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EXAMINING THE COMORBIDITY OF BORDERLINE PERSONALITY DISORDER AND POSTTRAUMATIC STRESS DISORDER IN A COMMUNITY SAMPLE

A Thesis

presented to

the Faculty of the Graduate School

at the University of Missouri-Columbia

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

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by

EMILY M. SCHEIDERER

Timothy J. Trull, PhD, Thesis Supervisor

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The undersigned, appointed by the dean of the Graduate School, have examined the thesis or dissertation entitled

Examining the Comorbidity of Borderline Personality Disorder and Posttraumatic Stress Disorder in a Community Sample

presented by Emily Scheiderer,

a candidate for the degree of Master of Arts,

and hereby certify that, in their opinion, it is worthy of acceptance.

Tim Trull, PhD

Phil Wood, PhD

Niels Beck, PhD

Denis McCarthy, PhD

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ABSTRACT

Borderline Personality Disorder (BPD) and Posttraumatic Stress Disorder (PTSD), both relatively prevalent disorders in our society, overlap and/or co-occur in ways that are not yet well understood, especially outside of clinical samples. Despite methodological and sampling differences among existing studies, ample evidence exists to suggest that this comorbidity is frequent and presents a variety of difficulties for the individual, the clinician, and the researcher. This comorbidity also raises many questions, most of which remain unanswered. The present study aimed to address some of these questions in a large, community sample. In particular, the question of the importance of childhood sexual abuse (CSA) as a potential etiological factor and predictor of general functioning was addressed, along with other important factors, such as gender and age. CSA has been a focus of prior clinical studies and theoretical literature, but empirical evidence to generalize this focus to the broader population has been lacking. Results from the present study suggest that, in the general population: This comorbidity is more deleterious than either BPD or PTSD alone; CSA should continue to be considered an important factor; and the factors of gender, age, and CSA exhibit interactions and main effects in the prediction of this comorbidity and its associated decrements in health-related functioning, calling for continued research as well as attention to these factors in the treatment context.

INTRODUCTION

The frequent co-occurrence of borderline personality disorder (BPD) and posttraumatic stress disorder (PTSD) presents a variety of difficulties for the individual, the clinician, and the researcher. It also raises many questions, most of which remain unanswered. The present study aims to address some of these questions in a large community sample, and to do so from a pragmatic perspective, so as to work toward better understanding and alleviation of the particular difficulties of this comorbidity.

BPD and PTSD, both relatively prevalent disorders in our society, overlap and/or co-occur in ways that are not yet well understood, especially outside of clinical samples. As currently defined, the diagnostic criteria for these two disorders do not overlap substantially; yet patients with either of these two disorders can present with confusingly similar clinical pictures. Clinician orientation appears to add yet another twist to this confusion, significantly influencing clinicians' diagnostic differentiation between BPD and PTSD when the presenting features are mixed or ambiguous (Woodward, Taft, Gordon, & Meis, 2009). Moreover, fundamental questions remain unsettled regarding whether these two disorders share some common construct (i.e., some type of true 'overlap' or even a subsumption, in which one is a variant of the other), whether they simply get confused frequently due to our biases and the imperfections of our diagnostic criteria, or whether they often co-occur as two legitimately separate disorders. In fact, the general concept of comorbidity, itself, remains controversial: The term *comorbidity* may confer more meaning than is appropriate on something that is merely a co-occurrence of disorders or syndromes; and some of what we call *comorbidity* or co-

occurrence may be merely an artifact of an imperfect diagnostic framework (e.g., Lilienfeld, Waldman, & Israel, 1994; Aragona, 2009). For the purposes of this paper, the term *comorbidity* will be used to mean *co-occurrence*, without any specific theoretical intent. The fundamental debate on comorbidity and the questions regarding whether the current *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000) criteria capture optimal diagnostic configurations of BPD and PTSD, though interesting and important, lie beyond the scope of the present study. Topics and findings addressed herein may be applicable to this debate in various ways, but neither a complete nor explicit treatment of this topic will be attempted.

As our current diagnostic system stands (i.e., as set forth by the DSM-IV-TR)—despite the lack of clarity regarding the clinical and theoretical boundaries between BPD and PTSD, and despite the fundamental contention surrounding concept of comorbidity—many people distinctly experience one or both of these disorders. Reported comorbidity rates range vastly, depending on sampling characteristics and other relevant factors that vary across studies (e.g., differential use of diagnostic guidelines and assessment methods). These numbers, nonetheless, unanimously reflect a significant overlap between the two disorders. For example, studies relying on treatment-seeking clinical samples, which comprise the majority of existing studies of BPD-PTSD comorbidity, have presented comorbidity rates of PTSD among individuals with BPD ranging from 25% to 58%, and comorbidity rates of BPD among individuals with PTSD ranging from 10% to 78% (Pagura et al., 2010). And most epidemiological studies, employing community samples, have reported that 60% to 70% of BPD patients have

comorbid PTSD (Schmahl et al., 2009); though some report much lower numbers, for example 46.9% (McGlashan et al., 2000) or even 17% (Lenzenweger et al., 2007).

Though notably limited in generalizability to the non-clinical population, recent studies examining mostly small, clinical, treatment-seeking samples have made important contributions to our current understanding of BPD-PTSD comorbidity. Such studies have highlighted the greater frequency and/or severity of problems associated with this comorbidity and have pointed out potentially important differences (e.g., in brain functioning and behavior) between individuals with the comorbidity and those with just one of the two disorders. For example, findings from small, clinical studies have suggested that individuals with comorbid BPD-PTSD have different neurological reactions to pain (e.g., Kraus et al., 2009), and that BPD-PTSD comorbidity tends to result in lower general functioning and more frequent hospitalizations than BPD and PTSD alone (Zlotnick et al., 2003; Heffernan & Cloitre, 2000). From the perspective of public health and the general population, however, these contributions remain hindered by their limited generalizability. Clinical samples tend to be different from non-clinical samples in that they are generally treatment-seeking, more severely impaired, disproportionately female, and potentially impacted by various other sample-specific biases (e.g., by clinician expectations or diagnosis-related stigma). The potential for such differences to affect mental and physical health outcomes must be considered. Although the findings reported in clinical samples could generalize to the broader population, the lack of empirical support for such generalization and the potential for such sample differences to affect outcomes (and thus affect our understanding of this comorbidity) call for increased efforts toward a community-based or epidemiological approach.

Looking then to existing, large, non-clinical samples, some community epidemiological studies have included examination of BPD-PTSD comorbidity (usually along with other DSM Axis I and Axis II comorbidities; e.g., Grant et al., 2008; Lenzenweger, Lane, Loranger, & Kessler, 2007), generally reporting high prevalence rates. Different implementations of diagnostic criteria, sampling methods, and approaches to handling extraneous variables (e.g., demographics and other comorbid disorders), however, contribute to substantial variation in these reported numbers and to a lack of clarity with regard to their implications. Moreover, very little is understood about the fundamental 'why' and 'how' behind these numbers. For example, why is the BPD-PTSD comorbidity so prevalent? How does it impact individuals? How is it different from having just one of the two disorders? And what implications might this all have for treatment? The substantial prevalence of this comorbidity and its seeming potential for particularly deleterious outcomes, together with our lack of understanding, warrant continued research. The present study utilizes data from the *National Epidemiological Survey on Alcohol and Related Conditions* (NESARC) to further explore and characterize this BPD-PTSD comorbidity and its implications in a large, representative community sample.

Comparing BPD and PTSD

Consulting the *Diagnostic and Statistical Manual of Mental Disorders* (DSM IV-TR; American Psychiatric Association, 2000), the phenomenological descriptions of BPD and PTSD (found on Axis II and Axis I, respectively) appear quite different.[1] So, why

[1] Not all agree with this diagnostic separation employed by the DSM-IV. Lenzenweger et al. (2007) asserted that high Axis I comorbidity rates among personality disorders (PDs) call into question the separation of PDs from Axis I disorders in the

and how is it that we see these two disorders overlapping or co-occurring so often? Consideration of the literature and theory on this topic points to possible overlaps in etiology. Multidimensional, or biopsychosocial, approaches to BPD posit childhood trauma as an important category of psychological risk factor (e.g., Paris, 1994). In this view, at least one psychological risk factor is necessary—along with biological and social risk factors—for the development of BPD. Of the psychological risk factors that have been examined, trauma appears to be the most specific to BPD, but no factor appears specific enough to draw firm conclusions about its etiological role (Paris, 1994). As reflected by 'criterion A' of the DSM-IV criteria for PTSD, a significant trauma could also open the door to PTSD, allowing for the possibility of a common point of etiology of BPD and PTSD in a given individual. Alternatively, or perhaps additionally, the impulsivity and emotional reactivity characteristic of BPD could increase an individual's risk for encountering traumatic experiences throughout life, thereby increasing that person's chances of developing PTSD.

From the clinician's perspective, the question of the boundary between BPD and PTSD often arises with the presentation of patients whose symptoms blur the lines (e.g., when a depressed, self-mutilative, and impulsive patient presents with a history that prominently features childhood trauma). Differentiation of diagnosis can be extremely difficult in this complex interface between BPD and PTSD (Gunderson, 2001), perhaps rendering the impact of biases, such as those related to clinician orientation (Woodward et al., 2009), almost inevitable. From this diagnostic complexity, various perspectives

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DSM—a separation not made by the *International Classification of Diseases* (ICD; published by the World Health Organization)—and, further, suggested that PDs are possibly "variants on processes common to Axis I disorders" (p.562).

have emerged. Some believe BPD and PTSD to be variants of a single construct (e.g., that BPD may be a chronic form of PTSD; Kroll, 1993). Many others view BPD and PTSD as two distinct and independent disorders, or simply do not confront the issue at all. And certainly, various more nuanced perspectives have been voiced, as researchers and clinicians have tried to make sense of their findings and patients.

Some compelling evidence from recent clinical studies argues for the consideration of BPD and PTSD as distinct and separate constructs that exhibit relatively independent constellations of symptoms. For example, studies by Zlotnick et al. (2003) and Heffernan and Cloitre (2000) found that while this comorbidity did not impact the core clinical features of either individual disorder, it did impact some more general outcomes (i.e., outside of each disorder's core features), including in Zlotnick et al. (2003), lower global functioning scores and higher frequency of hospitalization; and in Heffernan and Cloitre (2000), higher general distress, worse treatment compliance, and a trend toward more hospitalizations. Overall, such findings suggest that BPD-PTSD comorbidity tends to present as an addition of BPD features, plus PTSD features, plus some additional general difficulties beyond what one might expect in someone not dealing with this comorbidity (Zlotnick et al., 2003). Such findings fall in line with the general treatment outcome findings in the field of BPD comorbidity research, in which comorbidity on Axis I is associated with BPD stability (i.e., lack of improvement over time; Skodol et al., 2002a), and presence of PD at intake predicts poor short- and long-term outcome of an Axis I disorder, even among patients matched on Axis I symptom severity at intake (e.g., Skodol et al., 2002b).

Approaching the question of the distinction between BPD and PTSD from another area of the psychological discipline, recent physiological and neuroimaging studies have provided preliminary evidence for the separate existence of BPD and PTSD. Schmahl et al. (2004) provided preliminary evidence for divergence in the pathophysiology of BPD and PTSD. In this study, individuals with PTSD and a history of childhood sexual and/or physical abuse showed greatest systolic blood pressure responses to traumatic scripts, whereas those with this childhood trauma history and BPD tended toward greater skin conductance responses to abandonment scripts (Schmahl et al., 2004). Problems of small sample size and PTSD diagnoses present in the BPD group, however, limited these findings, calling for a replication with more rigorous sampling and methodology. Recent neuroimaging findings have further elucidated some of the biological facets of BPD and PTSD, in some cases, implicating a significant influence of comorbid PTSD on prior findings in the BPD literature. For example, the finding of Kraus et al. (2009) suggested that the co-occurrence of PTSD alters the neural processing of pain in BPD patients; and thus, amygdala deactivation seen in previous BPD findings may be an artifact of high rates of co-occurring PTSD in many BPD samples. Similarly, the findings of Schmahl et al. (2009) suggested that the comorbidity of PTSD with BPD may explain prior findings of reduced hippocampi size in BPD patient samples. Additionally, Schmahl et al. (2009) found a significant positive correlation between hippocampal volume and impulsiveness (as assessed by the Barratt Impulsiveness Scale, 10th revision (BIS-10); Patton, Stanford, & Barratt, 1995) that disappeared once their (all-BPD) sample was split into those with and without PTSD, suggesting that the potential impact of PTSD comorbidity on central features of BPD should continue to be explored, and suggesting that PTSD comorbidity

could be a variable of interest in the ongoing discussion of BPD 'subtypes' (see Leihener et al. (2003) for a review of the varied conceptual and empirical arguments for BPD subtying). Taken together, these physiological and neuroimaging findings are suggestive, but inconclusive, on the topic of interaction of BPD and PTSD with regard to volumetric changes and pain reactivity in the brain's limbic system (Schmahl et al., 2009). The authors advocate further investigation with larger samples and more careful attention to comorbidity (e.g., implementation of subgroup analyses) to address whether comorbid BPD and PTSD have independent additive neurological effects on the limbic system, have supra-additive effects, or whether the effects seen are driven primarily by PTSD and/or traumatization (e.g., Schmahl et al., 2009; Kraus et al., 2009; Weniger et al., 2009). Although the present study does not employ neurological assessments, its large and heterogeneous sample provides a valuable opportunity for examining some of the phenomenological outcomes and clinical implications that may be related to the still unfolding neurological picture of BPD, PTSD, and BPD-PTSD comorbidity.

Unique and Common Etiological Influences

Shifting from the phenomenological perspective to the etiological, it bears noting that the growing body of evidence in support of the separate classification of BPD and PTSD does not preclude the possibility of significant overlaps in etiology between the two disorders: Certainly, the prevalent comorbidity of BPD and PTSD could result from etiological overlap without necessarily implicating a shared construct. Much of the literature on the overlap between BPD and PTSD has focused on the etiological factor of childhood sexual abuse (CSA), which has been recognized as a significant predisposing factor for BPD (e.g., Gunderson, 2001; Kroll, 1993; Paris, 1994; Linehan, 1993) and is

also, undoubtedly, a potential 'criterion A' event in the development of PTSD (APA, 2000). CSA, in particular (as opposed to childhood trauma in general), epitomizes the type of invalidating childhood environment theorized to be an important factor in the etiology of BPD (e.g., Linehan, 1993). However, authors on the topic of BPD and its overlap with PTSD rarely make explicit the full reasoning behind the focus on this particular type of trauma. To some extent, this focus likely grew out of recurrence of CSA as a feature of the confounded clinical presentation (that which blurs the diagnostic lines between BPD and PTSD). As such, this focus may be, itself, confounded with features particular to clinical samples, such as the overrepresentation of women and/or greater severity of impairment.

Though it may be difficult to discern which came first, the focus on CSA or the empirical findings to support it, numerous findings do support a particular importance of CSA in the co-occurrence of BPD and PTSD. Studies tend to report higher rates of CSA in BPD patient samples than in other patient samples. Among ten empirical studies, out of various research centers, the rate of CSA found in BPD patients was about 70% and was significantly greater than the rates found in any of the other patient control groups (Paris, 1994). Findings from a variety of studies looking at the differential predictive relationships (or lack thereof) of childhood physical and sexual abuse with later BPD diagnosis, together, suggest that childhood sexual abuse, as distinct from other types of abuse, may be uniquely associated with BPD (Linehan, 1993). The extent to which CSA interacts with other potential psychological risk factors for BPD may contribute to the CSA-BPD association. For example, CSA may interact with pathological family environment—with disrupted family dynamics in the context of incest, or with family

neglect in the context of extrafamilial CSA (Paris, 1994). From a theoretical perspective, the CSA-BPD association makes sense in that the severe disruption of the close relationships that children otherwise look to for nurturance and security—the paradox of caregiver-abuser—somewhat foreshadows the paradoxical idealization-devaluation characteristic of BPD patients' close relationships in adulthood. CSA, in fact, may be one of the clearest examples of the extreme invalidation theorized to be a key environmental factor in the development of BPD (Linehan, 1993).

