Published in final edited form as:
J Psychopathol Behav Assess. 2013 June 1; 35(2): 254–263. doi:10.1007/s10862-012-9327-0.

Trauma Exposure in Anxious Primary Care Patients

J. Bomyea, MS[1,*], **A.J. Lang, PhD, MPH**[2,3], **D. Golinelli, PhD**[4], **M.G. Craske, PhD**[5], **D.A. Chavira, PhD**[2], **C.D. Sherbourne, PhD**[4], **R.D. Rose, PhD**[5], **L. Campbell-Sills, PhD**[2], **S.S. Welch, PhD**[6], **G. Sullivan, MD**[4,7,8], **A. Bystritsky, MD**[9], **P. Roy-Byrne, MD**[6], and **M.B. Stein, MD, MPH**[2,10]

[1]SDSU/UCSD Joint Doctoral Program in Clinical Psychology, University of California, San Diego

[2]Department of Psychiatry, University of California, San Diego

[3]VA San Diego Health Care System Center of Excellence for Stress and Mental Health, San Diego, California

[4]RAND Corporation, Santa Monica, California

[5]Department of Psychology, University of California, Los Angeles

[6]Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine and Harborview Center for Healthcare Improvement for Addictions, Mental Illness, and Medically Vulnerable Populations (CHAMMP), Seattle, Washington

[7]Department of Psychiatry University of Arkansas for Medical Sciences, Little Rock

[8]VA South Central Mental Illness Research, Education, and Clinical Center University of Arkansas for Medical Sciences, Little Rock

[9]Psychiatry and Biobehavioral Sciences, David Geffen School of Medicine, University of California, Los Angeles

[10]Family and Preventive Medicine, University of California, San Diego

Abstract

The present study examined rates of trauma exposure, clinical characteristics associated with trauma exposure, and the effect of trauma exposure on treatment outcome in a large sample of primary care patients without posttraumatic stress disorder (PTSD). Individuals without PTSD (N = 1263) treated as part of the CALM program (Roy-Byrne et al., 2010) were assessed for presence of trauma exposure. Those with and without trauma exposure were compared on baseline

[*]Corresponding author: Jessica Bomyea UCSD Department of Psychiatry 8939 Villa La Jolla Dr. Suite 200 San Diego, CA 92037 Phone: 858-534-6438 Fax: 858-534-6460 bomyea@hotmail.com.
Ariel J. Lang, Ph.D., MPH, 8939 Villa La Jolla Dr. Suite 200, San Diego, CA 92037, Phone: 858-246-0631
Danielle Gollineli, 1776 Main Street, Santa Monica, CA, USA 90401, Phone: (310) 393-0411
Michelle Craske, UCLA Anxiety Disorders Research Center, Department of Psychology, Franz Hall - Box 951563, Los Angeles, CA 90094-1563, Phone: (310) 206-9191
Denise Chavira, 8939 Villa La Jolla Dr. Suite 200, San Diego, CA 92037, Phone: 858-246-0623
Cathy Sherbourne, 1776 Main Street, Santa Monica, CA, USA 90401, Phone: (310) 393-0411
Raphael Rose, UCLA Anxiety Disorders Research Center, Department of Psychology, Franz Hall - Box 951563, Los Angeles, CA 90094-1563, Phone: (310) 206-9191
Laura Campbell-Sills, 8939 Villa La Jolla Dr. Suite 200, San Diego, CA 92037, Phone: 858-534-6448
Stacy Shaw Welch, 1200 5th Avenue Suite 800, Seattle WA 92101, 206-374-0109
Greer Sullivan, 4301 W. Markham # 554, Little Rock, AR 72205, 501-526-8100
Alexander Bystritsky, UCLA Psych and Behav Sciences, Box 956969 200 Medical Plaza Ste 2200, Los Angeles CA 90095, (310) 206-5133
Peter Roy-Byrne, 325 9th Ave PSB – 5020, Box 359911, Seattle WA 98104, 206-897-4201
Murray B. Stein, M.D., MPH, UCSD Department of Psychiatry, 8939 Villa La Jolla Dr. Suite 200, San Diego, CA 92037, Phone: 858-534-6451, Fax: 858-534-6460, mstein@ucsd.edu

demographic and diagnostic information, symptom severity, and responder status six months after beginning treatment. Trauma-exposed individuals (N = 662, 53%) were more likely to meet diagnostic criteria for Obsessive Compulsive Disorder and had higher levels of somatic symptoms at baseline. Individuals with and without trauma exposure did not differ significantly on severity of anxiety, depression, or mental health functioning at baseline. Trauma exposure did not significantly impact treatment response. Findings suggest that adverse effects of trauma exposure in those without PTSD may include OCD and somatic anxiety symptoms. Treatment did not appear to be adversely impacted by trauma exposure. Thus, although trauma exposure is prevalent in primary care samples, results suggest that treatment of the presenting anxiety disorder is effective irrespective of trauma history.

Keywords

anxiety; treatment; primary care; trauma

Estimated rates of trauma exposure (i.e., exposure to events such as serious accidents, assault, terrorist attacks, being held hostage or kidnapped, fire, discovering a body, sudden death of someone close, war, or natural disaster) in primary care patients range from 65% to almost 88% (Alim et al., 2006; Gillespie et al., 2009; Kartha et al., 2008). Trauma exposure is associated with poorer mental health outcomes in the general population (Breslau, Chilcoat, Kessler, & Davis, 1999; Breslau, Davis, & Schultz, 2003; Bryant et al., 2010; Cougle, Timpano, Sachs-Ericsson, Keogh, & Riccardi, 2010; Green et al., 2000; Simon et al., 2009; Simpson & Miller, 2002) and in primary care (Bruce et al., 2001; Coid et al., 2003; Gillespie et al., 2009; Glover, Olfson, Gameroff, & Neria, 2010; Khoury, Tang, Bradley, Cubells, & Ressler, 2010; Löew et al., 2011; McQuaid, Pedrelli, McCahill, & Stein, 2001; Mellman, Aigbogun, Graves, Lawson, & Alim, 2008), leading to greater mental health care utilization and costs (Simpson & Miller, 2002). Poorer functioning and greater symptom severity are associated with trauma exposure, even in those who do not have posttraumatic stress disorder (PTSD; Bruce et al., 2001; Löew et al., 2011), thus raising the question of the role of trauma exposure in other anxiety disorders and depression. Anxiety and depression are very common in primary care (Kroenke, Spitzer, Williams, Monahan, & Lowe, 2007; Pini, Perkonnig, Tansella, & Wittchen, 1999; Serrano-Blanco et al., 2010); over 10% of adults who consult a primary care physician have had an episode of an anxiety or depressive disorder in the past month (Kirmayer, Robbins, Dworkind, & Yaffe, 1993). In addition, anxious and depressed individuals who seek services typically rely on treatment options provided in primary care rather than specialty mental health services (Bijl & Ravelli, 2000; Kroenke et al., 2007; Wang et al., 2005; Weisberg, Dyck, Culpepper, & Keller, 2007), underscoring the importance of understanding how trauma exposure may impact outcomes for this frequently-treated population.

