Abstract

Existing literature indicates significant comorbidity between posttraumatic stress disorder (PTSD) and major depression. We examined whether PTSD's dysphoria and mood/cognitions factors, conceptualized by the empirically supported four-factor *DSM-5* PTSD models, account for PTSD's inherent relationship with depression. We hypothesized that depression's somatic and non-somatic factors would be more related to PTSD's dysphoria and mood/cognitions factors than other PTSD model factors. Further, we hypothesized that PTSD's arousal would significantly mediate relations between PTSD's dysphoria and somatic/non-somatic depression. Using 181 trauma-exposed primary care patients, confirmatory factor analyses (CFA) indicated a well-fitting *DSM-5* PTSD dysphoria model, *DSM-5* numbing model and two-factor depression model. Both somatic and non-somatic depression factors were more related to PTSD's dysphoria and mood/cognitions factors than to re-experiencing and avoidance factors; non-somatic depression was more related to PTSD's dysphoria than PTSD's arousal factor. PTSD's arousal did not mediate the relationship between PTSD's dysphoria and somatic/non-somatic depression. Implications are discussed.

Keywords: PTSD, depression, *DSM-5*, primary care sample, structural equation modeling, mediation, confirmatory factor analyses

1. Introduction

About 48-55% of the general population diagnosed with posttraumatic stress disorder (PTSD) have comorbid major depressive disorder (MDD) in their lifetime (Kessler et al., 1995; Elhai et al., 2008). However, little is known about the latent factors of PTSD and depression that may be contributing to their comorbidity. In this study, we assess if PTSD's new *DSM-5* mood/cognitions and dysphoria factors capture depression's underlying dimensions compared to other PTSD factors.

1.1. PTSD's factor structure

Several models have been proposed to represent PTSD's underlying symptom structure, based on empirically testing inter-correlation patterns of PTSD symptoms through factor analysis. One long-standing model is the *DSM-IV* model, categorizing PTSD using three symptom clusters of re-experiencing, avoidance/numbing, and arousal. Elaborating on symptom clusters, re-experiencing symptoms may be linked to a need to integrate the new trauma-related information with prior knowledge, and are triggered usually by trauma-related cues (reviewed in Brewin and Holmes, 2003). Avoidance of traumatic reminders, although reducing the short-term emotional impact of exposure to traumatic event-related cues, maintain and contribute to PTSD severity by preventing the emotional processing of traumatic memories (Foa and Kozak, 1986; Foa et al., 1989). PTSD's arousal represents a person's need to be vigilant about any traumatic reminders to avoid the associated distress, and reactions to the traumatic experiences (e.g., anger, sleep disturbances) (reviewed in Brewin and Holmes, 2003). Most research indicates a lack of substantial empirical support for this tripartite model (Yufik and Simms, 2010; reviewed in Elhai and Palmieri, 2011); however a recent study highlights that the PTSD

item response order may be a methodological reason for the inferior fit of the this model compared to the alternative four-factor models developed later (Marshall et al., 2013).

The four-factor emotional numbing model (King et al., 1998) splits PTSD's avoidance/numbing factor into discrete factors (i.e., effortful avoidance and emotional numbing), with research supporting their construct distinctiveness (Asmundson et al., 2004). The four-factor dysphoria model (Simms et al., 2002) further alters the numbing model by combining several arousal (PTSD symptom criteria D1-D3) and numbing symptoms (C3-C7) to create a dysphoria factor, distinct from an arousal factor (items D4-D5). This model is based on support for a dysphoria dimension common to mood and anxiety disorders (Watson, 2009). A meta-analysis found that the dysphoria model has a slightly better fit than the numbing model (reviewed in Yufik and Simms, 2010; Miller et al., in press).

There have been several changes to the organization and structure of PTSD symptoms in PTSD's diagnostic criteria for *DSM-5*. First, four instead of three symptom clusters are outlined (Friedman et al., 2011). Second, the new cluster similar to the numbing cluster of the emotional numbing model is relabeled as "negative alterations in mood and cognition," adding new dysphoria-related symptoms. Finally, the arousal cluster adds a reckless behavior symptom.

1.2. Depression's factor structure

Research on the underlying structure of MDD is limited (Elhai et al., 2012a), with less research using measures mapping onto *DSM-IV*'s MDD criteria. One such measure is the Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001). Previous research has utilized exploratory (EFA) and confirmatory factor analyses (CFA) to investigate

depression's dimensionality using the PHQ-9 (e.g., Krause et al., 2010). Most support has been found for a two-factor model of depression with somatic and affective/non-somatic factors (Krause et al., 2010; Elhai et al., 2012a). Item mappings are indicated in Table 2. Understanding the relation between these two depression factors and PTSD's factors could indicate mechanisms of comorbidity, specifically if driven by depression's somatic or affective components. This may have implications for future DSM revisions as well.

1.3. Common underlying dimensions of PTSD and depression

Explanations for the PTSD-depression comorbidity have incorporated a symptom-level and a latent-level approach. As a fairly recent explanation of comorbidity is the network approach indicating that comorbid disorders are represented by the individualized causal interrelationships of symptoms as in a network. For example, the fatigue symptom in a Major Depressive Episode (MDE) could casually influence the concentration problems in Generalized Anxiety Disorder (GAD). Thus, there are individualized pathways to comorbidity between disorders (Borsboom et al., 2011). Given the controversy regarding whether symptoms are manifest or latent-level constructs (McFarland and Malta, 2010), the idea that the network approach does not completely contradict the latent-level approach (Humphry and McGrane, 2010), and possibility of incorporating the symptom and latent-level approaches (Molenaar, 2010) necessitates consideration of the latent-level approach.