Looking more closely at specific parameters of CSA (e.g., age at onset, number of abusers/incidents, duration of abusive relationship) may also be important, as many studies have found such parameters to be influential factors in the relationship between CSA and psychopathology. Findings relevant to BPD and PTSD, in particular, have highlighted the predictive nature of such parameters (Paris, 1994; Kroll, 1993; Pagura et al., 2010). For example, in a study by Van Den Bosch, Verheul, Langeland, and Van Den Brink (2003), prevalence of PTSD among 64 female BPD patients with childhood traumatic experiences was associated with severity of CSA in terms of greater physical extent of CSA, intrafamilial CSA, duration of CSA for more than one year, and abuse by multiple perpetrators. Looking at the comorbidity from the other angle, Heffernan and Cloitre (2000) found that the additional BPD diagnosis in PTSD patients with history of CSA was associated with earlier age of onset of CSA.

Any thorough focus on CSA in this line of research should not only address these severity parameters, but also must be tempered by the almost certain fact that many people who experience CSA do not develop BPD or PTSD; and many who do develop BPD, PTSD, or both disorders did not experience CSA. Any true relationships among

CSA, BPD, and PTSD are likely to be complex and certainly not absolute. Other classifications of traumatic childhood experiences (e.g., physical abuse, verbal abuse, separation and loss) have also shown associations with BPD (Paris, 1994). Although a host of multivariate findings suggest that CSA makes an independent contribution to BPD diagnosis beyond these other factors, we do not have sufficient evidence to conclude that CSA is the most important factor in most cases (Paris, 1994). A thorough epidemiological examination of the BPD-PTSD comorbidity, thus, should not assume a singular focus on CSA, but rather should examine the associations with CSA among other potentially important risk factors (e.g., other types of traumatic experiences).

The possibility of a gender difference in the BPD-PTSD comorbidity presents another issue potentially related to CSA. Greater prevalence of CSA among women than among men could contribute to a greater prevalence of BPD-PTSD comorbidity among women. Alternatively, greater prevalence of the comorbidity among women for some reason not at all related to CSA could spuriously lead to the apparent association between this comorbidity and CSA due to the mere fact that CSA occurs more in females than males (e.g., Briere & Elliott, 2003). Interestingly, neither the gender difference in prevalence of the BPD-PTSD comorbidity nor the gender difference in CSA prevalence has emerged uniformly across BPD-PTSD studies: Some studies have found both differences, some neither, and at least one study (Johnson et al., 2003) found the expected gender difference in the comorbidity but not in the prevalence of CSA. If this gender difference in prevalence of the comorbidity does exist, factors other than CSA prevalence may also, or alternatively, be involved. For instance, consistent with gender role theories of affect regulation, epidemiological studies tend to find gender differences in the Axis I

comorbidities of BPD patients such that men are more prone to 'externalizing' disorder comorbidities (e.g., alcohol dependency), whereas women are more prone to 'internalizing' disorder comorbidities, including PTSD (e.g., Kessler, Berglund, Demler, Jin, & Walters, 2005; Johnson et al., 2003; Tadic et al., 2009).

Outcomes and Impact of BPD-PTSD Comorbidity

Much as the etiologies of individuals with BPD and PTSD, respectively, appear heterogeneous, so too are the clinical courses or outcomes within both BPD and PTSD patient samples. BPD-PTSD comorbidity may be an important factor in this heterogeneity of clinical course/outcome, presenting particular difficulties for affected individuals, clinicians, researchers, and society at large (i.e., greater public health burden) and perhaps—though the existing evidence is inconclusive and, in some cases, conflicting, (e.g., Zlotnick et al., 2003; Heffernan & Cloitre, 2000; Schmahl et al., 2009; Pagura et al., 2010)—affecting the expression of certain core features of these disorders. Further examination of the possible risk factors and outcomes associated with this comorbidity stands to facilitate important future gains in the areas of clinical treatment, research, and public health. Better understanding of this relatively common comorbidity could improve treatment efficiency and efficacy by better informing clinicians', patients', and researchers' decisions, potentially leading to more specific and promising treatment approaches (Tadic et al., 2009). Hopefully, by better understanding the trends and phenomena that underlie this comorbidity and its particular difficulties, we can better anticipate and address them not only in treatment and research, but also at the levels of public health and management of societal costs.

Looking to Community Studies

Two recent studies (Connor, Davidson, Hughes, Swartz, Blazer, & George, 2002; Pagura, Stein, Bolton, Cox, Grant, & Sareen, 2010) have presented the first community studies to focus specifically on the topic of BPD-PTSD comorbidity. The first of these, by Connor and colleagues (2002), suffered limited generalizability due to factors including a regionally bound sample, inclusion of only 15 respondents who had both BDP and posttraumatic stress symptoms, no BPD-only group, between-group comparisons obfuscated by criterion contamination, and use of "posttraumatic stress symptoms (PTSS)"—a diagnosis that the authors assigned to any person endorsing at least one post-trauma symptom, without any direct question about the trauma or traumas that may, or may not, have preceded the symptom—as a proxy for PTSD. Given these limitations, some of the findings of Connor et al. are still relevant to the present discussion, but must be weighted relatively lightly. Namely, Connor et al. found that those in the PTSS-BPD group rated their own health more poorly than those in the PTSS-only group; more frequently endorsed suicidal thoughts; showed greater rates of benzodiazepine, anxiolytic, sedative, and antidepressant usage; exhibited greater impairment in occupational and social domains (e.g., marital discord); reported more parental abuse, discord, and separation before age 10; were more likely to have been raised in poverty; and were more likely to have experienced sexual assault before age 16 or at some point in their lives than those in the PTSS-only group.

The second known community study, a recent contribution from Pagura, Stein, Bolton, Cox, Grant, and Sareen (2010), provided a much greater contribution toward the understanding of BPD-PTSD comorbidity. These authors presented the first examination

of BPD-PTSD comorbidity in a large, nationally representative sample using reliable and valid diagnostic methods. Data from Wave II (N = 34,653; response rate 70.2%) of the *National Epidemiological Survey on Alcohol and Related Conditions* (NESARC; Grant, Kaplan, Shepard, & Moore, 2003; Grant & Kaplan, 2005) were used to examine, via multiple regression models, the differences in psychopathology, traumatic events, and health-related quality of life across individuals with BPD only (n = 1290), PTSD only (n = 1820), and comorbid BPD-PTSD (n =643). This study by Pagura et al., which was published during preparation of the present study proposal, bore some striking resemblances to the present study, but also featured some important differences—most notably in use of personality disorder (PD) diagnostic criteria—which will be described below.

As reported by Pagura et al. (2010), NESARC psychiatric diagnoses were assessed by trained lay interviewers using the fully structured Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV Version (AUDADIS-IV; Grant et al., 2003; Ruan et al., 2008). Wave II Axis I and II AUDADIS-IV diagnoses demonstrated fair-to-good test-retest and inter-rater reliability, using a subsample of 1,899 respondents (Grant et al., 2003; Ruan et al., 2008); kappas were 0.77 and 0.64 for past-year and lifetime diagnoses of PTSD, and 0.71 for BPD, indicating fair-to-good agreement; and internal consistencies also were good (0.84 for PTSD and 0.83 for BPD). Lifetime prevalence of BPD, PTSD, and BPD-PTSD comorbidity were 5.9% , 6.6%, and 1.6%, respectively; 30.2% of individuals diagnosed with BPD were also diagnosed with PTSD; and 24.2% of individuals diagnosed with PTSD were also diagnosed with BPD (Pagura et al., 2010).

Results from the multiple regression models employed by Pagura et al. (2010) suggested that individuals with the comorbid BPD-PTSD diagnosis had a poorer health-related quality of life, more Axis I comorbidity, increased odds of a lifetime suicide attempt, and a higher prevalence of repeated traumatic events in childhood than did individuals with either diagnosis alone. In terms of demographic characteristics, Pagura and colleagues also found that individuals with PTSD-only were more likely than those with BPD-only or BPD-PTSD comorbidity to be female. Individuals in the comorbid BPD-PTSD group were significantly more likely than those in either the BPD-only or the PTSD-only groups to have most lifetime mood, anxiety, and substance use disorders. Those in the BPD-PTSD comorbidity group were also most likely to have made a lifetime suicide attempt, followed by those in the BPD-only group, and then those in PTSD-only group, with significant differences between all. In analyses assessing health-related quality of life, Pagura and colleagues used past-year diagnosis of PTSD rather than lifetime and found that significant differences persisted after adjusting for sociodemographic factors and number of past-year Axis I mental disorders. Individuals with the comorbidity had significantly lower mental health-related quality of life scores than those with BPD-only or PTSD-only; and individuals with PTSD-only and the comorbidity had significantly lower physical health-related quality of life scores than those with BPD-only. Interactions between the BPD-PTSD group and gender were not significant for mental or physical health-related quality of life. In terms of what Pagura et al. refer to as BPD and PTSD "symptom severity," those in the comorbid BPD-PTSD group endorsed the highest number of BPD symptom items (M = 9.44, out of a total of 18 possible), followed by those in the BPD-only group (M = 8.08), and then the PTSD-only

group (*M* = 1.95). Similarly, those in the BPD-PTSD group endorsed the highest number of PTSD symptom items (*M* = 14.58, out of a total of 19 possible), followed by the PTSD-only group (*M* = 13.03), and then the BPD-only group (*M* = 5.42).

In analyses of reports of childhood traumatic events, Pagura et al. (2010) found several notable group differences. Individuals with BPD-only were significantly more likely to report having experienced sexual versus nonsexual trauma as compared to individuals with PTSD-only. Individuals with BPD-PTSD comorbidity were significantly more likely than individuals in either of the other groups to report having experienced repeated traumatic events in childhood. In terms of single-occurrence events, those with the comorbidity were: significantly more likely than those in the other two groups to have experienced a single episode of neglect, significantly more likely than those in the PTSD-only group to have experienced a sexual (versus nonsexual) trauma, and significantly more likely than those in the BPD-only group to have experienced a single episode of physical attack or abuse by someone other than a parent or caretaker. There was a significant interaction between gender and the PTSD-BPD group for this last childhood trauma variable, such that females in the comorbidity group were significantly more likely than those in the other two groups to report having experienced a single episode of physical attack or abuse by someone other than a parent or caretaker; and there were no significant differences among males.

Synthesizing their results, Pagura et al. (2010) emphasized the conclusion that individuals with comorbid BPD-PTSD shoulder a significantly greater burden of illness than individuals with either disorder alone. On the conceptual, diagnostic level, these authors concluded that, although the symptomatic and, perhaps, etiologic overlaps

between BPD and PTSD are substantial, their data clarify that the two disorders are not redundant. Rather, the differential impact of these disorders occurring alone versus in comorbid form argues for the importance of diagnosing both BPD and PTSD, as appropriate (i.e., when the appropriate criteria for each diagnosis are met). Additionally, Pagura et al. reasoned that the mismatch between their findings of significantly greater BPD and PTSD symptoms in the comorbid group and the findings of previous studies (e.g., Zlotnick et al., 2003; Heffernan & Cloitre, 2000) possibly reflects some of the differences between small, clinical samples and their large, community sample, particularly differences of disorder severity and statistical power.

Another potential reason for differences between previous findings and those of Pagura et al. (2010) lies in their use of the personality disorder (PD) diagnostic criteria. Pagura et al. (2010) utilized data from a large, representative community sample, affording themselves greater statistical power and, likely, a broader view of the complete spectrum of severity of psychopathology and functioning present in the population. However, in assessment of PD diagnoses, these authors employed diagnostic rules that do not line up with the current consensus among PD researchers and clinicians (e.g., Trull, Jahng, Tomko, Wood, & Sher, 2010). Most widely accepted PD diagnosis guidelines (e.g., the DSM-IV-TR; APA, 2000) stipulate that each criterion causes significant distress and/or impairment in order to count toward the diagnosis. The diagnostic data used by Pagura and colleagues, however, were compiled using guidelines that only required distress and/or impairment to be caused by one of the requisite criteria in order to achieve the diagnosis, thus resulting in potential over-diagnosis and prevalence rates that were considerably higher than those seen in other community studies (Trull et al., 2010). From

this perspective, the findings of Pagura et al. may not accurately characterize the diagnostic groups under study: Some individuals in the Pagura et al. comorbid group might otherwise fall into a PTSD-only group; and some in their BPD-only group would not have enough distress/impairment to meet criteria for inclusion in the analyses at all, falling into a 'neither disorder' group. The present study addresses this concern by employing the more conventional PD diagnostic rules, requiring endorsement of distress/impairment on each PD criterion in order for it to count toward the diagnosis, in further examination of BPD-PTSD comorbidity in the NESARC Wave 2 data collection.

PRESENT STUDY

The present study aims to further examine the risk factors and outcomes associated with BPD-PTSD comorbidity in a large, representative community sample. Similar to Pagura et al. (2010), the present study utilized data from Wave 2 of the *National Epidemiological Survey on Alcohol and Related Conditions* (NESARC; Grant et al., 2003; Grant & Kaplan, 2005) to examine those associations suggested by the theoretical and empirical work discussed above. Particularly, the present study focuses on potential associations between and among BPD-PTSD comorbidity and CSA, other trauma types, gender differences, healthcare usage behaviors, and mental and physical health-related functioning outcomes. Diverging from Pagura et al., the present study employed Trull et al.'s (2010) re-analysis of the NESARC PD data, which, in line with the predominant view on PD diagnosis, requires that each criterion be associated with distress/impairment in order to count toward the diagnosis. This change in diagnostic rules shifts the diagnostic group membership of a number of individuals formerly in the comorbid group (now in the PTSD-only group) and formerly in the BPD-only group (now in the mass of individuals with neither diagnosis, who were not included in the present study), thus changing the composition of all 3 of the diagnostic groups included in the present study. Examination of how much and in what ways this change would affect marked changes in the pattern of results from those of Pagura et al. (2010) was an important secondary aim of the present study. It was hypothesized that some discernable differences will emerge, and that these differences will better reflect the true character of

BPD-PTSD comorbidity in the general U.S. population. Given a review of the findings discussed above, the present study specifically hypothesized the following:

1) BPD-PTSD comorbidity will be associated with worse mental and physical health-related functioning outcomes than BPD or PTSD alone.
2) CSA will be associated with worse mental and physical health-related functioning outcomes than no-CSA, but this association will not be as robust as that with diagnostic status (BPD-PTSD comorbidity vs. BPD or PTSD alone).
3) BPD-PTSD comorbidity will be associated with greater and/or more intensive use of health care services (e.g., more ER visits, overnight hospitalizations) than either BPD or PTSD alone.
4) There may be a gender difference in the association of BPD with PTSD. Specifically, the odds ratios for BPD-PTSD comorbidity may be different for women and men; and multiple factors, including gender differences in CSA, may contribute to this gender difference in the comorbidity.
5) In assessment of trauma type prevalence rates, CSA will be more prevalent among both men and women with the comorbidity than among those with one of the two diagnoses; and among men and women with BPD-PTSD comorbidity, CSA will be among the most prevalent types of traumatic experiences.
6) Among people with CSA and one or both BPD and PTSD, parameters of abuse severity (younger age at onset of abuse, frequency of abuse) will be associated with the diagnosis of BPD-PTSD comorbidity.
7) Findings from the present study will generally replicate, but differ somewhat from, the findings reported by Pagura et al. (2010). Diagnosis prevalence and

comorbidity rates will differ based on revision of the BPD diagnostic rules in the present study. This change in numbers will also be accompanied by a change in severity, altering the composition of the diagnosis groups under consideration, and as such, may impact diagnostic group differences, e.g., in health-related functioning and in symptom count comparisons.

