Research report

Relations between the underlying dimensions of PTSD and major depression using an epidemiological survey of deployed Ohio National Guard soldiers

Tracey L. Biehn, Ateka Contractor, Jon D. Elhai, Marijo Tamburrino, Thomas H. Fine, Marta R. Prescott, Edwin Shirley, Philip K. Chan, Renee Slembarski, Israel Liberzon, Joseph R. Calabrese, Sandro Galea

Article info

Article history:
Received 16 May 2012
Accepted 12 June 2012
Available online 10 September 2012

Keywords:
Posttraumatic stress disorder
Major depressive disorder
Factor analysis
Military veterans
Comorbidity

1. Introduction

There are substantial rates of comorbidity between posttraumatic stress disorder (PTSD) and major depression. For example, the National Comorbidity Survey (NCS) found that 48–55% of individuals diagnosed with PTSD were also diagnosed with major depression in their lifetimes (Kessler et al., 1995). While the comorbidity between PTSD and depression is well established (Elhai et al., 2011a; Keane and Kaloupek, 1997), few studies have examined how the underlying dimensions of PTSD and depression are most related to each other in order to further understand the high comorbidity rates.

Several hypotheses have been proposed to explain the significant comorbidity between PTSD and major depressive disorder. We focus on two hypotheses in particular. First, several symptoms overlap between DSM-IV major depressive disorder (MDD) and PTSD (i.e., difficulties with sleep, concentration and anhedonia). As a consequence, satisfying criteria for one of these two disorders places an individual at substantial risk of being diagnosed with the other disorder merely by virtue of these overlapping symptoms (Spitzer et al., 2007). Second, there may be a shared underlying latent association between depression and PTSD. Watson (2005) proposed that mood and anxiety disorders are defined by a higher order negative affect factor which subsumes a broad range of negative emotional states including fear, anger, and sadness. Watson argued that this higher order...
factor accounts for the high rates of comorbidity among mood and anxiety disorders, such as major depression and PTSD.

Recently, researchers have investigated the overlapping symptoms hypothesis of PTSD’s comorbidity. Spitzer et al. (2007) suggested that removing the overlapping PTSD–depression items should result in a more pure, less comorbid PTSD diagnostic construct. However, a study by Elhai et al. (2008) found instead that removing overlapping items had negligible effects on PTSD’s comorbidity rates (after removing overlapping items, comorbidity rates dropped nominally from 54.72% to 54.41% in the NCS Replication dataset). Similar results were found in a study of military veterans by Grubaugh et al. (2010) and a study of nationally representative, trauma-exposed adolescents conducted by Ford et al. (2009). Thus symptom overlap does not seem to primarily account for the PTSD–MDD comorbidity.

The shared latent mechanism hypothesis has been investigated as well, by examining PTSD’s robust, empirically supported latent factor of dysphoria that is conceptually similar to the general negative affect construct (Simms et al., 2002). We discuss the dysphoria construct more extensively below in the context of factor analysis. Factor analysis is a relatively under-utilized approach to examining issues of comorbidity between mental disorders. The use of factor analysis can help to elucidate the nature of comorbidity by examining which underlying factors of a disorder are more highly correlated with factors of another similar mental disorder. This method can be used to test the construct validity of a disorder (e.g., its uniqueness as a disorder) in a more refined manner than by examining comorbidity between crudely measured, observed diagnostic variables. Furthermore, this research is particularly timely given that a new edition of the DSM is currently being developed.

