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Dimensional Structure and Correlates of Posttraumatic Stress Symptoms Following Suspected Acute Coronary Syndrome

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Abstract

Background—Posttraumatic stress disorder (PTSD) is a heterogeneous construct, and some have suggested that PTSD triggered by acute coronary syndrome (ACS) may differ from PTSD due to prototypical traumas.

Methods—We conducted the first examination of the latent structure of PTSD symptoms after suspected ACS in 399 adults in the REactions to Acute Care and Hospitalization (REACH) study, an observational cohort study of patients recruited from the emergency department during evaluation for ACS. Using confirmatory factor analysis, we compared the 4-factor dysphoria, 4-factor numbing, and 5-factor dysphoric arousal models of PTSD.

Results—Although all models fit well, the dysphoria model was selected as the best-fitting model. Further, there was measurement invariance of the dysphoria model by sex. PTSD dimensions evidenced differential associations with indicators of threat perception during ACS evaluation and adherence to cardioprotective medication.

Limitations—One limitation of this investigation is the use of self-report measures. In addition, only one-third of the sample was diagnosed with ACS at discharge; the remaining participants

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received diagnoses such as chest pain without a cardiac diagnosis, another symptom/disease process (e.g., hypertensive chronic kidney disease), or another cardiac disease.

Conclusions—Findings suggest that suspected ACS-related PTSD symptoms are best-represented by a 4-factor structure distinguishing between specific (e.g., re-experiencing) and non-specific (dysphoria) symptoms of PTSD that has received support in the broader PTSD literature.

Keywords

Posttraumatic stress disorder; acute coronary syndrome; latent structure; confirmatory factor analysis

1. Introduction

Unlike most psychiatric disorders, posttraumatic stress disorder (PTSD) is contingent upon exposure to a traumatic event [American Psychiatric Association (APA), 2013]. In recent years, PTSD that develops in response to acute life-threatening medical events, such as acute coronary syndrome (ACS), has received increasing attention as an important public health concern (see Edmondson, 2014, for a review). ACS encompasses a variety of conditions in which the blood supply to the heart is suddenly blocked, including acute myocardial infarction and unstable angina (Ruff and Braunwald, 2011), and over 1 million individuals are hospitalized in the United States each year for ACS (Mozaffarian et al., 2015). ACS patients frequently report peritraumatic experiences that are associated with risk for developing PTSD, including intense fear, perceived life threat, helplessness, and a lack of control (Edmondson, 2014; Holbrook et al., 2001). Meta-analytic evidence suggests that PTSD due to ACS is common, with approximately 12% of individuals developing significant PTSD symptoms in response to ACS (Edmondson et al., 2012). Moreover, elevated ACS-related PTSD symptoms have been associated with double the risk of ACS recurrence and all-cause mortality (Edmondson et al., 2012), as well as with lower adherence to recommended health behaviors, such as adherence to cardiovascular medications (Kronish, Edmondson et al., 2012).

Although PTSD has often been treated as a homogeneous diagnostic entity, a well-established literature indicates that this disorder is a heterogeneous construct (Forbes et al., 2010; Zoellner et al., 2014). A growing body of factor analytic studies has supported 4- and 5-factor models of PTSD symptoms. The 4-factor dysphoria model has factors for re-experiencing, avoidance, hyperarousal, and dysphoria symptoms, with the dysphoria factor defined by symptoms reflecting non-specific aspects of emotional disorders, such as insomnia and irritability (Simms et al., 2002). The 4-factor numbing model separates avoidance and emotional numbing symptoms into distinct factors, resulting in re-experiencing, avoidance, numbing, and hyperarousal factors (King et al., 1998). The main distinction between these models is whether three hyperarousal items, namely sleep disturbance, irritability/anger, and difficulty concentrating, are indicators of dysphoria (as in the dysphoria model) or hyperarousal (as in the numbing model). Meta-analytic evidence suggests that both 4-factor models characterize PTSD symptoms well (Yufik and Simms, 2010), and this work influenced the number of symptom clusters included in the revised PTSD diagnosis for the fifth edition of the *Diagnostic and Statistical Manual of Mental*

Disorders (*DSM-5*; APA, 2013). Indeed, the *DSM-5* PTSD criteria include a 4-factor model of symptoms that is most similar to the numbing model. A 5-factor dysphoric arousal model has also been developed that separates hyperarousal symptoms into those reflecting dysphoric arousal (i.e., symptoms of agitation and restlessness) and anxious arousal (i.e., fear-based arousal symptoms), resulting in re-experiencing, avoidance, numbing, dysphoric arousal, and anxious arousal factors (Elhai et al., 2011). The dysphoric arousal model has been found to be superior to the two 4-factor models in several samples (e.g., Armour et al., 2013; Harpaz-Rotem et al., 2014; Pietrzak et al., 2012).