The NESARC Wave 2 data offer a particularly valuable opportunity for this investigation for reasons including a very large sample size (N = 34,653) and careful use of a representative community sample (Grant & Kaplan, 2005). With this large and representative community sample and an approach that borrows from the discipline of epidemiology, this study is well suited to address questions of risk, prevalence, and outcome in the general United States population, thus making an important contribution to the existing field of BPD and PTSD-focused studies, many of which have employed only clinical or treatment-seeking samples, samples composed of predominantly or only women, and very small sample sizes. Utilizing a community sample avoids biases introduced by the use of only participants who have sought out or been placed in treatment, for example, helping to avoiding the overrepresentation of women compared to men and the limited representation of only a portion of the population distribution of mental health impairment (Lenzenweger, 2008; Zimmerman, Chelminski, &Young, 2008).

METHOD

Epidemiological Survey

The *National Epidemiological Survey on Alcohol and Related Conditions* (NESARC), one of the largest epidemiological surveys assessing mental illness and its correlates to date, was a nationally representative face-to-face survey evaluating mental health in the non-institutionalized population of the United States (Trull, Jahng, Tomko, Wood, & Sher, 2010).

Wave 2, which included both BPD and PTSD assessments, was conducted in 2004-2005 with a sample of 34,653 completed interviews (Grant & Kaplan, 2005). Oversampling of African-Americans, Latinos, and young adults (age 18-24) was implemented. The data were weighted according to this oversampling and to reflect design characteristics of the survey. Adjustments were made for nonresponse across sociodemographic characteristics; and the weighted Wave 2 data were then adjusted—based on the 2000 Decennial Census—to represent the civilian population on sociodemographic variables including region, age, race, and gender (Grant et al., 2008).

Measures

DSM-IV Diagnoses. BPD was assessed on a lifetime basis using the Alcohol Use Disorder and Associated Disabilities Interview Schedule—DSM-IV Version (AUDADIS-IV; Grant et al., 2008). PTSD was assessed with regard to past-year and prior-to-past-year diagnoses, also using the AUDADIS-IV (Grant et al., 2008). The AUDADIS-IV is a fully structured diagnostic interview designed to assess alcohol, drug, and other mental disorders in both general and clinical populations according to DSM-IV

criteria (Grant, Dawson, & Hasin, 2001). To establish the pervasiveness that is a critical feature of BPD, and all other personality disorders, the AUDADIS-IV requires that personality disorder symptoms should occur "most of the time throughout your life, regardless of the situation and who you were with" (Pagura et al., 2010). Using a subsample of 1,899 respondents, fair to good test-retest and inter-rater reliability have been demonstrated for Wave 2 Axis I and II AUDADIS-IV diagnoses (Ruan et al., 2008). Kappas indicated fair to good agreement: For PTSD past-year and lifetime diagnoses, kappas were 0.77 and 0.64, respectively; and for BPD, 0.71. Internal consistency of symptom scales associated with BPD and PTSD fell within the good range (alpha = 0.75−0.89); and reliability of risk factor measures fell in the good-to-excellent range (intraclass correlations = 0.50−0.94; alpha = 0.64−0.90), further indicating the usefulness of the AUDADIS-IV diagnostic measures (Ruan et al., 2008).

The present study employs Trull et al.'s (2010) re-analysis of the NESARC PD data, which, in line with the predominant view on PD diagnosis, requires that each criterion be associated with distress/impairment in order to count toward the diagnosis. This re-analysis significantly reduces the PD prevalence rates, bringing them much more into line with recent epidemiological studies in the U.S. and Britain. It should, in turn, help to paint a clearer picture of the PDs, their correlates, and comorbidities actually present in the United States population.

Childhood Sexual Abuse. History of childhood sexual abuse (CSA) was assessed based on response to the NESARC interview item, "Were you ever sexually assaulted, molested, or raped, or did you ever experience unwanted sexual activity?" along with the following item about age at onset, such that endorsement of the former

together with an answer less than 16 (years of age) on the latter counted as positive endorsement of CSA. This age cut-off was chosen based on its previous use in the literature (see Pagura et al., 2010). Items assessing age at onset, frequency (number of times happened), and physical extent/severity of abuse (i.e., whether the reported experience of CSA involved inappropriate touching, attempted intercourse, or completed intercourse) will be utilized as parameters of CSA severity.

Other Traumatic Experiences. Other traumatic experiences assessed in the NESARC interview and utilized in the present study include those related to: military combat, military peacekeeping missions, civilian experience of war, experience as a refugee, life-threatening accident/illness, natural disaster, physical assault by parent/caretaker, neglect by parent/caretaker, witnessing serious fights at home, physical assault by spouse/romantic partner, physical assault by anyone else, kidnapping/being held hostage or as a POW, being stalked, being held up with a weapon, death/injury of someone close in a terrorist attack, injury (self) in a terrorist attack, direct/indirect experience of terrorist attack, witnessing a severe injury/death, unexpected death of someone close, and serious illness/injury/traumatic experience of someone close. Of these 'other traumatic experiences,' 4 are childhood experiences: physical abuse by a parent or caretaker, neglect by a parent or caretaker, witnessing violence in the home, and physical attack or abuse by someone other than a parent or caretaker. Thus, together with childhood sexual abuse, 5 total childhood traumatic event types were examined.

Physical and Mental Health-Related Functioning. The SF-12v2 Health Survey (Ware, Kosinski, Turner-Bowker, & Gandek, 2007) was employed to assess participants' physical and mental health-related functioning. Eight scales, 4 physical (General Health,

Physical Functioning, Role Physical, and Bodily Pain) and 4 mental (Mental Health, Social Functioning, Role Emotional, and Vitality), comprise this 12-item short-form health survey. The SF-12v2 has been used in a wide variety of studies—particularly those that monitor population health, compare and analyze disease burden, and/or predict medical expenses—as a measure of perceived physical and mental health and is variously referred to as a measure of overall health status, outcome, functioning, well-being, and/or health-related quality of life (QualityMetric, 2011). The standard version of the SF-12v2, used here (as opposed to the acute, one-week recall version), asks respondents to recall over the last 4 weeks. For example, "During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities like visiting with friends, relatives, and so forth?" Because the health-related functioning perspective and content of the SF-12v2 does not overlap much, if at all, with the diagnostic criteria of BPD and PTSD, its use as an outcome measure in the present study should not be invalidated by criterion contamination. Scoring was conducted using techniques described in the SF-12v2 user's manual (Ware, Kosinski, Turner, Bowker, & Gandek, 2002), resulting in norm-based scores with a standardized range (0 to 100) and mean (50), and thus facilitating comparisons across populations and studies.

Healthcare Usage. NESARC interview items assessing number of overnight hospitalizations, days spent in the hospital, and number of times treated in a hospital emergency room in the last 12 months all were used to examine aspects of general treatment usage.

Analyses

Data analyses were conducted in SAS 9.2 and Mplus. In order to make statistically valid population-based inferences, the Wave 2 stratification and weighting systems that were part of the *NESARC*'s complex survey design were incorporated into these analyses. In SAS 9.2, such complex survey sample analyses required use of procedures equipped specifically for incorporation of complex sampling design (i.e., PROC SURVEYMEANS, PROC SURVEYREG, PROC SURVEYFREQ). For example, in instances in which an analysis of variance (ANOVA) typically would be used to answer the research question, PROC SURVEYREG was used to answer the same questions about contributions to variance in outcomes, but with the capacity to incorporate the complex survey design into the analyses.

Descriptives. Descriptive analyses were conducted to characterize the groups employed throughout the analyses in this study (i.e., groups designated based on diagnoses, gender, and CSA status) in terms of diagnostic status, CSA prevalence, and basic sociodemographic characteristics, including age, gender, U.S. region, race/ethnicity, marital status, education, and household income. All variables used in these analyses were categorical except for *age*, which was grouped to be categorical in the present study based on the results of loess regressions. Both this decision to make *age* categorical and the cutoff points chosen for the age groups matched the decisions of prior NESARC studies (e.g., Pagura et al. , 2010). SAS proc SURVEYFREQ was used to calculate disorder (BPD-only, PTSD-only, and BPD-PTSD comorbidity) and CSA prevalence, sociodemographic characteristics, and cross tabulations of these variables. Additionally, Wald Chi-square tests were run within the SURVEYFREQ procedure to

test for independence of the row and column variables in the two-way tables crossing the diagnostic group variable (BPD-only vs. PTSD-only vs. Comorbid BPD-PTSD) with each of the sociodemographic variables, thus testing for whether the pattern of demographic characteristics across the diagnostic groups varied significantly from that which would be expected by chance.

In a large community sample, is BPD-PTSD comorbidity associated with significantly worse physical and mental health-related functioning than BPD or PTSD alone? To address Hypothesis 1, a series of SAS SURVEYREG procedures, which perform linear regression or ANOVA analyses for complex survey sample designs, was conducted, assessing the variance in scores on each SF-12v2 scale accounted for by diagnostic status (BPD-only, PTSD-only, or comorbid BPD-PTSD), by gender, and by their interaction. Separate analyses were run including different levels of the diagnostic status variable: one including all 3 levels (BPD-only, PTSD-only, and comorbid BPD-PTSD), one including just comorbid vs. BPD-only, and one including comorbid vs. PTSD-only. The SAS SURVEYMEANS procedure was also used to calculate the means for each of the SF-12v2 subscale scores across the different levels of the diagnostic status and gender.

Is history of CSA associated with worse physical and mental health-related functioning among people with one or both of these disorders? If related, how does this relationship between CSA and functioning compare to any relationship found between BPD-PTSD comorbidity and functioning? To address Hypothesis 2, a series of SAS SURVEYREG procedures was conducted in the same fashion as above (for Hypotheses 1). The simplest of these assessed the variance in SF-12v2 subscale scores

accounted for by CSA status (endorsement of history of CSA vs. none), by gender, and by their interaction. The rest replicated those conducted to address Hypothesis 1, but with the addition of CSA status as a predictor as well as the 3-way interaction among CSA status, gender, and diagnostic status, thus assessing the contributions of diagnostic status, gender, and CSA status in the same models. The SAS SURVEYMEANS procedure was also used to calculate the means for each of the SF-12v2 subscale scores across the 2 levels of CSA status (CSA and no-CSA).

Is BPD-PTSD comorbidity associated with different patterns of health care usage (e.g., more frequent overnight hospitalizations) than BPD or PTSD alone? To address Hypothesis 3, a series of SAS SURVEYREG procedures was conducted, in the same manner as above (for Hypotheses 1 and 2) but with number of overnight hospitalizations, days spent in the hospital, and number of times treated in a hospital emergency room in the last 12 months as the outcome variables, thus assessing the variance in these healthcare usage variables accounted for by diagnostic status (BPD-only, PTSD-only, or comorbid), by gender, by CSA status, and all 2- and 3-way interactions. The SAS SURVEYMEANS procedure was also used to calculate the means for each of the healthcare usage variables across the different levels of the diagnostic status, gender, and CSA status variables.

Is there a gender difference in the association of BPD with PTSD? If so, do other potentially predictive factors (e.g., CSA) contribute to this difference by operating differently across the genders? Specifically, is the odds ratio for BPD-PTSD comorbidity different for women and men; and if so, will logistic regression analyses indicate that gender differences in CSA as well as in other potential risk factors

contribute to this gender difference in the comorbidity? To address Hypothesis 4, a series of logistic regressions were conducted using the Mplus program to determine odds ratios for receiving a diagnosis of BPD-only, PTSD-only, or comorbid BPD-PTSD in this sample based on the potential risk factors of gender, CSA status, and age group. These logistic regressions were additionally run separately for women and men to examine whether the other risk factors had a different impact across the genders, and thus could be considered as contributing factors in any gender differences.

Is CSA one of the most prevalent types of trauma for people with this comorbidity? If not, which type(s) of trauma is/are most prevalent? Is this answer the same for men as for women? How do the answers for the men and women with BPD-PTSD comorbidity compare to those with BPD only and those with PTSD only? In addressing Hypothesis 5, SAS proc SURVEYFREQ was used to calculate prevalence rates for the different trauma types for each gender and diagnostic group. A Wald's Chi-square test was conducted using the SAS SURVEYFREQ procedure to test for significant difference in CSA prevalence across the diagnostic groups.

Among people with CSA and one or both of the disorders (BPD, PTSD, or both), are certain parameters of abuse severity associated with the BPD-PTSD comorbidity? To address Hypothesis 6, logistic regressions were conducted in Mplus to assess whether the parameters of age at onset and frequency of sexual assault/abuse were significantly associated with increased risk for receiving the comorbid BPD-PTSD diagnosis. These logistic regressions were additionally run separately for women and men to examine whether the other risk factors had a similar or different impact across the genders.

Do the findings of the present study differ slightly, as expected, from those of Pagura et al. (2010)? If so, how do they differ? Results of the analyses described above—particularly diagnosis prevalence rates and diagnostic group differences in health-related quality of life (i.e., SF-12v2 scores)—were examined in comparison to those published by Pagura et al. For example, Pagura et al. (2010) found that individuals with BPD-PTSD comorbidity had significantly lower mental health-related quality of life scores than those with BPD-only or PTSD-only; but their results for the physical health-related quality of life scores were slightly different: Individuals with the comorbidity and individuals with PTSD-only had significantly lower scores than those with BPD-only. Also, Pagura et al. (2010) found that interactions between the BPD-PTSD group and gender were not significant for mental or physical health-related quality of life. The present study hypothesized that the comorbid group would score significantly lower than both single-disorder groups on both the mental and physical health-related quality of life measures (SF-12v2 scores), and that some diagnostic group-by-gender interactions would be significant. Additionally, the present study replicated Pagura et al.'s analysis comparing BPD and PTSD symptom counts among the diagnostic groups (comorbid BPD-PTSD, BPD-only, and PTSD-only). Pagura et al. suggested that the discrepancy between their results and those of prior studies with regard to core disorder-specific symptoms arose from the differences between their large, community sample and the small, clinical samples used in prior studies. Using the same sample as Pagura et al. but more widely accepted diagnostic rules, the present study stands to help clarify this discrepancy. To attempt to replicate the symptom count analyses of Pagura et al., in the present study, SAS proc SURVEYMEANS was used to calculate mean number of BPD

and PTSD symptoms endorsed by each diagnostic group. For further clarity, these means were also calculated for number of BPD symptoms endorsed with distress/impairment; and the mean difference between number of BPD symptoms endorsed without and with distress/impairment was calculated across the diagnostic groups. (Although Pagura et al. do not explicitly clarify whether they required distress/impairment for the BPD symptoms that were counted in their symptom count analyses, it seems safe to assume—based on their use of diagnostic rules that downplay the importance of distress/impairment—that they only required endorsement of the symptom content for these analyses.) Additionally, in the present study, SAS proc SURVEYREGs were conducted to examine whether mean differences in the number of PTSD symptoms endorsed by individuals with PTSD-only and those with BPD-PTSD comorbidity were significant (i.e., whether comorbid BPD significantly contributed to the variance in PTSD symptom counts among those with PTSD). Likewise, proc SURVEYREGs were conducted to examine whether mean differences in BPD symptom counts (both with and without distress/impairment) were significantly accounted for by the distinction between having the BPD-only diagnosis versus the BPD-PTSD comorbidity.