1.1. PTSD’s factor structure

There are two models of underlying PTSD symptoms that have received substantial empirical support. One model proposed by King et al. (1998) comprises the following four intercorrelated factors: re-experiencing, effortful avoidance, emotional numbing, and hyperarousal. This model is essentially identical to DSM-IV’s conceptualization of PTSD except that the avoidance and numbing symptom cluster (PTSD’s Criterion C) is separated into distinct avoidance and numbing factors, supported by empirical research (reviewed by Asmundson et al., 2004). The other empirically supported PTSD model is the dysphoria model proposed by Simms et al. (2002), comprising the following four intercorrelated factors: re-experiencing, avoidance, dysphoria, and hyperarousal. In this model, the numbing items are combined with three hyperarousal items (difficulties with sleep, concentration difficulties, and irritability) to form a dysphoria factor which involves symptoms of emotional distress common to mood and anxiety disorders (reviewed in Watson, 2005). In this model of PTSD, the hyperarousal factor only contains anxious- arousal items (hyper-vigilance and exaggerated startle response) which are more characteristic of fear based disorders. Both models have been extensively studied using confirmatory factor analysis (CFA), demonstrating good fit among different trauma exposed samples and using a variety of PTSD instruments (reviewed in Elhai and Palmieri, 2011; Yufik and Simms, 2010).

One specific research question that recent empirical studies have tested is the notion that PTSD’s dysphoria factor drives PTSD’s association with external measures of depression and general emotional distress. Several studies have found support for this notion (Elklit et al., 2010; Forbes et al., 2010; Simms et al., 2002). However, other studies have found that in contrast to other factors of PTSD, PTSD’s dysphoria is no more related to depression and distress (Marshall et al., 2010; Miller et al., 2010). Importantly, these studies used crude, unitary measures of depression and distress rather than examining these constructs in a more refined manner to better understand the PTSD–depression relationship.

1.2. Depression’s factor structure

There is less research regarding the factor structure of major depressive disorder, and the resulting factor structure often differs depending on the specific depression instrument used to assess depressive symptoms. In the present study, the Patient Health Questionnaire-9 (PHQ-9) was used to examine depression’s factor structure. The PHQ-9 is a widely used self-report measure of depression and maps directly onto DSM-IV symptom criteria for a major depressive episode (MDE) (Kroenke et al., 2001). Although the PHQ-9 has been empirically well-researched, few studies have analyzed its factor structure using CFA. Prior studies have used exploratory factor analyses (EFA) (Cameron et al., 2008; Dum et al., 2008; Huang et al., 2006; Kalpakjian et al., 2009; Krause et al., 2010) and CFA (Baas et al., 2011; Krause et al., 2008) to assess the PHQ-9’s symptom structure. Most PHQ-9 factor analytic studies either support a unidimensional depression model (Baas et al., 2011; Cameron et al., 2008; Dum et al., 2008; Kalpakjian et al., 2009) or a two-factor model of somatic and non-somatic/affective dimensions (Krause et al., 2008; 2010; Richardson and Richards, 2008).

The only study, to our knowledge, that has empirically tested several PHQ-9 depression factor models simultaneously with objective statistical criteria using CFA was a study conducted by Elhai et al. (in press). Overall, this and other studies have found the most support for a two-factor model, with one factor comprising five somatic items (sleep changes, appetite disturbances and feeling tired, difficulty concentrating and psychomotor changes) and the other factor primarily based on non-somatic or affective items (anhedonia, depressed mood, suicidal thoughts, and feeling bad about oneself) (Elhai et al., in press; Krause et al., 2010; Richardson and Richards, 2008).

1.3. Relationship between the factor structure of depression and PTSD

Despite PTSD’s high comorbidity with major depressive disorder, lacking in the literature is a more refined analysis of the PTSD–depression relationship by exploring relations between the latent factors of PTSD and MDD. In only one recent study, evidence demonstrated that PTSD’s dysphoria factor was strongly related to the Center for Epidemiologic Studies-Depression Scale’s (CES-D) factors of somatic problems and negative affect, indicating that the shared variance between the two comorbid disorders may be best accounted for by the dysphoria symptoms present in the PTSD diagnosis (Elhai et al., 2011b). Specifically, this study found that the PTSD dysphoria factor demonstrated a strong relationship with the CES-D’s depressive affect (r=.77) and somatic problems (r=.84) factors, compared to the positive affect (r=−.45) and interpersonal problems (r=.65) factors. Although this study provides a better understanding of the relationship between the latent factors of PTSD and MDD, the CES-D is a 20-item depression instrument that does not directly map onto DSM-IV’s major depressive disorder criteria. Therefore, it is unknown whether these study results are generalizable to a DSM-IV major depression-based instrument that would be more likely used to support a MDD diagnosis.