Studying the measurement invariance of PTSD symptom structure is important because it can indicate whether model features remain stable as a function of clinically-relevant factors, such as sample characteristics (e.g., age; Sumner et al., 2014) or conditions (endorsement of the subjective fear/helplessness/horror Criterion A2; Armour, Layne et al., 2011). In the broader literature, PTSD symptoms have been found to be more common and severe in women than in men (Kessler et al., 1995; Tolin and Foa, 2006), and several studies have examined sex differences in the factor structure of PTSD symptoms (e.g., Armour, Elhai et al., 2011; Hall et al., 2012; Sumner et al., 2014; Wang et al., 2013). Although there is variability in the degree of support for measurement invariance in the dysphoria, numbing, and dysphoric arousal models of PTSD symptoms across sex, a number of investigations have documented higher item intercepts and/or factor means (suggestive of greater PTSD severity) for female than male participants (Armour, Elhai et al., 2011; Sumner et al., 2014; Wang et al., 2013; although see Hall et al., 2012, for an exception).

In addition, it is of interest to examine correlates of PTSD symptom dimensions. Indeed, researchers have emphasized the importance of validating distinct PTSD dimensions against functional correlates rather than solely relying on model fit statistics (Elhai and Palmieri, 2011). Accordingly, evidence of differential associations between symptom dimensions of the dysphoric arousal model with neurobiological correlates of PTSD (e.g., serotonergic receptor density and norepinephrine transporter availability in the brain; Pietrzak, Gallezot et al., 2013; Pietrzak, Henry et al., 2013) suggests that studying PTSD symptom dimensions may help to refine understanding of how underlying factors relate to manifestations of the PTSD phenotype.

Despite extensive research on the dimensional structure of PTSD in veterans (e.g., Harpaz-Rotem et al., 2014; Pietrzak et al., 2012) and nationally representative samples exposed to a variety of traumas (Armour et al., 2013), to date, no study of which we are aware has examined the latent structure of PTSD symptoms that develop in response to ACS. Elucidating the dimensions underlying this disorder can further the understanding of manifestations of PTSD symptoms induced by ACS. Some researchers have hypothesized that PTSD triggered by discrete external traumatic events such as combat exposure and physical assault is distinct from PTSD triggered by acute manifestations of chronic disease due, in part, to differences in the nature of certain PTSD symptoms. For example, the re-experiencing symptoms of individuals with PTSD triggered by an acute presentation of a chronic illness may be focused on enduring threats of recurrence and functional decline as opposed to a discrete past event (Edmondson, 2014). Tests of whether the latent structure of PTSD induced by ACS is similar to that of PTSD that develops in response to other types of

traumatic events can help to address whether PTSD due to acute illness best fits within the traditional PTSD framework. Furthermore, delineating how dimensions of ACS-related PTSD symptoms relate to clinically-relevant factors, and demonstrating evidence of differential associations, can refine understanding and improve knowledge of mechanisms of risk of the development of and consequences from PTSD.

In this study, we conducted the first known evaluation of the dimensional structure of PTSD symptoms after suspected ACS using data from the REactions to Acute Care and Hospitalization (REACH) study, an observational cohort study of emergency department (ED) predictors of medical and psychological outcomes after evaluation for ACS. Using confirmatory factor analysis (CFA), we compared the 4-factor dysphoria, the 4-factor numbing, and the 5-factor dysphoric arousal models. In addition, we tested for measurement invariance of the best-fitting model across sex. Furthermore, we examined predictors and correlates of PTSD symptom dimensions after suspected ACS, namely perceived life threat and personal vulnerability in the ED (a risk factor for developing PTSD; Holbrook et al., 2001), and aspirin adherence one month after ACS evaluation (a functional correlate with relevance to cardiovascular health outcomes; Kronish, Edmondson et al., 2012).