RESULTS

Demographic characteristics. Results from the descriptive analyses of diagnostic status and sociodemographic characteristics are presented in Table 1. A total of 3074 individuals met criteria for the PTSD-only group, 483 met for the BPD-only group, and 547 for the comorbid group, comprising the full subsample of *NESARC* Wave 2 respondents ($N = 4104$) whose data were examined in the present study. These numbers indicate the following comorbidity rates: 53.11% of those who met criteria for BPD also met criteria for lifetime PTSD; and 14.69% of those who met for PTSD also met for BPD. Results of the Wald chi-square tests for independence of the row and column variables in the two-way tables crossing the diagnostic group variable (BPD-only vs. PTSD-only vs. Comorbid BPD-PTSD) with each of the sociodemographic variables (age group, gender, U.S. region, marital status, education level, household income, and race/ethnicity) indicated that the null hypothesis of independence should be rejected for the following demographic variables: age group ($\chi^2[6, n = 4104] = 122.21$, Wald $F^*[6, 3687] = 20.34$, $p < .0001$), gender ($\chi^2[2, n = 4104] = 47.47$, Wald $F^*[2, 3691] = 23.73$, $p < .0001$), marital status ($\chi^2[4, n = 4104] = 45.83$, Wald $F^*[4, 3689] = 11.45$, $p < .0001$), household income ($\chi^2[6, n = 4104] = 35.84$, Wald $F^*[6, 3687] = 5.97$, $p < .0001$), and race/ethnicity ($\chi^2[8, n = 4104] = 15.85$, Wald $F^*[8, 3685] = 1.98$, $p = .045$). Although the adjusted and unadjusted Wald *F* statistics were virtually the same for all tests, the adjusted statistics and corresponding significance levels are reported in the present study, as these have been shown to be a more stable test statistic, given that the number of sample clusters is large enough to preclude concerns about low power (e.g., Thomas &

Rao, 1984; as cited in SAS Institute Inc., 2011). Wald chi-square tests indicated that the null hypothesis of independence should be retained for the following: U.S. region of residence (p = .087) and level of education (p = .079). To summarize, these tests indicated that the distribution of age, gender, marital status, household income, and ethnicity/race across the BPD-only, PTSD-only, and comorbid groups varied significantly from that which would be expected by chance; whereas the distribution of U.S. region and education did not.

Specifically, looking at the descriptive statistics presented in Table 1—for example, comparing the percentages of representation of each diagnostic group within each sociodemographic group to the percentage breakdown of the diagnostic groups overall (i.e., 74.89% PTSD-only, 12.83% BPD-only, and 12.27% comorbid)—one can roughly see the nature of these sociodemographic differences among the diagnostic groups: Women tended to be overrepresented in the PTSD-only group and men in the BPD-only group. The BPD-only and comorbid groups tended to be overrepresented in the 'never married' category, whereas PTSD-only was overrepresented in the 'married or living with someone as if married' category. The PTSD-only group was overrepresented at the higher education (though the Wald's Chi-square test indicated that the overall group differences for education level were not significantly different from chance) and household income levels; whereas the comorbid group was overrepresented at the lowest levels of these 2 variables. And finally, in terms of race/ethnicity, PTSD-only was slightly underrepresented and the comorbidity was notably overrepresented in the American Indian group. Nearly the opposite was true for the Asian group, in which PTSD-only was overrepresented and the comorbidity was underrepresented. In the

Hispanic group, the comorbidity was slightly overrepresented as compared to the two single-disorder diagnoses.

Hypothesis 1: BPD-PTSD comorbidity will be associated with worse general functioning outcomes than BPD or PTSD alone.

Main effects of diagnostic group. Results from the series of SAS SURVEYREG procedures conducted to assess the variance in scores on each SF-12v2 scale accounted for by diagnostic status (BPD-only, PTSD-only, or comorbid), by gender, and by their interaction (reported in full in Tables 2-4) supported this hypothesis. The diagnostic group variable had a significant main effect ($p < .01$) on each of the 8 SF-12v2 scales when all 3 levels of the diagnostic status variable were included (BPD-only, PTSD-only, and comorbid; Table 2) and when PTSD-only was contrasted against the comorbid group (2 levels: PTSD-only vs. comorbid; Table 3). When BPD-only was contrasted against the comorbid group (2 levels: BPD-only vs. comorbid; Table 4), the diagnostic group variable had a significant main effect ($p < .01$) on each of the 8 SF-12v2 scales except for the Vitality scale ($p = .28$). Mean SF-12v2 scale scores calculated for each diagnostic group using the SAS SURVEYMEANS procedure (Table 5) helped to clarify these results: The comorbid group showed the lowest mean score on each of the 8 scales. Averaging the mean scores across all 8 scales yielded 41.08 for the comorbid group, 44.71 for the BPD-only group, and 46.64 for the PTSD-only group. Thus, the main effect of the diagnostic group variable on SF-12v2 outcomes can be characterized as an association between BPD-PTSD comorbidity and deficits in health-related life functioning that are significantly greater than those deficits seen in individuals with PTSD or BPD alone.

Main effects of gender. Across these 3- and 2-level analyses, a main effect of gender ($p < .01$) was seen on 3 scales: Social Functioning, Mental Health, and Vitality. Mean SF-12v2 scale scores calculated for each gender using the SAS SURVEYMEANS procedure (Table 6) helped to clarify these results: On each of the scales for which there was a significant main effect of gender, the women (n = 2,903) scored lower than the men (n = 1201), indicating that the main effect of gender seen here can be characterized as significantly greater deficits for women than for men in these diagnostic groups in health-related quality of life in the areas of Social Functioning, Mental Health, and Vitality. Looking across all of the SF-12v2 scale means, the women in this 3-diagnostic group subsample of NESARC Wave 2 respondents scored lower than the men in this subsample on 5 of the 8 scales (all but General Health, Role Physical Functioning, and Bodily Pain). The mean score for all men averaged across all 8 scales was 45.89; whereas the mean score for all women across all 8 scales was 45.62, suggesting a somewhat inconsistent and weak (small in magnitude and only significant for 3 scales) trend for the women to score lower on the SF-12v2 scales than the men. The inconsistencies (i.e., scales on which men, rather than women, scored lower) and small magnitude of this gender trend distinguish it from that seen across the whole NESARC Wave 2 sample, in which women scored consistently lower on all 8 scales of the SF-12v2, and the overall means for women (n = 20089) and men (n = 14564) were, respectively, 49.98 and 51.75. Although not addressed directly in the present study, this apparent difference between the diagnostic groups analyzed here and the whole Wave 2 sample suggests that these disorders may have more of an impact on the health-related functioning for men than for

women. Questions regarding whether this is true and what other factors may be involved remain to be examined.

Diagnostic group-by-gender interactions. In the 3-level analyses, one diagnostic group-by-gender interaction approached significance on the Social Functioning scale ($p = .056$). In the analyses comparing PTSD-only with the comorbidity, significant diagnostic group-by-gender interactions emerged on the Social Functioning ($p = .022$) and Mental Health ($p = .037$) scales. No significant interactions emerged in the analyses comparing BPD-only with the comorbidity. Mean SF-12v2 scale scores calculated using the SAS SURVEYMEANS procedure (Table 7) helped to clarify these results: On each of the scales for which there were significant or marginally significant interactions, the association of lower functioning scores with the comorbidity, as compared to PTSD-only or both PTSD- and BPD-only, was greater for the women than for the men. That is, whereas the direction of the main effect of diagnostic group was the same for both genders, the magnitude of the mean Social Functioning and Mental Health score difference for men across the diagnostic groups was less pronounced than that for women, particularly when contrasting the PTSD-only men and women with the comorbid men and women (see Figures 1 and 2).

Hypothesis 2: CSA will be associated with worse mental and physical health-related functioning outcomes than no-CSA, but these associations will not be as robust as those with diagnostic status (BPD-PTSD comorbidity vs. BPD or PTSD alone).

Results from the series of SAS SURVEYREG procedures conducted to assess the variance in scores on each SF-12v2 scale accounted for by CSA status, along with gender, diagnostic status, and all 2- and 3-way interactions (reported in Tables 8 and 9)

supported this hypothesis. In those analyses including just the CSA status variable and gender as predictors (Table 8), CSA had a significant main effect ($p < .01$) on each of the 8 SF-12v2 scales, a significant main effect of gender ($p < .05$) emerged for all scales, and no significant interactions between CSA status and gender occurred. When CSA status was entered into the models with diagnostic status (3 levels: Comorbid, BPD-only, and PTSD-only) and all 2- and 3-way interactions (among diagnosis, CSA, and gender), the main effect of CSA status no longer emerged on any of the SF-12v2 scales; the diagnostic group variable maintained a significant main effect ($p < .01$) on all scales; gender had a significant main effect ($p < .01$) on the Mental Health and Vitality scales; and the interaction of CSA and diagnostic group approached significance ($p = .053$) on the Vitality scale (Table 9; Figure 3). Mean SF-12v2 subscale scores calculated using the SAS SURVEYMEANS procedure (Table 10) helped to clarify these results: The mean score for those who reported CSA was lower than that for the no-CSA group on each of the 8 scales. Averaging the mean scores across all 8 scales yielded 47.80 for the CSA group and 51.04 for the no-CSA group, suggesting that where the main effect of CSA did emerge, it reflected an association between history of CSA and lower health-related functioning. Mean Vitality scale scores calculated separately for those with and without CSA within each diagnostic group (Table 11, Figure 3) helped to clarify the marginally significant diagnostic group-by-CSA interaction on this scale. As seen in the plot in Figure 3, those who reported CSA showed an even greater association between diagnostic status and deficits in Vitality scores than did those who did not report CSA.

Hypothesis 3: BPD-PTSD comorbidity will be associated with greater and/or more intensive use of health care services than either BPD or PTSD alone.

Results from the series of SAS SURVEYREG procedures conducted to assess the variance in these healthcare usage variables (number of overnight hospitalizations, days spent in hospital, and number of times treated in a hospital emergency room in the last 12 months) accounted for by diagnostic status (BPD-only, PTSD-only, or comorbid), by gender, by CSA status, and all 2- and 3-way interactions largely supported this hypothesis. Diagnostic status had a significant main effect on both *number of days spent in hospital*, $F(2, 3664) = 3.07$, $p = .046$, and *times treated in ER*, $F(2, 3663) = 6.19$, $p < .01$, and a marginally significant effect on *overnight hospitalizations*, $F(2, 3665) = 2.70$, $p = .068$. Gender also had a marginally significant main effect on *times treated in ER*, $F(1, 3663) = 3.36$, $p = .067$. No other main effects, nor any interactions, achieved significance. Means for each of the 3 healthcare usage variables, calculated using the SAS SURVEYMEANS procedure, helped to clarify these results: each of the means for women ($M_{overnight} = .209$, $SE = .006$; $M_{days} = .944$, $SE = .045$; $M_{ER} = .404$, $SE = .010$) were greater than those for men ($M_{overnight} = .186$, $SE = .008$; $M_{days} = .868$, $SE = .051$; $M_{ER} = .341$, $SE = .009$); and each of the means for the comorbid group ($M_{overnight} = .448$, $SE = .060$; $M_{days} = 2.752$, $SE = .563$; $M_{ER} = 1.188$, $SE = .132$) were greater than those for either of the single-disorder groups (BPD-only: $M_{overnight} = .360$, $SE = .080$; $M_{days} = 1.963$, $SE = .355$; $M_{ER} = .753$, $SE = .089$; PTSD-only: $M_{overnight} = .298$, $SE = .020$; $M_{days} = 1.538$, $SE = .158$; $M_{ER} = .653$, $SE = .035$).

Hypothesis 4: There may be a gender difference in the association of BPD with PTSD. Specifically, the odds ratios for BPD-PTSD comorbidity may be different for women and men; and multiple factors, including gender differences in CSA, may contribute to this gender difference in the comorbidity.

Results from the logistic regressions of diagnostic status on CSA status, age group, and gender, generally supported this hypothesis. Logistic regression odds ratios examining the prediction of diagnostic group based on gender, CSA status, and age group indicated that these predictors did have differential associations with diagnostic status, that some gender differences occurred in the prediction, and that some predictors were operating differently across the genders. Specifically, those who reported history of CSA (vs. those who did not report CSA) had significantly greater odds (more than 2 times the odds) of being in the comorbid group than in either the BPD-only (*OR* = 2.259, 99% CI [1.373, 3.717]) or the PTSD-only group (*OR* = 2.359, 99% CI [1.681, 3.310]). Those in the older age groups (as compared to the younger age groups) had significantly greater odds of being in the PTSD-only group than in the comorbid group (*OR* = 1.493, 99% CI [1.255, 1.776]) or in the BPD-only group (*OR* = 1.664, 99% CI [1.399, 1.979]). Being a woman significantly increased the odds of being in the PTSD-only group as compared to the BPD-only group (*OR* = 2.549, 99% CI [1.791, 3.628]) or the comorbid group (*OR* = 1.580, 99% CI [1.124, 2.222]), and being a woman increased the odds of being in the comorbid group as compared to the BPD-only group (*OR* = 1.613, 99% CI [1.044, 2.493]). Conversely, being a man (as opposed to a woman) significantly increased the odds of being in the BPD-only group as compared to the comorbid group (*OR* = 1.613, 99% CI [1.044, 2.493]), significantly increased the odds of being in the BPD-only group as compared to the PTSD-only group (*OR* = 2.549, 99% CI [1.791, 3.628]), and significantly increased the odds of being in the comorbid group as compared to the PTSD-only group (*OR* = 1.580, 99% CI [1.124, 2.222]).

Conducting these logistic regressions separately for women and men helped to clarify whether the risk factors had a similar or different impact across the genders. The pattern of significant odds ratios was very similar across the genders, with only 2 notable differences: (1) Among women, older age conferred slightly but significantly greater odds of being in the comorbid group than in the BPD-only group (*OR* = 1.236, 95% CI [1.013, 1.509]); whereas in men, this older age-comorbidity association was not indicated (*OR* = 1.005, 95% CI [0.756, 1.335]). (2) For men with CSA, the odds ratio for having the comorbid diagnosis versus PTSD-only was even greater (approximately 3-fold: *OR* = 3.176, 99% CI [1.366, 7.383]) than that for women with CSA (approximately 2-fold: *OR* = 2.207, 99% CI [1.507, 3.231]).

Hypothesis 5: In assessment of trauma type prevalence rates, CSA will be more prevalent among both men and women with the comorbidity than among those with one of the two diagnoses; and CSA will be one of the most prevalent types of trauma among individuals with the comorbidity.

Calculation of the prevalence rates of different traumatic experiences across the diagnostic groups and genders in this study, using the SAS SURVEYFREQ procedure, largely supported this hypothesis. 35.83% (196 out of 547) of those in the comorbid group, 18.43% (89 out of 483) of those in the BPD-only group, and 19.52% (600 out of 3074) of those in the PTSD-only group reported CSA. Whereas the CSA prevalence rate for women was slightly more than double that for men in the comorbid group (43.42% vs. 19.14%), the gender difference was closer to 4-fold in the BPD-only (28.24% vs. 7.62%) and PTSD-only (24.33% vs. 6.51%) groups. For both women and men, the comorbid group had close to double the CSA prevalence rate seen in either the BPD-only

or the PTSD-only group. A Wald's Chi-square test conducted using the SAS SURVEYFREQ procedure indicated that the difference in CSA prevalence across the diagnostic groups was significant (χ^2[2, n = 4082] = 35.7594, Wald F*[2, 3669] = 17.87, $p < .0001$). Looking at the full range of different traumatic experience prevalence rates (Table 12), CSA was the 6th most prevalent traumatic experience, out of 23 different types of traumatic experiences, in the comorbid group. In the BPD-only group, CSA ranked 10th; and in the PTSD-only group CSA ranked 9th most prevalent. All three groups shared the same top 2 most prevalent traumatic experiences: ranking 1st was the unexpected death of someone close (comorbid group: 70.57%, BPD-only group: 55.69%, and PTSD-only group: 63.01%), and 2nd was the experience of seeing someone badly injured or killed (comorbid group: 46.98%, BPD-only group: 33.95%, and PTSD-only group: 37.35%).

