PTSD and Suicidal Ideation: An Application of the Interpersonal-Psychological Theory of Suicide

by

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Abstract

The relationship between posttraumatic stress disorder (PTSD) and passive suicidal ideation (P-SI) is well established, but the mechanisms underlying it are not well understood (Panagioti, Gooding, & Tarrier, 2012).This study sought to enhance understanding of the PTSD/P-SI relationship using the interpersonal theory of suicide (IPTS; Joiner, 2005) as a conceptual framework. It was hypothesized that the strongest relationship between individual PTSD clusters and P-SI would be found for the emotional numbing symptoms, but that this relationship would be fully mediated by one or both IPTS constructs specified as proximal causes for SI: thwarted belongingness and perceived burdensomeness. Participants were trauma-exposed college students (N = 334). Hypotheses were tested using structural equation modeling while controlling for gender and sadness. Although the numbing cluster had the strongest bivariate relationship with P-SI, full mediation through one or both IPTS constructs was not observed. Instead, numbing and reexperiencing were both directly related to P-SI, and all PTSD clusters had indirect relationships with P-SI, partially mediated through one or both IPTS constructs. Results, while not entirely consistent with hypotheses, support the utility of the IPTS for understanding P-SI among those with PTSD, and point to a unique relationship between P-SI and numbing.

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Introduction

A growing body of research has established a positive relationship between posttraumatic stress disorder (PTSD) and all forms of suicidal behavior, including suicidal ideation (SI; Panagioti, Gooding, & Tarrier, 2012). Several large epidemiological studies, including the European Study of Mental Disorders (ESMED; Bernal et al., 2007) and World Health Organization (WHO) International Mental Health survey (Nock et al., 2009) have identified PTSD diagnostic status as a significant risk factor for SI in both developed and developing countries. More recently, meta-analyses by Panagioti, Gooding, and Tarrier (2012) and Krysinska and Lester (2010) have confirmed the link between PTSD and SI, finding it to be consistent across age, gender, and trauma type. Krysinska and Lester further found that the relationship between PTSD and SI is non-spurious in that it is not fully accounted for by psychiatric comorbidity, including, most relevantly, major depressive disorder (MDD). This finding is particularly important because PTSD and MDD are highly comorbid (Breslau, Davis, Peterson, & Schultz, 2000), leading some researchers to argue that comorbid MDD accounts for the association between PTSD and SI (e.g., Oquendo et al., 2005).

Although the relationship between PTSD and SI has been widely studied, the underlying mechanisms are not well understood (Panagioti, Gooding, Dunn, & Tarrier, 2011). Most research in this area has been descriptive, examining the presence, magnitude, and consistency of the PTSD-SI relationship but not addressing its causes. Two key limitations have hampered the investigation of causal factors. First, most studies have examined PTSD as a syndrome, despite consensus in the trauma field that PTSD is a heterogeneous disorder characterized by several

distinct but related clusters of symptoms (Wilson, 2004; Elhai & Palmieri, 2011). Failure to consider PTSD at the symptom cluster level may obscure a pattern of differential associations between SI and specific PTSD symptoms clusters (Guerra & Calhoun, 2010). Second, research on the PTSD-SI relationship has been conducted outside the framework of existing theory capable of explaining the development of SI (Panagioti et al., 2009).

Unresolved questions regarding the core symptoms and latent structure of PTSD (Elhai & Palmieri, 2011) complicate study of both the disorder and its relationship to correlates such as SI. The *Diagnostic and Statistical Manual, Fourth Edition, Text Revision* (*DSM-IV-TR;* APA, 2002) implies a three-factor model of PTSD, grouping symptoms into three clusters: reexperiencing the trauma, avoidance of trauma-related cues and numbing of general responsiveness, and hyperarousal. However, confirmatory factor analyses (CFAs) support two competing four-factor models (Elhai & Palmieri, 2011). The first four-factor model, the numbing model (King, Leskin, King, & Weathers, 1998), separates avoidance and numbing into two clusters. Avoidance and numbing have been shown to be differentially related to treatment responsiveness and a variety of other types of psychopathology including SI (Asmundson, Stapleton, & Taylor, 2004), and are now separated into distinct clusters in *DSM-5* PTSD criteria (APA, 2013). The second four-factor model, the dysphoria model, also separates avoidance from numbing, but combines numbing symptoms with three hyperarousal symptoms to form a dysphoria cluster (Simms et al, 2002). Recently, in an effort to resolve differences between the numbing and dysphoria models, Elhai and colleagues (2011) proposed a five-factor model, the dysphoric arousal model, which separates avoidance and numbing but also divides the hyperarousal cluster into anxious and dysphoric arousal clusters. A number of studies have shown that the dysphoric arousal model

provides superior fit compared to the numbing and dysphoria models (e.g., Carter, 2011; Wang, Long, Li, & Armour, 2011; Reddy, Anderson, Liebschultz, & Stein, in press).

Several recent studies have examined the link between specific PTSD symptom clusters and SI with contradictory results. Tarrier and Gregg (2004) found that both hyperarousal and reexperiencing were positively associated with a composite variable used as an index of suicidal behavior (including SI, plans, and attempts). Others have found a positive association between SI or composite variables and either hyperarousal (Ben-Ya'acov & Amir, 2004; Steryn, Vawda, Wyatt, Williams, & Madu, 2013) or re-experiencing (Bell & Nye, 2007), but not both. Recently, Panagioti, Gooding, Dunn, and Tarrier (2011) re-analyzed the data from Tarrier and Gregg (2004), incorporating additional variables into their model (e.g., social and occupational impairment). They found that avoidance/numbing was indirectly positively associated with a composite variable of suicidal behavior through its association with impairment in social functioning. No other individual cluster was associated with suicidal behavior.

In contrast, two studies utilizing multiple regression have found a negative relationship between avoidance/numbing and SI (Monteith, Mnefee, Pettit, Leopoulos, & Vincent, 2013), or a composite variable including SI (Ben-Ya'acov & Amir, 2004). However, their findings may be attributable to multicollinearity: In both studies, the regression coefficient corresponding to avoidance/numbing was negative in the prediction of SI, whereas the zero-order correlation between avoidance/numbing and SI was moderate and positive. Avoidance/numbing was also highly correlated with other variables included in the regression models in both studies, which may have caused the observed flipping of signs between the bivariate relationships and structural coefficients. This suggests that results may be biased by the presence of multicollinearity and thus should not be interpreted (Cheung & Lau, 2008).

Although the findings concerning the relationship between PTSD clusters and SI are mixed, none of the noted studies found support for a direct, positive relationship between avoidance/numbing and SI. This may due to use of the *DSM-IV-TR* three-factor model which conflates avoidance and numbing. Consistent with that possibility, two other studies that used the numbing model found that only numbing was associated with SI when all other PTSD symptom clusters and depressive symptoms were controlled for (Guerra & Calhoun, 2011; Hellmuth, Stappenbeck, Hoerster, & Jakupcak, 2012). Moreover, one of these studies (Guerra & Calhoun, 2011) found that the numbing cluster had the largest bivariate relationship with SI, whereas the avoidance cluster had the smallest. Additionally, a currently unpublished study (Davis, Witte, & Weathers, 2013) examined the relationship between individual PTSD symptoms and SI. Numbing symptoms had the highest bivariate associations with SI. Further, the numbing symptoms *feelings of detachment or estrangement from others* and *sense of foreshortened future* were the only PTSD symptoms with a significant positive association with SI after controlling for depressive symptoms, response bias, and all other PTSD symptoms. Thus, distinguishing between numbing and avoidance may be especially important when examining the relationship between PTSD symptoms and SI. However, deeper understanding of how and why high levels of numbing, or any other PTSD cluster might lead to the development of SI requires reference to theory capable of explaining how and why SI develops (Panagioti et al., 2009).

One model with the potential to guide a theoretically informed investigation of the relationship between PTSD and suicidal behavior is the interpersonal-psychological theory of suicide (IPTS; Joiner, 2005; Van Orden et al., 2010). Unlike other models, the IPTS corresponds with the observed taxonomy of suicidal behavior and proposes distinct constructs to account for the prediction of different forms of suicidal behavior, including SI, suicide attempts (SA), and

death by suicide. These constructs are conceptualized as causal processes mediating the relationship between many specific risk factors (e.g., guilt and lack of social support in those with PTSD) and suicidal behavior. The theory postulates two constructs, perceived burdensomeness (PB) and thwarted belongingness (TB), as sufficient, proximal causes of SI. Perceived burdensomeness is defined as the perception or belief that one is a burden to others and that others would be better off without oneself. Likely contributors to the development of PB include loss of employment and functional impairment (Inoue et al., 2007; Kaplan, McFarland, Huguet, & Newsom, 2007). The second construct, TB, is hypothesized to develop when the fundamental human need to belong socially and to be meaningfully connected to others is not satisfied (Baumeister & Leary, 1995). Indicators of TB include isolation, loneliness, and lack of social support (Van Orden et al., 2010).

The IPTS holds that PB and TB constitute distinct but related constructs, each of which serves as a proximal cause for the development of passive SI (i.e., thoughts of suicide without intent) when present in isolation. The simultaneous presence of PB, TB, and hopelessness serves as the proximal cause for active SI (Van Orden et al. 2010). In a review paper describing the IPTS, Van Orden and colleagues (2010) noted that the theory currently describes IPTS constructs, including PB and TB, as sufficient but not necessary for the development of various forms of suicidal behavior. They also describe an alternative testable hypothesis: IPTS constructs are necessary for the development of suicidal behavior (Van Orden et al., 2010). If this alternative were true, that would imply that the relationship between any suicide risk factor and suicidal behavior is fully accounted for by the IPTS constructs.

Compared to other theoretical models attempting to account for suicidal behavior, the IPTS has advantages in terms of its ability to predict suicidal outcomes with maximal sensitivity

and specificity. First, the IPTS provides precise definitions of both its proposed constructs and the hypothesized causal pathways of those constructs to specific suicidal behavior. The level of detailed explanation provided enhances both the theory's testability and clinical usefulness. Second, the IPTS differentially accounts for various forms of suicidal behavior and does so using hypothesized constructs whose rarity, or rarity in combination, is consistent with the observed frequency of the corresponding suicidal behaviors they predict (Van Orden et al., 2010). For example, the experience of passive SI is more common than active SI, and thus correspondingly in the IPTS experiencing either PB or TB is more common than experiencing PB, TB, and hopelessness together.

In addition to its general advantages, the IPTS seems well suited for explaining SI among individuals with PTSD. In contrast to the inconsistent results found in studies examining relationships between the *DSM-IV* three-factor model and SI, the few studies that have not conflated avoidance with numbing have demonstrated a unique association between numbing symptoms and SI (Davis et al., 2013; Guerra & Calhoun, 2011; Hellmuth et al., 2012). This pattern of results fits within the IPTS framework in that numbing symptoms seem theoretically consistent as indicators of the presence of PB and TB. For example, detachment/estrangement, which is part of the numbing cluster, is the only PTSD symptom that directly refers to disrupted interpersonal connection, which both PB and TB reflect. Indeed, Davis et al. (2013) found that detachment/estrangement had a stronger association with SI than any other PTSD symptom.