2. Methods

2.1 Participants and Procedure

The REACH study is an ongoing observational cohort study of patients recruited during evaluation for ACS at the New York-Presbyterian Hospital ED. Patients were included if the treating ED physician indicated they had “probable ACS.” Patients were excluded if they had ST elevations on their electrocardiogram, as this triggers a rapid emergency protocol and transfer to the cardiac catheterization laboratory such that enrollment in the ED was not possible. Patients were also excluded from participation if they were deemed unable to comply with the protocol (e.g., due to dementia or substance abuse), were deemed in need of immediate psychiatric intervention, or were unavailable for follow-up (e.g., due to terminal non-cardiovascular illness). In the ED, participants completed measures of their ED experience, including perceived life threat and personal vulnerability in response to the acute cardiac event that brought them to the hospital. Diagnosis at discharge was determined by review of the medical record by a board-certified cardiologist. Approximately one month after ED enrollment, participants completed a follow-up phone interview that assessed PTSD symptoms that developed in response to the “heart problem, ED visit, and hospitalization” that occurred when they enrolled in the study. Adherence to aspirin medication in the past month was also assessed via self-report at this follow-up assessment.

Of those deemed eligible for the REACH study, 75% enrolled, and the participant retention rate for the one-month follow-up assessment has been 93%. The analytic sample for the current study comprised 399 individuals who completed the one-month follow-up assessment. Participant characteristics are presented in Table 1. All participants provided informed consent. The study was approved by the Institutional Review Board at the Columbia University Medical Center and conducted in accordance with the Helsinki Declaration as revised in 1989.

2.2 Measures

2.2.1 Perceived Life Threat and Personal Vulnerability—We assessed participants' perceptions of life threat and personal vulnerability in response to the acute cardiac event in the ED with 12 items (e.g., "I am in pain," "I am afraid," "I feel helpless," "I feel vulnerable," "I worry that I am not in control of my situation") based on Ozer et al. (2003). Patients rated the extent to which these statements reflected their experience in the ED on a 4-point Likert scale ranging from "Not at all" to "Extremely." Responses to these items had good internal consistency (Cronbach's α=.79). Previous research (e.g., Wiedemar et al., 2008) has utilized similar items to assess perceived vulnerability after acute cardiovascular events.

2.2.2 PTSD Symptoms in Response to Suspected ACS—PTSD symptoms were assessed at one-month follow-up with the PTSD Checklist for a Specific Stressful Experience (PCL-S; Weathers et al., 1993), a PTSD screening instrument developed by the National Center for PTSD that assesses the degree to which individuals are bothered by the 17 *DSM-IV* diagnostic criteria for PTSD (APA, 2000). Participants rated the extent to which they were bothered by PTSD symptoms in the past month in response to the heart problem, emergency room visit, and hospitalization that occurred one month ago when they enrolled in the study. Responses were rated on a scale from 1 ("Not at all") to 5 ("Extremely"). Internal consistency of the PCL-S in the current sample was excellent (Cronbach's α=.92). Prior research has demonstrated that the PCL is a reliable and valid measure of PTSD (e.g., Blanchard et al., 1996; Weathers et al., 1993). *DSM-IV* PTSD symptoms were assessed in the current study, as the study was started prior to the publication of *DSM 5*. PCL-S items were used as indicators in the confirmatory factor analyses. However, for descriptive purposes, we report the percentage of the sample with probable PTSD based on a cut-off score of 35 or greater on the PCL-S, as has been suggested for civilian medical settings (VA National Center for PTSD, 2012).

2.2.3 Aspirin Adherence—Post-discharge aspirin adherence was assessed at the one-month follow-up interview

Participants were asked if their doctor recommended that they take aspirin. If participants reported that their doctor had prescribed aspirin, they then were asked to indicate how often they took their aspirin as recommended in the past month. Responses were made on a 0–5 scale that ranged from "Less than half the time (<50%)" to "All the time (100%)." Adherence was dichotomized as being 100% adherent or not (Cutter et al., 1991; Gehi et al., 2005).

2.3 Data Analysis

Analyses proceeded in three steps. First, we compared structural models of suspected ACS-related PTSD symptoms using CFAs conducted with Mplus version 6.11 (Muthén and Muthén, 1998–2010); Table 2 shows item mappings of all of the models evaluated in this study. The PCL-S items were treated as ordinal variables, and a robust (mean- and variance-adjusted) method of weighted least squares estimation (WLSMV; Muthén and Muthén, 1998–2010) was used. The WLSMV estimator in Mplus invokes a polychoric correlation

matrix, and probit coefficients for the factor loadings are estimated (Muthén and Muthén, 1998–2010). Mplus can produce maximum likelihood estimations under missing at random conditions. Model fit was evaluated based on conventional fit statistics, including χ^2, Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and root mean square error of approximation (RMSEA). The following cutoffs were used to identify good-to-excellent model fit: CFI ≥ .95, TLI ≥ .95, and RMSEA ≤ .06 (Hu and Bentler, 1999). We also conducted χ^2 difference tests for nested models (using the DIFFTEST option in Mplus) to evaluate which model provided the best representation of the symptom structure of PTSD after suspected ACS.