Hypothesis 6: Among people with CSA and one or both BPD and PTSD, parameters of abuse severity (younger age at onset of abuse, frequency of abuse) will be associated with the diagnosis of BPD-PTSD comorbidity.

Results of the logistic regressions conducted to address this hypothesis were not supportive of the notion that younger age of onset or frequency of abuse/assault significantly predict diagnosis of BPD-PTSD comorbidity vs. PTSD-only vs. BPD-only. No odds ratios based on these CSA severity parameters achieved significance, whether men and women were analyzed together or separately. One interesting result, however, did emerge: in the logistic regressions in which women and men were analyzed together, being a woman increased the odds of having a PTSD-only diagnosis as compared to a comorbid diagnosis (*OR* = 2.460, 99% CI [1.052, 5.752]) or a BPD-only diagnosis (*OR* =

3.699, 99% CI [1.446, 9.463]). This replicated the result described above (Hypothesis 4; Table 9) for the full sample, i.e., not only those men and women who reported CSA. So, among those reporting CSA, the same gender difference was indicated in the prediction of PTSD-only vs. comorbidity and vs. BPD-only as was indicated in the broader sample (i.e., all men and women with at least one of the two disorders).

Hypothesis 7: Findings from the present study will generally replicate, but differ somewhat from, the findings reported by Pagura et al. (2010). Diagnosis prevalence and comorbidity rates will differ based on revision of the BPD diagnostic rules in the present study. This change in numbers will also be accompanied by a change in severity, altering the composition of the diagnosis groups under consideration, and as such, may impact diagnostic group differences, e.g., in health-related functioning and in symptom count comparisons.

Results of the analyses described above were examined in comparison to those published by Pagura et al. (2010).

Differences in prevalence rates. As expected based on the use of more stringent rules for BPD diagnosis (i.e., requiring more distress/impairment, or arguably, greater severity) in the present study, the numbers and composition of the diagnostic groups under study did change. The tightening of the BPD criteria did, in fact, reduce numbers in the BPD-only group and comorbid groups, and increased the number in the PTSD-only group (as this group gained those who were previously in the comorbid group but lost their BPD diagnosis with the revision in diagnostic rules). Specifically, the number of individuals in the BPD-only group shifted from 1290 in Pagura et al.'s findings to 483 in the present study; the number of those in the PTSD-only group shifted from 1820 to

3074; and the number of those in the comorbid group shifted from 643 to 547. Accordingly, in the present study, as compared to Pagura et al., those diagnosed with BPD had a higher rate of comorbidity with PTSD, and those diagnosed with PTSD had a lower rate of comorbidity with BPD. Pagura et al. (2010) found that 30.2% of individuals diagnosed with BPD were also diagnosed with PTSD; and 24.2% of individuals diagnosed with PTSD were also diagnosed with BPD. Using the same *NESARC* Wave 2 sample, the present study found the following: 53.11% of those who met criteria for BPD also met criteria for lifetime PTSD; and 14.69% of those who met for PTSD also met for BPD.

With regard to sociodemographic group differences, Pagura et al. (2010) found significant relationships between each of the sociodemographic variables and the diagnostic group variable; whereas the present study found significant relationships for all but education level and U.S. region (Pagura et al. did not report on U.S. region). Looking at the specific prevalence of each diagnostic group within each sociodemographic group (Table 1) indicated largely the same patterns (described above) in the present study as reported in the findings of Pagura et al., including in the case of education level, despite the relationship between education level and diagnostic group not achieving significance the present study. A few differences in these sociodemographic differences did, however, appear: Pagura et al. reported that individuals with PTSD alone were most likely to be married, while those with BPD alone were most likely to have never been married, and those with the comorbidity were most likely to have been widowed, separated or divorced. In the present study, prevalence rates indicated that both the BPD-only group and the comorbid group were most overrepresented in the

'never married' group. The comorbid group was also slightly overrepresented in the 'widowed, divorced, or separated' group, but they were more so in the 'never married' group. Lastly, Pagura et al. reported that more individuals with PTSD were Caucasian, while higher proportions of those with BPD and those with the comorbidity fell into the other race/ethnicity categories. The patterns for race/ethnicity found in the present study and described above were more nuanced than this and did not include a notable trend for the PTSD-only group to be overrepresented in the Caucasian category, as compared to the other diagnostic groups. It is unclear whether Pagura et al. made this race/ethnicity comparison based on the 3 mutually exclusive diagnostic groups (BPD-only, PTSD-only, BPD-PTSD comorbidity), which were used in the present study, or whether they were allowing overlap between the comorbid group and each of the single-disorder groups. Thus, this last difference may relate to not only the different composition of the diagnostic groups in the present study, but also to a slight difference in the approach of this particular analysis.

Differences in health-related quality of life (SF-12v2) findings. In analyses assessing health-related quality of life, Pagura and colleagues found that individuals with the comorbidity had significantly lower *mental* health-related quality of life scores than those with BPD-only or PTSD-only. For *physical* health-related quality of life (the other half of the SF-12v2 scales), their findings showed a slightly different pattern: Individuals with PTSD-only and the comorbidity had significantly lower than those with BPD-only. In other words, in the findings of Pagura et al., the comorbid diagnosis was not associated with significantly lower scores than PTSD-only on the *physical* health-related quality of life scales. As hypothesized in the present study (H1, H7), results from the SF-12v2

analyses described above upheld this diagnostic group difference in *mental* health-related quality of life scores, and diverged from the results of Pagura et al. with regard to the *physical* scales. The present study, indeed, found that the comorbid group had significantly lower SF-12v2 scores than either single disorder group on both the *mental* and *physical* scales. Although differences in SF-12v2 scores between the two single disorder groups were not directly tested in the present study, inspecting the mean SF-12v2 scale scores for each diagnostic group, moreover, indicates further distinction from the findings of Pagura et al.: Rather than the comorbid and PTSD-only groups showing lower functioning than the BPD-only group, the mean scores in the present study would place the PTSD-only group at a higher level of functioning than the BPD-only group, which was, in turn, at a higher level of functioning than the comorbid group. The more stringent diagnostic criteria implemented for BPD in the present study, as well as the use of lifetime, as opposed to past-year, diagnosis of PTSD most likely contributed to, if not caused, this partial inversion in the rank order of the means as well as the significant main effect in the SURVEYREG analyses comparing PTSD-only with the comorbidity in terms of the *physical* SF-12v2 scales.

Additionally, the results of Pagura et al. (2010) indicated that interactions between the BPD-PTSD group and gender were not significant for mental or physical health-related quality of life. The present study hypothesized that some diagnostic group-by-gender interactions would be significant, and in fact found a diagnostic group-by-gender interaction that approached significance on the Social Functioning scale ($p = .056$) when comparing all three diagnostic groups (comorbid vs. PTSD-only vs. BPD-only); and when comparing PTSD-only vs. the comorbidity, found significant diagnostic group-

by-gender interactions on the Social Functioning (p = .022) and Mental Health (p = .037) scales. These interactions and the associated means for these diagnostic group-by-gender subgroups, as discussed above and illustrated in Figures 1 and 2, suggested that the BPD-PTSD comorbidity has a greater detrimental impact on the Social Functioning and Mental Health scale scores of women than for men—a finding not present in Pagura et al.

Symptom count analyses. In the study by Pagura et al. (2010), those in the comorbid BPD-PTSD group endorsed the highest number of BPD symptom items (M = 9.44, out of a total of 18 possible), followed by those in the BPD-only group (M = 8.08), and then the PTSD-only group (M = 1.95). Similarly, those in the BPD-PTSD group endorsed the highest number of PTSD symptom items (M = 14.58, out of a total of 19 possible), followed by the PTSD-only group (M = 13.03), and then the BPD-only group (M = 5.42). Roughly following the approach used by Pagura et al. (2010) in their "symptom severity" analyses, mean numbers of BPD and PTSD items endorsed on the were calculated for each diagnostic group using the SAS SURVEYMEANS procedure (Table 11). In the present study, two separate counts were calculated for BPD: One count, "BPD symptoms," required endorsement of distress/impairment in order for endorsement of the BPD item to count. The other count, "BPD items," merely required endorsement of the item with or without distress/impairment also being endorsed for that item. Those in the comorbid BPD-PTSD group endorsed the highest number of BPD symptoms and BPD items (M = 8.98 and M = 10.40, respectively, out of a total of 18 possible), followed by those in the BPD-only group (M = 8.09, M = *9.49*), and then the PTSD-only group (M = 1.05, M = *2.74*). Similarly, those in the comorbid group endorsed the highest number of PTSD symptom items (M = 14.50, out of a total of 19

possible), followed by the PTSD-only group (*M* = 12.85), and then the BPD-only group (*M* = 5.95). The mean difference between the number of BPD symptoms and BPD items endorsed ("BPD symptom-item difference") was similar for all groups (PTSD-only: *M* = 1.68, BPD-only: *M* = 1.40, comorbid: *M* = 1.41). SAS SURVEYREG procedures conducted to test the significance of the differences (Table 12) between the comorbid and the BPD-only groups in terms of the BPD symptom and item counts indicated that these differences were, in fact, significant ($p < .0001$), as was the difference in PTSD symptoms between the comorbid and PTSD-only groups ($p < .0001$).

DISCUSSION

Overall, the results of the present study supported many but not all of the original hypotheses. An association was found between BPD-PTSD comorbidity and lower general functioning, or health-related quality of life, than seen in BPD alone or PTSD alone. This finding generally echoes the functioning findings presented in previous clinical studies (e.g., Zlotnick et al., 2003; Heffernan & Cloitre, 2000) and community studies (e.g., Pagura et al., 2010). Also as hypothesized, childhood sexual abuse (CSA) showed an association with lower health-related general functioning levels, and this association was not as robust as that between the comorbid diagnosis and functioning. These results suggest that CSA may, in fact, have a lasting impact on the lives of individuals with BPD, PTSD, or both; but this impact pales in comparison to the impact of these disorders in their own right. In other words, the deficits in health-related quality of life, associated with the BPD-PTSD comorbidity cannot be explained away based on this, albeit very impactful, childhood trauma.

Several potentially important interactions also emerged in the health-related functioning (SF-12v2) analyses in the present study. Though no causality can be inferred from these analyses, the pattern of interaction results found here does suggest that, in the areas of Social Functioning and Mental Health, women with BPD-PTSD comorbidity may suffer the consequences of the association between this comorbidity and deficits in health-related quality of life to a greater extent than do the men with this comorbidity. Whereas being in the 3-diagnostic group subsample analyzed in the present study verses in the general NESARC Wave 2 sample seems to make a greater difference for men's

functioning than for women's (as discussed above and seen in the gender difference in mean SF-12v2 scores for the general Wave 2 sample vs. the diagnostic group subsample analyzed here), the distinction among the 3 diagnostic groups—particularly between the PTSD-only group and the comorbid group—appears to make a greater difference for women. Additionally, those who reported CSA showed an even greater association between diagnostic status and deficits in Vitality scores than did those who did not report CSA, suggesting that those with history of CSA and BPD-PTSD comorbidity may be particularly at risk for deficits in this particular area of health-related quality of life. Thus, in treatment or research on the topic of health-related functioning, it may be of particular importance to carefully assess for and diagnose BPD-PTSD comorbidity (i.e., not just one of the 2 when both may be present) in women and in those who report history of CSA. Likewise, among those diagnosed with this comorbidity, being female and/or reporting history of CSA may be important factors to take into account when considering risk for deficits in health-related functioning. The results of the present study clearly support the importance of carefully assessing for and diagnosing BPD-PTSD comorbidity in any individual or (sub)sample, but consideration of particular risk associated with gender and CSA status could be instructive in future research and treatment policy/planning.

In the present study, BPD-PTSD comorbidity was also associated with patterns of greater healthcare usage, namely more days spent in the hospital, greater frequency of treatment in a hospital ER, and (though marginally significant) a trend toward more overnight hospitalizations, echoing findings of previous clinical studies (e.g., Zlotnick et al., 2003; Heffernan & Cloitre, 2000) comparing much smaller patient samples.

Examination of gender, CSA status, and age group as predictors of diagnosis of BPD-only, PTSD-only, and BPD-PTSD comorbidity generally supported the hypothesis that there would be gender differences in the association of BPD with PTSD and presented some additional important and interesting results across both genders. The finding that those who reported history of CSA had significantly greater odds of being in the comorbid group than either the BPD-only or the PTSD-only group, provides empirical, general population-based support for the focus on CSA as a potential precursor to BPD-PTSD comorbidity, as traditionally has been seen in much of the theoretical and some of the clinical literature on the topic of this comorbidity. The finding that those in the older age groups had significantly greater odds of being in the PTSD-only group than in the comorbid group or in the BPD-only group mirrored the findings of Pagura et al. (2010) and, arguably, calls attention to the need for further examination of BPD, PTSD, and their comorbidity from a developmental lifespan perspective. Some researchers have begun to look at the stability of the BPD diagnosis and changes in symptomatology later in life, for example examining the possibility that changes related to personality later in life (e.g., less clinically significant impulsivity) place some of those who once met criteria for BPD outside of the diagnosis and into a subthreshold and/or qualitatively distinct category (for a review of relevant findings, see Oltmanns & Balsis, 2011). In their review of current findings on the trajectory of personality pathology in later life, Oltmanns and Balsis report that the most consistently (across studies) stable symptoms of BPD across the lifespan seem to be problems with managing anger, low mood, and interpersonal difficulties related to fear of abandonment. Least stable symptoms, or those showing the most improvement over time, include self-harm and suicidal behaviors

(Oltmanns & Balsis, 2011). Oltmanns and Balsis also point out that changing life contexts (e.g., demands) in later life, and thus changes in the impact of personality disorders (PDs), mean that not only the changing symptomatology, but also how these changes relate to other aspects of adjustment in later life, need be considered in order to understand changes in PD pathology across the lifespan. Given the importance of age as a predictor of disorder in the present study and its importance in the trajectory and impact of BPD (e.g., see Oltmanns & Balsis, 2011), further examination of what such age-related trends mean, if anything, for the characteristics of BPD-PTSD comorbidity later in life may be an important future direction in the study of this comorbidity.

Although the findings were not as straight-forward and uniform as hypothesized—that is, being a woman did not increase the odds of being in the comorbid group vs. the either of the single disorder groups—gender differences in the prediction of diagnostic group did emerge. Being a woman significantly increased the odds of being in the PTSD-only group as compared to the BPD-only group or the comorbid group; and being a woman increased the odds of being in the comorbid group as compared to the BPD-only group. This pattern of results suggests the possibility that the gender difference in PTSD prevalence (i.e., the fact that whereas the gender breakdown of the BPD group was close to even, there were many more women in the PTSD group than men) may drive the gender difference in the comorbidity as compared to BPD-only.