As noted earlier, most existing research on the relationship between PTSD and SI has been atheorethical, with a few recent exceptions. To date, six published studies have used the IPTS framework to examine the relationship between PTSD and suicidal behavior. The first two studies -- Brenner et al. (2008) and Anestis, Bryan, Cornette, and Joiner (2009) -- used

qualitative data to test applicability of the theory's SI-relevant constructs in returning veterans. Both found support for the utility of the IPTS in understanding suicidal behavior among those with PTSD. Of the existing quantitative studies, two (Bryan, Cukrowicz, West, & Morrow, 2010; Bryan, Hernandez, Allison, & Clemans, 2012) are limited in that they focused on overall PTSD symptom severity, as opposed to PTSD symptom clusters. The two studies that have considered PTSD symptom clusters are also limited in that one (i.e., Monteith et al., 2013) utilized the *DSM-IV* three-factor model, and the other (i.e., Bryan & Anestis, 2011) only reported results for the reexperiencing symptom cluster.

Despite their limitations, the aforementioned studies provide mixed support for the notion that PTSD symptoms are associated with PB (Bryan et al., 2010; Monteith et al., 2013) and TB (Bryan et al., 2010; Bryan & Anestis, 2011; Bryan et al., 2012, Study 1; Monteith et al., 2013) . Further, there is some evidence that PB (Bryan et al., 2010; Bryan et al., 2012, Study 2; Monteith et al., 2013) and TB (Bryan et al., 2010; Bryan et al., 2012, Study 2) are associated with suicidal behavior even after accounting for depressive and PTSD symptoms. Regarding the question of whether PB and TB fully account for the relationship between PTSD symptoms and suicidal behavior, one study found that PTSD symptoms are still associated with suicidal behavior after accounting for PB and TB (Bryan et al., 2010), whereas both studies reported in Bryan et al. (2012) found no association between PTSD symptoms and suicidal behavior after accounting for PB and TB.

In sum, a growing body of research has examined the utility of the IPTS in explaining the development of SI in a PTSD population, but with significant limitations. The aim of this study was to address gaps in the current literature by using the IPTS to identify mechanisms underlying the relationship between PTSD and SI. More specifically, the primary objective of the present

study was to examine the differential relationships between specific PTSD symptom clusters, PB, TB, and SI. Like the study by Monteith and colleagues (2013), the present study examined the relationship between IPTS constructs (PB and TB), SI, and specific PTSD symptom clusters in a trauma-exposed population. However, the study design differed in several important ways.

First, Monteith and colleagues used a regression framework, whereas the present study used structural equation modeling (SEM) to test hypotheses. Use of SEM allowed for simultaneous testing a priori hypotheses regarding direct and indirect relationships between specific PTSD symptom clusters, interpersonal model constructs (PB and TB), and SI. Second, Monteith and colleagues combined passive SI (thoughts of suicide) and active SI (thoughts, desire, intent, and plans) in their outcome variable, whereas the present study focused exclusively on passive SI (P-SI). Combining the two types of SI is inconsistent with the IPTS, which states that each type is predicted by different conditions. Given interest in the individual relationships between PB, TB and PTSD clusters, hypotheses and analyses in this study focused exclusively on P-SI, which according to the theory does not require the simultaneous presence of PB and TB. Third, Monteith and colleagues used the three-factor *DSM-IV* model of PTSD, whereas the current study used CFAs to evaluate fit of the three PTSD models with the most empirical support (i.e., the numbing, dysphoria, and dysphoric arousal models). Finally, as noted earlier, the results of Monteith and colleagues reflect the likely occurrence of multicollinearity, possibly due in part to the inclusion of depressive symptoms in their analyses. The present study sought to avoid multicollinearity by identifying and eliminating content overlap between symptoms of depression and PTSD. As a result, only sadness (Beck Depression Inventory- Second Edition Item 1) was controlled for in structural analyses (see *Method* section for a more detailed explanation and justification). This decision was made to minimize multicollinearity while still

accounting for an aspect of depression that was likely to be both relevant to P-SI and conceptually distinct from other constructs included in the model.

The present study tested three hypotheses designed to address noted gaps in existing literature on the relationship between PTSD and SI. Consistent with existing factor analytic literature, the first hypothesis was that the dysphoric arousal model would show the best fit for PTSD symptom structure compared to the numbing and dysphoria models. The second hypothesis was that relative to other PTSD symptom clusters the numbing cluster would have the strongest relationship with PB, TB, and P-SI. The third hypothesis was that PB, TB, or both would fully mediate the relationship between numbing symptoms and P-SI when other relevant correlates (i.e., gender, sadness, and all other PTSD clusters) were controlled for. To test the second and third hypotheses, measurement and structural models were specified to include individual PTSD symptom clusters, gender, sadness, PB, TB, and P-SI. This was seen as providing a rigorous test of hypotheses concerning the relationships between numbing, PB, and TB in a trauma-exposed population.

Method

Participants and Procedure

Participants were undergraduates enrolled in psychology courses at a large public university in the southeastern United States. With the approval of the university's internal review board, 512 participants who self-identified as experiencing a "very stressful life event" were recruited between Spring 2011 and Spring 2012. Study participation involved completion of a battery of self-report questionnaires during proctored research sessions conducted in a university computer lab. Each session accommodated up to 20 participants with one clinical psychology graduate student and one undergraduate research assistant present to supervise. Participants were included in analyses if their index event met *DSM-IV* Criterion A1 as classified by a trained team of graduate students; this process is described in further detail below. The final sample included 334 individuals ranging in age from 17 to 54 (M = 19.65 years, SD = 3.30). The sample was predominantly female (72.20 %, n = 241), and the ethnic breakdown was 79.30 % Caucasian, 12.60 % African-American, 3.60 % Latino/Hispanic, 3.00 % Asian, and 1.50 % Other.

Measures

Trauma exposure and PTSD. The Life Events Checklist (LEC) was used to assess trauma type and exposure level. The LEC is the self-report trauma assessment portion of the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995; Weathers, Keane, & Davidson, 2001) and assesses exposure to 17 types of traumatic events (e.g., natural disaster, sexual assault). The LEC has been demonstrated to have good psychometric properties (Gray, Litz, Hsu, & Lombardo, 2004). Based on responses to the LEC the three most commonly reported

traumatic event types were car accidents (23.70 %, n = 79), sudden and unexpected death of a loved one (18.00 %, n = 60), and life threatening illness or injury (12.9%, n = 43).

After completing the LEC, participants wrote a brief narrative description of the worst traumatic event they had experienced, and this served as the index event for PTSD symptom assessment. Narrative event descriptions were coded by a team of two graduate students, supervised by Dr. Weathers, to determine whether participants met *DSM-IV* Criterion A1 for PTSD. Only participants whose index event met Criterion A1, which specifies exposure to an event that "involved actual or threatened death or serious injury or a threat to the physical integrity of self or others," were included in analyses (APA, 1994, p. 427). Interrater reliability for Criterion A1 coding was in the acceptable range (κ = .77; Landis and Koch, 1977). Discrepancies were resolved by discussion among the two raters and Dr. Weathers.

The PTSD Checklist – Specific Version (PCL-S; Weathers, Litz, Herman, Huska, & Keane, 1993) was used to measure PTSD symptom severity. The PCL-S is a widely used, well-validated self-report measure that assesses each of the 17 *DSM-IV* symptoms of PTSD. Respondents first identify an index stressful life event, then indicated how much they have been bothered by each PTSD symptom in the past month using a 5-point scale (1 = *not at all* to 5 = *extremely*). The PCL has excellent psychometric properties (McDonald & Calhoun, 2010; Wilkins, Lang, & Norman, 2011). In the current sample, internal consistency for the PCL-S was high (α = .93), mean total score was 29.86 (*SD* = 12.45), and 13.80 % of the sample (*n* = 46) met criteria for likely PTSD diagnosis based on a cutoff score of 44 established for diagnosing PTSD among treatment-seeking trauma survivors (Ruggiero, Del Ben, Scotti, & Rabalais, 2003).

Suicidal ideation. The Beck Suicide Scale (BSS; Beck & Steer, 1991) is a 21-item self-report measure designed to assess the presence and severity of SI. The first 19 items assess

suicidal ideation, planning, and preparation and are presented with variable response options which range in severity on a scale from 0 to 2. Items 20 and 21 assess history of suicide attempts and were therefore not included in study analyses. The BSS has been widely used and has strong psychometric properties (Beck, Brown, & Steer, 1997). The decision to include only select BSS items in structural analyses is discussed below in the *Results* section

IPTS constructs, The Interpersonal Needs Questionnaire (INQ) is a 15-item self-report measure designed to assess PB (6 items) and TB (9 items). Items are presented on a 7-point Likert scale with response options ranging from 0 (*not at all true for me*), to 6 (*very true for me*). The INQ has good psychometric properties (Van Orden et al., 2012). In the current sample, internal consistency for the INQ was excellent for both subscales (α = .90 for PB; α = .89 for TB), mean score for PB was 8.30 (*SD* = 4.48), and mean score for TB 19.91 (*SD* = 11.07).

Depressive symptomatology. Symptoms of depression were assessed using the Beck Depression Inventory-II (BDI-II), a widely used measure which has good psychometric properties (Beck, Steer, & Brown, 1996; Osmond, Downs, Barrios, Kopper, Gutierrez, & Chiros, 2008). On the BDI-II, respondents are presented with 21 groups of statements describing depressive symptoms and are asked to select the one statement from each group that most closely matches how they have been feeling in the past two weeks. The statements range in severity on a scale from 0 to 3. In the current sample, internal consistency for BDI-II was excellent (α = .93), and mean total score on the BDI-II was 10.56 (*SD* = 9.40).

Of concern, Monteith and colleagues (2013) reported a strong positive zero-order correlation between the avoidance/numbing PTSD symptom cluster and depression as measured by the BDI-II (*r* = .53). This information suggests that the inclusion of depressive symptoms may have contributed to the occurrence of multicollinearity in their study. To further investigate this

possibility, BDI-II item content was compared with that of other measures in the present study to identify areas of potential conceptual overlap. Multiple instances of content overlap were identified between the BDI-II and items on the PTSD Checklist (PCL). For example, BDI-II items 4 and 12 (loss of interest and loss of pleasure respectively) closely resemble PCL item 9 which refers to "loss of interest in activities that you used to enjoy." Similarly, BDI-II items 17 and 19 (reflecting irritability and difficulty concentrating) are virtually identical to PCL items 14 *feeling irritable or having angry outbursts* and 15 *having difficulty concentrating*. Each pair of BDI-II and PCL items mentioned was correlated between .57 and .60. Tables C1 and C2 contain content descriptors for all study items. Thus, as noted, to avoid potential conceptual overlap and related concerns regarding multicollinearity, only BDI-II item 1 (sadness) was controlled for in structural analyses.