1.4. Study aims

The purpose of the current study was to replicate and extend findings by Elhai et al. (2011b) to further examine the
relationship between the latent factors of PTSD and depression in a sample of war-exposed military veterans. At present, this is only the second known study to address the comorbidity of PTSD and depression by analysis at the latent level. The Simms et al. (2002) dysphoria model was used to model PTSD symptoms given that this model comprises a general negative affect component which is conceptually similar to depression. Krause et al.’s (2008) two-factor depression model of somatic and non-somatic factors was used to analyze the depression factor structure, given that this model has received the most empirical support (Elhai et al., in press). We used the PTSD Checklist (PCL) to measure PTSD symptoms, and the PHQ-9 to measure major depression symptoms.

We were interested in testing if the dysphoria and hyperarousal factors of PTSD were more related to depression’s somatic vs. non-somatic factor. Four specific hypotheses were tested in particular. The first hypothesis was that PTSD’s dysphoria factor would correlate most strongly with depression’s somatic than non-somatic factor. The dysphoria factor comprises both somatic items and non-somatic items (e.g., difficulty concentrating, feeling emotionally numb). We hypothesized that dysphoria would correlate more strongly with the somatic factor of depression given that a previous study found that PTSD’s dysphoria correlated more strongly with depression’s somatic complaints factor (Elhai et al., 2011b). The second hypothesis was that the two-item PTSD hyperarousal factor (i.e., exaggerated startle response, hypervigilance) would correlate more strongly with MDD’s somatic than non-somatic factor given that these items are somatic in nature. However, in Hypothesis 3, we expected that depression’s somatic factor would more strongly correlate with PTSD’s dysphoria factor than with the hyperarousal factor given that the dysphoria factor contains more somatic-related items (PTSD items D1–D3). Finally, as our fourth hypothesis, we expected that neither re-experiencing nor avoidance factors of PTSD would be differentially related to the somatic or non-somatic factors of depression, given the lack of conceptual similarities between these constructs.

2. Method

2.1. Participants and procedure

The present study was part of the Ohio Army National Guard Mental Health Initiative (OHARNG MHI), a longitudinal prospective study of mental health among National Guard soldiers in Ohio. All members of the Ohio National Guard who served between July 2008 and February 2009 were invited to participate in the telephone interview portion of the study. There were 12,225 Guard members with a valid mailing address who were invited to participate (345 individuals were excluded for having no mailing address). Among the pool of potential subjects, 1013 (8.3%) declined to participate, 1130 (10.1%) did not have a telephone number listed with the Guard, and 4568 (31.8%) did not have a correct or working phone number. Among the remaining 6514 Guard members (58.1%), the following individuals were excluded: 187 (2.8%) based on the age eligibility restrictions, 1364 (20.9%) declined to participate, 31 (.4%) for having English language or hearing difficulties, and 2316 (35.5%) for not being contacted before the cohort was closed to new recruitment. Of the remaining 2616 subjects, one subject was excluded for missing as many as six items on the PHQ-9, leaving 2615 participants. Finally, we only included participants who reported being deployed and further reported a deployment related trauma, leaving an effective dataset of 1266.

Study enrollment began in November 2008 and ended in November 2009. The National Guard Bureau, Office of Human Research Protections of the U.S. Army Medical Research and Materiel Command, along with the institutional review boards of University Hospitals Case Medical Center, University of Toledo, and Columbia University, approved the study with written informed consent waived in lieu of verbal consent by telephone.