Second, we examined whether the best-fitting model was invariant by sex using a comprehensive invariance testing approach (Meredith and Teresi, 2006). We tested a series of models that imposed increasingly stringent restrictions across men and women: 1) Configural invariance model (same factor structure across groups but all parameters allowed to vary); 2) Weak factorial/metric invariance model (factor loadings constrained to be equal across groups); 3) Strong factorial/scalar invariance model (observed variable thresholds constrained to be equal across groups to assess equivalence in item severity); 4) Strict factorial invariance model (residual error variances constrained to be equal across groups to assess equivalence in measurement error across groups); 5) Invariance of factor variances and covariances (factor variances and covariances constrained to be equal across groups); and 6) Invariance of factor means (factor means constrained to be equal across groups). We investigated whether each of the progressively more restricted models was associated with a statistically significant decrement in model fit using χ^2 difference tests (using the DIFFTEST option in Mplus). Two participants were missing data on sex and were excluded from tests of measurement invariance across sex, resulting in sub-samples of 212 men and 185 women.

Third, using a latent variable framework, we examined correlates of the suspected ACS-related PTSD symptom dimensions by correlating PTSD dimensions from the best-fitting model with a factor representing perceived life threat and personal vulnerability in the ED (defined by the 12 items administered in the ED) and an indicator representing 100% aspirin adherence in the month after ACS evaluation. We assessed differences in the magnitude of these correlations by computing Wald χ^2 tests of parameter constraints (Muthén and Muthén, 1998–2010). Wald tests were computed to assess whether the difference between each pair of correlations was statistically significantly different than zero.

3. Results

3.1 Descriptive Statistics

Descriptive statistics are presented in Table 1. Men (M=24.67, SD=6.69) and women (M=25.31, SD=7.13) reported similar mean levels of perceived life threat and personal vulnerability in the ED, $t(395)=0.92$, $p=.36$. Although all participants presented to the ED with acute cardiac symptoms consistent with probable ACS according to the treating ED physician's determination, only 34.1% were diagnosed with ACS at hospital discharge based on medical record review. The remaining participants were given diagnoses such as chest pain without a cardiac diagnosis, another symptom/disease process (e.g., hypertensive

chronic kidney disease, Tietze's disease), or another cardiac disease (e.g., intermediate coronary syndrome). Neither perceived life threat and personal vulnerability in the ED nor PTSD total severity scores (calculated by summing the responses to the PCL-S administered at one-month follow-up) differed by confirmed ACS status, *p*s>.32. Men (*M*=24.10, *SD*=10.05) and women (*M*=24.56, *SD*=10.71) did not differ significantly in their mean PTSD severity scores, $t(395)=0.44$, $p=.66$. The percentages of men (15.6%) and women (16.2%) with a positive screen for probable PTSD (PCL-S≥35) also did not differ significantly, $\chi^2(1)=0.03$, $p=.86$. The majority of participants reported at one-month follow-up that their doctor had recommended that they take aspirin, and most of those individuals endorsed full adherence in the past month. A higher percentage of men (90.2%) than women (78.4%) reported 100% aspirin adherence, $\chi^2(1)=7.73$, $p=.01$.

3.2 Model Fit Comparison

Fit statistics for the 4- and 5-factor models are presented in Table 3. All models fit the PCL-S data well. Chi-square difference tests indicated that the 5-factor dysphoric arousal model fit significantly better than the 4-factor numbing model [$\chi^2(4) = 12.44$, $p = .01$] but not significantly better than the 4-factor dysphoria model [$\chi^2(4) = 3.59$, $p = .46$]. Thus, based on model fit statistics and parsimony, we selected the 4-factor dysphoria model as the best-fitting model of PTSD symptoms after suspected ACS.