Results

Descriptive Statistics

All analyses were performed in Mplus version 7.0 (Muthèn & Muthèn, 1998-2012) and IBM SPSS version 20.0. Descriptive statistics including means, standard deviations, and skew can be found in Table A1. Item-level zero-order correlations between study variables are displayed in Tables A2 - A4. In keeping with recommendations from Bentler and Chou (1987) the distributions of responses to study variables were examined at the item level to determine if items should be treated as continuous or ordered categorical. Most of the items that constitute the PCL, INQ, BSS, and item 1 of the BDI-II were found to be positively skewed (skewness values between 0.84 and 8.60), and visual inspection of the item distributions revealed that none closely approximated a normal distribution (Table A1 contains skewness and kurtosis values for all items used in analyses). Therefore, all variables were treated as ordered-categorical in structural analyses. Given this decision, a mean- and variance-adjusted weighted least squares (WLSMV) estimator was implemented for all analyses, which has been described as the preferred estimation method for analyses involving ordered-categorical variables (Muthén, du Toit, & Spisic, 1997; Flora & Curran, 2004). In Mplus, the WLSMV estimator produces probit coefficients for both factor loadings and structural coefficients, which cannot be interpreted as linear regression coefficients would be. However, correlations between latent variables in such models are equivalent to standard linear regression coefficients (Muthèn & Muthèn, 1998-2012).

Missing data were handled using pairwise present deletion, the default strategy used in Mplus when the WLSMV estimator is employed (Asparouhov & Muthèn, 2010; Muthèn &

Muthèn, 1998-2012). To evaluate the appropriateness of this strategy, the pattern and degree of missing data were examined. Little's Test was significant (χ^2 [855] = 1249.18, $p < .01$) suggesting that data were not missing completely at random (MCAR), and therefore a robust strategy for handling missing data would be more appropriate. However, use of full information maximum likelihood is not possible when a WLSMV estimator is used, and multiple imputation (MI) was attempted but could not be implemented due to difficulties with convergence (Kline, 2011). Given that only a small proportion of the data was missing (covariance coverage values were all between .96 and .99 for all pairs of items included in analyses) use of pairwise deletion was deemed acceptable, despite recognition that this strategy may lead to biased results when data are not MCAR (Brown, 2006).

Across analyses, evaluation of model fit was accomplished by applying cutoff guidelines recommended by Kline (2011) to various model fit indices. First, non-significant χ^2 values were considered an indication of good model fit (Hayduk, 1996). Second, Root Mean Square Error of Approximation (RMSEA) estimates were evaluated according to two hypotheses. Estimates lower than or equal to .05 (or the presence of that value within the RMSEA 90 % confidence interval) resulted in a failure to reject the hypothesis that the model was a "close fit," while estimates greater than or equal to .10 (or the presence of that value within the RMSEA 90 % confidence interval) resulted in failure to reject the hypothesis that the model was a "poor fit" (Browne & Cudeck, 1993). Finally, values greater than or equal to .95 on Bentler's Comparative Fit Index (CFI) and Tucker-Lewis Index were considered indicators of acceptable model fit (Hu & Bentler, 1999).

Preliminary Analyses

As a preliminary step, a CFA was performed to confirm the latent structure of the BSS and to inform decisions on which items to include in structural analyses. Review of existing literature on BSS factor structure supported a two-factor solution, dividing items reflecting suicidal ideation, desire, and intent (SI), and resolved plans and preparation (RPP; Steer, 1993: Holden & DeLisle, 2005; Joiner, Rudd, & Rajab, 1997; Witte et al., 2006). Both because study hypotheses focused on P-SI only, and given the extreme restriction of range in responses to RPP items, all RPP items (BSS item 7-BSS item 19) were removed and, a one-factor model including BSS items 1-6 was specified and tested using a WLSMV estimator. BSS items 4 (suicidal desire) and 6 (length/ duration of SI) had a tetrachoric correlation of 1.00 which prevented the model from running when both were included. That is, although not all respondents answered the two questions identically (their bivariate correlation was r = .77), 100% of individuals who responded with a "1" or a "2" on BSS item 6 responded with a "1" on BSS item 4. Based on recommendations by Mplus software developers (L. Muthén, personal communication, 2008), the items were combined into one variable such that endorsing a 0 on BSS item 4 and a 0 on BSS item 6 resulted in a 0 on the combined variable, endorsing a 0 on BSS item 4 and a 1 on BSS item 6 resulted in a 1 on the combined variable, endorsing a 1 on both variables resulted in a 2 on the combined variable, and endorsing a 1 on the BSS item 4 and a 2 on the BSS item 6 resulted in a 3 on the combined variable The modified model, specified to load BSS items 1, 2, 3, 5, and the combined BSS 4 and 6 item on one latent factor (SI) showed good fit (χ^2 [5]= 6.58, p = .25, TLI = .99, CFI = .99, RMSEA [90 % CI] = .03 [.00 - .08]) and was therefore incorporated into structural analyses. All structural results made use of the resultant P-SI variable. Based on

summed responses to the remaining 5 items comprising P-SI, 14.97 % (n = 50) of participants reported experiencing some level of P-SI as indicated by a summed score of 1 or greater.

Hypothesis 1: Evaluation of PTSD Factor Structure

To test Hypothesis 1, three CFAs were conducted to evaluate the fit of competing models of the latent factor structure of PTSD symptoms in our sample. CFAs were specified to evaluate the three models of PTSD symptom structure with the strongest empirical support in the literature: the four-factor numbing and dysphoria model, and five-factor dysphoric arousal model (Elhai & Palmieri, 2011). All three models were determined to have acceptable fit based on evaluation of standard fit indices according to the referenced cutoff guidelines. Fit information for all three models is presented in Table A5. Because the numbing and dysphoria models are not nested, a formal comparison of model fit using the χ^2 difference test was not possible. However, χ^2 difference tests were conducted to determine whether the dysphoric arousal model provided a superior fit to each of the four-factor models. A two-step alternative procedure for χ^2 difference testing was employed using the 'DIFFTEST' command in Mplus to account for the fact that the WLSMV estimator was used (Brown, 2006; Muthén & Muthén, 1998-2012). Consistent with Hypothesis 1, results indicated that the five-factor model provided significantly better fit than either the numbing (χ^2 [4] = 29.85, p < .01) or dysphoria (χ^2 [4] = 72.86, p < .01) four-factor models. As such, the five-factor model, which divides PTSD symptoms into clusters reflecting reexperiencing, avoidance, numbing, dysphoric arousal, and anxious arousal respectively, was used in structural analyses.

Hypotheses 2 and 3: Evaluation of the Full Model

Measurement and structural models were specified to assess the hypothesized relationships between all individual PTSD symptom clusters, PB, TB, and P-SI (Hypotheses 2

and 3). The measurement model was specified first with covariances estimated between all latent variables and the two observed variables-gender and sadness. The covariance between the residual terms of INQ items 11 and 12, both of which were specified to load on the TB latent factor, was also estimated based on guidance from the literature (Van Orden et al, 2012). Examination of model-fit statistics revealed evidence of acceptable overall model fit (χ^2 [696] = 1009.48, $p < .01$, TLI = .98, CFI = .98, RMSEA [90 % CI] = .04 [.03 - .04]), and all standardized factor loadings were above the recommended guideline of .40 (Brown, 2006). It is important to note that the chi-square test of model fit in this and other models presented here was significant and therefore not suggestive of adequate model fit. However, this index has been shown to be sensitive to large sample sizes, and as such may not provide an accurate estimate of model fit in this case (Brown, 2006). Recently some have called the practice of discounting significant chi-square values into question. Kline (2011), for example, argued that the presence of a significant chi-square value should be taken as an indication of a problem with the specification of the model, and recommends further diagnostics be performed to determine how the model should be respecified. Examination of the pattern of residuals to identify potential sources of strain affecting model fit did not point to any theoretically justifiable modifications to model specification for any of the models presented. Modification indices were unavailable given use of the WLSMV estimator.

Standardized and unstandardized parameter estimates from the full measurement model are available in Table A6. These values were examined to evaluate Hypothesis 2 concerning the differential relationships between numbing and other PTSD clusters with PB, TB, and P-SI. Consistent with Hypothesis 2, numbing had stronger bivariate relationships (i.e., standardized covariance estimates) with PB, TB, and P-SI than any other PTSD symptom cluster in the

measurement model. Further, examination of the 95% confidence intervals surrounding each estimate confirmed that the bivariate relationship between numbing and PB was significantly larger than that of any other cluster except dysphoric arousal. Similarly, the relationship between numbing and TB was significantly larger than that of any other cluster. Finally, the relationship between numbing and P-SI was significantly larger than the relationships between other PTSD clusters and P-SI with two exceptions: anxious arousal and dysphoric arousal. Interestingly, the bivariate relationship between numbing and P-SI was also larger than the relationship between TB and P-SI, although not significantly so. Taken together these findings support the hypothesis that numbing would have a stronger relationship with PB, TB, and P-SI than any other cluster. Notably, gender had significant positive bivariate relationships with and both avoidance and sadness, suggesting higher values of both constructs among females.

Next, to evaluate Hypothesis 3, a corresponding structural model was specified with covariances estimated between the latent variables for all PTSD symptom cluster, sadness, and gender, and between PB and TB. As in the measurement model, INQ items 11 and 12 were also allowed to covary. Direct paths were specified from all PTSD symptom clusters, sadness, and gender to PB, TB, and SI, and from PB and TB to SI. See Figure B1 for depiction of the full structural model and specified direct paths. Examination of fit indices for the structural model also suggested acceptable model fit (χ^2 [697] = 1273.73, $p < .01$, TLI = .96, CFI = .99, RMSEA [90 % CI] = .05 [.04 – .05], with all standardized factor loadings above .40. However, two indications of multicollinearity were also observed. First, the standardized coefficients corresponding to the paths from numbing to P-SI and numbing to TB were both larger than 1.0 (β = 1.24 in both cases), which may indicate the presence of severe multicollinearity (e.g., Jöreskog, 1999; Kline, 2011). Second, a number of structural coefficients (specifically the paths

from TB, avoidance, and dysphoric arousal to SI, and from various PTSD symptom clusters to both PB and TB) were negative in the structural model, despite moderate, positive associations between the same pairs of variables in the corresponding measurement model. For example, dysphoric arousal and TB had a negative association in the structural model ($\beta = -.53$, $p = .01$), despite their positive association in the corresponding measurement model ($r = .49$, $p < .01$). All standardized and unstandardized parameter estimates from the full measurement (Table A6) and structural (Table A7) models are presented to illustrate the referenced discrepancies. As noted earlier, this flipping of signs between the measurement and structural models is a key marker of multicollinearity when predictors are highly intercorrelated (Cheung & Lau, 2008). Of particular concern, dysphoric arousal and numbing were correlated .89, and reexperiencing and avoidance were correlated .91 in the measurement model (inter-item correlations between all PCL items are also available for review in Table C2). Because the presence of multicollinearity can bias parameter estimates, the results of this model were not interpreted.

Based on these findings and recommendations by Kline (2011) and others concerning the appropriate handling of multicollinearity in structural equation modeling, a decision was made to re-specify the model (Grewal, Cote, & Baumgartner, 2004). To determine what changes were appropriate, the pattern of correlations in the measurement model was examined. It was determined that the observed presence of multicollinearity was at least partially accounted for by the fact that many of the PTSD symptoms clusters were strongly positively associated (as noted above). To avoid problems associated with multicollinearity, five separate models were specified, each containing only one PTSD symptom cluster along with PB, TB, gender, sadness and SI.