Despite evidence that trauma exposure is highly prevalent and detrimental for mental health outcomes, the effect of trauma exposure on treatment response for anxiety disorders other than PTSD is poorly understood. Trauma exposure might affect treatment response in a number of ways. Individuals with panic disorder and trauma exposure appear to have greater symptom severity at baseline (Michelson, June, Vives, Testa, & Marchione, 1998). Given that greater symptom severity and comorbidity have been found to be negative prognostic indicators of anxiety treatment in some instances (Forbes, Creamer, Hawthorne, Allen, & McHugh, 2003; Scholing & Emmelkamp, 1999; Seivewright, Tyrer, & Johnson, 1998; Solvason, Ernst, & Roth, 2003; Stein, Montgomery, Kasper, & Tanghoj, 2001 - although not all studies document this effect, see Brown, Antony, & Barlow, 1995; Tsao, Lewin, & Craske, 1998), trauma exposed individuals might be relatively treatment refractory due to greater severity or complexity. For example, exposure to traumatic events might strengthen

beliefs that maintain anxiety (e.g., seeing the world as a dangerous, uncontrollable place) and are often the focus of psychosocial interventions (James, Southam, & Blackburn, 2004).

Research to date evaluating the relationship between trauma exposure and response to anxiety treatment is sparse, relatively equivocal, and limited methodologically. Michaelson et June, Vives, Testa, & Marchione (1998) collected retrospective data on trauma exposure from participants with panic disorder who had previously completed randomized controlled trials. Earlier age at the time of trauma and higher level of violence during interpersonal trauma were negatively associated with baseline depression severity scores, and greater trauma frequency was associated with depression, anxiety, and general psychopathology at post-treatment controlling for baseline severity. However, Kipper et al. (2007), in a study examining pharmacotherapy treatment response in panic disorder, did not find an association between endorsement of trauma and symptom outcomes when controlling for age of onset, which was associated with positive trauma history and treatment response. Shavitt et al. (2010) found that individuals with obsessive compulsive disorder (OCD) who had a history of trauma showed equivalent treatment response to cognitive behavioral therapy and *better* response to pharmacotherapy than those without trauma exposure. Subsequent analyses also indicated that those within the trauma-exposed group with PTSD showed greater symptom reduction across both treatment types than those with OCD alone (Shavitt et al., 2010).

Methodological limitations in these studies preclude firm conclusions about the role of trauma exposure in treatment response. For example, trauma history was assessed with a single question in Kipper et al. (2007) and retrospectively after treatment completion by Michaelson et al. (1998). These studies also differed in how they operationalized improvement (e.g., self versus clinician report, disorder specific versus general outcome measures). These differences, in conjunction with the limited evidence currently available suggest that further research is needed to better understand the effect of trauma history on treatment outcomes in anxious individuals. Studies with more comprehensive trauma assessment and broader samples of anxiety disordered patients will allow us to draw inferences about the potential relationship between trauma history and treatment prognosis.

The purpose of the present analyses is to further examine the effect of trauma exposure on anxiety. Trauma exposure is a necessary condition for PTSD, and many studies have examined exposure factors that increase risk of PTSD. There is increasing recognition, however, that PTSD is not the only pathological response to trauma (Galea et al., 2002; North et al., 1999). Thus, we were specifically interested in isolating the effect of trauma exposure in cases where PTSD is not present to determine the impact of trauma exposure on both baseline presentation and treatment response. Although some evidence suggests that trauma exposure may be associated with greater severity and lower functioning independent of PTSD diagnosis (Marshall et al., 2001), relatively few studies have addressed this question in primary care samples and no published studies have examined the effect on treatment response in this population. With these aims in mind we first examined demographic characteristics, prevalence of psychiatric conditions, and symptom severity of those with and without trauma exposure histories but who did not have PTSD. Second, we evaluated the effect of trauma exposure on response to anxiety treatment provided in the primary care setting. Specifically, we examined whether or not baseline trauma exposure was a moderator (i.e., has an interactive effect with intervention arm on outcome, also referred to as a prescriptive variable) or predictor variable of treatment outcome (i.e., predicts treatment outcome irrespective of intervention assignment, also known as a prognostic variable; Fournier et al., 2009; Kraemer, Wilson, Fairburn, & Agras, 2002).

To this end, we utilized treatment outcome data from a randomized controlled trial in which participants were assigned to complete a Coordinated Anxiety Learning and Management (CALM) intervention or usual care (UC; Craske et al., 2011; Roy-Byrne et al., 2010). This sample includes a large ethnically diverse sample of primary care patients with one or more anxiety disorders (with the exception of those with PTSD, who were excluded for the present analyses), in contrast to a number of prior studies examining specific populations (e.g., low income inner city patients; patients with specific anxiety disorders like OCD; veteran samples). Moreover, the data include a broad range of clinician-assessed diagnostic data and self-reported severity outcomes and measures of mental health, including trauma exposure assessed prior to commencing treatment. Consistent with prior literature, we hypothesized that trauma exposure would be associated with greater symptom severity (anxiety, depression, and functional impairment) relative to those without trauma exposure. We also hypothesized that trauma exposure would be a prescriptive variable, such that individuals with trauma exposure would show a diminished treatment response relative to those without trauma exposure, particularly in the UC group.