A latent-level based empirically supported explanation for the high PTSD-depression comorbidity is that PTSD and depression share common underlying dimensions (Gros et al., 2010; Elhai et al., 2011a). The latest of the "common factors model" is the quantitative hierarchical model (Watson, 2005, 2009). This model proposes

that a higher-order factor of emotional disorders includes subclasses of 1) bipolar disorders 2) distress disorders (e.g., MDD, PTSD, dysthymia), and 3) fear disorders (panic disorder, phobias). Thus, PTSD was conceptualized as belonging to the same distress disorders category as depression, with the shared component being a greater amount of emotional distress.

An unresolved debate relates to whether PTSD's dysphoria factor captures PTSD's distress and thereby comorbidity with depressive disorders. Some studies support PTSD's dysphoria dimension as responsible for PTSD's significant relationship with emotional distress including depression (Simms et al., 2002; Armour and Shevlin, 2010; Gootzeit and Markon, 2011); other data challenge this proposition (Elhai et al., 2008; Marshall et al., 2010; Miller et al., 2010; Armour and Shevlin, in press). Conflicting results could be attributed to differences in samples used, differing instruments, and different methodologies. Further, these studies measured distress using observed variables as summed scores. However, examining structural relations between PTSD's and depression's latent factors would have several advantages such as ability to capture heterogeneity within disorders (Watson, 2009).

Although the two PTSD four-factor models are slightly different, the dysphoria and numbing factors (relabeled as the mood/cognitions factor in the *DSM-5*) have similarities in relations with depression/distress. First, although both PTSD models when adapted for *DSM-5* equally reduce the PTSD prevalence rates compared to the *DSM-IV* model (Elhai et al., 2009), they have similar comorbidity rates of about 54% with MDE (Elhai et al., 2009). Second, PTSD's dysphoria and numbing factors have evidence of similar correlations ranging from 0.72 to 0.76 with depression symptoms across several

studies (Watson, 2009), and one of the strongest predictive relations with depression in their respective PTSD models (Gootzeit and Markon, 2011). Third, there is evidence of dysphoria's and numbing's higher correlations with depression compared to their respective PTSD model factors (Palmieri et al., 2007; Watson, 2009; Gootzeit and Markon, 2011). Lastly, both seem to equally capture PTSD's non-specific distress accordingly to majority of studies (Miller et al., 2010; Gootzeit and Markon, 2011). There seem to be non-significant differences in the distress variance accounted for by PTSD's dysphoria factor and numbing factor, however PTSD's dysphoria factor relates more strongly to "internalizing" symptoms including depression (Miller et al., 2010). In regard to *DSM-5*, recent studies indicate that the newly proposed mood/cognitions factor shows similar relations with depression as did the previous numbing factor (Elhai et al., 2012b; Koffel et al., 2012). Thus, the current study also assessed the comparative relation of PTSD's mood/cognitions factor with somatic/non-somatic depression.

1.4. PTSD's arousal factor

The present study focused primarily on PTSD's arousal factor, specifically its relationship with depression latent factors, based on two reasons. PTSD's arousal plays a dominant role in the maintenance, severity and recovery of individuals with PTSD; those with more severe arousal symptoms demonstrate poorer recovery and more severe PTSD symptomology (Schell et al., 2004; Marshall et al., 2006; Stein et al., 2012). Additionally, numerous studies demonstrate an existing relationship between arousal and depression (Hellmuth et al., 2012; Biehn et al., 2013). To elaborate, Pérez et al. (2012) recently found arousal and depression to independently mediate the relationship between trauma exposure and physical health more so than other PTSD factors (Pérez et al., 2012);

however this finding is inconsistent in the literature (Eadie et al., 2008; Baker et al., 2009). Furthermore, PTSD's arousal factor relates to aggression through depression as a mediator (Hellmuth et al., 2012).

Evidence for a relationship between PTSD's arousal and dysphoria factors is indicated by the role of PTSD's arousal symptoms (e.g., hypervigilance) in avoiding distress possibly represented by PTSD's dysphoria (reviewed in Brewin and Holmes, 2003), arousal's relationship with external distress measures (Marshall et al., 2010), and arousal's predictive relationship of other PTSD symptoms' severity (Schell et al., 2004; Marshall et al., 2006; Stein et al., 2012). Further, prior research has indicated a significant relationship between PTSD's arousal factor and physical health problems (Pérez et al., 2012), possibly represented by somatic depression, and relationship between PTSD's arousal factor and other emotional reactions such as affective depression and anger (Hellmuth et al., 2012; Biehn et al., 2013), possibly represented by non-somatic depression. Thus, one could conceptualize the dysphoria and arousal factors as concurrently influencing somatic/non-somatic depression symptoms; specifically the relationship between PTSD's dysphoria and somatic/non-somatic depression is possibly augmented by concurrent arousal symptoms of PTSD. This may indicate arousal's possible role in PTSD's comorbidity with depression. Notable is that we assess the dysphoria and arousal factors per the dysphoria model, to enable a more refined measure of distress non-specific to PTSD and physiological arousal specific to PTSD.

1.5. Study aims

The present study differs from prior relevant studies in two major ways. First, because similar studies that have focused on military samples (Elhai et al., 2011b; Biehn

et al., 2013), we utilized a civilian trauma-exposed sample. Given the large relationship between PTSD and medical problems (reviewed in Pacella et al., 2013) as well as healthcare use (reviewed in Elhai et al., 2005) we sampled civilian primary care patients. Second, we assessed for PTSD based on new *DSM-5* diagnostic criteria. In addition to using the four-factor *DSM-5* PTSD model, we also used a modified model conceptually and structurally similar to the *DSM-IV* PTSD dysphoria model (Miller et al., in press). We additionally used the *DSM-5* model adapted in line with the *DSM-IV* dysphoria model because of a slightly better fit of the *DSM-IV* dysphoria model compared to other *DSM-IV* PTSD models (reviewed in Yufik and Simms, 2010), and its ability to separate the specific and non-specific PTSD factors (Simms et al., 2002). In addition, changes in criteria for the PTSD diagnosis that were implemented in this study have been examined in only a few studies thus far (Elhai et al., 2012b; Koffel et al., 2012; Miller et al., in press). Moreover, no known empirical studies have been published regarding the relationship between *DSM-5* PTSD symptom dimensions and depression.