3.3 Measurement Invariance by Sex

Factorial invariance testing results are presented in Table 4. Fit statistics for models with constraints at the different levels tested indicated very good fit to the data. There was evidence of measurement invariance in factor loadings, item thresholds, residual error variances, factor variances and covariances, and factor means for the dysphoria model across men and women. Chi-square difference tests comparing nested models suggested that constraining these different parameters to be equal across sex was not associated with statistically significant decrements in model fit. Together, these results supported invariance in the meaning of the PTSD factors, item severity, residual error, factor score variation, correlations between the PTSD factors, and latent mean scores across men and women.

3.3 Correlates of PTSD Symptom Dimensions

Table 5 presents the intercorrelations of the factors of the dysphoria model and Table 6 presents correlations between the PTSD symptom dimensions with a factor representing perceived life threat and personal vulnerability in the ED and an indicator representing 100% aspirin adherence in the month after ACS evaluation. Consistent with the broader literature on the latent structure of PTSD (e.g., Sumner et al., 2014; Wang et al., 2011), the PTSD symptom dimensions were highly correlated with one another. Despite these high correlations, the relative distinctiveness of the PTSD symptom dimensions was suggested by differential strength of associations between the dimensions with indicators of PTSD risk and cardiovascular health. Specifically, re-experiencing and dysphoria symptoms had the largest positive correlations with perceived life threat and personal vulnerability in the ED, whereas avoidance symptoms had the smallest positive correlation with this ED predictor. Results of the Wald tests indicated that the re-experiencing-perceived threat and dysphoria-

perceived threat correlations were significantly larger than the corresponding correlations with avoidance. We investigated what might be driving the dysphoria-perceived threat correlation by examining correlations between perceived threat with separate factors defined by the five numbing items of the dysphoria dimension (e.g., loss of interest, feeling as if the future will be cut short) and by the three dysphoric arousal items of the dysphoria dimension (e.g., trouble falling or staying asleep, irritability). The numbing-perceived threat correlation (r=.535, p<.0001) was larger than the dysphoric arousal-perceived threat correlation (r=.455, p<.0001), although the difference in magnitude was not statistically significant, Wald test(1)=3.491, p=.062. Moreover, only the numbing-perceived threat correlation was significantly larger than the avoidance-perceived threat correlation [comparison for numbing: Wald test (1)=6.749, p=.009; comparison for dysphoric arousal: Wald test (1)=1.684, p=.194].

The re-experiencing-aspirin adherence correlation was the smallest in magnitude for this correlate; re-experiencing symptoms were the only dimension that was not significantly negatively correlated with 100% aspirin adherence at one-month follow-up. Results of the Wald tests suggested that the avoidance-aspirin adherence and dysphoria-aspirin adherence correlations were larger than the re-experiencing-aspirin adherence correlation, although these tests were not statistically significant (ps<.08). As above, we further probed the dysphoria-aspirin adherence association by computing separate correlations for the numbing and dysphoric arousal symptoms of the dysphoria dimension. The numbing-aspirin adherence correlation (r=−.266, p=.006) and dysphoric arousal-aspirin adherence correlation (r=−.256, p=.011) were similar in magnitude, Wald test(1)=0.013, p=.911. Neither correlation was significantly larger than the re-experiencing-aspirin adherence correlation [comparison for numbing: Wald test (1)=2.426, p=.119; comparison for dysphoric arousal: Wald test (1)=2.294, p=.130].

4. Discussion

In recent years, acute life-threatening medical events have received increasing attention as traumatic events that can induce PTSD symptoms (see Edmondson, 2014, for a review). However, the extant literature has generally treated PTSD as a dichotomous diagnosis or total symptom score, which fails to capture the heterogeneous clinical manifestation of this disorder. In this first known examination of the dimensional structure of PTSD after an acute medical event, we found that the 4-factor dysphoria model provided the best representation of PTSD symptoms after suspected ACS. There was strong evidence of measurement invariance of the dysphoria model by sex. Moreover, findings suggested that the symptom dimensions of the dysphoria model had differential strengths of associations with variables with relevance to PTSD risk—perceived life threat and personal vulnerability in the ED—as well as cardiovascular health—post-discharge aspirin adherence, which provides support for the distinctiveness of these dimensions and their external validity.

As is common in the literature on the dimensional structure of PTSD symptoms (e.g., Armour et al., 2013; Sumner et al., 2014), the dysphoria, numbing, and dysphoric arousal models all fit the data well. However, we identified the dysphoria model as the best-fitting model based on fit statistics and parsimony. The dysphoria model distinguishes between

specific (i.e., re-experiencing, active avoidance, hypervigilance, exaggerated startle) and non-specific (i.e., dysphoria) symptoms of PTSD, and it has received substantial support in the broader PTSD literature (Yufik and Simms, 2010). Whereas symptoms more closely related to fear responses (e.g., re-experiencing, exaggerated startle) are thought to represent core aspects of PTSD, non-specific symptoms of dysphoria and general distress are also characteristic of other psychological symptoms, such as depression and generalized anxiety (Simms et al., 2002; Zoellner et al., 2014).