Models with Individual Symptom Clusters

Figure B2 depicts the structural model specified for each individual symptom cluster including all specified direct paths. Both measurement and structural models for each of the five individual symptom clusters showed acceptable fit (See Table A8 for fit statistics associated with each model). None of the individual model results were suggestive of multicollinearity. Table A9 lists both standardized and unstandardized parameter estimates for each model. Both control variables (gender and sadness) performed consistently across models. Specifically, in all five structural models sadness had positive direct paths to TB, PB, and SI. None of the direct paths from gender to TB, PB, or P-SI were significant, and only one variable (avoidance) was significantly correlated with gender (r = .14, p = .03, suggesting that females reported slightly higher levels of avoidance).

Given that study hypotheses involve multiple mediators, a bias-corrected (BC) bootstrapping procedure recommended for use with multiple mediators by Preacher and Hayes (2008) was used to estimate indirect effects in each model. BC bootstrapping involves resampling repeatedly from the original data set with replacement and calculating an estimate of the population indirect effects in each sample. Five-thousand resamplings were performed for each model based on recommendations from the literature (Preacher & Hayes, 2008). Specific indirect effects and the associated 95 % confidence intervals for each symptom cluster through both PB and TB are presented in Table A10.

Given that the full structural model containing all PTSD symptom clusters could not be interpreted, Hypothesis 3 could not be evaluated as intended (with all other PTSD symptoms controlled for). However, contrary to Hypothesis 3, results of the model containing numbing alone did not support full mediation of the relationship between numbing and P-SI through either

PB or TB. Further, also contrary to Hypothesis 3, results for the models containing numbing and reexperiencing were quite similar. Both numbing and reexperiencing had direct paths to both PB and TB. PB, in turn, had a significant direct path to P-SI in both models, whereas TB had a significant direct path to P-SI in the reexperiencing model but not the numbing model. Finally, contrary to Hypothesis 3, numbing and reexperiencing both had significant direct paths to P-SI, as well as significant indirect effects on P-SI through PB, but not TB. Thus, although full mediation was not present, PB partially accounted for the relationship between numbing, reexperiencing, and P-SI in their respective models.

Although Hypothesis 3 was not supported, results from the structural model containing numbing did provide additional support for Hypothesis 2. Specifically, the magnitudes of the standardized direct path coefficients from numbing to PB, TB, and P-SI were larger than the standardized coefficients for corresponding paths in any other individual cluster model (see Table A9). For example, the value for the direct path coefficient from numbing to TB was $\beta = .62$, compared to $\beta = .38$ for the path from dysphoric arousal to TB, which was the next largest (full results from the dysphoric arousal model are discussed below). Additionally, a slightly greater proportion of the variance in P-SI was accounted for in the numbing model ($R^2 = .64$) compared to any other individual cluster models (R^2 values of .60-.62, see Table A8). This suggests the relationship between numbing and both P-SI and the IPTS constructs was stronger than any other cluster, including reexperiencing.

Results from the models containing anxious arousal, dysphoric arousal, and avoidance were also similar to one another. Each symptom cluster had significant direct paths to both PB and TB, and in each model, both PB and TB had significant direct paths to SI. Unlike numbing and reexperiencing, anxious arousal, dysphoric arousal, and avoidance did not have significant

direct paths to SI. However, each was indirectly related to P-SI through either both PB and TB (dysphoric arousal and avoidance), or PB alone (anxious arousal). These findings were again partially consistent with study hypotheses. Specifically, the relationships between P-SI and all three symptom clusters were partially mediated by one or both IPTS constructs. As with reexperiencing, this finding is inconsistent with Hypothesis 3, which specified that of the PTSD clusters only numbing would have a significant indirect relationship with SI.

Discussion

This study examined differential relationships between specific PTSD symptom clusters and P-SI, using the IPTS as a theoretical framework. This is the first study to simultaneously address two key limitations of previous research. First, in contrast with previous studies that either examined PTSD as a syndrome, used the rationally derived three-factor *DSM-IV* model of PTSD symptoms, or failed to consider all PTSD symptom clusters, this study examined all clusters based on the empirically derived dysphoric arousal model. Second, in contrast with previous studies that were primarily atheoretical, this study involved systematic application of the IPTS, a comprehensive and specific model of suicidal behavior with considerable potential to account for the association between PTSD and P-SI. In addition, this study is the first to use SEM to simultaneously assess relationships between PTSD clusters, IPTS constructs, and P-SI, and to explicitly assess for potential indirect effects of PTSD symptoms on SI through PB and TB.

Based on the IPTS assumption that PB and TB are sufficient and possibly necessary proximal causes of P-SI, the primary study hypothesis (Hypothesis 3) was that PB and TB would fully mediate the relationship between numbing and P-SI. As noted, due to multicollinearity this hypothesis could not be evaluated as intended. Specifically, instead of a single structural model containing all PTSD symptom clusters, it was necessary to run separate structural models for each symptom cluster. Contrary to expectations, full mediation was not observed in the structural model involving numbing. Further, an implied hypothesis was that numbing would have a significant (indirect) association with P-SI while other PTSD clusters would not. Instead, both

numbing and reexperiencing had significant direct paths to P-SI in their respective structural models. Additionally, the relationship between each PTSD symptom cluster and P-SI was partially mediated by one or both of the IPTS constructs. Thus, in this sample, every PTSD cluster was indirectly related to P-SI.

The IPTS provides some explanation for the fact that this hypothesis was not supported. Specifically, the IPTS acknowledges that the nature of the relationship between PB, TB and P-SI is somewhat unclear. The theory conservatively describes PB and TB as sufficient proximal causes for the occurrence of P-SI. A more extreme competing hypothesis is that the presence of PB, TB, or both is not only sufficient but necessary for the development of P-SI, i.e., that PB and TB lead to P-SI across situations (Van Orden et al., 2010). If proven, this hypothesis would greatly enhance the clinical usefulness of the theory. This study attempted to assess the validity of this competing hypothesis, and failed to support it. Instead, both the noted presence of partial mediation through PB and TB, and direct relationships between PB, TB, and P-SI in all but the numbing model support the original IPTS assertion that both constructs are proximally related to P-SI. One possible explanation is that the INQ did not adequately measure PB and TB. However, in their review paper on the IPTS, Van Orden and colleagues (2010) noted that the majority of existing theory (e.g., Baechler's taxonomy of suicide) suggests that causes of various forms of suicidal behavior (including P-SI) are situationally dependent. This implies that no construct or set of constructs should be capable of universally predicting SI. Thus, this study's findings are actually consistent with the majority of existing theory and not explicitly inconsistent with the IPTS itself.

At least two explanations may account for the inaccuracy of predictions concerning numbing and other PTSD clusters. First, results may have been affected by the inability to

consider all clusters simultaneously in a single model. That is, the relationships between other PTSD symptom clusters and P-SI might not have been significant in a model that included numbing as a covariate. Second, given previous research, these findings should arguably not have been unexpected. Study hypotheses assumed that PB and TB were the sole proximal causes of SI. As such, symptom clusters that did not fit theoretically as indicators of either construct were not focused on, even though in some cases (i.e. reexperiencing and hyperarousal), mixed research demonstrated a relationship between those clusters and SI (Bell & Nye, 2007; Tarrier & Gregg, 2004; Ben Yaakov & Amir, 2004). Reexperiencing in particular was both directly and indirectly related to P-SI. Similar future studies should broaden focus and consider clusters that are less theoretically consistent with IPTS constructs and potentially search for explanations for the relationship between those clusters and P-SI outside the IPTS.

Notably, other findings were more consistent with study hypotheses. Specifically, as hypothesized the dysphoric arousal model had significantly better fit than both numbing and dysphoria models, adding to a growing body of research supporting this model. Additionally, various findings supported the hypothesis that numbing would have stronger relationships with PB, TB, and SI than other PTSD clusters. In the full measurement model, numbing had stronger bivariate relationship with TB, PB, and P-SI than any other cluster, and in all but three cases (i.e., dysphoric and anxious arousal with P-SI; dysphoric arousal with PB), those differences were statistically significant. Further, the magnitudes of the relationships between numbing and TB, PB, and P-SI were larger than corresponding values in any other individual cluster structural model. The magnitude of the relationship between numbing and TB in the individual structural model in particular seemed to confirm the hypothesized theoretical consistency between the two. Finally, the lack of a significant direct relationship between TB and P-SI in the numbing model

only may in part have been due to conceptual overlap between numbing and TB. Taken together these findings support the idea that compared to other PTSD clusters, numbing has a unique and important relationship with SI.

This study had several methodological limitations. First, and most crucially, apparent multicollinearity prevented the consideration of all PTSD symptom clusters together as part of a single model. In addition to preventing evaluation of study hypotheses as intended, the need to model clusters separately limits the validity and generalizability of these findings. PTSD clusters are distinct but highly intercorrelated, and would be unlikely to present in isolation (e.g., the modal response to trauma would not be to experience only symptoms of reexperiencing, or only symptoms of hyperarousal; Wilson, 2004). Without examining a model containing all symptom clusters, it is impossible to speak to the unique relationship between specific clusters and SI as the disorder typically presents. For example, although in the individual model reexperiencing was both directly and indirectly related to SI, inclusion of numbing and other clusters might have rendered those relationships non-significant. Notably, it is possible that the occurrence of multicollinearity was affected by a second limitation, which was the non-clinical nature of the sample. Specifically, limited variability in symptom endorsement may have contributed to the unusually high inter-item and inter-cluster correlations in the sample (i.e. r = .91 between reexperiencing and avoidance), which led to muticollinearity.

A third limitation concerned the choice to control for sadness rather than all depressive symptoms in study analyses. According to the IPTS, PB and TB should be directly related to SI even when controlling for MDD (Van Orden et al., 2010). The inclusion of all depressive symptoms might arguably have constituted a more rigorous test of the theory. Further, although consensus holds that co-occurrence of PTSD and MDD is associated with increased SI compared

to PTSD alone, researchers disagree on why (i.e. whether comorbid MDD mediates or moderates the relationship between PTSD and SI; Panagioti et al., 2012). Excluding all depressive symptoms prevented this study from contributing to the ongoing debate. That said, some researchers have suggested that the high rate of comorbidity between PTSD and MDD is attributable to symptom overlap, which can contribute to multicollinearity (Brady, Kileen, Brewerton, & Lucerini, 2000). Research supporting this hypothesis is currently mixed (Tabet, Abed, & Vostanis, 2004; Taft, Resick, Watkins, & Panuzio, 2009). However, given the aims of this study the majority of depressive symptoms were excluded to avoid multicollinearity apparent in similar studies (e.g., Monteith et al., 2013).

A fourth limitation was that this study was cross-sectional and correlational in nature, which prevents inferences concerning causal relationships between study variables (Pearl, 2000), and data were collected exclusively via self-report. Ideally, future research would be longitudinal to provide information about temporal relationships between variables and would rely more heavily on clinician-administered measures. Fifth, our sample was, predominantly Caucasian, and predominantly female. Although gender was controlled for in all analyses, findings should be replicated in samples with greater ethnic diversity to determine generalizability. Sixth, for the purposes of this study all trauma types were combined, masking potential differences in the presentation of SI or IPTS constructs across type of trauma. Finally, the use of pairwise deletion as a strategy for handling missing data is a potential limitation. Specifically, this strategy can lead to biased parameter estimates, particularly when data are missing at random rather than MCAR, as was the case in our study. Ideally, a more robust strategy, such as multiple imputation in the case of a WLSMV estimator, would be used (Brown, 2006).