We tested specific hypotheses about the relationship between PTSD's *DSM-5-*adapted dysphoria and numbing model factors and depression's two-factor model. Subsequent to comparing the fit of two *DSM-5* four-factor models, we tested three main research questions. First we address if PTSD's dysphoria factor better accounts for the variance in somatic and non-somatic depression compared to other PTSD factors (*DSM-5* dysphoria model). We hypothesized that both depression's somatic and non-somatic factors would be more related to PTSD's dysphoria factor than to PTSD's re-experiencing (hypothesis 1), avoidance (hypothesis 2), and arousal (hypothesis 3) factors, which is consistent with both theory (Watson, 2009), and prior empirical evidence

(Simms et al., 2002; Elhai et al., 2011b). Second, we assess if PTSD's mood/cognitions factor better accounts for the variance in somatic and non-somatic depression compared to other PTSD factors (*DSM-5* emotional numbing model). We hypothesized that both depression's somatic and non-somatic factors would be more related to PTSD's mood/cognitions factor than to PTSD's re-experiencing (hypothesis 4), avoidance (hypothesis 5), and arousal (hypothesis 6) factors. Lastly, we assess the mediational role of PTSD's arousal in the relationship between PTSD's dysphoria and somatic/non-somatic depression factors, predicting a significant mediation effect as discussed above (hypotheses 7 and 8).

2. Method

2.1. Procedure and participants

We recruited medical patients from a primary care clinic affiliated with a community hospital and Midwestern university's medical school. Individuals were consecutively invited to participate upon presenting for appointments to the clinic by trained student research assistants. We recruited participants primarily in fall 2011 and spring 2012. Individuals who agreed to participate completed questionnaires in the clinic waiting room. Eligibility criteria for participation included proficiency in English and being 18-70 years old. Among the pool of potential participants who met eligibility requirements ($N = 411$), 293 individuals agreed to participate (response rate = 71.3%). Three participants did not answer queries about trauma exposure, leaving 290 participants. The project received IRB approval from relevant institutions.

2.2. Exclusions and treatment of missing data

The sample of 290 participants was restricted to those endorsing a worst trauma (as per below), reducing the sample to 194 participants. Among participants, 13 missed

more than 8 PTSD items and were excluded, resulting in an effective sample of 181 participants. To ensure sufficient items for missing value estimation, we restricted the sample to participants not missing more than 30% of items on at least one measure (Schafer and Graham, 2002; Graham, 2009). In this effective sample, six participants missed one and one participant missed eight PTSD items; five participants missed one depression item. Missing data were imputed using maximum likelihood (ML) estimation.

2.3. Effective sample characteristics

The effective sample had a mean age of 40.2 years ($SD = 13.64$), with the majority being female ($n = 144$, 79.6%). Most respondents were married ($n = 74$, 41.1%), and employed, either full-time ($n = 72$, 40%) or part-time ($n = 38$, 21.1%). Years of schooling ranged from 7 to 20 years ($M = 13.72$, $SD = 2.08$). Most participants had income less than $34,999 - more specifically, less than $15,000 ($n = 49$, 27.7%), $15,000-$24,999 ($n = 31$, 17.5%), and $25,000-$34,999 ($n = 30$, 16.9%). Further, most reported their race as Caucasian ($n = 136$, 75.1%), or African American ($n = 38$, 21.0%). Hispanic/Latino ethnicity was reported by 13 participants (7.4%). The most prevalent worst traumatic events (upon which PTSD ratings were assessed) were unexpected death of a family member/close friend ($n = 63$, 34.8%), life-threatening illness ($n = 23$, 12.7%), physical harm by partner/date ($n = 18$, 9.9%), attempted/actual physical force to have sex ($n = 17$, 9.4%), inappropriate touch during childhood ($n = 14$, 7.7%), and being threatened by a weapon ($n = 11$, 6.1%). The average time elapsed since the experience of the traumatic event was 12.89 years ($SD = 12.63$).

2.4. Instrumentation

2.4.1. Patient Heath Questionnaire-9 (PHQ-9). The PHQ-9 is a 9-item self-report measure assessing *DSM-IV* MDE symptoms over the past two weeks. Four response options range from 0 indicating "*Not at all*" to 3 indicating "*Nearly everyday*" (Kroenke et al., 2001). The MDE scoring algorithm is consistent with *DSM-IV* diagnostic criteria, with at least 5 out of 9 statements endorsed minimally as "more than half the days," and at least one of the symptoms being depressed mood or anhedonia. Kroenke et al. (2001) found acceptable internal consistency ranging from 0.86-0.89 (alpha = 0.93 in our study) and good test-retest reliability within 48 hours ($r = 0.84$). Diagnostic validity was demonstrated by detecting MDD diagnoses per structured diagnostic interviews (Kroenke et al., 2001).

2.4.2. Stressful Life Events Screening Questionnaire (SLESQ). The SLESQ is a 13-item self-report screening measure of lifetime exposure to 12 broad categories of trauma exposure meeting Criterion A1 of *DSM-IV* PTSD criteria. Some adaptations were made in accordance with the *DSM-5* PTSD criteria. Specifically, we added a supplementary question to ascertain if the endorsed traumatic even was witnessed through electronic media, or by virtue of one's occupational responsibilities (Elhai et al., 2012b). Test-retest reliability kappa coefficients for a 2-week period at the item level ranged from 0.31 to 1.00 (averaging 0.73, with only four below 0.60) for the 11 *DSM-IV* based traumatic events and overall test-retest reliability of 0.89 across events. Further, it has good concurrent and convergent validity with a more extensive trauma exposure interview with a mean kappa of 0.64 (Goodman et al., 1998). Lastly, a question asking participants to indicate their most distressing event (if endorsing more than one event) and to provide details on that event was added. Subsequently, participants were instructed

to respond to subsequent PTSD symptoms with reference to the index/most distressing traumatic event.