Method

Design

Data for the present analyses were drawn from the randomized controlled effectiveness trial comparing the CALM intervention (described below) to usual care (UC; clinicaltrials.gov Identifier NCT00347269; Craske et al., 2011; Roy-Byrne et al., 2010). Individuals with at least one anxiety disorder (panic disorder, social phobia (SP), generalized anxiety disorder (GAD), and/or PTSD) were recruited through primary care physicians in 17 primary care clinics from 4 U.S. cities (Seattle WA, San Diego CA, Los Angeles CA, Little Rock, AR). Intervention efficacy was determined by blinded assessments conducted at 6, 12, and 18 months after the baseline assessment.

Participants

Initial eligibility assessments were conducted on 1620 individuals. A total of 1004 primary care patients were randomized between June 1, 2006 and April 1, 2008. Randomization was conducted by an automated program at RAND using a stratified randomized blocked design (stratified by clinic and presence of comorbid depression). Eligible participants were between the ages of 18 and 75, met DSM-IV criteria for at least 1 of the previously mentioned anxiety disorders, and indicated a score of 8 or greater on the Overall Anxiety Severity and Impairment Scale (OASIS; Campbell-Sills et al., 2009). Exclusion criteria included the presence of life-threatening medical conditions, marked cognitive impairment, active suicidal intent or plan, Bipolar I disorder, current substance dependence (except alcohol and marijuana), ongoing treatment in a cognitive behavioral intervention program, and inability to speak either English or Spanish. In order to determine the effects of trauma exposure other than PTSD, individuals with PTSD were excluded from the present analyses. For examination of the relationship between trauma exposure and diagnosis at baseline, data were used from 1263 individuals who completed a clinical interview to determine eligibility for the study. For examination of the relationship between trauma exposure and demographic and clinical characteristics, and response to treatment, data from 696 individuals with complete symptom measures at baseline and 6 months were utilized (for a complete description of this data set, including missing data, see Roy-Byrne, et al., 2010).

Potential participants were referred from primary care providers at one of the participating clinic locations. In addition, some sites conducted screening with the OASIS to identify individuals who might be eligible to participate. Final eligibility was determined by a

clinician. All procedures were approved by the Institutional Review Board at each institution.

Intervention

CALM—Participants receiving the CALM intervention could opt to receive CBT, medication, or both. The CBT program consisted of 5 generic modules for anxiety treatment (self-monitoring, psychoeducation, fear hierarchy, breathing retraining, and relapse prevention), a cognitive module and 3 exposure modules that were disorder-specific (for a detailed review of treatment components see Craske et al., 2009) that were presented in a computer-guided format that directed the clinician and patient. In the case of comorbid conditions, participants completed the modules for the disorder that they indicated was most distressing and/or disabling during their first treatment session. For participants selecting medication management, medication was prescribed by the individual's primary care provider with consultation from local study psychiatrists. The algorithm for medication management was based on first-line use of selective serotonin reuptake inhibitor or serotonin noradrenalin reuptake inhibitory antidepressants. Non- or suboptimal response to medication was addressed by medication change, addition of another antidepressant or a benzodiazepine. In the case of medication management, a study clinician provided adherence monitoring and counseling on minimizing alcohol and caffeine, sleep hygiene, and behavioral activation.

Usual Care—Participants randomized to the usual care (UC) condition continued to be managed by their primary care physician. Treatment received by these individuals possibly included medication, counseling by the primary care physician, or counseling by mental health specialists based on physician referral. Contact with study personnel took place only for completion of assessments.

Assessments

Diagnostic status—The Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) was administered by a trained study clinician to determine diagnostic status and eligibility for study entry. In the course of completing this assessment, individuals were asked about exposure to potentially traumatic events involving actual or threatened death or injury (i.e., "Have you ever experienced or witnessed or had to deal with an extremely traumatic event that included actual or threatened death or serious injury to you or someone else?"). The clinicians were trained to solicit information to assure that a PTSD criterion A event was being reported. Consistent with prior studies of trauma exposure in primary care, individuals who indicated that they had experienced at least one such event were considered to be positive for trauma exposure (Bruce et al., 2001; Glover et al., 2010).

Demographics and symptom severity—After completing the MINI to determine eligibility, participants were contacted via telephone by the RAND Corporation to complete a baseline assessment. This assessment included questions about demographic characteristics including age, education, and ethnicity/race (categorized as Caucasian, Black, Hispanic, or other). General symptom severity was assessed using 12 items from the anxiety and somatization subscales of the 18-item Brief Symptom Inventory (BSI-18; we will refer to the subset used in this project as the BSI-12; Derogatis & Savitz, 2000). This assessment was the primary dependent outcome variable. The BSI and its subscales possess adequate psychometric properties, including sensitivity to change (e.g., Piersma, Reaume, & Boes, 1994). Other symptom severity measures administered that were relevant to the objectives of this study included the OASIS (Campbell-Sills et al., 2009), Patient Health Questionnaire-8 (PHQ-8, the Patient Health Questinnaire-9 excluding the suicide item; Kroenke, Spitzer, & Williams, 2001), the Sheehan Disability Scale for assessing functional impairment (SDS;

Sheehan, Harnett-Sheehan, & Raj, 1996), and the 12-item short form Heath Survey version 2 (SF-12; Ware, Kosinski, Turner-Bowker, & Gandek, 2002).

Statistical analysis

We first examined the frequency of trauma exposure and compared demographic and clinical characteristics between trauma exposed and non-trauma exposed individuals. Continuous variables were analyzed using t-tests. Categorical variables were compared using χ^2 tests. To describe the association between trauma exposure and treatment prognosis, multiple logistic regression was conducted predicting treatment response at the 6-month assessment. To compensate for multiple comparisons, we set our critical p-value in all analyses to $p < .01$.

Results

Baseline rates of trauma exposure and exposure rates by demographic variables

Of 1263 patients, 662 (53%) reported exposure to a traumatic event as defined by DSM-IV Criterion A. Demographic characteristics of trauma and no-trauma groups are in Table 1 (total N for these analyses = 696). There were no significant differences in trauma exposure based on ethnicity/race ($\chi^2(3) = 2.53$, $p = .47$), educational attainment ($\chi^2(2) = .73$ $p = .69$), or age ($t(695) = -1.85$, $p = .07$). Although the trauma exposed group tended to have a higher proportion of females, this difference did not meet the $p < .01$ threshold for statistical significance set for the analyses ($p = .048$)[i].