Despite these limitations, this study represents a significant contribution to literature on the relationship between PTSD and SI both methodologically and practically. Focusing on methodology, this was the first study to study the relationships between PTSD clusters and SI using SEM. Although this approach has clear advantages, it would ideally be used to consider PTSD symptom clusters as they occur phenomenologically, together. Future research should focus on identifying and eliminating specific causes of multicollinearity that prevent the simultaneous consideration of PTSD clusters. The choice to focus only on P-SI was another important advance. Both research and theory suggest that various forms of suicidal behavior (e.g. P-SI, desire, intent, attempts) occur at different rates, and for different reasons (Van Orden et al., 2010). Thus, deliberate focus on identifying predictors for specific forms of suicidal behavior should provide more useful and specific information.

The relationship between PB, TB, and P-SI in this sample was clear, which implies the utility of the IPTS in predicting P-SI among those with PTSD. Clinicians should consider assessing for the presence of PB and TB in this population to identify patients at risk for developing SI. Additionally, future research should investigate the applicability of the IPTS in this population with other forms of suicidal behavior (i.e., suicidal intent and attempts). However, contrary to hypotheses, PB and TB were not the only variables proximally related to P-SI. This suggests that although the IPTS is helpful, it does not provide a comprehensive explanation for the occurrence of P-SI in this population. Findings also implicated PTSD's numbing and reexperiencing clusters and sadness as variables with direct relationships to SI. Assuming that these findings are replicated, future research should focus on providing empirically and theoretically justifiable explanations for the relationship between these variables and SI. The finding concerning numbing also highlights the importance of separating avoidance

and numbing symptoms. Notably, with the recent publication of the *DSM-5* the core symptoms of PTSD have changed (APA, 2013). While these changes must be taken into account, they should not be considered to invalidate existing findings. *DSM-5* clusters corresponding to reexperiencing and numbing should be prioritized as the focus of future research, and likely intervention. More broadly, this study provides additional confirmation for the idea that differential relationships exist between specific PTSD symptom clusters and SI. Future research should continue to consider PTSD and its correlates at symptom cluster level after suitable investigation of the latent structure of the *DSM-5* symptoms.

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Appendix A: Tables

Table A1

Descriptive Statistics for Items Used in Structural Analyses.

Variable	Minimum	Maximum	Mean	SD	Skew	Kurtosis
Reexperiencing						
PCL item 1	1.00	5.00	1.90	1.02	1.27	1.20
PCL item 2	1.00	5.00	1.67	0.97	1.63	2.34
PCL item 3	1.00	5.00	1.52	0.89	1.81	2.67
PCL item 4	1.00	5.00	2.17	1.17	0.84	-0.14
PCL item 5	1.00	5.00	1.74	1.07	1.48	1.50
Avoidance						
PCL item 6	1.00	5.00	2.11	1.25	0.96	-0.19
PCL item 7	1.00	5.00	1.87	1.16	1.25	0.50
Emotional Numbing						
PCL item 8	1.00	5.00	1.71	1.07	1.53	1.54
PCL item 9	1.00	5.00	1.49	0.92	0.95	4.10
PCL item 10	1.00	5.00	1.64	1.05	1.23	1.67
PCL item 11	1.00	5.00	1.59	1.05	1.73	1.96
PCL item 12	1.00	5.00	1.47	0.92	2.05	3.54
Dysphoric Arousal						
PCL item 13	1.00	5.00	1.95	1.27	1.20	0.24
PCL item 14	1.00	5.00	1.62	1.02	1.62	1.71
PCL item 15	1.00	5.00	1.98	1.24	1.05	-0.10

(continued)

Variable	Minimum	Maximum	Mean	SD	Skew	Kurtosis
Anxious Arousal						
PCL item 16	1.00	5.00	1.86	1.24	1.34	0.60
PCL item 17	1.00	5.00	1.67	1.08	1.58	1.54
Sadness (BDI-II item 1)	1.00	4.00	1.39	0.56	1.23	1.11
Perceived Burdensomeness						
INQ item 1	1.00	6.00	1.43	0.97	2.60	6.54
INQ item 2	1.00	6.00	1.39	0.92	2.56	6.17
INQ item 4	1.00	6.00	1.20	0.64	3.84	15.80
INQ item 5	1.00	6.00	1.29	0.77	3.30	11.80
INQ item 6	1.00	7.00	1.64	1.24	2.28	5.06
Thwarted Belongingness						
INQ item 7	1.00	7.00	1.90	1.41	1.80	3.70
INQ item 8	1.00	7.00	2.52	1.78	1.10	0.23
INQ item 9	1.00	7.00	1.91	1.47	1.83	2.63
INQ item 10	1.00	7.00	1.93	1.57	1.92	3.00
INQ item 11	1.00	7.00	2.58	1.86	0.94	-0.37
INQ item 12	1.00	7.00	2.65	1.91	0.90	-0.50
INQ item 13	1.00	7.00	2.05	1.69	1.74	2.10
INQ item 14	1.00	7.00	2.29	1.72	1.30	0.70
INQ item 15	1.00	7.00	2.11	1.69	1.55	1.44

(continued)

Variable	Minimum	Maximum	Mean	SD	Skew	Kurtosis
Suicidal Ideation						
BSS item 1	1.00	1.00	0.02	0.20	6.22	36.90
BSS item 2	1.00	2.00	0.09	0.30	3.20	9.65
BSS item 3	0.00	1.00	0.04	0.20	5.00	22.82
BSS item 4/6 combined	0.00	3.00	0.03	0.26	9.00	78.13
BSS item 5	0.00	2.00	0.07	0.26	4.01	16.34

Note. SD = Standard Deviation; PCL= PTSD Checklist – Specific Version; PB = perceived burdensomeness; TB = thwarted belongingness; BDI-II = Beck Depression Inventory-Second Edition; BSS = Beck Suicide Scale.

Table A2

PTSD Checklist Inter-item Correlations

Variable	PCL1	PCL2	PCL3	PCL4	PCL5	PCL6	PCL7	PCL8	PCL9	PCL10	PCL11	PCL12	PCL13	PCL14	PCL15	PCL16
PCL 1	1.00															
PCL 2	.70*	1.00														
PCL 3	.53*	.52*	1.00													
PCL 4	.65*	.59*	.50*	1.00												
PCL 5	.59*	.58*	.49*	.69*	1.00											
PCL 6	.53*	.51*	.34*	.64*	.54*	1.00										
PCL 7	.52*	.52*	.45*	.63*	.57*	.59*	1.00									
PCL 8	.26*	.31*	.23*	.31*	.36*	.41*	.33*	1.00								
PCL 9	.36*	.32*	.34*	.38*	.39*	.44*	.44*	.27*	1.00							
PCL 10	.32*	.29*	.22*	.36*	.32*	.44*	.34*	.29*	.61*	1.00						
PCL 11	.31*	.28*	.26*	.32*	.30*	.43*	.32*	.28*	.60*	.71*	1.00					
PCL 12	.26*	.29*	.38*	.38*	.37*	.33*	.36*	.31*	.49*	.52*	.55*	1.00				
PCL 13	.31*	.33*	.31*	.39*	.32*	.37*	.35*	.32*	.55*	.53*	.52*	.49*	1.00			
PCL 14	.40*	.36*	.41*	.48*	.42*	.47*	.49*	.32*	.60*	.56*	.65*	.53*	.68*	1.00		
PCL 15	.38*	.39*	.37*	.44*	.41*	.46*	.44*	.32*	.58*	.60*	.54*	.50*	.42*	.68*	1.00	
PCL 16	.39*	.38*	.46*	.38*	.43*	.35*	.36*	.26*	.30*	.36*	.30*	.33*	.50*	.36*	.38*	1.00
PCL 17	.39*	.38*	.42*	.35*	.41*	.35*	.39*	.27*	.31*	.36*	.33*	.34*	.43*	.47*	.52*	.66*

Note. PCL= PTSD Checklist – Specific Version (PCL-S).
* $p \leq .05$.

Table A3

Bivariate Correlations Between PTSD Checklist Items and Other Study Variables

Variable	PCL1	PCL2	PCL3	PCL4	PCL5	PCL6	PCL7	PCL8	PCL9	PCL10	PCL11	PCL12	PCL13	PCL14	PCL15	PCL16	PCL17
INQ 1	.17*	.28*	.23*	.22*	.22*	.28*	.26*	.23*	.38*	.50*	.49*	.40*	.47*	.47*	.42*	.24*	.25*
INQ 2	.08	.17*	.17*	.19*	.12*	.23*	.23*	.16*	.28*	.47*	.43*	.36*	.40*	.40*	.33*	.15*	.19*
INQ 3	.10	.17*	.13*	.10	.12*	.20*	.15*	.15*	.26*	.41*	.35*	.32*	.35*	.32*	.33*	.17*	.24*
INQ 4	.11	.18*	.20*	.20*	.15*	.21*	.23*	.21*	.27*	.42*	.38*	.45*	.38*	.39*	.34*	.20*	.30*
INQ 5	.13*	.19*	.20*	.20*	.16*	.17*	.29*	.20*	.23*	.32*	.35*	.39*	.30*	.31*	.26*	.13*	.14*
INQ 6	.10	.18*	.16*	.12*	.14*	.14*	.15*	.10	.29*	.39*	.37*	.31*	.37*	.35*	.32*	.23*	.20*
INQ 7	.07	.10	.10	.12*	.06	.14*	.15*	.13*	.32*	.36*	.40*	.37*	.28*	.27*	.21*	.06	.08
INQ 8	.11*	.10	.07	.12*	.10	.17*	.17*	.11*	.33*	.50*	.43*	.37*	.28*	.29*	.26*	.17*	.13*
INQ 9	.06	.06	.14*	.08	.09	.19*	.17*	.01	.31*	.28*	.33*	.23*	.29*	.27*	.22*	.16*	.16*
INQ 10	.15*	.15*	.10	.13*	.11	.17*	.24*	.13*	.34*	.39*	.31*	.27*	.18*	.33*	.19*	.05	.11*
INQ 11	.18*	.11	.15*	.24*	.20*	.29*	.24*	.10	.39*	.51*	.47*	.34*	.34*	.37*	.37*	.26*	.26*
INQ 12	.16*	.11	.10	.21*	.21*	.22*	.24*	.11*	.33*	.46*	.40*	.28*	.28*	.31*	.34*	.26*	.30*
INQ 13	.13*	.14*	.10	.10	.04	.13*	.17*	.10	.30*	.35*	.32*	.30*	.21*	.26*	.19*	.06	.06
INQ 14	.18*	.15*	.11	.18*	.10	.17*	.21*	.11	.33*	.45*	.43*	.31*	.25*	.34*	.21*	.13*	.11*
INQ 15	.16*	.17*	.10*	.11	.09	.16*	.20*	.10	.30*	.28*	.40*	.24*	.22*	.30*	.20*	.10	.10
BSS 1	.10	.12*	.14*	.13*	.15*	.13*	.10	.14*	.22*	.23*	.23*	.27*	.20*	.14*	.23*	.20*	.13*
BSS 2	.15*	.21*	.15*	.16*	.13*	.20*	.15*	.12*	.30*	.31*	.34*	.40*	.22*	.26*	.23*	.24*	.12*
BSS 3	.10	.20*	.13*	.08	.14*	.05	.10	.10	.20*	.22*	.20*	.20*	.15*	.17*	.15*	.09	.11*
BSS 4/6	.15*	.20*	.14*	.14*	.14*	.12*	.20*	.20*	.15*	.23*	.15*	.23*	.17*	.13*	.15*	.14*	.14*
BSS 5	.10	.16*	.05	.10	.10	.05*	.07	.11*	.20*	.22*	.29*	.24*	.13*	.12*	.14*	.03	-.01
Sadness	.19*	.18*	.21*	.25*	.17*	.21*	.23*	.08	.34*	.38*	.45*	.40*	.31*	.38*	.34*	.24*	.24*

Note. PCL= PTSD Checklist – Specific Version (PCL-S); INQ = Interpersonal Needs Questionnaire; BSS = Beck Suicide Scale; Sadness = Beck Depression Inventory-Second Edition (Item 1).
* $p \leq .05$.