We believe that our findings can help to inform the conceptualization of PTSD triggered by an acute life-threatening medical event. Some researchers have questioned whether PTSD that develops after a traumatic experience like ACS or cancer is qualitatively distinct from PTSD triggered by a discrete external trauma, such as combat exposure or a natural disaster (see Edmondson, 2014, for a review). Our results indicated that the *DSM-IV* PTSD symptoms that developed in response to suspected ACS exhibited a similar structure to the same symptoms induced by other traumatic experiences (e.g., military experiences; Simms et al., 2002), which suggests that both types of PTSD can be conceptualized within this similar framework. Nevertheless, it is possible that differences may emerge between PTSD triggered by an acute life-threatening medical event vs. other traumatic events with more nuanced assessments (e.g., by assessing ongoing present- and future-oriented intrusions in those with medically-induced PTSD given the enduring somatic threat that may exist for these individuals; Edmondson, 2014).

We found that the dysphoria model fit well in men and women. Moreover, results suggested that men and women did not differ significantly in PTSD factor meaning, item severity, residual error, factor score variance, factor intercorrelations, and factor means. This set of findings is in contrast to some results from the broader PTSD literature. For example, epidemiologic research has found that lifetime PTSD is twice as common in women than in men (Kessler et al., 1995), and meta-analytic evidence suggests that women report greater PTSD symptom severity than men in response to a variety of traumatic events, although acute medical events were not included in this analysis (Tolin and Foa, 2006). Furthermore, several studies in the broader PTSD literature have documented sex differences in aspects of PTSD factor structure that suggested greater severity for female than male participants (e.g., Armour, Elhai et al., 2011; Sumner et al., 2014; Wang et al., 2013). However, our current findings of measurement invariance of the dysphoria model across sex are consistent with some initial results from research on ACS-induced PTSD. In a meta-analysis of the prevalence of PTSD in ACS patients, the percentage of male participants in study samples was not significantly associated with PTSD prevalence estimates (Edmondson et al., 2012). More research on possible sex differences in the presentation of PTSD triggered by acute medical events is needed, but our findings suggest that PTSD symptoms after suspected ACS manifested similarly in men and women in our sample.

In addition to elucidating the factor structure of suspected ACS-related PTSD symptoms, we examined whether perceived life threat and personal vulnerability, a risk factor for the development of PTSD (Holbrook et al., 2001), was differentially associated with PTSD symptom dimensions. Although perceived threat/vulnerability was positively correlated with all symptom dimensions, greater perceived threat/vulnerability in the ED was a significantly

stronger predictor of re-experiencing and dysphoria symptoms than avoidance symptoms. This pattern of results is consistent with theoretical and empirical work on the progression of PTSD symptoms. Specifically, Creamer et al. (1992) proposed that processing of the threatening aspects of a traumatic event contributes to the formation of a fear memory network that leads to re-experiencing symptoms, such as intrusions, and that avoidance symptoms develop secondarily (and hence, less directly) as a strategy to cope with the re-experiencing symptoms. We also found that perceived threat/vulnerability in the ED was a significantly stronger predictor of dysphoria than avoidance symptoms, which may suggest that perceptions of threat in the early stages of an acute cardiac event may be associated with experiencing greater levels of non-specific general distress after suspected ACS. Alternatively, more general dysphoria also may have preceded the development of PTSD and have been a risk factor for PTSD onset (Edmondson et al., 2014). Furthermore, results suggested that the numbing symptoms of dysphoria (e.g., loss of interest, feeling as if the future will be cut short) were especially related to perceived threat in the ED. There was a trend for the association between perceived threat in the ED with numbing symptoms to be stronger than the corresponding correlation with dysphoric arousal symptoms, and the numbing-perceived threat correlation was significantly larger than the avoidance-perceived threat correlation. Thus, a heightened sense of personal vulnerability in the acute aftermath of suspected ACS may underlie, in part, the development of symptoms of emotional withdrawal and disengagement. Qualitative interviews with patients may help to better understand how these particular aspects of PTSD develop after suspected ACS.