Among the 1266 remaining subjects, the average age of participants was 33 years ($SD=8.81$) and ranged from 18 to 60 years. The majority of participants were male ($n=1144, 90.4%$) and identified their racial background as primarily Caucasian ($n=1127, 89.0%$) or African–American ($n=81, 6.4%$). Only 16 participants identified themselves as Hispanic (1.3%). The majority of participants had at a minimum received a high school diploma or its equivalent ($n=1260, 99.5%$). Many had attended some college or technical training ($n=636, 50.2%$) or graduated from college ($n=243, 19.2%$). There were 873 participants working full-time (69.0%), 103 participants working part-time (8.1%), 185 unemployed (14.6%), and 80 who were of student status (6.3%). There were 333 participants (26.3%) who had a household income greater than $80,000. Income of $20,000 or less was reported by 84 participants (6.6%), between $20,001 and $40,000 by 276 respondents (21.8%), $40,001 to $60,000 by 311 participants (24.6%), and between $60,001 and $80,000 by 236 (18.6%). The average length of time participants served in the military was 12.7 years ($SD=7.81$). All participants had deployed at least once, with the average number of deployments being 2.02 ($SD=1.76$). For the majority of participants ($n=742, 58.6%$), the most recent deployment was in support of Operation Iraqi Freedom (OIF) or Operation Enduring Freedom (OEF). There were 503 (39.7%) who most recently deployed to an area of non-Conflict.

The most prevalent traumatic events experienced by participants were exposure to combat ($n=999, 78.8%$), sudden and unexpected death of a loved one or close friend ($n=874, 69.0%$), witnessing someone being killed or injured ($n=968, 55.1%$), and witnessing severe human suffering ($n=598, 47.2%$). There were 240 participants (19.0%) who reported that receiving incoming hostile fire was their worst deployment related trauma. The other most frequently nominated worst deployment-related traumas included experiencing combat or exposure to a war zone ($n=87, 6.9%$), and experiencing a sudden death of a close friend or loved one ($n=69, 5.5%$).

2.2. Instrumentation

2.2.1. Computer-assisted telephone interview (CATI)

A CATI was conducted for all participants by trained professionals at the survey research firm Abt SRBI, Inc., to assess demographic characteristics and mental health functioning using standardized questionnaires.

2.2.2. PHQ-9

Participants completed the PHQ-9 (Spitzer et al., 1994). Traditionally, the PHQ-9 measures depression symptoms over the previous two weeks based on the DSM-IV major depressive episode symptom criteria, but for this study, the instructions were modified in order to query depression symptoms over the course of the respondent’s lifetime, as done by Cannon et al. (2007). The PHQ-9 uses a Likert-type scale with four response options ranging from 0 = “Not at all” to 3 = “Nearly every day” to assess symptom severity. A study by Kroenke et al. (2001) examined the validity of using the PHQ-9 to detect and assess for depression, and found that internal consistency ranged from .86 to .89. Diagnostic validity was demonstrated in detecting an MDD diagnosis based on structured diagnostic interviews. Lastly, construct validity was reflected in the association of PHQ-9 severity scores and measures of functional
status, number of disability days and difficulties based on symptoms (Kroenke et al., 2001).

2.2.3. PCL-C

Participants also completed the PTSD Checklist – Civilian Version (PCL-C). The PCL-C was adapted so that participants were asked to anchor their PTSD ratings to one’s self-nominated worst deployment trauma. The PCL is a self-report measure which maps onto the DSM-IV symptom criteria for PTSD. There are 17 symptoms assessed by the PCL, and respondents indicate how distressed they were by each symptom over the past month by rating items on a five-point Likert-type scale (1 = “not at all” to 5 = “extremely”); however, in our study we assessed lifetime symptoms since the worst trauma. The PCL has demonstrated adequate reliability (α = .94; test–retest r = .88) in various trauma-exposed populations (Ruggiero et al., 2003), and total scores were found to highly correlate (r range = .8–.9) with those from structured PTSD diagnostic interviews in military veterans (Forbes et al., 2001). The psychometric properties of the PCL are reviewed in articles by McDonald and Calhoun (2010) and Wilkins et al. (2011).

