betrayal trauma (DePrince & Freyd, 2004) warrant scientific and clinical examination in the treatment of patients diagnosed with BPD who have histories of TPC in childhood.

Affect dysregulation is a core component of what has been described as complex PTSD or disorders of extreme stress not otherwise specified (DESNOS; Herman, 1992; Pelcovitz, Van der Kolk, Roth, Mandel, & Resick, 1997; Roth et al., 1997; Van der Kolk, 1996). Women (McLean, Toner, Jackson, Desrocher, & Stuckless, 2006) and men (Ford, 1999) in a psychiatric treatment samples who met diagnostic criteria for BPD were found to be likely to have high levels of complex PTSD/DESNOS symptoms and histories of childhood psychological trauma. However, no study has assessed the association of the more specific forms of psychological trauma (childhood TPC) and affect dysregulation (underregulation of affect) identified in the present study to complex PTSD/DESNOS symptom severity.

The relationship between complex PTSD/DESNOS and BPD has been controversial because complex PTSD was first formally described (Herman, 1992), and research clarifying how the two syndromes' symptoms and etiological factors are similar or different is needed. TPC in childhood and underregulation of affect may provide a basis for identifying a subgroup of BPD patients for whom chronic traumatic stress reactions and subsequent affect dysregulation are primary etiological factors—versus others diagnosed with BPD for whom affect dysregulation secondary to attachment disruptions may not play an etiological role. Such a distinction could guide not only the development of more individualized treatment methods for adults diagnosed with BPD, but could also serve as a basis for both clinical and scientific testing of the benefits of targeting different treatment approaches for BPD depending on the likely role of attachment-related childhood trauma.

It is also important to note that the absence of a correlation between TPC in childhood and overregulation of affect does not mean that either those individuals with childhood TPC histories do not experience difficulties with overregulation or that overregulation is not of concern in the assessment and treatment of BPD. Further research is needed to determine if there is a subgroup of patients in psychiatric treatment who have childhood TPC histories and problems with underregulation of affect. Given prior findings from this sample that overregulation co-occurs with underregulation in a distinct subgroup, most of whom met criteria for somatoform disorders as well as BPD (Van Dijke, Ford et al., 2010), scientific and clinical studies should examine the role that TPC may play when both somatic and relational dysregulation are problems (e.g., comorbid somatoform and BPD) and both over- as well as underregulation of affect require treatment.

The role of dissociation, which has been found to be a substantial problem for adults in psychiatric treatment for both somatic and relational dysregulation (Van Dijke, Van der Hart et al., 2010), was not tested in these analyses. TPC in childhood has been found to be linked to problems with dissociation in adulthood in a prospective study (Lyons-Ruth et al., 2006). Dissociation and affect dysregulation may lead to chronic excitatory (underregulated) or inhibitory (overregulated) states that are consistent with features of complex PTSD/DESNOS (Van Dijke et al., 2012) and a number of the sequelae of childhood psychological trauma (D'Andrea et al., in press). Lanius and colleagues (2010) also recognized undermodulation and overmodulation in traumatized patients and they proposed a dissociative subtype of PTSD. However, they concluded that overmodulation was dissociative, thereby regarding emotional numbing to be dissociative (and not alexithymic). Also they were, by concluding overmodulation to be dissociative, acknowledging only negative dissociative phenomena instead of differentiating positive and negative psychoform and somatoform dissociative symptoms (e.g., Van der Hart, Nijenhuis, & Steele, 2006). Intrusive PTSD symptoms that some authors (Van der Hart et al., 2006) regarded as positive dissociative symptoms (e.g., the *DSM–IV–TR* speaks of dissociative flashback episodes as one of PTSD's symptoms; APA, 2004), were viewed not as dissociative but instead as anxiety-based PTSD symptoms.

In contrast, Van Dijke and colleagues (Van Dijke, Van der Hart, Ford, Van Son, Van der Heijden, & Bühring, 2010) differentiated both inhibitory and excitatory symptoms for affect dysregulation (i.e., underregulation and overregulation of affect) and for psychoform and somatoform dissociation (i.e., positive and negative symptoms). Results showed that although inhibitory (negative psychoform and somatoform dissociative symptoms and overregulation of affect) and excitatory (positive psychoform and somatoform dissociative symptoms and underregulation) symptoms of posttraumatic self-dysregulation are related, these inhibitory and excitatory symptoms of affect dysregulation and dissociation are distinct phenomena (Van Dijke, Van der Hart, et al., 2010). Research is needed therefore to examine the potential interaction of posttraumatic affect dysregulation and both positive and negative types of dissociation in patients with psychiatric disorders.