 2.4.3. Posttraumatic Stress Disorder Symptom Scale-Self Report (PSS-SR). The PSS-SR is a 17-item self-report measure of PTSD's symptom severity. Symptoms are defined using PTSD's *DSM-IV* diagnostic criteria. It utilizes a four-point Likert-type method (0 = "*Not at all*" to 3 = "*5 times or more per week/very much/almost always*") for reporting symptom severity (Foa et al., 1993). The measure has adequate internal consistency ranging from 0.65-0.71 (alpha = 0.96 in our study) and test-retest reliability coefficients ranging from 0.66 to 0.77 (Foa and Tolin, 2000). We included items in the *DSM-5* PTSD criteria (Friedman et al., 2011), reflecting a pervasive negative emotional state, excessive trauma-related blame, and reckless behavior; we also added an item of negative perceptions of oneself, the future or the world that replaces the foreshortened future item in *DSM-5* diagnostic criteria for PTSD. These added items were used in a recent study by Elhai et al. (2012b).

2.5. Analyses

 All primary analyses conducted with the Mplus 6.12 software entailed three steps. First, CFA separately estimated the fit of PTSD's four-factor *DSM-5* dysphoria and numbing models (PSS-SR items), and depression's two-factor model (PHQ-9 items). Factor variances and error terms were scaled to "1." We used an alpha level of 0.05 and two-tailed tests. PSS-SR and PHQ-9 items were treated as ordinal items (four response options), using a polychoric covariance matrix, robust weighted least squares estimation with a mean-and variance-adjusted chi-square (WLSMV), and probit regression coefficients (Flora and Curran, 2004). A well-fitting (adequate) model has comparative

fit index (CFI) and Tucker Lewis Index (TLI) values $\geq$ 0.95 (0.90-0.94), and root mean

square error of approximation (RMSEA) value $\leq$ 0.06 (0.07-0.08) as indicated by Hu and

Bentler's criteria (1999). These criteria for fit indices are most sensitive to model fit (Hu

and Bentler, 1999). Additionally, Bayesian Information Criterion (BIC) values (using ML

estimation) were used for comparing the two PTSD non-nested models. A 10-point BIC

difference represents a 150:1 likelihood and "very strong" ($p < 0.05$) support that the

model with the smaller BIC value fits best; a difference of 6-9 points indicates "strong"

support (Kass and Raftery, 1995).

The second step entailed computing Wald chi-square tests of parameter

constraints to test hypothesized latent-level relations between PTSD's *DSM-5* dysphoria

and numbing models and depression's two-factor model. This test assesses the null

hypothesis that the difference between two correlation paths is zero, using alpha of 0.05.

Specifically, Wald tests assessed if depression's somatic and non-somatic factors were

more related to PTSD's dysphoria factor than to PTSD's re-experiencing (hypothesis 1),

avoidance (hypothesis 2), and arousal factors (hypothesis 3). Wald tests assessed if

depression's somatic and non-somatic factors were more related to PTSD's

mood/cognitions factor than to PTSD's re-experiencing (hypothesis 4), avoidance

(hypothesis 5), and arousal factors (hypothesis 6).

The third step included mediation analyses, with PTSD's arousal (per the *DSM-5*

dysphoria model) as a mediator between PTSD's dysphoria factor and depression's

somatic/non-somatic factors. We used the product of path coefficients approach

estimated using the delta method (MacKinnon, 2008). We used 1000 bootstrapped

samples to estimate standard errors. Direct effects in the current study included the

relationship between PTSD's dysphoria and depression's somatic/non-somatic factors, between PTSD's arousal and depression's somatic/non-somatic factors, and between PTSD's dysphoria and arousal factors. Indirect effects included the effect of PTSD's dysphoria factor on depression's somatic/non-somatic factors through PTSD's arousal factor. Notable are two points: a significant direct effect between the independent and dependent variables is not required to retain the power to detect a significant mediation effect (MacKinnon, 2008), and current study's "mediation" terminology refers to a mechanism explaining the relation between variables, given the lack of temporal precedence for variables (Kraemer et al., 2008).

3. Results

Total PSS-SR scores (*DSM-IV* symptoms) averaged 12.28 (*SD* = 13.21), with 66 (or 22.75% of the 290 eligible) participants having a probable PTSD diagnosis using the *DSM-IV* diagnostic algorithm of at least one re-experiencing symptom, three avoidance/numbing symptoms and two arousal symptoms endorsed as "1" or higher (Foa et al., 1993). Using the *DSM-5* PTSD algorithm (at least one re-experiencing symptom, one avoidance symptom, two cognition/mood symptoms, two arousal symptoms endorsed as "1" or higher), 62 (or 21.37% of the 290 eligible participants) had a probable PTSD diagnosis. These PTSD prevalence rates are higher than that found in the general population (Kessler et al., 1995), but expected in a sample such as medical patients, given the large relationship between trauma/PTSD and health and healthcare use (Elhai et al., 2005; Pacella et al., 2013). Further, we focused on the dimensional aspect of PTSD symptoms rather than a categorical yes/no endorsement given evidence of PTSD being a dimensional construct (Forbes et al., 2005). Total PHQ-9 scores averaged 6.76 (*SD* =

6.73), with 10.43% ($n = 29$) of the trauma-exposed sample having a probable MDE diagnosis using the *DSM-IV* depression diagnostic algorithm.