Looking at the prediction of diagnostic status separately for women and men, produced similar patterns of prediction save for 2 interesting discrepancies: (1) The association between older age and the comorbidity vs. BPD-only (another difference that may be driven by the demographic differences between PTSD and BPD) was only

significant for women. Such a finding reiterates the notion that examination of the developmental factors at play may help to further elucidate this comorbidity. (2) Among those with CSA, the odds of having the comorbidity were significantly greater than the odds of PTSD-only for both genders, but this difference was approximately 3-fold for men and 2-fold for women. Though such a conclusion would require further investigation, this finding suggests that CSA may be an even more specific predictor of BPD-PTSD comorbidity among men than among women. Interestingly, this gender difference was not reported by Pagura et al. (2010). The only gender difference in diagnosis group odds ratio reported by these authors was the finding that, among women, those with the comorbidity were significantly more likely to report experience of a single episode (but not repeated episodes) of physical attack or abuse in childhood relative to those with PTSD-only or BPD-only; and among men, there was no significant difference (Pagura et al., 2010). This contrast with Pagura et al. and the potential importance (e.g., implications for future directions in treatment/intervention and research) of the present study's finding that CSA may be a greater risk factor for BPD-PTSD comorbidity among men than women (though a significant risk factor for both) highlight the need for further investigation of gender differences in the association of BPD-PTSD comorbidity with CSA and other potential etiological factors.

That CSA was in the upper range of reported traumas (ranking 6th out of 23) in the comorbid group, when a broad array of 23 different types of traumatic experiences were compared, further supports notion of CSA as a predictor of BPD-PTSD comorbidity. Still, 6th out of 23 is not quite even in the top quarter. This could result at least partly from the relative inevitability of some of the top-most reported traumatic

experiences—for example, once someone has reached adulthood, the unexpected death of a close loved one is a relatively very common experience regardless of any conceivable differences among individuals. Nevertheless, the ranking of 5 other types of traumatic experiences above CSA in the comorbid group serves to remind us that this specific type of trauma cannot be the whole story, especially on the individual level. Other types of trauma and other types of predictive factors will likely continue to be an important part of the broader picture of BPD-PTSD comorbidity as we continue to learn more about it.

In the present study, comparing the trauma type prevalence rates across diagnostic groups and breaking down the prevalence rates by gender was, perhaps, more instructive than rank ordering. The comorbid group had more than double the prevalence rate of CSA than that seen in either of the single-disorder groups. And, lining up with the suggestion made above that CSA may be a more specific predictor of BPD-PTSD comorbidity for men than for women, the percentage of comorbid men (19.14%) who reported CSA was greater than that of BPD-only men (7.62%) or PTSD-only men (6.51%) who reported CSA; and the difference for women across the diagnostic groups was less pronounced (43.42%, 28.24%, and 24.33%, respectively). Accordingly, the gender disparity in CSA prevalence rate was much smaller in the comorbid group (19.14% of men vs. 43.42% of women) than in either of the single-disorder groups (BPD-only: 7.62% vs. 28.24% and PTSD-only: 6.51% vs. 24.33%).

Interestingly, results from the present study did not support the importance of the CSA severity parameters of age at onset and frequency of abuse/assault in the prediction of BPD-only vs. PTSD-only vs. the comorbidity of these two disorders. However, this finding does not necessarily provide evidence for the non-importance of such severity

parameters and deserves at least 2 important qualifications: (1) The available data allowed for examination of only two parameters, *frequency* and *age of onset*. There exists no known empirical or theoretic justification for the importance if these two parameters over any others (e.g., relationship with abuser); these were simply the only ones that the data provided. Moreover, the *frequency* measured here did not distinguish between instances of CSA and instances of sexual abuse/assault throughout the lifespan. Thus, in cases in which individuals who reported CSA also experienced sexual abuse/assault as adults, these adult experiences were included in their *frequency* counts, rendering the use of this frequency as a "CSA severity parameter" less than ideal. (2) The importance of severity parameters in the prediction of BPD-only vs. PTSD-only vs. the comorbidity should not be confused with the potential importance of CSA severity parameters in the prediction of disorder vs. no disorder, or the prediction any of the disorders examined in the present study vs. disorders not examined here, or the prediction general functioning outcomes (e.g., health-related quality of life), etc.. None of these other outcomes were examined in the preliminary CSA severity parameter analyses in the present study; as such, they offer potential directions for further examination, both within this data set and in others.

As described above, most differences between the findings of the present study and those of Pagura et al. (2010)—including the slight but important differences in the results of health-related quality of life findings—could be attributed largely to the difference in personality disorder diagnostic rules between the 2 studies. It intuitively makes sense that outcomes would change in many of the ways that they did, given the raising of the bar for BPD diagnosis. In the present study, requiring distress/impairment

on each BPD criterion in order for it to count toward the diagnosis arguably increased the severity of both the BPD-only group and the comorbid group, as well as changing the PTSD-only group as it absorbed all those who were previously in the comorbid group but lost their BPD diagnosis due to lack of distress/impairment on some or all of the BPD criteria. While the differences between the findings of Pagura et al. and the present study do, in fact, reflect increased severity in the BPD-only and comorbid groups, the difference in the composition of the PTSD-only group between the two studies is less readily observable or interpretable, suggesting that individuals with PTSD and some relatively mild BPD features may be more similar than different from those with PTSD and no or few BPD features.

The present study replicated Pagura et al.'s analysis comparing BPD and PTSD symptom counts among the diagnostic groups (comorbid BPD-PTSD, BPD-only, and PTSD-only), aiming to help clarify the discrepancy between their results and those of prior studies with regard to core disorder-specific symptoms. Pagura et al. concluded that their findings indicative of an impact of BPD-PTSD comorbidity on central features of each individual disorder—something examined and not found in previous clinical studies—arose from the differences between their large, community sample and the small, clinical samples used in prior studies. Using the same sample as Pagura et al. but more widely accepted diagnostic rules, the present study did, in fact, replicate the findings of Pagura et al., although the difference between the comorbid and BPD-only group was of a notably smaller magnitude in the present study. Thus, the findings of the present study generally support the notion that the impact of the comorbidity on core features of the single disorders seen in the *NESARC* Wave 2 sample may speak to the

differences between a large, community sample and those smaller clinical samples that preceded it. At the very least, the present findings leave open the possibility of this conclusion. Future examination across these two different types of samples, employing the same measures of core symptom severity or core symptom counts will be necessary to more firmly support such a conclusion.

Some broader methodological limitations also merit consideration in the interpretation of results from the present study. Partial reliance on retrospective self-report (most notably in the reporting of trauma experiences), as always, must be regarded with caution. In this investigation, the biases and inaccuracies typical of autobiographical recall and self-report (arguably including additional concerns related to the reporting of traumatic experiences) may be magnified by the lens of BPD. That is, with its characteristic distorted perceptions of adult relationships, BPD may contribute some additional inaccuracy to some participants' retrospective self-reports. However, to the knowledge of myself and colleagues, there exists no empirical evidence of this effect; whereas there is some evidence to support the accuracy of BPD patients' reports of childhood experiences, for example via sibling concordance (Paris, 1994). Additionally, some have criticized the *NESARC* for its use of PD interview items that were not drawn directly from a traditional, validated Axis II diagnostic instrument (e.g., Lenzenweger, 2008). However, no single PD interview has been shown to be superior to others; and these other interviews have been shown to disagree with each other at times (Trull et al., 2010). Thus, although notable, it is not clear whether this is actually a significant limitation. Lastly, diverging from the more pure epidemiological approach of isolating disorders under investigation via statistical controls (e.g., for sociodemographics and

other diagnoses), the present study conducted analyses without additional statistical controls. At no point in the present study were other comorbid Axis I or Axis II diagnoses taken into account or statistically controlled. There are myriad pros and cons for controlling for such other factors or not. Coming from a hybrid clinical-epidemiological perspective, the present study adopted the stance that overly controlling for other factors could reduce the meaning of results such as to represent patterns of comorbidity and outcomes that are rarely or never seen in real life (e.g., BPD-PTSD comorbidity with no other Axis I or II disorder). Thus, statistical controls for sociodemographic factors or other disorders were not included in the analyses of the present study. Rather, the sociodemographic variables were examined via descriptive analyses and integrated, as appropriate, into some of the central analyses of this study (e.g., inclusion of the age group and gender variables in the logistic regressions and subsequent separation of the logistic regressions by gender). Examination of the prevalence rates and potential impacts of other Axis I and Axis II comorbidities among the diagnostic groups that were examined in this study presents another, potentially very important future direction, but was beyond the scope of the present study.

Overall, findings from this study emphasize the role of BPD-PTSD comorbidity as an important factor in the study and treatment of both BPD and PTSD and as a source of even greater difficulty and suffering for many of those people living with and/or treating these disorders, as well as for those tasked with the design and implementation of public health policy. In line with the findings of Pagura et al. (2010), among others, the findings of the present study reflect a differential impact of these disorders occurring alone versus in comorbid form, which argues for the importance of diagnosing both BPD

and PTSD, as appropriate (i.e., when the appropriate criteria for each diagnosis are met). The present findings further support the continued emphasis on CSA as one of the prominent trauma types present in and predictive of this comorbidity. Additionally, as described above, the present findings suggest several promising jumping-off points for future research. These include further investigation of the differences between large, community samples and smaller, clinical samples, particularly with regard to the impact of this comorbidity on core "symptom severity", as well as further investigation of different patterns of association seen across genders and age groups.

REFERENCES

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders, 4th ed., text revision.* Washington, DC: American Psychiatric Publishing, Inc.

Aragona, M. (2009). The Role of comorbidity in the crisis of the current psychiatric classification system. *Philosophy, Psychiatry, & Psychology, 16,* 1-11.

Bremner, J. D., Staib, L. H., Kaloupek, D., Southwick, S. M., Soufer, R., & Charney, D. S. (1999). Neural correlates of exposure to traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder: A Positron emission tomography study. *Biological Psychiatry, 45,* 806-816.

Brendel, G. R., Stern, E., & Silbersweig, D. A. (2005). Defining the neurocircuitry of borderline personality disorder: Functional neuroimaging approaches. *Development and Psychopathology, 17,* 1197-1206.

Briere, J., & Elliott, D. M. (2003). Prevalence and psychological sequelae of self-reported childhood physical and sexual abuse in a general population sample of men and women. *Child Abuse and Neglect, 27,* 1205-1222.

Connor, K. M., Davidson, J. R. T., Hughs, D. C., Swartz, M. S., Blazer, D. G., & George, L. K. (2002). The Impact of borderline personality disorder on post-traumatic stress in the community: A Study of health status, health utilization, and functioning. *Comprehensive Psychiatry, 43,* 41-48.

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., et al. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: Results from wave 2 National Epidemiological Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 69,* 533-545.

Grant, B. F., Dawson, D. A., & Hasin, D. S. (2001). The Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV Version. National Institute on Alcohol Abuse and Alcoholism: Bethesda, MD.

Grant, B. F., Dawson, D. A., Stinson, F. S., Chou, S. P., Kay, W., & Pickering, R. P. (2003). The Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV (AUDADIS-IV): Reliability of alcohol consumption, tobacco use, family history of depression and psychiatric diagnostic modules in a general population sample. *Drug and Alcohol Dependence, 71,* 7-16.

Grant, B. F. & Kaplan, K. D. (2005). Source and Accuracy Statement for the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). National Institute on Alcohol Abuse and Alcoholism: Rockville, MD.

Gunderson, J. G. (2001). *Borderline Personality Disorder.* Washington, DC: American Psychiatric Publishing, Inc.

Heffernan, K. & Cloitre, M. (2000). A Comparison of posttraumatic stress disorder with and without borderline personality disorder among women with a history of childhood sexual abuse: Etiological and clinical characteristics. *The Journal of Nervous and Mental Disease, 188,* 589-595.

Hoerst, M., Weber-Fahr, W., Tunc-Skarka, N., Ruf, M., Bohus, M., Schmahl, C., & Ende, G. (2010). Metabolic alterations in the amygdala in borderline personality disorder: A proton magnetic resonance spectroscopy study. *Biological Psychiatry, 67,* 399-405.

Johnson, D. M., Shea, M. T., Yen, S., Battle, C. L., Zlotnick, C., Sanislow, C. A., et al. (2003). Gender differences in borderline personality disorder: Findings from the Collaborative Longitudinal Personality Disorders Study. *Comprehensive Psychiatry, 44,* 284-292.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV Disorders in the National Comorbidity Survey Replication. *Archive of General Psychiatry, 62,* 593-602.

Kraus, A., Esposito, F., Seifritz, E., Di Salle, F., Ruf, M., Valerius, G., . . . Schmahl, C.(2009). Amygdala deactivation as a neural correlate of pain processing in patients with borderline personality disorder and co-occurrent posttraumatic stress disorder. *Biological Psychiatry, 65,* 819-822.

Kroll, J. (1993). *PTSD/Borderlines in Therapy*. New York: W. W. Norton & Company, Inc.

Leihener, F., Wagner, A., Haaf, B., Schmidt, C., Lieb, K., Stieglitz, R., & Bohus, M. (2003). Subtype differentiation of patients with borderline personality disorder using a circumplex model of interpersonal behavior. *The Journal of Nervous and Mental Disease, 191,* 248-254.

Lenzenweger, M. F. (2008). Epidemiology of personality disorders. *Psychiatric Clinics of North America, 31,* 395-401.

Lenzenweger, M. F., Lane, M., Loranger, A. W., & Kessler, R. C. (2007). DSM-IV personality disorders in the National Comorbidity Survey Replication (NCS-R). *Biological Psychiatry, 62,* 553-564.

Lilienfeld, S. O., Waldman, I. D., & Israel A. C. (1994). A Critical examination of the use of the term and concept of comorbidity in psychopathology research. *Clinical Psychology Science and Practice, 1,* 71-83.

Linehan, M. M. (1993). *Cognitive-Behavioral Therapy of Borderline Personality Disorder*. New York: Guilford Press.

McGlashan, T. H., Grilo, C. M., Skodol, A. E., Gunderson, J. G., Shea, M. T., Morey, L. C., . . . Stout R. L. (2000). The Collaborative Longitudinal Personality Disorders Study: Baseline Axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatrica Scandinavica, 102,* 256-264.

Oltmanns, T. F. & Balsis, S. (2011). Personality disorders in later life: Questions about the measurement, course, and impact of disorders. *Annual Review of Clinical Psychology, 7,* 321-349.

Pagura, J., Stein, M. B., Bolton, J. M., Cox, B. J., Grant, B., & Sareen, J. (2010). Comorbidity of borderline personality disorder and posttraumatic stress disorder in the U.S. population. *Journal of Psychiatric Research, 44,* 1190-1198.

Paris, J. (1994). *Borderline Personality Disorder, A Multidimensional Approach.* Washington, DC: American Psychiatric Publishing, Inc.

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology, 51,* 768-774.

Pietrzak, R. H., Goldstein, R. B., Southwick, S. M., & Grant, B. F. (2011). Personality disorders associated with full and partial posttraumatic stress disorder in the U.S. population: Results from Wave 2 of the National Epidemiological Survey on Alcohol and Related Conditions. *Journal of Psychiatric Research, 25,* 456-465.

QualityMetric, (2011). *SF-12v2 Health Survey*. Retrieved from http://www.qualitymetric.com /WhatWeDo /GenericHealthSurveys/SF12v2HealthSurvey/tabid/186/Default.aspx.

Ruan, W. J., Goldstein, R. B., Chou, P. S., Smith, S. M., Saha, T. D., Pickering, R. P., et al. (2008). The Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV (AUDADIS-IV): Reliability of new psychiatric diagnostic modules and risk factors in a general population sample. *Drug and Alcohol Dependence, 92,* 26-37.