Mental health impact of trauma – Rates of emotional disorders

For the full sample of individuals who received a diagnostic interview (N = 1263), we examined whether or not individuals with and without trauma history differed in rates of the following disorders: agoraphobia without a history of panic disorder, panic with and without agoraphobia, OCD, major depressive disorder, dysthymia, manic or hypomanic episode, alcohol abuse or dependence, and substance abuse or dependence. Trauma exposure was significantly associated only with OCD ($\chi^2(1) = 11.53$, $p = .001$; see Table 2).

Mental health impact of trauma – Severity of emotional symptoms

For the sample of individuals with complete symptom severity data (N = 696), as compared to individuals who did not endorse trauma exposure, those who had experienced one or more traumas scored higher on the BSI somatization subscale ($t(964) = -2.71$, $p = .01$). No group differences were found for the BSI anxiety subscale ($t(964) = -2.16$, $p = .03$, ns), PHQ-8 ($t(964) = -1.14$, $p = .26$, ns), OASIS ($t(694 = -.14$, $p = .85$), Sheehan Disability Scale ($t(688 = -.14$, $p = .89$, ns) or the mental health subscale of the SF-12 ($t(694) = -1.88$, $p = .06$, ns).

Relationship between trauma exposure and treatment response at six-months

To examine the relationship between trauma exposure (those who had experienced one or more traumas compared to those with no trauma history) and treatment outcome, treatment response was defined as a 50% or more decrease in symptoms from baseline to 6-month assessment on the BSI-12, which is the threshold used in analysis of data from the main trial (Roy-Byrne et al., 2010). We examined whether or not trauma exposure was associated with differential response to CALM relative to UC (i.e., trauma exposure as a moderator of response to the CALM or UC intervention). Intervention was entered as a predictor in the first step, followed by trauma exposure in the second step and the interaction between intervention and trauma exposure in the third step. Results revealed that there was no significant relationship between trauma exposure or the interaction between trauma

exposure and intervention on outcomes (see Table 3)[1]. Means and standard deviations for pre and post-treatment BSI-12 scores are provided in Table 1.

Discussion

We first sought to examine potential demographic and clinical differences between primary care patients who had and had not been exposed to trauma (excluding those with PTSD). Findings were consistent with prior studies indicating that trauma exposure is highly prevalent in anxious primary care patients. The prevalence in this sample is similar to that reported in population surveys (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995) and other primary care samples (Alim et al., 2006). Results revealed that among the diagnoses assessed in this study, only OCD was more prevalent in trauma exposed patients than in non-exposed patients. Consistent with prior studies of the effects of trauma exposure in primary care samples (Löew et al., 2011), individuals who had experienced trauma reported more somatic symptoms than those who had not. However, no group differences were found for severity of anxiety, depression, or functional impairment. We also examined whether or not trauma exposure differentially impacted response to CALM versus UC. No interactive effect of trauma exposure and intervention condition was found on responder status at 6-months.

The present data are the first to suggest greater rates of OCD in individuals with trauma exposure within a primary care sample. Prior literature suggests that stressors often precede the emergence of OCD (Cromer, Schmidt, & Murphy, 2007; Hollingsworth, Tanguay, Grossman, & Pabst, 1980; Khanna, Rajendra, & Channabasavanna, 1988) and a number of prior case studies have indicated a potential link between trauma exposure and OCD (see for example de Silva & Marks, 2001; Pitman, 1993; Sasson et al., 2005). Given these data, researchers have posited that OCD symptoms may be expression of maladaptive attempts to regulate negative, anxiety provoking cognitions and affect that may occur after stress (Kessler et al., 1995). Extant literature exploring the link between OCD symptoms and PTSD suggests that obsessions and compulsions may emerge after trauma and reflect exaggerated attempts to regain control over the environment and ensure safety (e.g., checking locks), or alternatively may function as avoidance behaviors in relation to trauma cues (e.g., mental or behavioral rituals such as washing to avoid trauma-related thoughts; see for example de Silva & Marks, 1998; Gershuny, Baer, Radomsky, Wilson, & Jenike, 2003).

However, larger-scale studies have produced equivocal support for this relationship (Huppert et al., 2005; Grabe et al., 2001; although see Grabe et al., 2008 for an exception). In attempting to account for these discrepancies some researchers have argued that the link between trauma and OCD may be an artifact of overlapping symptoms in OCD and PTSD (i.e., persistent intrusive thoughts and ritualistic behavior to prevent feared outcomes; Huppert et al., 2005). The present data add to this literature by examining the potential increased rates of OCD in those with trauma exposure who do *not* meet criteria for PTSD. These findings suggest that there may be a link between trauma exposure and OCD, consistent with models suggesting extreme stress as a catalyst for obsessions and compulsion symptoms in vulnerable individuals. However, future work controlling for the severity of subsyndromal PTSD (using continuous measures of symptoms in those with trauma exposure) is needed to determine whether symptom overlap may partially account for this relationship.

The present findings are partially consistent with prior studies (Kipper et al., 2007; Shavitt et al., 2010) that failed to find a relationship between presence of trauma exposure and poor

[1]Results of analyses using continuous BSI-12 scores as the dependent variable produced equivalent results.

treatment prognosis. There were no differential effects of trauma exposure in patients receiving the CALM intervention relative to UC condition across both anxiety and somatic symptoms. The replication of this finding in a large sample of individuals with multiple anxiety disorders increases confidence that these findings are not merely due to insufficient power to detect an effect.