CFA indicated a well-fitting four-factor *DSM-5* PTSD numbing model according to the majority of the fit indices, robust χ^2(164, $N = 181$) = 306.480, $p < 0.001$, CFI = 0.98, TLI = 0.97, RMSEA = 0.07 (90% CI: 0.057-0.081). Furthermore, CFA indicated a well-fitting four-factor *DSM-5* dysphoria model according to the majority of the fit indices, robust χ^2(164, $N = 181$) = 319.753, $p < 0.001$, CFI = 0.97, TLI = 0.97, RMSEA = 0.07 (90% CI: 0.061-0.084). Comparing both these non-nested models, BIC values for the four-factor *DSM-5* dysphoria model (7642.655) was more than 10 points greater than the four-factor *DSM-5* numbing model (7623.265), indicating the latter as a better-fitting model.

CFA indicated a well-fitting depression two-factor model according to the majority of the fit indices, robust χ^2(26, $N = 181$) = 51.633, $p = 0.002$, CFI = 0.99, TLI = 0.99, RMSEA = 0.07 (90% CI: 0.044-0.103). A combined CFA of PTSD's *DSM-5* dysphoria model and depression's two-factor model indicated a good fit according to most fit indices, robust χ^2(362, $N = 181$) = 582.160, $p < 0.001$, CFI = 0.98, TLI = 0.97, RMSEA = 0.06 (90% CI: 0.049-0.067).

Regarding Wald tests (see Table 3), consistent with the first and second hypotheses, depression's somatic factor was more related to PTSD's dysphoria ($r = 0.753$, $p < 0.001$) than PTSD's re-experiencing factor ($r = 0.626$, $p < 0.001$), Wald χ^2(1, $N = 181$) = 10.565, $p = 0.001$, and avoidance factor ($r = 0.578$, $p < 0.001$), Wald χ^2(1, $N = 181$) = 11.493, $p < 0.001$. Furthermore, depression's non-somatic factor was more related to PTSD's dysphoria factor ($r = 0.816$, $p < 0.001$) than to PTSD's re-experiencing factor

($r = 0.701$, $p < 0.001$), Wald $\chi^2(1, N = 181) = 8.314$, $p = 0.004$, and avoidance factor ($r =$ 0.654, $p < 0.001$), Wald $\chi^2(1, N = 181) = 9.925$, $p = 0.002$. Contradictory to the third hypothesis, depression's somatic factor was not more related to PTSD's dysphoria factor ($r = 0.753$, $p < 0.001$) than PTSD's arousal factor ($r = 0.677$, $p < 0.001$), Wald $\chi^2(1, N =$ 181$) = 3.652$, $p = 0.056$. Notable is that the alpha value of 0.056 was very close to significance. However, consistent with this hypothesis, depression's non-somatic factor was more related to PTSD's dysphoria factor ($r = 0.816$, $p < 0.001$) than to PTSD's arousal factor ($r = 0.736$, $p < 0.001$), Wald $\chi^2(1, N = 181) = 3.886$, $p = 0.049$.

We further conducted a similar set of analyses with the numbing model, finding a very similar set of results to the dysphoria model analyses. Consistent with the fourth and fifth hypotheses, depression's somatic factor was more related to PTSD's mood/cognitions ($r = 0.717$, $p < 0.001$) than PTSD's re-experiencing factor ($r = 0.626$, $p < 0.001$), Wald $\chi^2(1, N = 181) = 5.28$, $p = 0.02$, and avoidance factor ($r = 0.578$, $p <$ 0.001$), Wald $\chi^2(1, N = 181) = 6.86$, $p < 0.001$. Furthermore, depression's non-somatic factor was more related to PTSD's mood/cognitions factor ($r = 0.812$, $p < 0.001$) than to PTSD's re-experiencing factor ($r = 0.701$, $p < 0.001$), Wald $\chi^2(1, N = 181) = 7.204$, $p =$ 0.007, and avoidance factor ($r = 0.654$, $p < 0.001$), Wald $\chi^2(1, N = 181) = 9.751$, $p =$ 0.002. Contradictory to the sixth hypothesis, depression's somatic factor was not more related to PTSD's mood/cognitions factor ($r = 0.717$, $p < 0.001$) than PTSD's arousal factor ($r = 0.766$, $p < 0.001$), Wald $\chi^2(1, N = 181) = 2.642$, $p = 0.104$. Additionally, depression's non-somatic factor was not more related to PTSD's mood/cognitions factor ($r = 0.812$, $p < 0.001$) than to PTSD's arousal factor ($r = 0.787$, $p < 0.001$), Wald $\chi^2(1, N$

$= 181) = 0.656$, $p = 0.417$. The statistical values of the Wald test results are indicated in Table 4.

Contradictory to hypothesis 7, PTSD's arousal did not significantly mediate the relationship between PTSD's dysphoria factor and depression's non-somatic factor ($\beta = -0.30$, $SE = 0.16$, $p = 0.058$), again close to significance (see Figure 1). Results indicated significant direct effects of PTSD's dysphoria factor on PTSD's arousal factor ($B = 1.95$, $SE = 0.31$, $\beta = 0.89$, $p < 0.001$) and on depression's non-somatic factor ($B = 3.11$, $SE = 1.01$, $\beta = 1.21$, $p = 0.002$). However, the direct effect of PTSD's arousal factor on depression's non-somatic factor was not statistically significant ($B = -0.40$, $SE = 0.26$, $\beta = -0.34$, $p = 0.13$). Further, PTSD's arousal did not significantly mediate the relationship between PTSD's dysphoria factor and depression's somatic factor ($\beta = -0.30$, $SE = 0.19$, $p = 0.11$), contradictory to hypothesis 8 (see Figure 2). In addition, there were significant direct effects of PTSD's dysphoria factor on PTSD's arousal factor ($B = 1.97$, $SE = 0.33$, $\beta = 0.89$, $p < 0.001$) and on depression's somatic factor ($B = 2.19$, $SE = 0.65$, $\beta = 1.14$, $p = 0.001$). However, the direct effect of PTSD's arousal factor on somatic depression was not statistically significant ($B = -0.30$, $SE = 0.20$, $\beta = -0.34$, $p = 0.13$).