Table A4

Bivariate Correlations Between Sadness (BDI-II Item 1), and Items of the Interpersonal Needs Questionnaire, and Beck Suicide Scale

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. INQ 1	1.00																			
2. INQ 2	.83*	1.00																		
3. INQ 3	.72*	.64*	1.00																	
4. INQ 4	.76*	.73*	.60*	1.00																
5. INQ 5	.58*	.59*	.53*	.62*	1.00															
6. INQ 6	.59*	.54*	.64*	.46*	.49*	1.00														
7. INQ 7	.38*	.45*	.28*	.30*	.34*	.28*	1.00													
8. INQ 8	.36*	.44*	.36*	.30*	.32*	.40*	.68*	1.00												
9. INQ 9	.36*	.35*	.32*	.29*	.24*	.29*	.23*	.28*	1.00											
10.INQ 10	.27*	.32*	.27*	.24*	.22*	.26*	.61*	.60*	.26*	1.00										
11.INQ 11	.42*	.49*	.41*	.33*	.39*	.42*	.30*	.54*	.47*	.30*	1.00									
12.INQ 12	.36*	.39*	.36*	.31*	.35*	.36*	.27*	.48*	.38*	.26*	.81*	1.00								
13.INQ 13	.40*	.47*	.36*	.29*	.30*	.34*	.66*	.63*	.29*	.68*	.38*	.33*	1.00							
14.INQ 14	.37*	.42*	.33*	.27*	.30*	.31*	.64*	.65*	.36*	.65*	.44*	.39*	.78*	1.00						
15. INQ 15	.32*	.28*	.30*	.24*	.20*	.32*	.57*	.51*	.31*	.54*	.27*	.22*	.63*	.70*	1.00					
16. BSS 1	.34*	.28*	.35*	.32*	.17*	.26*	.13*	.12*	.25*	.18*	.24*	.21*	.12*	.19*	.13*	1.00				
17. BSS 2	.40*	.33*	.30*	.40*	.33*	.30*	.27*	.34*	.21*	.26*	.38*	.29*	.29*	.38*	.20*	.48*	1.00			
18. BSS 3	.32*	.22*	.25*	.35*	.27*	.23*	.18*	.18*	.10	.23*	.24*	.28*	.17*	.20*	.14*	.39*	.38*	1.00		
19. BSS 4/6	.30*	.29*	.30*	.40*	.35*	.18*	.10*	.11*	.10	.21*	.19*	.20*	.16*	.17*	.14*	.51*	.39*	.47*	1.00	
20. BSS 5	.19*	.18*	.16*	.21*	.25*	.22*	.24*	.24*	.09	.15*	.27*	.20*	.25*	.26*	.17*	.34*	.31*	.33*	.15*	1.00
21.Sadness	.39*	.38*	.35*	.40*	.35*	41*	30*	.37*	.27*	.24*	.47*	.43*	.32*	.35*	.29*	.32*	.40*	.20*	.23*	.29*

Note. INQ = Interpersonal Needs Questionnaire; BSS = Beck Suicide Scale.* $p \leq .05$.

Table A5

Fit Statistics for Three Empirically Supported Models of PTSD Symptom Structure.

Model	χ^2 (p-value)	Degrees of Freedom	RMSEA Estimate (90% CI)	CFI	TLI
Numbing Model (4-Factors)	359.16 ($p \leq .01$)	113	.08 (.07-.09)	.97	.96
Dysphoria Model (4-Factors)	252.16 ($p \leq .01$)	113	.06 (.05-.07)	.98	.98
Dysphoric Arousal Model (5-Factors)	216.78 ($p \leq .01$)	109	.06 (.04-.06)	.98	.98

Note. Numbing Model = four-factor numbing model proposed by King et al., 1995; Dysphoria model = four factor model proposed by Simms et al., 2002; Elhai model = five-factor model proposed by Elhai et al., 2011; RMSEA = Root Mean Square Error of Approximation; CFI = Bentler Comparative Fit Index; TLI = Tucker-Lewis Index.

Table A6

Standardized and Unstandardized Covariance Estimates for the Full Measurement Model

Covariance Estimated	Estimate /S.E. (p-value)	95 % CI	STDYX (p-value)	95 % CI
P-SI with Rxp	0.30 / 0.07 (p < .01)	(0.15 - 0.44)	.40 (p < .01)	(.23 - .57)
P-SI with Avoid	0.27 / 0.08 (p < .01)	(0.11 - 0.43)	.36 (p < .01)	(.17 - .56)
P-SI with Numb	0.32 / 0.05 (p < .01)	(0.21 - 0.42)	.70 (p < .01)	(.58 - .82)
P-SI with Anx arousal	0.32 / 0.08 (p < .01)	(0.15 - 0.48)	.40 (p < .01)	(.21 - .58)
P-SI with Dys arousal	0.39 / 0.09 (p < .01)	(0.21 - 0.56)	.50 (p < .01)	(.32 - .68)
P-SI with Gender	-0.03 / 0.10 (p = .74)	(-0.23 - 0.17)	-.04 (p = .74)	(-.26 - .18)
P-SI with Sadness	0.66 / 0.07 (p < .01)	(0.53 - 0.79)	.72 (p < .01)	(.62 - .81)
P-SI with TB	0.52 / 0.06 (p < .01)	(0.49 - 0.80)	.67 (p < .01)	(.56 - .78)
P-SI with PB	0.65 / 0.08 (p < .01)	(0.39 - 0.64)	.73 (p < .01)	(.63 - .83)
TB with Rxp	0.17 / 0.04 (p < .01)	(0.09 - 0.26)	.25 (p < .01)	(.14 - .37)
TB with Avoid	0.26 / 0.04 (p < .01)	(0.17 - 0.34)	.37 (p < .01)	(.26 - .49)
TB with Numb	0.28 / 0.04 (p < .01)	(0.21 - 0.36)	.68 (p < .01)	(.61 - .76)
TB with Anx arousal	0.23 / 0.05 (p < .01)	(0.15 - 0.32)	.33 (p < .01)	(.21 - .44)
TB with Dys arousal	0.35 / 0.04 (p < .01)	(0.27 - 0.42)	.49 (p < .01)	(.40 - .58)
TB with Gender	0.04 / 0.06 (p = .58)	(-0.09 - 0.16)	.04 (p = .58)	(-.23 - .20)
TB with Sadness	0.48 / 0.04 (p < .01)	(0.40 - 0.56)	.57 (p < .01)	(.48 - .66)
TB with PB	0.61 / 0.03 (p < .01)	(0.54 - 0.67)	.75 (p < .01)	(.69 - .80)
PB with Rxp	0.24 / 0.05 (p < .01)	(0.13 - 0.34)	.30 (p < .01)	(.17 - .43)
PB with Avoid	0.32 / 0.06 (p < .01)	(0.21 - 0.43)	.41 (p < .01)	(.37 - .54)
PB with Numb	0.33 / 0.04 (p < .01)	(0.24 - 0.43)	.70 (p < .01)	(.61 - .79)
PB with Anx arousal	0.31 / 0.06 (p < .01)	(0.18 - 0.43)	.37 (p < .01)	(.23 - .51)
PB with Dys arousal	0.50 / 0.05 (p < .01)	(0.41 - 0.60)	.62 (p < .01)	(.52 - .72)
PB with Gender	0.03 / 0.08 (p = .74)	(-0.14 - 0.19)	.03 (p = .74)	(-.14 - .20)

(continued)

Covariance Estimated	Estimate /S.E. (p-value)	95% CI	STDYX (p-value)	95% CI
PB with Sadness	0.60 / 0.05 (p < .01)	(0.50 - 0.69)	.62 (p < .01)	(.52 - .72)
Rxp with Avoid	0.61 / 0.04 (p < .01)	(0.54 - 0.68)	.91 (p < .01)	(.86 - .96)
Rxp with Numb	0.26 / 0.04 (p < .01)	(0.19 - 0.33)	.64 (p < .01)	(.56 - .73)
Rxp with Anx arousal	0.48 / 0.04 (p < .01)	(0.39 - 0.56)	.68 (p < .01)	(.59 - .76)
Rxp with Dys arousal	0.44 / 0.04 (p < .01)	(0.37 - 0.52)	.64 (p < .01)	(.55 - .72)
Rxp with Gender	0.12 / 0.06 (p = .05)	(0.00 - 0.25)	.15 (p = .05)	(.00 - .30)
Rxp with Sadness	0.26 / 0.05 (p < .01)	(0.15 - 0.36)	.31 (p < .01)	(.19 - .44)
Avoid with Numb	0.30 / 0.04 (p < .01)	(0.22 - 0.36)	.75 (p < .01)	(.67 - .83)
Avoid with Anx arousal	0.44 / 0.05 (p < .01)	(0.35 - 0.53)	.63 (p < .01)	(.53 - .73)
Avoid with Dys arousal	0.49 / 0.04 (p < .01)	(0.31 - 0.56)	.71 (p < .01)	(.62 - .79)
Avoid with Gender	0.16 / 0.07 (p = .03)	(0.02 - 0.30)	.20 (p = .03)	(.02 - .36)
Avoid with Sadness	0.30 / 0.06 (p < .01)	(0.19 - 0.41)	.37 (p < .01)	(.24 - .50)
Numb with Anx arousal	0.27 / 0.04 (p < .01)	(0.19 - 0.34)	.63 (p < .01)	(.53 - .72)
Numb with Dys arousal	0.37 / 0.04 (p < .01)	(0.28 - 0.46)	.89 (p < .01)	(.85 - .93)
Numb with Gender	0.17 / 0.04 (p = .69)	(-0.07 - 0.10)	.03 (p = .70)	(-.14 - .21)
Numb with Sadness	0.30 / 0.04 (p < .01)	(0.22 - 0.38)	.61 (p < .01)	(.50 - .71)
Anx arousal with	0.50 / 0.04 (p < .01)	(0.42 - 0.58)	.69 (p < .01)	(-.15 - .18)
Dys arousal				
Anx arousal with Gender	0.14 / 0.08 (p = .07)	(-0.01 - 0.29)	.16 (p = .07)	(-.01 - .33)
Anx arousal with Sadness	0.33 / 0.06 (p < .01)	(0.21 - 0.46)	.39 (p < .01)	(.25 - .52)
Dys arousal with Gender	0.01 / 0.07 (p = .84)	(-0.13 - 0.16)	.02 (p = .84)	(-.15 - .18)
Dys arousal with Sadness	0.43 / 0.06 (p < .01)	(0.31 - 0.53)	.50 (p < .01)	(.38 - .63)
Gender with Sadness	0.20 / 0.09 (p = .03)	(0.02 - 0.37)	.20 (p = .03)	(.02 - .37)

Note. CI = confidence interval; PB = perceived burdensomeness; TB = thwarted belongingness; P-SI = passive suicidal ideation, desire and intent; Rxp = reexperiencing; Avoid = avoidance; Numb = emotional numbing; Anx arousal = anxious arousal; Dys arousal = dysphoric arousal.