2.3. Analysis

There were nominal amounts of missing items from the PCL and PHQ measures. There were 1243 complete cases (98.2%) for the PCL and 1240 complete cases from the PHQ (97.9%). Missing data were estimated with multiple imputation procedures using an iterative Markov chain Monte Carlo method with the Gibbs Sampler procedure (in SPSS’s Version 17 Missing Value Analysis software) to estimate missing item-level PCL and PHQ data, generated across 10 imputed datasets.

The data were screened for univariate and multivariate non-normality. There were several PCL and PHQ items with skewness and kurtosis values greater than 2.0, which indicated a non-normal univariate distribution. Further, Mardia’s multivariate normality (Satorra and Bentler, 2001). In the first CFA, we examined PTSD’s dysphoria model, with PCL items 1–5 specified to load onto the re-experiencing factor, PCL items 6 and 7 on the avoidance factor, PCL items 8–15 on the dysphoria factor, and PCL items 16 and 17 on the hyperarousal factor. The PCL items were treated as continuously scaled items. Next, Krause’s depression model was examined using the PHQ items. PHQ items 1, 2, 6, and 9 were specified to load onto the non-somatic factor, and PHQ items 3, 4, 5, 7, and 8 on the somatic factor. The PHQ items were also treated as continuously scaled items, using MLM estimation. A CFA was then conducted to examine the combined PTSD dysphoria and depression model, with all factors allowed to correlate.

Wald’s chi-square test of parameter constraints was used, which tests the null hypothesis that the difference between two correlations would be zero; we used an alpha level of .01 to control for Type I error. These analyses were conducted to determine if specific PTSD dysphoria model factors were more highly correlated with either the non-somatic or somatic factors of the depression model. Specifically, we tested whether the PTSD’s dysphoria factor was more related to depression’s somatic than non-somatic factor (Hypothesis 1). Likewise, we next tested whether the PTSD’s hyperarousal factor was more related to depression’s somatic than non-somatic factor (Hypothesis 2). Additionally, we tested whether depression’s somatic factor would be more related to PTSD’s dysphoria than hyperarousal factor (Hypothesis 3). Furthermore, we tested whether PTSD’s re-experiencing and avoidance factors (separately) were differentially related to depression’s somatic vs. non-somatic factors (Hypothesis 4).

3. Results

The average PHQ total score among participants was 5.85 (SD = 6.18), and the average PCL score was 29.86 (SD = 14.60). A PCL cutoff score of 50 in military veterans best discriminates between those with and without PTSD (McDonald and Calhoun, 2010). Kroenke et al. (2001) reported that PHQ-9 scores greater than 10 result in a sensitivity of 88% and specificity of 88% for detecting major depressive disorder.

CFA results from the PTSD dysphoria model indicate that the model fit well, S–B $\chi^2(113, N = 1266)$ = 569.53, p < .001, CFI = .95, TLI = .93. RMSEA = .06, SRMR = .03. Similar well-fitting results were obtained from Krause’s 2-factor depression model, $\chi^2(286) = 131.93, p < .001, CFI = .95, TLI = .94, RMSEA = .06, SRMR = .04$. The 6-factor combined model also fit the data well, $\chi^2(284, N = 1266)$ = 1115.407, p < .001, CFI = .93, TLI = .93, RMSEA = .05, SRMR = .04.

Wald’s tests of parameter constraints were conducted in order to test our hypotheses regarding the differential relationship between the somatic and non-somatic factors of depression with the four PTSD factors. Results indicated that PTSD’s dysphoria factor was more strongly correlated with depression’s somatic factor ($r = .69$) than with the non-somatic factor ($r = .59$), $\chi^2(1) = 3.323, p < .048$, as expected (Hypothesis 1). PTSD’s hyperarousal factor was also more correlated with depression’s somatic factor ($r = .51$) than with the non-somatic factor ($r = .34$), $\chi^2(1) = 21.731, p < .001$ (Hypothesis 2) (Table 1).