4. Discussion

We assessed latent-level relations between both *DSM-5* PTSD dysphoria and numbing model factors with depression's two-factor model factors, which has been rarely addressed. The meditational role of PTSD's arousal factor (*DSM-5* dysphoria model) referencing the relationship between PTSD's dysphoria and somatic/non-somatic depression was also assessed. Using a primary care sample, most proposed hypotheses were supported as further elaborated.

4.1. Relationship between PTSD's dysphoria and mood/cognitions factors and somatic/non-somatic depression

Results indicated that somatic and non-somatic depression factors were more related to PTSD's dysphoria and mood/cognitions factors than to their respective model's re-experiencing and avoidance factors, consistent with prior results with the *DSM-IV* PTSD models (e.g., Watson, 2009; Gootzeit and Markon, 2011). One explanation is that somatic symptoms are part of the non-specific emotional distress (possibly represented by PTSD's dysphoria and mood/cognitions factors) consequent to trauma exposure (Ursano et al., 2009). Regarding the relation between non-somatic depression and PTSD's dysphoria/mood and cognitions factors, PTSD's dual-representation theory's proposes that chronic processing and constant pre-occupation with traumatic memories could contribute to non-somatic depression, or subsequent feelings of loss/powerlessness related to the traumatic event (Brewin et al., 1996), again possibly better captured by PTSD's dysphoria and mood/cognitions factors.

Overall, the current study results indicate that both PTSD's dysphoria and mood/cognitions factors may capture PTSD's non-specific distress and thus possibly account for the PTSD-depression comorbidity for the most part. What this means for the depression construct is that non-somatic depression may be a predominant contributor to the PTSD-depression comorbidity for two reasons: (1) in addition to a greater relation between somatic/ non-somatic depression and PTSD's dysphoria and mood/cognition factors compared to PTSD's re-experiencing and avoidance symptoms in their respective models, non-somatic depression was more related to PTSD's dysphoria when compared to PTSD's arousal, and (2) both the dysphoria and mood/cognitions factors had a greater

relationship with non-somatic depression compared to somatic depression; although future research needs to test the significance of these path differences.

4.2. Relationship between PTSD's arousal factor and somatic/non-somatic depression

Contradictory to proposed hypotheses and prior study results (Elhai et al., 2011b; Biehn et al., 2013), PTSD's dysphoria and mood/cognitions factors were not more related to somatic depression compared to PTSD's arousal factor. There are two possible explanations for these discrepant findings. First, prior studies used *DSM-IV* PTSD criteria (Elhai et al., 2011b; Biehn et al., 2013), rather than the current study's *DSM-5* PTSD criteria which has several modified and edited items. Building on this, the new *DSM-5* arousal criterion has an additional item of reckless behavior compared to the prior *DSM-IV* arousal criterion. Although it has an adequate factor loading in the current study (0.81), it has been shown to be a weak indicator of its proposed factor, and less able to discriminate between people high vs. low in PTSD severity (Miller et al., in press). Thus, differential latent-factor conceptualizations (for both the dysphoria and mood/cognitions factors) and item loadings for the latent factors may have contributed to the discrepant current study findings. Second, per *DSM-IV* PTSD studies, PTSD's mood/cognitions and arousal factors seem to have an equal and the strongest correlation with depression symptoms (Gootzeit and Markon, 2011), which was consistent with our findings. Thus, PTSD's mood/cognitions and arousal factors may both capture somatic depression symptoms equally.

Focusing on somatic depression, PTSD's hypervigilance and startle responses may mimic the somatic discomfort (depression's somatic factor), explaining previously established greater correlation between PTSD's arousal factor with somatic than non-

somatic depression (Biehn et al., 2013). Additionally, given some overlap between somatic depression and anxiety symptoms, a significant relationship between PTSD's arousal and anxiety (Gootzeit and Markon, 2011) may explain the relationship between PTSD's arousal and somatic depression. Overall, results are consistent with evidence indicating arousal's significant relationship with depression (King et al., 1998; Simms et al., 2002). Additionally, results support the idea that dysphoria may not be PTSD's *only* non-specific factor (Elhai et al., 2008; Elklit et al., 2010; Marshall et al., 2010; Miller et al., 2010), with PTSD's arousal capturing some PTSD-related distress (Gootzeit and Markon, 2011). Lastly, the mood/cognitions factor may be equivalent to PTSD's arousal in capturing PTSD's relation with depression (Gootzeit and Markon, 2011).

4.3. Mediating role of PTSD's arousal factor

Results indicated that PTSD's arousal factor did not significantly mediate relations between PTSD's dysphoria and depression's somatic and non-somatic factors. This means that PTSD's arousal may not contribute to the relationship between PTSD's dysphoria factor and somatic/non-somatic depression as proposed in the current study. Although PTSD's arousal may still play its purported role of increasing PTSD symptoms severity longitudinally based on results of prior studies (Marshall et al., 2006), it may not increase comorbid PTSD symptomatology over time. The current study used different PTSD diagnostic criteria (*DSM-5)* and a primary care sample different from the sample in prior empirical studies (e.g., survivors of orofacial injury and community violence) (Marshall et al., 2006); these may have contributed to the difference between the current study results and those expected based on results of prior studies. Although there is an established significant relationship between PTSD's arousal and depression in the

literature (Simms et al., 2002; Gootzeit and Markon, 2011), there was a non-significant direct effect of PTSD's arousal on somatic/non-somatic depression in the current study.

4.4. Implications

The current study has several important conceptual and practical implications. First, for the most part, results indicate dysphoria and mood/cognitions as PTSD's non-specific factors (e.g., Watson, 2009) when using a more statistically refined manner of analyzing latent-level relations. Thus, comorbidity pathways between PTSD and depression seem to be at least partly driven by PTSD's mood/cognitions and dysphoria factors. Regarding the debate over PTSD's dysphoria being a non-specific factor, it could be said that PTSD's dysphoria may/may not account for PTSD's entire inherent distress contingent on: the PTSD factor it is compared with, and the latent dimensions of the relevant psychopathology construct. Lack of latent-level analyses in prior studies could have contributed to contradictory results regarding dysphoria's accounting for *all* of PTSD's shared distress (e.g., Marshall et al., 2010; Miller et al., 2010).