*p ≤ .05.**p≤ .01.

Table A7

Standardized and Unstandardized Parameter Estimates for the Full Structural Model

Path Estimated	Estimate /S.E. (p-value)	STDYX (p-value)
TB on Gender	-0.02 / 0.22 (p = .81)	.02 (p = .81)
P-SI on Gender	-0.02 / 0.16 (p = .69)	-.04 (p = .67)
PB on Gender	-0.06 / 0.10 (p = .85)	.01 (p = .85)
TB on Reexperiencing	-0.27 / 0.25 (p = .28)	-.26 (p = .28)
P-SI on Reexperiencing	0.86 / 0.52 (p = .10)	.77 (p = .09)
PB on Reexperiencing	-0.23 / 0.30 (p = .43)	.38 (p = .43)
TB on Avoidance	-0.02 / 0.30 (p = .96)	-.02 (p = .96)
P-SI on Avoidance	-1.09 / 0.62 (p = .08)	-.97 (p = .07)
PB on Avoidance	-0.05 / 0.36 (p = .89)	-.04 (p = .89)
TB on Emotional Numbing	2.11 / 0.45 (p < .01)	1.24 (p < .01)
P-SI on Emotional Numbing	2.32 / 0.93 (p = .01)	1.24 (p = .02)
PB on Emotional Numbing	1.41 / 0.49 (p < .01)	.72 (p < .01)
TB on Anxious Arousal	0.06 / 0.11 (p = .57)	.06 (p = .67)
P-SI on Anxious Arousal	0.02 / 0.20 (p = .91)	.02 (p = .91)
PB on Anxious Arousal	-0.04 / 0.12 (p = .71)	-.04 (p = .71)
TB on Dysphoric Arousal	-0.52 / 0.20 (p < .01)	-.53 (p = .01)
SI on Dysphoric Arousal	-0.80 / 0.44 (p = .07)	-.73 (p = .08)
PB on Dysphoric Arousal	0.10 / 0.24 (p = .69)	.09 (p = .69)
TB on Sadness	0.19 / 0.08 (p = .02)	.13 (p = .02)
SI on Sadness	0.30 / 0.12 (p = .02)	.18 (p = .02)
PB on Sadness	0.32 / 0.09 (p <.01)	.19 (p <.01)
P-SI on TB	-0.02 / 0.22 (p = .92)	-.02 (p = .92)
P-SI on PB	0.36 / 0.16 (p = .03)	.38 (p = .02)
Numbing with Gender	0.01 / 0.02 (p = .70)	.03 (p = .70)
Numbing with Dysphoric Arousal	0.37 / 0.05 (p <.01)	.89 (p <.01)

(continued)

Path Estimated	Estimate /S.E. (p-value)	STDYX (p-value)
Numbing with Reexperiencing	0.26 / 0.04 (p <.01)	.64 (p < .01)
Numbing with Avoidance	0.30 / 0.20 (p <.01)	.75 (p < .01)
Numbing with Anxious Arousal	0.27 / 0.04 (p <.01)	.63 (p < .01)
Numbing with Sadness	0.13 / 0.02 (p <.01)	.47 (p < .01)
Gender with Dysphoric Arousal	0.01 / 0.02 (p = .85)	.01 (p = .85)
Gender with Reexperiencing	0.04 / 0.02 (p = .05)	.11 (p = .05)
Gender with Avoidance	0.05 / 0.02 (p = .03)	.14 (p = .03)
Gender with Anxious Arousal	0.05 / 0.03 (p = .08)	.12 (p = .08)
Dysphoric Arousal with Reexperiencing	0.44 / 0.04 (p <.01)	.12 (p < .01)
Dysphoric Arousal with Avoidance	0.49 / 0.04 (p <.01)	.71 (p < .01)
Dysphoric Arousal with Anxious Arousal	0.50 / 0.04 (p <.01)	-.04 (p < .01)
Dysphoric Arousal with Sadness	0.19 / 0.03 (p <.01)	.40 (p < .01)
Reexperiencing with Avoidance	0.61 / 0.04 (p <.01)	.91 (p < .01)
Reexperiencing with Anxious Arousal	0.48 / 0.04 (p <.01)	.68 (p < .01)
Reexperiencing with Sadness	0.12 / 0.03 (p <.01)	.25 (p <.01)
Avoidance with Anxious Arousal	0.44 / 0.05 (p <.01)	.63 (p < .01)
Avoidance with Sadness	0.13 / 0.03 (p <.01)	.29 (p < .01)
Anxious Arousal with Sadness	0.15 / 0.03 (p <.01)	.31 (p < .01)
PB with TB	0.17 / 0.04 (p <.01)	.49 (p < .01)
Gender with Sadness	0.03 / 0.02 (p = .05)	.11 (p = .05)

Note. PCL= PTSD Checklist – Specific Version; PB = perceived burdensomeness; TB = thwarted belongingness; P-SI = passive suicidal ideation.
* $p \leq .05$. ** $p \leq .01$.

Table A8

Model Fit Statistics for Models Including Single PTSD Symptom Clusters.

PTSD Symptom Cluster Included	χ^2 (p-value)	Degrees of Freedom	RMSEA Estimate (90% CI)	CFI	TLI	R^2 (TB)	R^2 (PB)	R^2 (SI)
Reexperiencing	561.63 ($p \leq .01$)	310	.05 (.04-.06)	.98	.98	.22	.27	.62
Avoidance	526.75 ($p \leq .01$)	238	.06 (.05-.07)	.97	.97	.26	.31	.60
Numbing	598.09 ($p \leq .001$)	310	.05 (.05-.06)	.97	.97	.49	.52	.64
Dysphoric Arousal	554.70 ($p \leq .01$)	261	.06 (.05-.07)	.97	.97	.32	.45	.60
Anxious Arousal	542.66 ($p \leq .01$)	238	.06 (.06-.07)	.97	.96	.23	.29	.61

Note. RMSEA = root mean square error of approximation; CFI = Bentler comparative fit index; TLI = Tucker-Lewis index; R^2 = coefficient of determination; TB = thwarted belongingness; PB = perceived burdensomeness.

Table A9

Standardized and Unstandardized Parameter Estimates for Structural Models Containing Individual PTSD Symptom Clusters

Parameter Estimated	Reexpereincing Model		Avoidance Model		Numbing Model		Anxious Arousal Model		Dysphoric Arousal Model	
	Estimate (S.E.)	STDYX	Estimate (S.E.)	STDYX	Estimate (S.E.)	STDYX	Estimate (S.E.)	STDYX	Estimate (S.E.)	STDYX
TB on Gender	-0.06 (0.10)	-.03	-0.09 (0.10)	-.05	-0.01 (0.10)	-.01	-0.07 (0.10)	-.04	-0.01 (0.01)	-.01
P-SI on Gender	-0.20 (0.16)	-.10	-0.18 (0.16)	-.09	-0.17 (0.15)	-.08	-0.19 (0.16)	-.09	-0.17 (0.15)	-.08
PB on Gender	-0.11 (0.13)	-.05	-0.15 (0.13)	-.07	-0.04 (0.13)	-.02	-0.12 (0.13)	-.06	-0.04 (0.12)	-.02
TB on PTSD Sx Cluster	0.16 (0.06)*	.16	0.29 (0.07)**	.28	1.33 (0.24)**	.62	0.21 (0.06)**	.21	0.36 (0.05)**	.38
P-SI on PTSD Sx Cluster	0.17 (0.09)*	.16	0.04 (0.12)	.03	0.73 (0.30)*	.31	0.11 (0.10)	.10	0.03 (0.13)	.03
PB on PTSD Sx Cluster	0.22 (0.07)*	.20	0.35 (0.09)**	.30	1.49 (0.27)**	.61	0.28 (0.08)**	.25	0.55 (0.06)**	.51
TB on Sadness	0.61 (0.06)**	.41	0.55 (0.06)*	.37	0.22 (0.07)*	.15	0.57 (0.07)**	.38	0.44 (0.07)**	.29
P-SI on Sadness	0.39 (0.07)**	.24	0.41 (0.08)**	.26	0.35 (0.08)**	.21	0.40 (0.08)**	.24	0.41 (0.08)**	.25
PB on Sadness	0.75 (0.07)**	.44	0.69 (0.07)**	.41	0.33 (0.08)**	.20	0.71 (0.08)**	.42	0.49 (0.08)**	.28
P-SI on TB	0.29 (0.11)**	.26	0.29 (0.11)*	.27	0.17 (0.13)	.16	0.29 (0.11)*	.26	0.29 (0.11)*	.27
P-SI on PB	0.33 (0.10)*	.35	0.36 (0.10)**	.37	0.25 (0.11)*	.27	0.34 (0.10)**	.36	0.35 (0.10)**	.37
TB with PB	0.41 (0.03)**	.67	0.37 (0.04)**	.65	0.19 (0.04)**	.49	0.39 (0.04)**	.66	0.30 (0.10)**	.61
Gender with PTSD Cluster	0.04 (0.02)	.11	0.05 (0.03)*	.14	0.01 (0.01)	.03	0.05 (0.03)	.12	0.01 (0.03)	.01
Gender with Sadness	0.03 (0.02)	.11	0.03 (0.02)	.11	0.03 (0.02)	.11	0.03 (0.02)	.11	0.03 (0.02)	.11
PTSD Cluster with Sadness	0.12 (0.03)**	.25	0.13 (0.03)**	.29	0.10 (0.02)**	.48	0.14 (0.03)**	.31	0.20 (0.03)**	.40

Note. TB = thwarted belongingness; P-SI = passive suicidal ideation; PB = perceived burdensomeness; STDYX = standardized solution outputted by Mplus; S.E. = standard error; Sx = symptom. * $p \leq .05$. ** $p \leq .01$.

Table A10

Indirect Effects of Each PTSD symptom cluster on Suicidal Ideation Through Perceived Burdensomeness and Thwarted Belongingness.