Next, to assess Hypothesis 3, we assessed whether depression’s somatic factor was more related to PTSD’s dysphoria than hyperarousal factor. Results indicated that the depression somatic factor was more strongly correlated with PTSD’s dysphoria ($r = .69$) than with hyperarousal ($r = .51$), $\chi^2(1) = 64.482, p < .001$ (see Table 2).

<table>
<thead>
<tr>
<th>Correlation</th>
<th>Value</th>
<th>Wald chi-square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Re-experiencing and somatic</td>
<td>$r = .544$</td>
<td>$\chi^2(1) = 3.306, p = .069$</td>
</tr>
<tr>
<td>Re-experiencing and non-somatic</td>
<td>$r = .459$</td>
<td></td>
</tr>
<tr>
<td>Avoidance and somatic</td>
<td>$r = .537$</td>
<td>$\chi^2(1) = 6.502, p = .011$</td>
</tr>
<tr>
<td>Avoidance and non-somatic</td>
<td>$r = .425$</td>
<td></td>
</tr>
<tr>
<td>Dysphoria and somatic</td>
<td>$r = .689$</td>
<td>$\chi^2(1) = 3.323, p = .048$</td>
</tr>
<tr>
<td>Dysphoria and non-somatic</td>
<td>$r = .592$</td>
<td></td>
</tr>
<tr>
<td>Hyperarousal and somatic</td>
<td>$r = .505$</td>
<td>$\chi^2(1) = 21.731, p &lt; .001$</td>
</tr>
<tr>
<td>Hyperarousal and non-somatic</td>
<td>$r = .337$</td>
<td></td>
</tr>
</tbody>
</table>
Table 2  
Correlations for the Somatic factor with Dysphoria and Avoidance.

<table>
<thead>
<tr>
<th>Correlation between factors</th>
<th>r Value</th>
<th>Wald Chi-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic and dysphoria</td>
<td>r=.689</td>
<td>χ²(1)=64.482, p &lt; .001</td>
</tr>
<tr>
<td>Somatic and avoidance</td>
<td>r=.537</td>
<td></td>
</tr>
<tr>
<td>Somatic and dysphoria</td>
<td>r=.689</td>
<td>χ²(1)=53.330, p &lt; .001</td>
</tr>
<tr>
<td>Somatic and hyperarousal</td>
<td>r=.505</td>
<td></td>
</tr>
</tbody>
</table>

Table 3  
Correlations among PTSD and depression factors.

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Re-experiencing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoidance</td>
<td>.898</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysphoria</td>
<td>.846</td>
<td>.837</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperarousal</td>
<td>.772</td>
<td>.717</td>
<td>.740</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatic</td>
<td>.544</td>
<td>.537</td>
<td>.689</td>
<td>.505</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Somatic</td>
<td>.459</td>
<td>.425</td>
<td>.592</td>
<td>.337</td>
<td>.862</td>
<td></td>
</tr>
</tbody>
</table>

Unexpectedly, in contrast to Hypothesis 4, the PTSD's avoidance factor was more strongly correlated with depression's somatic factor (r=.54) than to the non-somatic factor (r=.425), χ²(1)=6.502, p < .011.