SAS Institute Inc. (2011). *SAS/STAT® 9.3 User's Guide*. Cary, NC: SAS Institute Inc.

Schmahl, C., Berne, K., Krause, A., Kleindienst, N., Valerius, G., Vermetten, E., & Bohus, M. (2009). Hippocampus and amygdala volumes in patients with borderline personality disorder with or without posttraumatic stress disorder. *Journal of Psychiatry and Neuroscience, 34,* 289-295.

Schmahl, C. G., Elzinga, B. M., Ebner, U. W., Simms, T., Sanislow, C., et al. (2004). Psychophyisiological reactivity to traumatic and abandonment scripts in

borderline personality and posttraumatic stress disorders: a preliminary report. *Psychiatry Research, 126,* 33-42.

Skodol, A. E., Gunderson, J. G., Pfohl, B., Widiger, T. A., Livesley, W. J., & Siever, L. J. (2002a). The borderline diagnosis I: Psychopathology, comorbidity, and personality structure. *Biological Psychiatry, 51,* 936-950.

Skodol, A. E., Siever, L. J., Livesley, W. J., Gunderson, J. G., Pfohl, B., & Widiger, T. A. (2002b). The borderline diagnosis II: Biology, genetics, and the clinical course. *Biological Psychiatry, 51,* 951-963.

Tadic, A., Wagner, S., Hoch, J., Baskaya, O., von Cube, R., Skaletz, C., et al. (2009). Gender differences in Axis I and Axis II comorbidity in patients with borderline personality disorder. *Psychopathology, 42,* 257-263.

Thomas, D. R. & Rao, J. N. K. (1984). A Monte Carlo study of exact levels of goodness-of-fit statistics under cluster sampling. Proceedings of the Survey Research Methods Section, American Statistical Association, 207–211.

Trull, T. J., Jahng, S., Tomko, R. L., Wood, P. K., & Sher, K. J. (2010). Revised NESARC personality disorder diagnoses: Gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders, 24,* 412-426.

Van Den Bosch, L., Verheul, R., Langeland, W., & Van Den Brink, W. (2003). Trauma, dissociation, and posttraumatic stress disorder in female borderline patients with and without substance abuse problems. *Australian and New Zealand Journal of Psychiatry, 37,* 549-555.

Ware, J. E., Kosinski, M., Turner-Bowker, D. M., & Gandek B. (2007). *User's Manual for the SF-12v2 Health Survey with a Supplement Documenting SF-12 Health Survey*. Lincoln, RI: Quality Metric, Inc.

Ware, J. E., Kosinski, M., Turner Bowker, D. M., & Gandek, B. (2002). *How to Score Version 2 of the SF-12 Health Survey*. Lincoln, RI: Quality Metrics, Inc.

Weniger, G., Lange, C., Sachsse, U., & Irle, E. (2009). Reduced amygdala and hippocampus size in trauma-exposed women with borderline personality disorder and without posttraumatic stress disorder. *Journal of Psychiatry and Neuroscience, 34,* 383-388.

Woodward, H. E., Taft, C. T., Gordon, R. A., & Meis, L. A. (2009). Clinician bias in the diagnosis of posttraumatic stress disorder and borderline personality disorder. *Psychological Trauma: Theory, Research, Practice, and Policy, 1,* 282-290.

Zimmerman, M., Chelminski, I., & Young, D. (2008). The Frequency of personality disorders in psychiatric patients. *Psychiatric Clinics of North America, 31,* 405-420.

Zlotnick, C., Johnson, D. M., Yen, S., Battle, C. L., Sanislow, C. A., Skodol, A. et al. (2003). Clinical features and impairment in women with borderline personality disorder (BPD) with posttraumatic stress disorder (PTSD), BPD without PTSD, and other personality disorders with PTSD. *The Journal of Nervous and Mental Disease, 191,* 706-713.

Table 1

Demographic characteristics of the sample

Characteristics	PTSD-only (*n* = 3074) *n* (column %, row %)	BPD-only (*n* = 483) *n* (column %, row %)	Comorbid (*n* = 547) *n* (column %, row %)
Gender			
Male	815 (26.5, 67.8)	224 (46.4, 18.7)	162 (29.6, 13.5)
Female	2259 (73.5, 77.8)	259 (53.6, 8.9)	385 (70.4, 13.3)
Age			
20-29	381 (12.4, 63.2)	111 (23.0, 18.4)	111 (20.3, 18.4)
30-44	1007 (32.8, 72.0)	181 (37.5, 12.9)	211 (38.6, 15.1)
45-64	1246 (40.5, 77.6)	156 (32.3, 9.7)	203 (37.1, 12.6)
65+	440 (14.3, 88.5)	35 (7.2, 7.0)	22 (4.0, 4.4)
Race/Ethnicity			
African American	661 (21.5, 75.1)	98 (8.8, 11.4)	121 (22.1, 13.8)
American Indian/Alaskan	68 (2.2, 68.0)	11 (2.3, 11.0)	21 (3.8, 21.0)
Asian/Pacific Islander	66 (2.1, 84.6)	9 (1.9, 11.5)	3 (0.5, 3.9)
Caucasian	1774 (57.7, 75.4)	288 (59.6,12.2)	290 (53.0, 12.3)
Hispanic	505 (16.4, 72.8)	77 (15.9, 11.1)	112 (20.5, 16.1)
Education			
<High school	538 (17.5, 74.0)	82 (17.0, 11.3)	107 (19.6, 14.7)
High school or equivalent	806 (26.2, 72.0)	156 (32.3, 13.9)	158 (28.9, 14.1)
Some college+	1730 (56.3, 76.7)	245 (50.7, 10.9)	282 (51.6, 12.5)
Marital status			
Married/cohabiting	1483 (48.2, 78.6)	197 (40.8, 10.4)	206 (37.7, 10.9)
Widowed/separated/divorced	1049 (34.1, 74.6)	159 (32.9, 11.3)	199 (36.4, 14.1)
Never married	542 (17.6, 66.8)	127 (26.3, 15.7)	142 (26.0, 17.5)
Household income			
$0-$19,999	890 (29.0, 70.7)	133 (27.5, 10.6)	236 (43.1, 18.7)
$20,000-$34,999	677 (22.0, 75.3)	106 (21.9, 11.8)	116 (21.2, 12.9)
$35,000-$59,999	681 (22.2, 73.8)	133 (27.5, 14.4)	109 (19.9, 11.8)
$60,000+	826 (26.9, 80.7)	111 (23.0, 10.9)	86 (15.7, 8.4)
U.S. region			
New England	134 (4.4, 74.9)	15 (3.1, 8.4)	30 (5.5, 16.8)
Mid Atlantic	414 (13.5, 74.6)	63 (13.0, 11.4)	78 (14.3, 14.1)
East North Central	419 (13.6, 73.6)	60 (12.4, 10.5)	90 (16.5, 15.8)
West North Central	173 (5.6, 80.1)	21 (4.3, 9.7)	22 (4.0, 10.2)
South Atlantic	643 (20.9, 75.8)	103 (21.3, 12.1)	102 (18.6, 12.0)
East South Central	212 (6.9, 75.7)	25 (5.2, 8.9)	43 (7.9, 15.4)
West South Central	294 (9.6, 72.8)	52 (10.8, 12.9)	58 (10.6, 14.4)
Mountain	230 (7.5, 75.2)	45 (9.3, 14.7)	31 (5.7, 10.1)
Pacific	555 (18.1, 74.3)	99 (20.5, 13.3)	93 (17.0, 12.4)

Table 2

Variance in SF-12v2 scores accounted for by diagnostic status and gender

3-Level Dx Status Models: Comorbid vs. PTSD-only vs. BPD-only			
Scale/Effect	df_{num}	*F*	*p*
General Health (df_{denom} = 3690)			
Dx status	2	11.32	**<.0001**
Gender	1	1.39	.2378
Dx status*Gender	2	0.27	.7665
Social Functioning (df_{denom} = 3687)			
Dx status	2	57.43	**<.0001**
Gender	1	9.28	**.0023**
Dx status*Gender	2	2.88	.0562
Role Emotional Functioning (df_{denom} = 3689)			
Dx status	2	56.04	**<.0001**
Gender	1	2.59	.1077
Dx status*Gender	2	0.67	.5133
Mental Health (df_{denom} = 3688)			
Dx status	2	80.95	**<.0001**
Gender	1	17.13	**<.0001**
Dx status*Gender	2	2.21	.1103
Physical Functioning (df_{denom} = 3691)			
Dx status	2	6.00	**.0025**
Gender	1	0.21	.6465
Dx status*Gender	2	0.23	.7955
Role Physical Functioning (df_{denom} = 3690)			
Dx status	2	9.87	**<.0001**
Gender	1	0.12	.7280
Dx status*Gender	2	0.34	.7142
Vitality (df_{denom} = 3688)			
Dx status	2	19.57	**<.0001**
Gender	1	18.50	**<.0001**
Dx status*Gender	2	1.77	.1701
Bodily Pain (df_{denom} = 3689)			
Dx status	2	9.53	**<.0001**
Gender	1	0.05	.8266
Dx status*Gender	2	1.24	.2895

Table 3

Variance in SF-12v2 scores accounted for by diagnostic status and gender

2-Level Dx Status Models: Comorbid vs. PTSD-only			
Scale/Effect	df_{num}	*F*	*p*
General Health (df_{denom} = 3212)			
Dx status	1	22.57	**<.0001**
Gender	1	1.04	.3090
Dx status*Gender	1	0.41	.5230
Social Functioning (df_{denom} = 3209)			
Dx status	1	97.42	**<.0001**
Gender	1	7.80	**.0053**
Dx status*Gender	1	5.23	**.0223**
Role Emotional Functioning (df_{denom} = 3211)			
Dx status	1	100.79	**<.0001**
Gender	1	1.87	.1718
Dx status*Gender	1	1.11	.2911
Mental Health (df_{denom} = 3210)			
Dx status	1	119.53	**<.0001**
Gender	1	16.30	**<.0001**
Dx status*Gender	1	4.38	**.0365**
Physical Functioning (df_{denom} = 3213)			
Dx status	1	6.01	**.0143**
Gender	1	0.03	.8668
Dx status*Gender	1	0.45	.5039
Role Physical Functioning (df_{denom} = 3212)			
Dx status	1	18.87	**<.0001**
Gender	1	0.01	.9301
Dx status*Gender	1	0.34	.5579
Vitality (df_{denom} = 3210)			
Dx status	1	26.74	**<.0001**
Gender	1	14.64	**<.0001**
Dx status*Gender	1	3.34	.0677
Bodily Pain (df_{denom} = 3211)			
Dx status	1	18.37	**<.0001**
Gender	1	0.12	.7309
Dx status*Gender	1	0.93	.3352

Table 4

Variance in SF-12v2 scores accounted for by diagnostic status and gender

2-Level Dx Status Models: Comorbid vs. BPD-only			
Scale/Effect	df_{num}	*F*	*p*
General Health (df_{denom} = 716)			
Dx status	1	11.56	**.0007**
Gender	1	0.31	.5797
Dx status*Gender	1	0.06	.8142
Social Functioning (df_{denom} = 716)			
Dx status	1	12.59	**.0004**
Gender	1	9.76	**.0019**
Dx status*Gender	1	1.03	.3116
Role Emotional Functioning (df_{denom} = 717)			
Dx status	1	19.52	**<.0001**
Gender	1	2.81	.0942
Dx status*Gender	1	0.14	.7036
Mental Health (df_{denom} = 717)			
Dx status	1	6.94	**.0086**
Gender	1	13.35	**.0003**
Dx status*Gender	1	1.73	.1893
Physical Functioning (df_{denom} = 717)			
Dx status	1	12.50	**.0004**
Gender	1	0.02	.8874
Dx status*Gender	1	0.32	.5714
Role Physical Functioning (df_{denom} = 717)			
Dx status	1	14.18	**.0002**
Gender	1	0.35	.5545
Dx status*Gender	1	0.00	.9810
Vitality (df_{denom} = 717)			
Dx status	1	1.16	.2821
Gender	1	15.52	**<.0001**
Dx status*Gender	1	0.93	.3348
Bodily Pain (df_{denom} = 717)			
Dx status	1	13.05	**.0003**
Gender	1	0.60	.4393
Dx status*Gender	1	0.07	.7921

Table 5

SF-12v2 Scale means by diagnostic group

	PTSD-only (n = 3074, $M_{overall}$ = 46.64) *M* (*SE*)	BPD-only (n = 483, $M_{overall}$ = 44.71) *M* (*SE*)	Comorbid (n = 547, $M_{overall}$ = 41.08) *M* (*SE*)
General Health	46.16 (0.29)	45.00 (0.66)	41.21 (0.73)
Social Functioning	46.85 (0.27)	42.63 (0.62)	38.31 (0.69)
Role Emotional Functioning	45.05 (0.27)	41.37 (0.57)	36.63 (0.64)
Mental Health	46.67 (0.25)	41.10 (0.59)	37.71 (0.64)
Physical Functioning	47.39 (0.27)	49.05 (0.63)	45.51 (0.65)
Role Physical Functioning	46.27 (0.25)	46.38 (0.61)	42.72 (0.60)
Vitality	48.56 (0.24)	46.02 (0.51)	44.38 (0.57)
Bodily Pain	46.17 (0.28)	46.15 (0.66)	42.14 (0.69)

Table 6

SF-12v2 Scale means by gender

	Men (n = 1201, $M_{overall}$ = 45.89) *M* (*SE*)	Women (n = 2903, $M_{overall}$ = 45.62) *M* (*SE*)
General Health	44.40 (0.48)	45.90 (0.30)
Social Functioning	**45.66 (0.42)**	**45.06 (0.30)**
Role Emotional Functioning	43.64 (0.43)	43.49 (0.29)
Mental Health	**45.75 (0.41)**	**44.41 (0.27)**
Physical Functioning	47.85 (0.43)	47.14 (0.28)
Role Physical Functioning	45.75 (0.40)	45.89 (0.26)
Vitality	**48.80 (0.40)**	**47.19 (0.25)**
Bodily Pain	45.27 (0.43)	45.87 (0.30)

Note. Bolded text reflects that a significant (p < .01) main effect of gender was found for these scales (Social Functioning, Mental Health, and Vitality).

Table 7

SF-12v2 Scale means by diagnostic group and gender

	PTSD-only (n = 3074, $M_{overall}$ = 46.64) M (SE)		BPD-only (n = 483, $M_{overall}$ = 44.71) M (SE)		Comorbid (n = 547, $M_{overall}$ = 41.08) M (SE)	
	Men ($M_{overall}$ = 46.75)	Women ($M_{overall}$ = 46.60)	Men ($M_{overall}$ = 45.29)	Women ($M_{overall}$ = 44.11)	Men ($M_{overall}$ = 42.32)	Women ($M_{overall}$ = 40.43)
General Health	45.01 (0.54)	46.65 (0.33)	44.55 (0.85)	45.47 (0.83)	40.96 (0.85)	41.33 (0.72)
Social Functioning	**47.14 (0.49)**	**46.73 (0.31)**	**43.65 (0.79)**	**41.57 (0.83)**	**41.00 (0.71)**	**36.92 (0.71)**
Role Emotional Functioning	45.23 (0.50)	44.98 (0.32)	41.97 (0.78)	40.74 (0.75)	37.92 (0.79)	35.97 (0.58)
Mental Health	**47.73 (0.44)**	**46.22 (0.28)**	42.21 (0.79)	39.95 (0.81)	**40.83 (0.76)**	**36.09 (0.65)**
Physical Functioning	47.89 (0.52)	47.18 (0.31)	49.40 (0.69)	48.69 (0.96)	45.23 (0.74)	45.66 (0.57)
Role Physical Functioning	46.00 (0.49)	46.38 (0.28)	46.66 (0.80)	46.09 (0.86)	43.07 (0.69)	42.54 (0.59)
Vitality	49.59 (0.48)	48.13 (0.27)	47.23 (0.68)	44.76 (0.59)	47.11 (0.70)	42.97 (0.51)
Bodily Pain	45.38 (0.50)	46.50 (0.33)	46.68 (0.78)	45.59 (0.93)	42.09 (0.85)	41.96 (0.72)

Note. Bolded text reflects the marginal diagnostic group-by-gender interaction found on the Social Functioning scale (p = .056), as well as the significant diagnostic group-by-gender interactions found for the PTSD-only vs. Comorbid group comparison on the Mental Health (p = .037) and Social Functioning (p = .022) scales.