We also examined associations between PTSD symptom dimensions and aspirin adherence at one-month follow-up (Kronish, Edmondson et al., 2012). Of the four dimensions of the dysphoria model, re-experiencing symptoms evidenced the smallest correlation with complete aspirin adherence in the past month, and there were trends for the negative correlations between avoidance and dysphoria symptoms with aspirin adherence to be larger than the corresponding correlation for re-experiencing symptoms. In addition, both the numbing and dysphoric arousal aspects of the dysphoria dimension were similarly related to poor aspirin adherence. Even though the differences between the re-experiencing, avoidance, and dysphoria associations with aspirin adherence were not statistically significant, they provide a preliminary indication that avoidance and dysphoria (but not re-experiencing) symptoms may be particularly important for understanding why some patients with suspected ACS-related PTSD symptoms do not follow through with the recommendations of their doctors to manage their health conditions. Ultimately, investigating how the underlying dimensions of PTSD after suspected ACS relate to functional correlates relevant to cardiovascular health may help to inform the development of more targeted and efficient intervention efforts. For example, if our initial findings receive further support in subsequent research, then it is possible that individuals with high avoidance and dysphoria symptoms might benefit most from approach-oriented interventions that incorporate behavioral activation and problem-solving techniques (e.g., Katon et al., 2010), which may reduce avoidance and withdrawal behaviors and encourage engagement with health management strategies (e.g., Katon et al., 1996; Lin et al., 2012; also see Kronish, Rieckmann et al., 2012).

3.1 Limitations

Four methodological limitations need to be acknowledged. First, although all participants were recruited during evaluation for ACS in the ED, nearly two-thirds of the sample was not diagnosed with ACS at discharge. This finding is consistent with results from other studies in which patients were recruited for suspected ACS in the ED (e.g., rates of confirmed ACS have ranged from 16–30% in recent studies; Chase et al., 2006; Kwong et al., 2003). Additionally, the non-ACS diagnoses were perceived as equally threatening and painful as confirmed ACS diagnoses. Furthermore, we assessed PTSD symptoms in response to the suspected ACS and related hospitalization that occurred when participants enrolled in the study. Thus, our work provides a broad perspective on PTSD symptoms that develop in response to suspected ACS, and it can serve as a foundation for more targeted research. Second, as in other work on PTSD factor structure, some factors were defined by only two items. Ideally, more indicators are used to define a latent construct in order to produce more reliable factors and stable parameter estimates (Marsh et al., 1998). It is also of interest to characterize these dimensions of PTSD across units of analysis that go beyond self-report, such as neural, physiological, and behavioral levels. Third, because this study was initiated before the *DSM-5* was published, our findings reflect the underlying dimensions of PTSD as defined by *DSM-IV*. Although changes were made in revising the PTSD diagnostic criteria from *DSM-IV* to *DSM-5* (e.g., the removal of the subjective fear/helplessness/horror Criterion A2; the addition of symptoms of persistent blame, persistent negative emotional state, and reckless/destructive behavior), most of the *DSM-5* and *DSM-IV* PTSD criteria are highly similar. Moreover, a 4-factor model of PTSD symptoms was included in the *DSM-5*. Nevertheless, further research is needed to investigate whether the dysphoria model provides a good fit to the *DSM-5* PTSD symptoms that develop after suspected ACS. Fourth, our data on aspirin adherence were based on self-report, and future work on this topic should include objective measures of adherence, such as electronic pill bottle monitors, as a way to overcome potential reporting biases. Moreover, data on aspirin adherence and PTSD symptoms were both assessed at one-month follow-up, so additional longitudinal research is needed to delineate temporal associations.

Despite these limitations, we believe that our study is characterized by several strengths that extend the extant literature on the dimensional structure of PTSD symptoms. Specifically, whereas most work on the dimensional structure of PTSD has been conducted in military samples (e.g., Harpaz-Rotem et al., 2014; King et al., 1998; Simms et al., 2002) or natural disaster victims (e.g., Sumner et al., 2014; Wang et al., 2011), we recruited patients from the ED during evaluation for ACS. Moreover, in this racially and ethnically diverse sample, we not only tested measurement invariance by sex but we also examined differential associations with correlates related to risk for developing PTSD and subsequent adherence to cardioprotective medication.