A number of limitations should be considered in interpreting these findings. First, the data pertaining to trauma exposure are cross-sectional in nature. Comorbid psychiatric conditions like OCD might precede or be a consequence of trauma exposure. For example, strict adherence to behavioral rituals or hyperfocus on the content of cognitions could potentially decrease attention to daily activities and interactions, paradoxically increasing the risk of danger in the environment. Thus, the mechanism underlying the relationship between trauma exposure and OCD is unclear at this time. Further information on chronology, as well as possible shared mechanisms like anxiety sensitivity, avoidant coping style, or dissociation, is needed to elucidate mechanisms and patterns of comorbidity in this population. Because OCD was not a treatment target in the present intervention and individuals with primary OCD were not included in this trial, it is not possible to determine the effect of trauma exposure on treatment specifically for this disorder. In addition, relatively limited information about the type, frequency, and duration of trauma exposure is available due to the methodology of the study. Thus, it is difficult to directly compare to other studies that have examined the psychiatric outcomes of specific trauma types like assault. It is possible that the overall patterns of findings may differ dependent on the type of trauma exposure experienced by the individual (e.g., interpersonal trauma versus accidents or combat). Future work in this area might include more detailed trauma assessment.

Additional limitations of the study relate to the use of data from a randomized clinical trial. The selected sample is biased in that it represents a group of individuals willing to participate in treatment and selected for specific eligibility criteria (rather than a random sample of anxious primary care patients). This sample selection may account for discrepancies found between these data and prior studies of anxious primary care patients. For example, although prior studies have found increased rates of substance use disorders in individuals with trauma exposure (Glover et al., 2010), the present analyses did not. This may be accounted for by differences in samples between this and prior studies (e.g., general trauma exposure vs. assault only in Glover et al., 2010, multiple sites of sampling vs. inner city populations, Gillespie et al., 2009). An additional explanation for discrepancies in initial severity analyses is that only individuals who enrolled in treatment and completed the 6-month follow-up were included. Thus, selection processes inherent in the completion of the clinical trial may have resulted in groups more equivalent in severity (anxiety, depression, functioning) than what would be seen if all patients initially screened had completed this assessment. The assessment of diagnostic status was completed using the MINI; although this measure corresponds well with other assessments such as the SCID, it is a brief assessment. Thus, not all Axis-I diagnoses are fully assessed, and participants are asked to retrospectively recall lifetime diagnoses which may lead to memory biases and other errors in recall. Finally, our measure of treatment-related change is limited to the BSI-12. Future studies should look at change in diagnostic status and disorder-specific measures.

Despite these limitations, findings highlight the high prevalence of trauma exposure in primary care settings. As noted by Bruce and colleagues (2001), increased awareness of the potential impact of trauma exposure and assessment by physicians is critical in detecting problems that may be associated with trauma (e.g., psychiatrically-based somatic complaints). Given the sensitive nature of trauma exposure and the high rates of avoidance and distress associated with trauma, proper screening procedures within primary care may provide access for individuals who would not otherwise seek specialty mental health care.

Moreover, findings suggest that for anxious patients who have experienced trauma but do not have PTSD, focusing on the traumatic event is not necessary for effective treatment. Instead, a clinical focus on the primary presenting problem appears sufficient to ameliorate anxiety symptoms regardless of trauma history. The lack of differential treatment response suggests that the presence of trauma does not necessarily indicate that a higher level of care is needed relative to other anxious individuals, nor that focusing on trauma is necessary for effective treatment in those with trauma exposure.

In summary, the present findings suggest that trauma exposure may be independently associated with OCD and greater somatic symptoms. Among individuals completing this randomized controlled trial of anxiety treatment, no differences in baseline severity (i.e., anxiety, depression, mental health functioning) between those with and without trauma exposure were noted. Moreover, trauma exposure did not appear to negatively affect treatment prognosis in individuals receiving the more intensive CALM program or usual care. This suggests that careful assessment of the specific presenting complaint rather than trauma exposure, per se, should dictate treatment planning.

Acknowledgments

This grand supported by funding from the National Institute of Mental Health, clinicaltrials.gov Identifier NCT00347269

References

Alim TN, Graves E, Mellman TA, Aigbogun N, Gray E, Lawson W, Charney DS. Trauma exposure, posttraumatic stress disorder and depression in an African american primary care population. Journal of the National Medical Association. 2006; 98(10):1630–1636. Retrieved from www.csa.com. [PubMed: 17052054]

Bijl RV, Ravelli A. Current and residual functional disability associated with psychopathology: Findings from the Netherlands mental health survey and incidence study (NEMESIS). Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences. 2000; 30(3): 657–668.10.1017/S0033291799001841

Breslau N, Chilcoat HD, Kessler RC, Davis GC. Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit area survey of trauma. The American Journal of Psychiatry. 1999; 156(6):902–907. Retrieved from www.csa.com. [PubMed: 10360130]

Breslau N, Davis GC, Schultz LR. Posttraumatic stress disorder and the incidence of nicotine, alcohol, and other drug disorders in persons who have experienced trauma. Archives of General Psychiatry. 2003; 60(3):289–294.10.1001/archpsyc.60.3.289 [PubMed: 12622662]

Brown TA, Antony MM, Barlow DH. Diagnostic comorbidity in panic disorder: Effect on treatment outcome and course of comorbid diagnoses following treatment. Journal of Consulting and Clinical Psychology. 1995; 63(3):408–418.10.1037/0022-006X.63.3.408 [PubMed: 7608353]

Bruce SE, Weisberg RB, Dolan RT, Machand JT, Kessler RC, Manchester G, Keller MB. Trauma and posttraumatic stress disorder in primary care patients. Primary Care Companion to the Journal of Clinical Psychiatry. 2001; 2:211–217.

Bryant RA, O'Donnell ML, Creamer M, McFarlane AC, Clark CR, Silove D. The psychiatric sequelae of traumatic injury. The American Journal of Psychiatry. 2010; 167(3):312–320.10.1176/appi.ajp.2009.09050617 [PubMed: 20048022]

Campbell-Sills L, Norman SB, Craske MG, Sullivan G, Lang AJ, Chavira DA, Stein MB. Validation of a brief measure of anxiety-related severity and impairment: The overall anxiety severity and impairment scale (OASIS). Journal of Affective Disorders. 2009; 112(1–3):92–101.10.1016/j.jad.2008.03.014 [PubMed: 18486238]

Coid J, Petruckevitch A, Chung W, Richardson J, Moorey S, Feder G. Abusive experiences and psychiatric morbidity in women primary care attenders. British Journal of Psychiatry. 2003; 183(4): 332–339.10.1192/bjp.183.4.332 [PubMed: 14519611]