Figure 1

Plot of mean SF-12v2 Social Functioning scores–diagnostic group-by-gender interaction

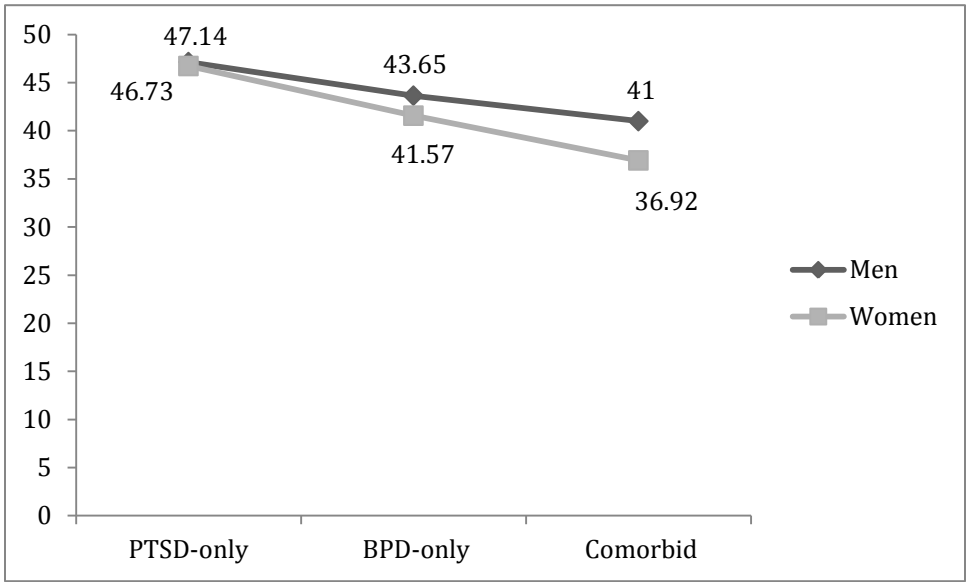

Figure 2

Plot of mean SF-12v2 Mental Health scores–diagnostic group-by-gender interaction

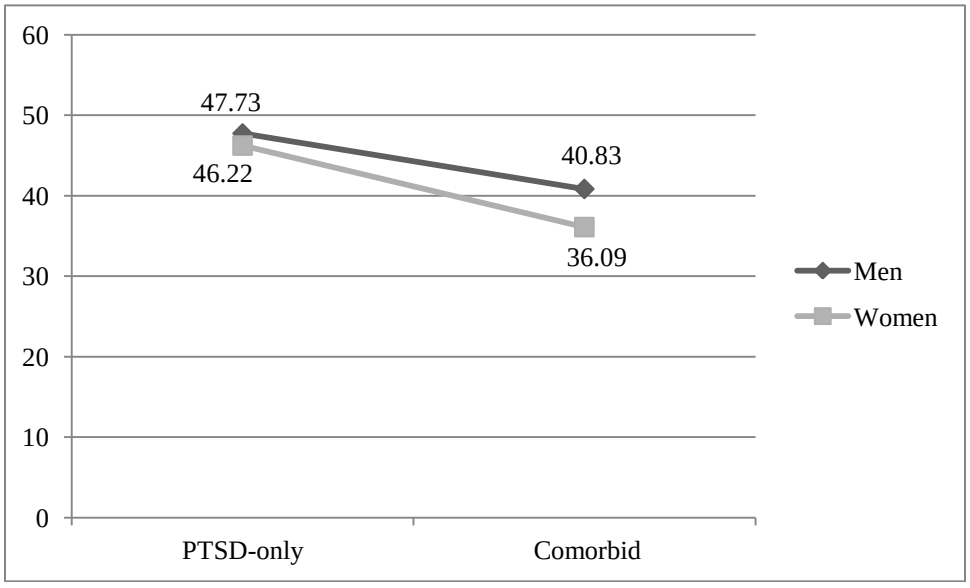

Table 8

Variance in SF-12v2 scores accounted for by CSA and gender

Scale/Effect	df_{num}	*F*	*p*
General Health (df_{denom} = 33914)			
CSA	1	13.94	**.0002**
Gender	1	5.51	**.0189**
CSA*Gender	1	0.93	.3341
Social Functioning (df_{denom} = 33902)			
CSA	1	77.82	**<.0001**
Gender	1	5.48	**.0193**
CSA*Gender	1	0.62	.4300
Role Emotional Functioning (df_{denom} = 33902)			
CSA	1	58.18	**<.0001**
Gender	1	14.97	**.0001**
CSA*Gender	1	0.22	.6381
Mental Health (df_{denom} = 33898)			
CSA	1	122.33	**<.0001**
Gender	1	16.43	**<.0001**
CSA*Gender	1	0.20	.6552
Physical Functioning (df_{denom} = 33914)			
CSA	1	12.16	**.0005**
Gender	1	23.63	**<.0001**
CSA*Gender	1	0.05	.8213
Role Physical Functioning (df_{denom} = 33903)			
CSA	1	16.04	**<.0001**
Gender	1	23.60	**<.0001**
CSA*Gender	1	0.42	.5181
Vitality (df_{denom} = 33901)			
CSA	1	43.85	**<.0001**
Gender	1	38.35	**<.0001**
CSA*Gender	1	0.39	.5332
Bodily Pain (df_{denom} = 33902)			
CSA	1	46.22	**<.0001**
Gender	1	10.74	**.0011**
CSA*Gender	1	0.08	.7743

Table 9

Variance in SF-12v2 scores accounted for by diagnostic status, CSA, and gender

Scale/Effect	df_{num}	*F*	*p*
General Health (df_{denom} = 3668)			
Dx status	2	10.55	**<.0001**
CSA	1	0.44	.5061
Gender	1	0.00	.9617
Dx status*CSA	2	0.40	.6732
Dx status*Gender	2	0.09	.9147
CSA*Gender	1	1.72	.1904
Dx status*CSA*Gender	2	0.06	.9377
Social Functioning (df_{denom} = 3665)			
Dx status	2	37.58	**<.0001**
CSA	1	1.62	.2026
Gender	1	2.94	.0863
Dx status*CSA	2	1.57	.2086
Dx status*Gender	2	0.58	.5607
CSA*Gender	1	0.00	.9579
Dx status*CSA*Gender	2	0.35	.7074
Role Emotional Functioning (df_{denom} = 3667)			
Dx status	2	42.63	**<.0001**
CSA	1	0.14	.7122
Gender	1	1.85	.1744
Dx status*CSA	2	0.24	.7839
Dx status*Gender	2	0.37	.6905
CSA*Gender	1	0.09	.7696
Dx status*CSA*Gender	2	0.44	.6435
Mental Health (df_{denom} = 3666)			
Dx status	2	42.17	**<.0001**
CSA	1	1.73	.1890
Gender	1	8.54	**.0035**
Dx status*CSA	2	0.82	.4410
Dx status*Gender	2	1.58	.2067
CSA*Gender	1	0.69	.4074
Dx status*CSA*Gender	2	0.21	.8112
Physical Functioning (df_{denom} = 3669)			
Dx status	2	5.69	**.0034**
CSA	1	2.09	.1488
Gender	1	1.39	.2380

Dx status*CSA	2	1.21	.2982
Dx status*Gender	2	1.29	.2750
CSA*Gender	1	0.84	.3597
Dx status*CSA*Gender	2	0.96	.3832
Role Physical Functioning (df_{denom} = 3668)			
Dx status	2	11.15	**<.0001**
CSA	1	0.01	.9411
Gender	1	0.14	.7074
Dx status*CSA	2	2.15	.1161
Dx status*Gender	2	0.85	.4285
CSA*Gender	1	0.17	.6813
Dx status*CSA*Gender	2	2.54	.0787
Vitality (df_{denom} = 3666)			
Dx status	2	19.65	**<.0001**
CSA	1	2.22	.1367
Gender	1	7.99	**.0047**
Dx status*CSA	2	2.93	**.0533**
Dx status*Gender	2	1.33	.2636
CSA*Gender	1	0.06	.8079
Dx status*CSA*Gender	2	2.09	.1236
Bodily Pain (df_{denom} = 3667)			
Dx status	2	7.90	**.0004**
CSA	1	1.40	.2363
Gender	1	0.00	.9829
Dx status*CSA	2	1.18	.3087
Dx status*Gender	2	0.22	.8033
CSA*Gender	1	0.04	.8444
Dx status*CSA*Gender	2	0.87	.4171

Table 10

SF-12v2 Scale means by CSA status

	CSA (n = 885, $M_{overall}$ = 44.97) *M* (*SE*)	No CSA (n = 3197, $M_{overall}$ = 45.92) *M* (*SE*)
General Health	44.93 (0.54)	45.53 (0.29)
Social Functioning	44.02 (0.56)	45.61 (0.27)
Role Emotional Functioning	42.43 (0.49)	43.86 (0.28)
Mental Health	43.16 (0.49)	45.30 (0.26)
Physical Functioning	47.74 (0.48)	47.25 (0.27)
Role Physical Functioning	45.60 (0.46)	45.90 (0.25)
Vitality	46.50 (0.46)	48.03 (0.24)
Bodily Pain	44.84 (0.51)	45.89 (0.28)

Table 11

SF-12v2 Vitality scale means by diagnostic group and CSA status

PTSD-only *M* (*SE*)		BPD-only *M* (*SE*)		Comorbid *M* (*SE*)	
CSA (n = 600)	No CSA (n = 2462)	CSA (n = 89)	No CSA (n = 389)	CSA (n = 196)	No CSA (n = 346)
48.14 (0.48)	48.66 (0.27)	43.49 (1.01)	46.47 (0.54)	42.52 (0.60)	45.23 (0.65)

Note. The Vitality scale was the only SF-12v2 scale on which evidence of a diagnostic group-by-CSA interaction emerged. This interaction was marginally significant (p = .0533).

Figure 3

Plot of mean SF-12v2 Vitality scores–marginally significant diagnostic group-by-CSA interaction

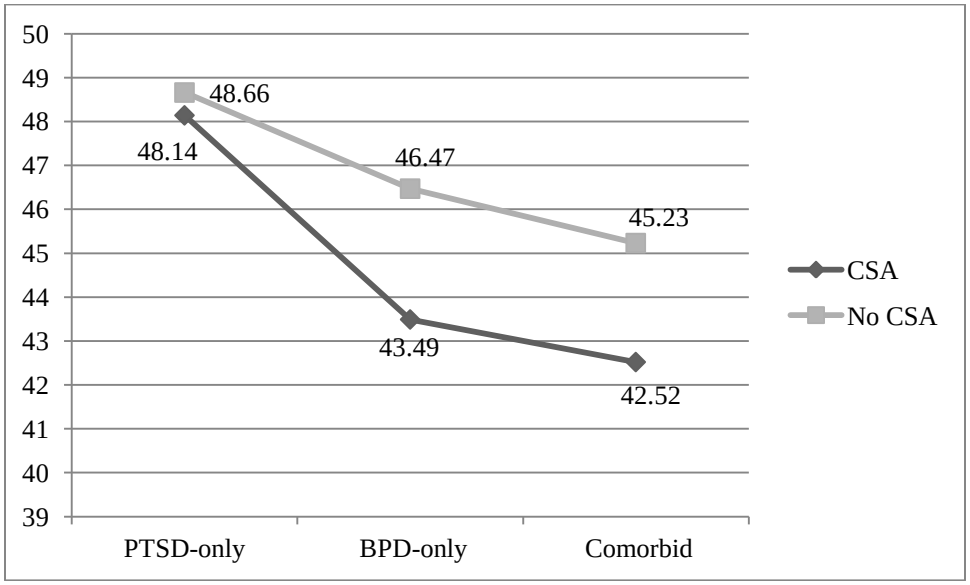

Table 12

Prevalence of different types of traumatic experience across the diagnostic groups

	PTSD-only (n = 3074)	BPD-only (n = 483)	Comorbid (n = 547)
	n (% within dx group, prevalence rank within dx group)	*n* (% within dx group, prevalence rank within dx group)	*n* (% within dx group, prevalence rank within dx group)
Childhood sexual abuse/assault (CSA)	600 (19.52%, 9)	89 (18.43%, 10)	196 (35.83%, 6)
Adult sexual abuse/assault (ASA)	175 (5.69%, 16)	17 (3.52%, 15)	39 (7.13%, 16)
Military combat	178 (5.79%, 15)	12 (2.48%, 17)	26 (4.75%, 17)
Peacekeeping/relief work in war zone	58 (1.89%, 21)	13 (2.69%, 16)	11 (2.01%, 19)
Civilian experience of War	92 (2.99%, 18)	8 (1.66%, 18)	15 (2.74%, 18)
Refugee experience	59 (1.92%, 20)	1 (0.21%, 20)	8 (1.46%, 20)
Serious/life-threatening accident	782 (25.44%, 5)	151 (31.26%, 3)	201 (36.75%, 5)
Serious/life-threatening illness	834 (27.13%, 3)	123 (25.47%, 6)	187 (34.19%, 8)
Natural disaster	686 (22.32%, 7)	115 (23.81%, 7)	150 (27.24%, 10)
Physical attack/abuse by caregiver before age 18	351 (11.42%, 13)	57 (11.80%, 12)	139 (25.41%, 12)
Neglect by caregiver before age 18	313 (10.18%, 14)	55 (11.39%, 13)	138 (25.23%, 13)
Witnessed fights between caregivers before age 18	787 (25.60%, 4)	141 (29.19%, 4)	224 (40.95%, 3)
Intimate partner violence	683 (22.22%, 8)	104 (21.53%, 9)	223 (40.77%, 4)
Physical attack by anyone else	467 (15.19%, 11)	113 (23.40%, 8)	166 (30.35%, 9)
Kidnapped/held hostage/POW	104 (3.38%, 17)	13 (2.69%, 16)	45 (8.23%, 15)
Stalked	585 (19.03%, 10)	81 (16.77%, 11)	142 (25.96%, 11)
Mugged	700 (22.77%, 6)	138 (28.57%, 5)	192 (35.10%, 7)
Death of someone close in a terrorist attack	58 (1.89%, 21)	5 (1.04%, 19)	8 (1.46%, 20)
Injured in terrorist attack	7 (0.23%, 22)	0 (0.0%, 21)	1 (0.18%, 22)
Direct experience of terrorist attack (without injury)	63 (2.05%, 19)	8 (1.66%, 18)	5 (0.19%, 21)
Saw someone badly injured/killed	1148 (37.35%, 2)	164 (33.95%, 2)	257 (46.98%, 2)
Unexpected death of someone close	1937 (63.01%, 1)	269 (55.69%, 1)	386 (70.57%, 1)
Any other traumatic experience	358 (11.65%, 12)	39 (8.07%, 14)	100 (18.28%, 14)