4. Discussion

The purpose of this study was to clarify the nature of the comorbid relationship between depression and PTSD by examining the relationships between one disorder's latent factors with those of the other disorder's latent factors. It was hypothesized that PTSD's dysphoria factor would be more related to depression's somatic than non-somatic factor (Hypothesis 1), and that PTSD's hyperarousal factor would correlate more strongly with depression's somatic than non-somatic factor (Hypothesis 2). Further, it was hypothesized that depression's somatic factor would be more related to PTSD's dysphoria than hyperarousal factor (Hypothesis 3), and that PTSD's re-experiencing and avoidance would not be differentially related to depression's somatic or non-somatic factors (Hypothesis 4). Results confirmed that despite PTSD's dysphoria factor evidencing a large relationship with non-somatic aspects of major depressive disorder (r=.59), dysphoria was significantly more related to somatic depression (r=.69). As hypothesized, PTSD's hyperarousal factor was also more strongly correlated with depression's somatic than non-somatic factor. However, hyperarousal was not as strongly correlated with the somatic factor as dysphoria was (Table 3).

Previous research has found mixed support for the notion that PTSD's dysphoria is especially related to depression and general emotional distress. While some studies (Eldkit et al., 2010; Forbes et al., 2010; Simms et al., 2002) have found that PTSD's dysphoria is uniquely related to emotional distress, other studies have failed to replicate that finding (Marshall et al., 2010; Miller et al., 2010). The present study clarifies these findings by more precisely analyzing depression not as a crude single variable but rather by analyzing its underlying factors. Thus perhaps mixed findings for dysphoria's external relationships resulted because depression and/or general emotional distress were analyzed as global external variables.

Rather based on our findings, analyzing depression based on its subcomponents, we find that PTSD's dysphoria is related to depression specifically by way of depression's somatic construct. Our findings corroborate those of a recent factor analytic study which examined the combined symptom structure of PTSD and depression symptoms. In that paper, Elhai et al. (2011b) also found that PTSD's dysphoria factor was more related to somatic symptoms of depression. It is possible that the shared somatic components to both PTSD and depression could account for the comorbidity between depression and PTSD. Perhaps PTSD is so highly comorbid with major depressive disorder because of the shared somatic component between these disorders.

Although PTSD's dysphoria factor shares two symptoms with the PHQ-9's somatic factor (sleep and concentration difficulties), which could account for its substantial relationship, the dysphoria factor has several other symptoms that are not shared by the somatic factor. Furthermore, it should be noted that research demonstrates that symptom overlap between PTSD and major depressive disorder is not solely responsible for the high rates of comorbidity between these two disorders (Elhai et al., 2008; Ford et al., 2009; Grubaugh et al., 2010).

The hypothesis that the somatic and non-somatic factors of depression would not be differentially related to either PTSD's re-experiencing or avoidance factors was not supported. Specifically, results indicated that depression's somatic factor was significantly more correlated with avoidance than the non-somatic factor was. It may be that individuals who use avoidance as a coping mechanism exhibit more somatic signs of distress (Morina et al., 2010). Avoidance in fact is considered a maladaptive emotion regulation strategy that leads to an increase in PTSD symptoms—especially physiological (somatic) manifestations of fear (Foos and Kozak, 1986).

Limitations of this study include using self-report instruments (in particular, by telephone) to assess PTSD and depression severity. Thus the limitations that are inherent in using self-report measures apply to this study, including potential problems with response validity, social desirability, memory recall, etc. Additionally, most respondents were men, and thus women were underrepresented. Furthermore, we assessed lifetime symptoms of depression and PTSD and thus we cannot generalize these findings to current assessment of PTSD and depression. Also, the relatively low mean scores on the PHQ-9 and PCL indicate relatively modest MDD and PTSD severity among respondents. Thus, the results of this study may not generalize to a sample of patients with more severe depression and PTSD. Finally, the results may not generalize to civilian trauma-exposed samples. Future studies should test these research questions using structured diagnostic interviews. Future studies should also attempt to use a more diverse sample exposed to a wider range of traumatic events.

Role of funding source

This project was funded by the Department of Defense Congressionally Directed Medical Research Program W81XWH-07-1-0409, the “Combat Mental Health Initiative.” The sponsor had no role in study design, data collection, analysis, interpretation of results, report writing, or manuscript submission.

Conflict of interest

No conflict declared.

References