Cougle JR, Timpano KR, Sachs-Ericsson N, Keough ME, Riccardi CJ. Examining the unique relationships between anxiety disorders and childhood physical and sexual abuse in the national comorbidity survey-replication. Psychiatry Research. 2010; 177(1–2):150–155.10.1016/j.psychres.2009.03.008 [PubMed: 20381878]

Craske MG, Roy-Byrne PP, Stein MB, Sullivan G, Sherbourne C, Bystritsky A. Treatment for anxiety disorders: Efficacy to effectiveness to implementation. Behaviour Research and Therapy. 2009; 47(11):931–937.10.1016/j.brat.2009.07.012 [PubMed: 19632667]

Craske MG, Stein MB, Sullivan G, Sherbourne C, Bystritsky A, Rose RD, Roy-Byrne P. Disorder-specific impact of coordinated anxiety learning and management treatment for anxiety disorders in primary care. Archives of General Psychiatry. 2011; 68(4):378–387.10.1001/archgenpsychiatry.2011.25 [PubMed: 21464362]

Cromer KR, Schmidt NB, Murphy DL. An investigation of traumatic life events and obsessive-compulsive disorder. Behaviour Research and Therapy. 2007; 45(7):1683–1691.10.1016/j.brat.2006.08.018 [PubMed: 17067548]

Derogatis, LR.; Savitz, KL. The SCL–90–R and brief symptom inventory (BSI) in primary care. Maruish, ME., editor. Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers; 2000. p. 297-334.

de Silva P, Marks M. The role of traumatic experiences in the genesis of obsessive compulsive disorder. Behaviour Research and Therapy. 1999; 37(10):41–51.

de Silva P, Marks M. Traumatic experiences, post-traumatic stress disorder and obsessive-compulsive disorder. International Review of Psychiatry. 2001; 13(3):172–180.10.1080/09540260120073993

Forbes D, Creamer M, Hawthorne G, Allen N, McHugh T. Comorbidity as a predictor of symptom change after treatment in combat-related posttraumatic stress disorder. Journal of Nervous and Mental Disease. 2003; 191(2):93–99.10.1097/00005053-200302000-00005 [PubMed: 12586962]

Fournier JC, DeRubeis RJ, Shelton RC, Hollon SD, Amsterdam JD, Gallop R. Prediction of response to medication and cognitive therapy in the treatment of moderate to severe depression. Journal of Consulting and Clinical Psychology. 2009; 77(4):775–787.10.1037/a0015401 [PubMed: 19634969]

Galea S, Ahern J, Resnick H, Kilpatrick D, Bucuvalas M, Gold J, Vlahov D. Psychological sequelae of the September 11 terrorist attacks in new york city. The New England Journal of Medicine. 2002; 346(13):982–987.10.1056/NEJMsa013404 [PubMed: 11919308]

Gershuny BS, Bear L, Radomsky AS, Wilson KA, Jenike MA. Connections among symptoms of obsessive-compulsive disorder and posttraumatic stress disorder: a case series. Behaviour Research and Therapy. 2003; 41(9):1029–41. [PubMed: 12914805]

Gillespie CF, Bradley B, Mercer K, Smith AK, Conneely K, Gapen M, Ressler KJ. Trauma exposure and stress-related disorders in inner city primary care patients. General Hospital Psychiatry. 2009; 31(6):505–514.10.1016/j.genhosppsych.2009.05.003 [PubMed: 19892208]

Glover K, Olfson M, Gameroff MJ, Neria Y. Assault and mental disorders: A cross sectional study of urban adult primary care patients. Psychiatric Services. 2010; 61(10):1018–1023.10.1176/appi.ps.61.10.1018 [PubMed: 20889641]

Grabe HJ, Meyer C, Hapke U, Rumpf H, Freyberger HJ, Dilling H, Ulrich J. Lifetime-comorbidity of obsessive-compulsive disorder and subclinical obsessive-compulsive disorder in northern Germany. European Archives of Psychiatry and Clinical Neuroscience. 2001; 251(3):130–135.10.1007/s004060170047 [PubMed: 11697574]

Grabe HJ, Ruhrmann S, Spitzer C, Josepeit J, Ettelt S, Buhtz F, Freyberger HJ. Obsessive compulsive disorder and posttraumatic stress disorder. Psychopathology. 2008; 41(2):129–134.10.1159/000112029 [PubMed: 18059115]

Green BL, Goodman LA, Krupnick JL, Corcoran CB, Petty RM, Stockton P, Stern NM. Outcomes of single versus multiple trauma exposure in a screening sample. Journal of Traumatic Stress. 2000; 13(2):271–286.10.1023/A:1007758711939 [PubMed: 10838675]

Hollingsworth CE, Tanguay PE, Grossman L, Pabst P. Long-term outcome of Obsessive-Compulsive Disorder in childhood. Journal of the American Academy of Child Psychiatry. 1980; 19(1):134–144.10.1016/S0002-7138(09)60658-0 [PubMed: 7365165]

Huppert JD, Moser JS, Gershuny BS, Riggs DS, Spokas M, Filip J, Foa EB. The relationship between obsessive-compulsive and posttraumatic stress symptoms in clinical and non-clinical samples. Journal of Anxiety Disorders. 2005; 19(1):127–136.10.1016/j.janxdis.2004.01.001 [PubMed: 15488372]

James IA, Southam L, Blackburn IM. Schemas revisited. Clinical Psychology & Psychotherapy. 2004; 11(6):369–377.10.1002/cpp.423

Kartha A, Brower V, Saitz R, Samet JH, Keane TM, Liebschutz J. The impact of trauma exposure and post-traumatic stress disorder on healthcare utilization among primary care patients. Medical Care. 2008; 46(4):388–393.10.1097/MLR.0b013e31815dc5d2 [PubMed: 18362818]

Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Archives of General Psychiatry. 1995; 52(12):1048–1606. [PubMed: 7492257]

Khanna S, Rajendra PN, Channabasavanna SM. Life events and onset of obsessive compulsive disorder. International Journal of Social Psychiatry. 1988; 34(4):305–309.10.1177/002076408803400408 [PubMed: 3235293]