Second, the results of the current study could map onto the "quantitative elements" proposal for each symptom cluster (Watson, 2009). To elaborate, it is proposed to quantify PTSD symptom dimensions per (1) amount of distress variance, and (2) PTSD specificity. The dysphoria and mood/cognitions factors could be viewed as factors with greater distress variance and lower PTSD specificity compared to PTSD's arousal conceptualized as having lower distress variance but higher PTSD specificity (Watson, 2009; Gootzeit and Markon, 2011). Additional research is needed to evaluate this notion.

Third, PTSD's mood/cognitions factor was equivalent to PTSD's dysphoria factor in capturing PTSD's non-specific distress for the most part similar to the results obtained

by Gootzeit and Markon (2011) with the *DSM-IV* factors. With some possible overlap in these two factor conceptualizations, it again bears the question of the latent-level differences in the two models. Future research would benefit by statistically testing convergent and discriminant validity of these two factors with external measures using the *DSM-5* models similar to prior studies with the *DSM-IV* models (Miller et al., 2010), even possibly using the network approach to comorbidity with symptom-level analyses (Borsboom et al., 2011).

Finally, results have implications for treatment of PTSD, providing further support for the importance of treating arousal (anxiety) *and* dysphoria/depression. Multicomponent treatments that target different aspects of the disorder (Beidel et al., 2011) may represent the treatment strategy with the highest likelihood of overall effectiveness for most patients compared to single component treatments, such as exposure therapy, that target only one aspect of the disorder. Use of multicomponent treatment strategies, with reliance on first-line treatment protocols and subsequently different evidence-based protocols if needed is consistent with some best practice recommendations (Forbes et al., 2010). Further, it would be beneficial to concurrently and longitudinally assess for PTSD's dysphoria/mood and cognition symptoms and somatic/non-somatic depression with clients presenting with comorbid PTSD-depression when evaluating the effectiveness of different types of treatment.

4.5. Limitations and future directions

First, use of self-report measures can lead to bias in terms of social desirability effects; thus it would be helpful to replicate the aforementioned results using multi-method assessments. Second, the study is limited to a specific primary care sample

restricting its generalizability. Further, there is no way to ascertain the primary reason for seeking care in the clinic and if the reason was related to PTSD symptoms. Third, the cross-sectional design does not rule out the possibility of bi-directional influences between the mediator and dependent/independent variables, rather than a causal unidirectional relationship (MacKinnon, 2008); this being better assessed using a longitudinal dataset. Additionally, arousal's conceptualization per the dysphoria model with just two items may have contributed to the non-significant mediation findings using PTSD's dysphoria model.

Fifth, with only a percentage of the sample having a probable PTSD and MDE diagnosis, the findings cannot be generalized to a sample with PTSD/MDE diagnosis and greater PTSD/MDE severity; rather they apply to samples with PTSD and depression symptoms. It is to be highlighted that the current study aims to address PTSD and depression symptoms dimensionally to assess their factor-level relations; hence the diagnostic prevalence of PTSD and depression was not the focus of the current study. Lastly, although the sample size may seem slightly small for such analyses, we considered evidence indicating that a sample size of 132 is adequate power for PTSD factor-analytic research given the extensive support for the four-factor PTSD models across studies (Elhai and Palmieri, 2011). Despite these limitations, this is the first study of *DSM-5* PTSD criteria examined in relation with depression's latent dimensions using robust data analyses.

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Table 1.PTSD Symptom Scale (PSS-SR) Item Mappings for Tested Models

DSM-IV Criteria from PSS-SR Items	DSM-5 Numbing		DSM-5 Dysphoria	
	Item mappings	Factor loadings	Item mappings	Factor loadings
B1: Intrusive thoughts (Item 1)	I	0.87	I	0.87
B2: Nightmares (Item 2)	I	0.83	I	0.83
B3: Reliving trauma (Item 3)	I	0.85	I	0.86
B4: Emotional cue reactivity (Item 4)	I	0.86	I	0.86
B5: Physiological cue reactivity (Item 5)	I	0.76	I	0.76
C1: Avoidance of thoughts (Item 6)	A	0.97	A	0.97
C2: Avoidance of reminders (Item 7)	A	0.85	A	0.85
D1: Trauma-related amnesia (Item 8)	M	0.67	D	0.66
D2: Negative expectations of self, world, others (Item 18)	M	0.90	D	0.89
D3: Blame of self or others for trauma (Item 19)	M	0.85	D	0.84
D4: Pervasive negative emotional state (Item 20)	M	0.95	D	0.94
D5: Loss of interest (Item 9)	M	0.90	D	0.89
D6: Feeling detached (Item 10)	M	0.91	D	0.90
D7: Feeling numb (Item 11)	M	0.83	D	0.81
E1: Irritable/angry (Item 14)	H	0.87	D	0.86
E2: Recklessness (Item 21)	H	0.76	D	0.81
E3: Overly alert (Item 16)	H	0.79	H	0.83
E4: Easily startled (Item 17)	H	0.86	D	0.91
E5: Difficulty concentrating (Item 15)	H	0.89	H	0.88
E6: Sleep problems (Item 13)	H	0.84	H	0.82

Note. PTSD = posttraumatic stress disorder; I = Intrusion; A = Avoidance;
M = Negative Alterations in Mood and Cognition; H = Hyperarousal; D = Dysphoria.