This study had several methodological limitations that should be considered when interpreting its findings. Data were obtained from a psychiatric population consisting of adult psychiatric inpatients, and the results cannot be generalized to other psychiatric (e.g., outpatient) populations without replication studies with a broader range of adults in psychiatric treatment. The sample primarily consisted of patients diagnosed with BPD and somatoform disorders, and although a general psychiatric inpatient cohort was included the findings need to be replicated with a fuller representation of other Axis I and II disorders.

The study design was cross-sectional, retrospective, and correlational; therefore no conclusions can be drawn about the etiolog-

ical significance of TPC in childhood or affect dysregulation for BPD. Prospective studies (e.g., Lyons-Ruth et al., 2006) are needed to assess TPC at the time of occurrence in childhood (or based on credible sources other than self-report; e.g., documented reports of abuse or neglect) and affect dysregulation over the developmental course of childhood, adolescence, and adulthood prior to the onset of BPD.

The self-report measures of affect dysregulation, although psychometrically sound, may not accurately describe the actual behavioral manifestations of participants' difficulties with affect regulation in vivo. Reports from other sources (e.g., family members, peers), direct observational data gathered using naturalistic or experimental paradigms, or ecologically valid self-report data (e.g., daily self-monitoring paradigms) are needed to replicate study findings regarding the nature and extent of participants' under- and overregulation of affect.

Although the accuracy of self-reported assessment of the types and times of exposure to psychological trauma is uncertain, there is evidence that adults with serious mental disorders can validly report trauma histories (Mueser & Taub, 2008). In addition, efforts were made (with patient consent) to confirm the reported traumatic events with close relatives (see van Dijke, 2008, for a more detailed description). Anecdotally, participants who reported potentially traumatic experiences involving a caregiver in childhood reported that this was difficult to disclose; therefore, there might have been more underreporting than overreporting of these events and the preliminary estimates of the likelihood of childhood TPC in this adult psychiatric population may actually be lower than the actual prevalence.

The finding of both a direct and an indirect (mediated by underregulation of affect) relationship of TPC to BPD symptom severity suggests that assessment and treatment of BPD may be enhanced for patients with histories of TPC by also addressing underregulation of affect as sequela of TPC rather than as only one feature of the disorder.

Treatment approaches to dysfunctional affect regulation have been developed for patients with severe mental disorders (Cloitre, Koenen, Cohen, & Han, 2002; Ford & Russo, 2006; McCullough et al., 2003; Wolfsdorf & Zlotnick, 2001), including dialectical behavior therapy (Linehan et al., 2006), transference focused psychotherapy (Levy et al., 2006), and mentalization based treatment (Bateman & Fonagy, 2008). However, these treatments address underregulation of affect more explicitly than overregulation of affect. For patients reporting TPC in childhood and BPD symptoms and who also report overregulation of affect, sensorimotor psychotherapy (Ogden, Minton, & Pain, 2006), accelerated experiential-dynamic psychotherapy (Fosha, 2003; Fosha, Paivio, Gleiser, & Ford, 2009) and emotion-focused therapy for trauma (Greenberg & Bolger, 2001; Paivio & Pascual-Leone, 2010) warrant testing to determine if they can ameliorate problems with overregulation by facilitating experiential processes of emotional awareness and emotional growth.

To the extent that affect dysregulation in BPD reflects fundamental problems with relational working models (e.g., disorganized attachment; Lyons-Ruth et al., 2006) resulting from psychobiological adaptations to TPC, treatment can focus on helping the patient to develop relational security (Allen, 2001; Courtois & Ford, 2009; Fosha, Paivio, Gleiser, & Ford, 2009; Kinniburgh, Blaustein, Spinazzola, & Van der Kolk, 2005) as well as to recognize, contain, and adaptively use and express emotions in their relationships.

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