Khoury L, Tang YL, Bradley B, Cubells JF, Ressler KJ. Substance use, childhood traumatic experience, and posttraumatic stress disorder in an urban civilian population. Depression and Anxiety. 2010; 27(12):1077–1086.10.1002/da.20751 [PubMed: 21049532]

Kipper L, Blaya C, Wachleski C, Dornelles M, Salum GA, Heldt E, Manfro GG. Trauma and defense style as response predictors of pharmacological treatment in panic patients. European Psychiatry. 2007; 22(2):87–91.10.1016/j.eurpsy.2006.09.009 [PubMed: 17188844]

Kirmayer LJ, Robbins JM, Dworkind M, Yaffe MJ. Somatization and the recognition of depression and anxiety in primary care. The American Journal of Psychiatry. 1993; 150(5):734–741. Retrieved from www.csa.com. [PubMed: 8480818]

Kraemer HC, Wilson GT, Fairburn CG, Agras WS. Mediators and moderators of treatment effects in randomized clinical trials. Archives of General Psychiatry. 2002; 59(10):877–883.10.1001/archpsyc.59.10.877 [PubMed: 12365874]

Kroenke K, Spitzer RL, Williams JBW. The PHQ-9: Validity of a brief depression severity measure. Journal of General Internal Medicine. 2001; 16(9):606–613.10.1046/j.1525-1497.2001.016009606.x [PubMed: 11556941]

Kroenke K, Spitzer RL, Williams JB, Monahan PO, Lowe B. Anxiety disorders in Primary care: prevalence, impairment, comorbidity, and detection. Annals of Intern Medicine. 2007; 146:317–325.

Löew B, Kroenke K, Spitzer RL, Williams JBW, Mussell M, Rose M, Spitzer C. Trauma exposure and Posttraumatic Stress Disorder in primary care patients: Cross sectional criterion standard study. Journal of Clinical Psychiatry. 2011; 72(3):304–12. [PubMed: 20584519]

Marshall RD, Olfson M, Hellman F, Blanco C, Guardino M, Struening EL. Comorbidity, impairment, and suicidality in subthreshold PTSD. The American Journal of Psychiatry. 2001; 158(9):1467–1473.10.1176/appi.ajp.158.9.1467 [PubMed: 11532733]

McQuaid JR, Pedrelli P, McCahill ME, Stein MB. Reported trauma, post traumatic stress disorder and major depression among primary care patients. Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences. 2001; 31(7):1249–1257.10.1017/S0033291701004202

Mellman TA, Aigbogun N, Graves RE, Lawson WB, Alim TN. Sleep paralysis and trauma, psychiatric symptoms and disorders in an adult african american population attending primary medical care. Depression and Anxiety. 2008; 25(5):435–440.10.1002/da.20311 [PubMed: 17607754]

Michelson L, June K, Vives A, Testa S, Marchione N. The role of trauma and dissociation in cognitive-behavioral psychotherapy outcome and maintenance for panic disorder with agoraphobia. Behaviour Research and Therapy. 1998; 36(11):1011–1050.10.1016/S0005-7967(98)00073-4 [PubMed: 9737056]

North CS, Nixon SJ, Shariat S, Mallonee S, McMillen JC, Spitznagel EL, Smith EM. Psychiatric disorders among survivors of the Oklahoma city bombing. JAMA: Journal of the American Medical Association. 1999; 282(8):755–762.10.1001/jama.282.8.755

Piersma HL, Reaume W, Boes MJL. The Brief Symptom Inventory (BSI) as an outcome measure for adult psychiatric inpatients. Journal of Clinical Psychology. 1994; 50(4):555–563. [PubMed: 7983203]

Pini S, Perkonnig A, Tansella M, Wittchen H. Prevalence and 12-month outcome of threshold and subthreshold mental disorders in primary care. Journal of Affective Disorders. 1999; 56(1):37–48.10.1016/S0165-0327(99)00141-X [PubMed: 10626778]

Pitman RK. Posttraumatic obsessive-compulsive disorder: A case study. Comprehensive Psychiatry. 1993; 34(2):102–107.10.1016/0010-440X(93)90054-8 [PubMed: 8485978]

Roy-Byrne P, Craske MG, Sullivan G, Rose RD, Edlund MJ, Lang AJ, Stein MB. Delivery of evidence-based treatment for multiple anxiety disorders in primary care: A randomized controlled trial. JAMA: Journal of the American Medical Association. 2010; 303(19):1921–1928.10.1001/jama.2010.608

Sasson Y, Dekel S, Nacasch N, Chopra M, Zinger Y, Amital D, Zohar J. Posttraumatic obsessive-compulsive disorder: A case series. Psychiatry Research. 2005; 135(2):145–152.10.1016/j.psychres.2004.05.026 [PubMed: 15922457]

Scholing A, Emmelkamp PMG. Prediction of treatment outcome in social phobia: A cross-validation. Behaviour Research and Therapy. 1999; 37(7):659–670.10.1016/S0005-7967(98)00175-2 [PubMed: 10402691]

Seivewright H, Tyrer P, Johnson T. Prediction of outcome in neurotic disorder: A 5 year prospective study. Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences. 1998; 28(5):1149–1157.10.1017/S0033291798007119

Serrano-Blanco A, Palao DJ, Luciano JV, Pinto-Meza A, Luján L, Fernández A, Haro JM. Prevalence of mental disorders in primary care: Results from the diagnosis and treatment of mental disorders in primary care study (DASMAP). Social Psychiatry and Psychiatric Epidemiology. 2010; 45(2):201–210.10.1007/s00127-009-0056-y [PubMed: 19452110]

Shavitt RG, Valério C, Fossaluza V, da Silva EM, Cordeiro Q, Diniz JB, Miguel EC. The impact of trauma and post-traumatic stress disorder on the treatment response of patients with obsessive-compulsive disorder. European Archives of Psychiatry and Clinical Neuroscience. 2010; 260(2):91–99.10.1007/s00406-009-0015-3 [PubMed: 20077119]

Sheehan DV, Harnett-Sheehan K, Raj BA. The measurement of disability. International Clinical Psychopharmacology. 1996; 11(S3):89–95.10.1097/00004850-199606003-00015 [PubMed: 8923116]

Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, Dunbar GC. The mini-international neuropsychiatric interview (M.I.N.I): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. Journal of Clinical Psychiatry. 1998; 59(S20):22–33. Retrieved from www.csa.com. [PubMed: 9881538]

Simon NM, Herlands NN, Marks EH, Mancini C, Letamendi A, Li Z, Stein MB. Childhood maltreatment linked to greater severity and poorer quality of life and function in social anxiety disorder. Depression and Anxiety. 2009; 26(11):1027–1032.10.1002/da.20604 [PubMed: 19750554]

Simpson TL, Miller WR. Concomitance between childhood sexual and physical abuse and substance use problems: A review. Clinical Psychology Review. 2002; 22(1):27–77.10.1016/S0272-7358(00)00088-X [PubMed: 11793578]

Solvason HB, Ernst H, Roth W. Predictors of response in anxiety disorders. Psychiatric Clinics of North America. Special Issue: Drug Therapy: Predictors of Response. 2003; 26(2):411–433.10.1016/S0193-953X(03)00027-3

Stein DJ, Montgomery SA, Kasper S, Tanghoj P. Predictors of response to pharmacotherapy with citalopram in obsessive-compulsive disorder. International Clinical Psychopharmacology. 2001; 16(6):357–361.10.1097/00004850-200111000-00007 [PubMed: 11712625]

Tsao JCI, Lewin MR, Craske MG. The effects of cognitive-behavior therapy for panic disorder on comorbid conditions. Journal of Anxiety Disorders. 1998; 12(4):357–371.10.1016/S0887-6185(98)00020-6 [PubMed: 9699119]

Wang PS, Lane M, Olfson M, Pincus HA, Wells KB, Kessler RC. Twelve month use of mental health services in the United States: Results from the national comorbidity survey replication. Archives of General Psychiatry. 2005; 62(6):629–640.10.1001/archpsyc.62.6.629 [PubMed: 15939840]

Ware, JE.; Kosinski, M.; Turner-Bowker, DM.; Gandek, B. How to Score Version 2 of the SF-12™ Health Survey (With a Supplement Documenting Version 1). Lincoln (RI): QualityMetric Incorporated; 2002.

Weisberg RB, Dyck I, Culpepper L, Keller MB. Psychiatric treatment in primary care patients with anxiety disorders: A comparison of care received from primary care providers and psychiatrists. The American Journal of Psychiatry. 2007; 164(2):276–282.10.1176/appi.ajp.164.2.276 [PubMed: 17267791]

Table 1

Baseline demographic variables and clinical characteristics by trauma exposure group

	Trauma Exposed (N = 378)	Not trauma exposed (N = 318)
Race:		
Hispanic	69 (18%)	59 (19%)
Black	30 (8%)	35 (11%)
White	230 (61%)	187 (59%)
Other	49 (13%)	37 (11%)
Years of education[+]:		
Less than 12 years	15	14
12 years	66	46
More than 12 years	297	258
Gender:		
Female	277	217
Male	101	101
Mean Age:	44.6 (13.6)	42.7 (13.1)
Baseline BSI-12		
CALM	16.4 (9.1)	13.8 (7.0)
UC	15.4 (8.2)	14.6 (8.8)
6-month BSI-12		
CALM	8.7 (7.9)[*]	8.3 (8.0)[*]
UC	10.8 (7.5)[*]	10.0 (8.8)[*]

[+] Education data missing for one patient.

Note: Table represents N in each cell for race, mean years of education, and gender. Cells represent means and standard deviation for age.

[*] Results of paired-samples t-tests comparing baseline to 6-month BSI-12 scores indicated a significant decrease over time for all groups, *p*s < .001

Table 2

Association of trauma exposure with emotional disorder (N = 1263)

Variable	% (N) trauma exposed	% (N) non-trauma exposed	Chi squared value	p-value
Current agoraphobia w/o panic	13 (82)	11 (64)	1.99	.37
Panic with agoraphobia	27 (179)	26 (155)	1.30	.52
Panic without agoraphobia	9 (59)	8 (49)	1.25	.54
Lifetime history of panic disorder	51 (336)	44 (263)	6.18	.013
Current OCD	9 (56)	4 (23)	11.53	.001
Current MDD	58 (383)	54 (324)	2.0	.16
Recurrent MDD	28 (183)	26 (158)	.29	.59
Current dysthymia	5 (33)	5 (30)	< .001	.99
Current hypomanic episode	3 (20)	3 (16)	.15	.70
Current manic episode	2 (13)	1 (7)	1.29	.26
Lifetime history of hypomanic episode(s)	6 (39)	5 (27)	1.24	.27
Lifetime history of manic episode(s)	3 (21)	2 (11)	2.30	.13
Current Alcohol Abuse	4 (29)	6 (34)	1.08	.30
Current Alcohol Dependence	5 (36)	5 (31)	.05	.83
Lifetime history of alcohol abuse	5 (32)	7 (39)	1.63	.20
Lifetime history of alcohol dependence	7 (45)	6 (35)	.50	.48
Current Substance Abuse	3 (17)	1 (7)	3.33	.07
Current Substance Dependence	2 (12)	1 (5)	2.28	.13
Lifetime history of substance abuse	4 (23)	2 (10)	4.06	.04
Lifetime history of substance dependence	3 (20)	2 (9)	3.26	.07

Table 3

Hierarchical Logistic Regression Analyses of Intervention (CALM, UC), Trauma Exposure, and the interaction of Intervention and Trauma Exposure Predicting Responder status on the BSI

Moderator				
	B	***SE B***	**OR [95% CI]**	**Nk R^2**
Step 1				.02***
Intervention	.54***	.164	1.72 [1.23, 2.33]	
Step 2				.02***
Intervention Status	.54***	.16	1.72 [1.27, 2.33]	
Trauma Exposure	.06	.16	1.06 [0.78, 1.44]	
Step 3				.02***
Intervention	.44	.23	1.55 [.99, 2.4]	
Trauma Exposure	−.05	.23	0.95 [.61, 1.49]	
Intervention x Trauma Exposure	.20	.39	1.22 [.66, 2.24]	

Note. OR = odds ratio, CI = confidence interval Nk = Nagerlkerke

** $p < .01$.

*** $p < 001$.