Table 2. Factors on Which Depression Items were Mapped

PHQ-9 Items	Depression Factor	Factor loadings
1. Anhedonia	Non-Somatic	0.93
2. Depressed mood	Non-Somatic	0.90
3. Sleep difficulties	Somatic	0.89
4. Fatigue	Somatic	0.94
5. Appetite changes	Somatic	0.81
6. Feeling of worthlessness	Non-Somatic	0.89
7. Concentration difficulties	Somatic	0.88
8. Psychomotor agitation/retardation	Somatic	0.81
9. Thoughts of death	Non-Somatic	0.79

Table 3. Results of the Wald tests of Parameter Constraints (*DSM-5* dysphoria model)

Path	*r*	Path	*r*	Wald test (*p* value)
Somatic with Dys	0.753***	Somatic with Reexp	0.626 ***	10.565 (*p* = 0.001)*
Somatic with Dys	0.753***	Somatic with Avoid	0.578 ***	11.493 (*p* = 0.0007)*
Somatic with Dys	0.753***	Somatic with Arousal	0.677 ***	3.652 (*p* = 0.056)
Non-Somatic with Dys	0.816***	Non-Somatic with Reexp	0.701 ***	8.314 (*p* = 0.004)*
Non-Somatic with Dys	0.816***	Non-Somatic with Avoid	0.654 ***	9.925 (*p* = 0.002)*
Non-Somatic with Dys	0.816***	Non-Somatic with Arousal	0.736 ***	3.886 (*p* = 0.049)**

Note. Somatic is depression's somatic factor; non-somatic is depression's non-somatic factor; Dys is PTSD's dysphoria factor; Avoid is PTSD's avoidance factor; Reexp is PTSD's reexperiencing factor.
* *p*< 0.01; ** *p*< 0 .05; ****p*< 0.001.

Table 4. Results of the Wald tests of Parameter Constraints (*DSM-5* Emotional Numbing Model)

Path	*r*	Path	*r*	Wald test (*p* value)
Somatic with Mood/cognitions	0.717 ***	Somatic with Reexp	0.626 ***	5.283 (*p* = 0.021)*
Somatic with Mood/cognitions	0.717 ***	Somatic with Avoid	0.578 ***	6.867 (*p* < 0.001)*
Somatic with Mood/cognitions	0.717 ***	Somatic with Arousal	0.766 ***	2.642 (*p* = 0.104)
Non-Som with Mood/cognitions	0.812 ***	Non-Som with Reexp	0.701 ***	7.204 (*p* = 0.007)*
Non-Som with Mood/cognitions	0.812 ***	Non-Som with Avoid	0.654 ***	9.751 (*p* = 0.002)*
Non-Som with Mood/cognitions	0.812 ***	Non-Som with Arousal	0.787 ***	0.656 (*p* = 0.417)

Note. Somatic is depression's somatic factor; non-som is depression's non-somatic factor; Avoid is PTSD's avoidance factor; Reexp is PTSD's reexperiencing factor; Mood/cognitions is PTSD's negative alterations in mood/cognitions factor.
* *p*< 0.01; ** *p*< 0.05; ****p*< 0.001.

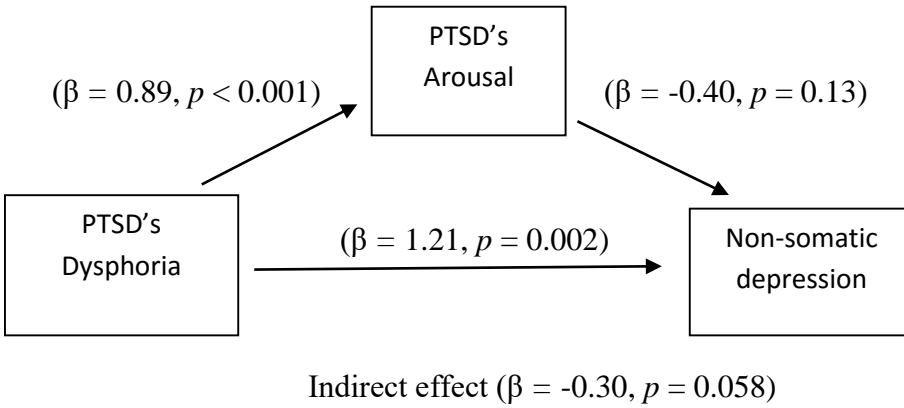Figure 1. Mediating role of PTSD's arousal in the relationship between PTSD's dysphoria and

non-somatic depression

PTSD's
Arousal

$(\beta = 0.89, p < 0.001)$

$(\beta = -0.34, p = 0.13)$

PTSD's
Dysphoria

$(\beta = 1.14, p = 0.001)$

Somatic
depression

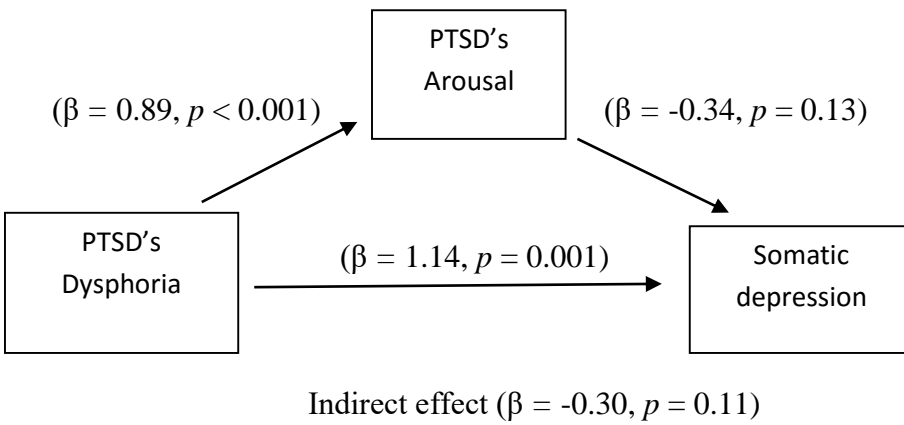Indirect effect $(\beta = -0.30, p = 0.11)$

Figure 2. Mediating role of PTSD's arousal in the relationship between PTSD's dysphoria and somatic depression