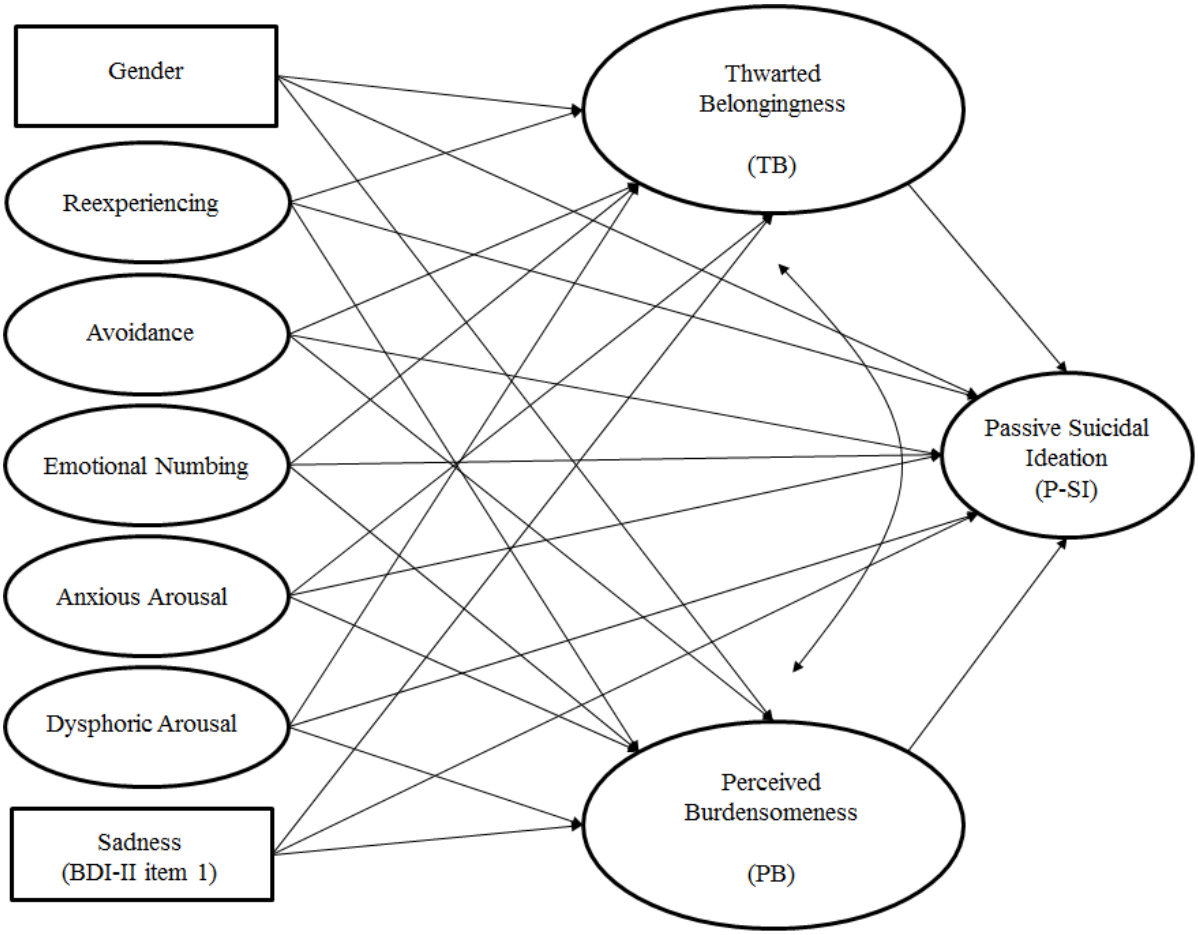
PTSD Symptom Cluster Included	Specific Indirect Effect on SI through PB		Specific Indirect Effect on SI through TB	
	Estimate (95 % CI)	STDYX (95 % CI)	Estimate (95 % CI)	STDYX (95 % CI)
Reexperiencing	0.07 (0.00 – 0.14)	0.07* (0.01 – 0.13)	0.04 (0.00 – 0.11)	0.04 (-0.01 – 0.09)
Avoidance	0.13 (0.01 – 0.23)	0.11* (0.02 – 0.20)	0.08 (0.02 – 0.20)	0.07* (0.01 – 0.14)
Numbing	0.38 (-0.10 – 0.76)	0.16* (0.00 – 0.32)	0.10 (-0.20 – 0.65)	0.10 (-0.05 – 0.25)
Dysphoric Arousal	0.19 (0.01 – 0.33)	0.19* (.04 – 0.33)	0.10 (0.02 – 0.21)	0.10* (.02 – 0.18)
Anxious Arousal	0.10 (0.01 – 0.19)	0.09* (0.01 – 0.17)	0.06 (0.01 – 0.16)	0.05 (0.00 – 0.12)

Note. PTSD = posttraumatic stress disorder; SI = suicidal ideation; PB = perceived burdensomeness; TB = thwarted belongingness. * $p \leq .05$. ** $p \leq .01$.

Appendix B: Figures

Figure B1

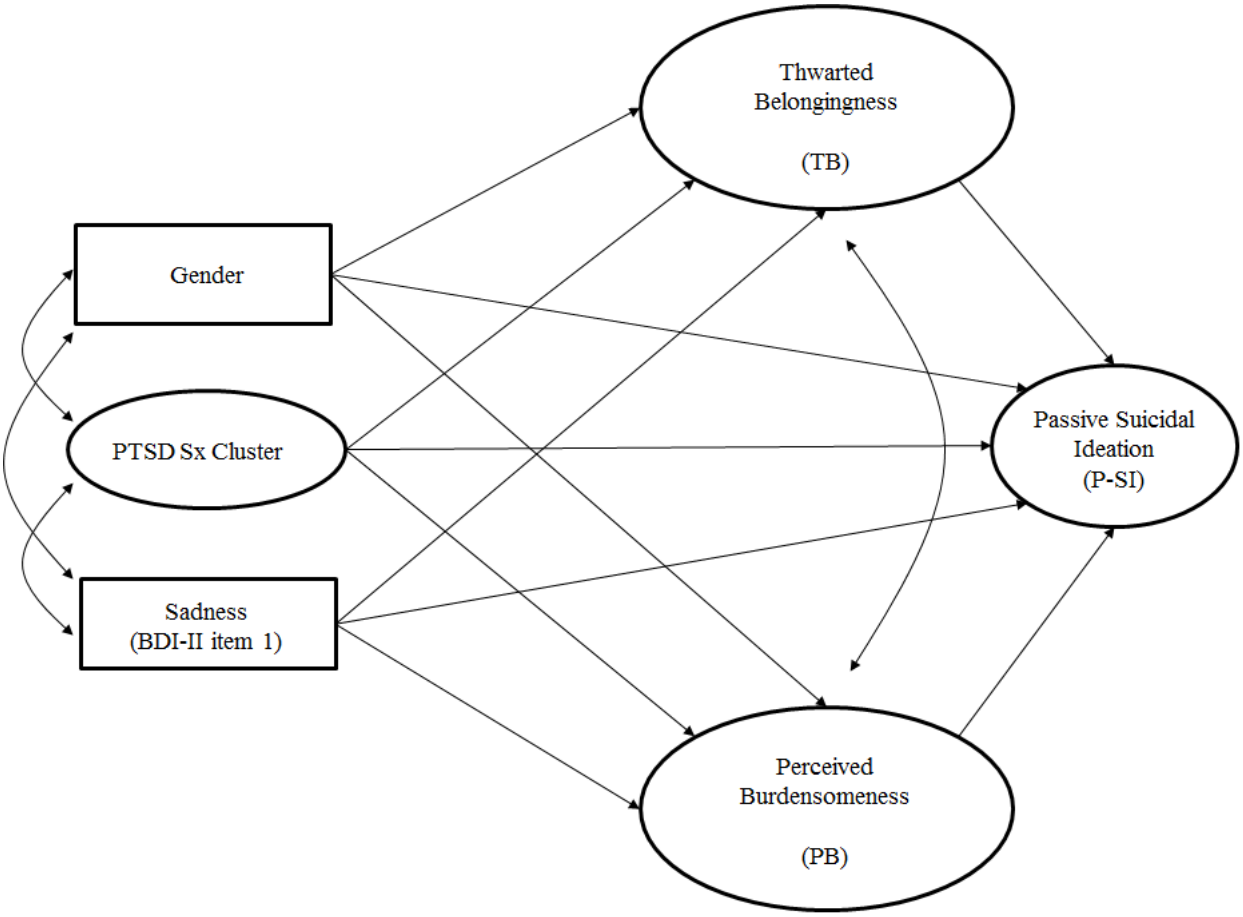
Full Structural Model



Note. Covariance values between all PTSD symptom clusters, gender, and sadness, and between PB and TB were also estimated, but are not depicted in this diagram to enhance readability. BDI-II = Beck Depression Inventory-Second Edition; SI = suicidal ideation.

Figure B2

Individual Symptom Cluster Models with all Specified Paths.



Note. BDI = Beck Depression Inventory-Second Edition; PTSD = posttraumatic stress disorder; Sx = symptom.

Appendix C: Item Content

Table C1

PCL Item Mapping for Numbing, Dysphoria, and Dysphoric Arousal CFA Models

PCL-S Item	DSM-IV PTSD Symptom Description		Numbing Model	Dysphoria Model	Dysphoric Arousal Model
1	B1	Intrusive thoughts of trauma	R	I	R
2	B2	Recurrent dreams of trauma	R	I	R
3	B3	Flashbacks	R	I	R
4	B4	Emotional reactivity to trauma cues	R	I	R
5	B5	Physiological reactivity to trauma cues	R	I	R
6	C1	Avoiding thoughts of trauma	A	A	A
7	C2	Avoiding reminders of trauma	A	A	A
8	C3	Inability to recall aspects of trauma	N	D	N
9	C4	Loss of interest	N	D	N
10	C5	Detachment	N	D	N
11	C6	Restricted affect	N	D	N
12	C7	Sense of foreshortened future	N	D	N
13	D1	Sleep disturbance	H	D	DA
14	D2	Irritability	H	D	DA
15	D3	Difficulty Concentrating	H	D	DA
16	D4	Hypervigilance	H	H	AA
17	D5	Exaggerated startle response	H	H	AA

Note. Numbing Model = four-factor numbing model proposed by King et al., 1995; Dysphoria model = four factor model proposed by Simms et al., 2002; Dysphoric Arousal model = five-factor model proposed by Elhai et al., 2011; PCL-S= PTSD Checklist, specific version; PTSD = posttraumatic stress disorder; I = Intrusions; R = Re-experiencing; A = Avoidance; N = Emotional Numbing; H = Hyperarousal; D = Dysphoria; DA = Dysphoric Arousal; AA = Anxious Arousal.

Table C2

Content Descriptors For All Item From the Interpersonal Need Questionnaire, Beck Suicide Scale, and Beck Depression Inventory (Second Edition) Used in Analyses.

Scale (Latent Factor)	Item Number	Item Content
INQ (Perceived Burdensomeness)		
	INQ item 1	Better off without me
	INQ item 2	Happier without me
	INQ item 3	Burden to society
	INQ item 4	Death as a relief
	INQ item 5	Rid of me
	INQ item 6	Makes things worse
INQ (Thwarted Belongingness)		
	INQ item 7*	Others care about me
	INQ item 8*	Feel like I belong
	INQ item 9	Rarely interact with caring others
	INQ item 10*	Have caring/supportive friends
	INQ item 11	Feel disconnected
	INQ item 12	Feel like an outsider
	INQ item 13*	People I can turn to
	INQ item 14*	Close to others
	INQ item 15*	One satisfying interaction/day

(continued)

Scale (Latent Factor)	Item Number	Item Content
BSS (Suicidal Ideation)		
	BSS item 1	Wish to live
	BSS item 2	Wish to die
	BSS item 3	Reasons for living (> for dying)
	BSS item 4/6	Desire for suicide/ Length and duration of SI
	BSS item 5	Passive desire for suicide
Sadness		
	BDI-II item 1	Sadness

Note. INQ = Interpersonal Needs Questionnaire; BSS = Beck Suicide Scale; BDI-II = Beck Depression Inventory (Second Edition); * Denotes items that were reverse coded for analyses.