3.2 Conclusions

ACS-related PTSD represents a substantial proportion of the PTSD burden in developed countries, and it has been estimated that over 150,000 ACS patients in the United States will develop PTSD symptoms in a given year (Edmondson, 2014). Our results suggest that considering underlying dimensions of PTSD, rather than treating PTSD as a homogenous

diagnostic entity, may help inform assessment and intervention efforts to promote emotional and cardiovascular health after suspected ACS. Longitudinal studies that examine the course of these dimensions are needed to better characterize these symptom dimensions and how they relate to aspects of emotional and physical functioning over time. Most importantly, future research should examine whether particular dimensions of PTSD are especially predictive of ACS recurrence and all-cause mortality.

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Highlights

- PTSD symptoms induced by acute coronary syndrome (ACS) are common (12% prevalence).
- We evaluated the dimensional structure of PTSD after suspected ACS.
- A 4-factor dysphoria model with specific and non-specific PTSD symptoms fit best.
- There was evidence of measurement invariance of this model by sex.
- PTSD dimensions related differentially to perceived threat and aspirin adherence.

Table 1

Participant characteristics, *N*=399.

	% (*n*)	Mean (*SD*)	Range
Demographics			
Male	53.1% (212)		
Race			
White	32.3% (129)		
Black/African American	23.1% (92)		
Other	44.6% (178)		
Hispanic ethnicity	51.4% (205)		
High school degree or greater	64.2% (256)		
Age		61.1 years (12.6)	27–95 years
Hospital Assessment			
Perceived life threat and personal vulnerability total score in the ED		24.96 (6.89)	12–46
Confirmed ACS upon discharge	34.1% (136)		
One-month Follow-up Assessment			
PTSD total severity score[a]		24.30 (10.33)	17–76
Positive screen for probable PTSD[b]	15.8% (63)		
Aspirin recommended by doctor	71.4% (285)		
Reported 100% adherence to aspirin in the past month[c]	85.6% (244)		

Note. ED = emergency department. ACS = acute coronary syndrome.

[a]PTSD total severity score calculated by summing responses on the PTSD Checklist for a Specific Stressful Experience (PCL-S).

[b]Positive screen based on a cut-off of total PCL-S scores greater than or equal to 35.

[c]Percentage is for those participants who reported that their doctor recommended aspirin.

Table 2

Item mappings of the dysphoria, numbing, and dysphoric arousal models.

***DSM-IV* PTSD symptom**	**Item Mappings**		
	Dysphoria	**Numbing**	**Dysphoric Arousal**
B1. Intrusive thoughts of trauma	R	R	R
B2. Recurrent dreams of trauma	R	R	R
B3. Flashbacks	R	R	R
B4. Emotional reactivity to trauma cues	R	R	R
B5. Physiological reactivity to trauma cues	R	R	R
C1. Avoiding thoughts of trauma	A	A	A
C2. Avoiding reminders of trauma	A	A	A
C3. Inability to recall aspects of trauma	D	N	N
C4. Loss of interest	D	N	N
C5. Detachment	D	N	N
C6. Restricted affect	D	N	N
C7. Sense of foreshortened future	D	N	N
D1. Sleep disturbance	D	H	DA
D2. Irritability	D	H	DA
D3. Difficulty concentrating	D	H	DA
D4. Hypervigilance	H	H	AA
D5. Exaggerated startle response	H	H	AA

Note. *DSM-IV* = Diagnostic and Statistical manual of Mental Disorders, 4th edition. R = re-experiencing; A = avoidance; H = hyperarousal; D = dysphoria; N = numbing; DA = dysphoric arousal; AA = anxious arousal.

Table 3

Fit statistics for confirmatory factor analyses.

Model	χ^2	df	CFI	TLI	RMSEA [90% CI]
Dysphoria	199.632***	113	.985	.982	.044 [.034, .054]
Numbing	206.155***	113	.984	.981	.045 [.035, .055]
Dysphoric arousal	196.948***	109	.985	.981	.045 [.035, .055]

Note. df = Degrees of freedom; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = root mean square error of approximation; CI = confidence interval.

*** $p < .0001$

Table 4

Results of factorial invariance testing for the dysphoria model by sex.

Type of invariance	χ^2	df	CFI	TLI	RMSEA [90% CI]	χ^2 difference test[a]	df	p
Configural	364.903**	276	.986	.986	.040 [.028, .051]	---	---	---
Weak factorial/matric	355.216**	289	.989	.990	.034 [.020, .045]	10.332	13	.667
Strong factorial/scalar	371.943**	302	.989	.990	.034 [.020, .045]	14.679	17	.619
Strict factorial[b]	371.943**	302	.989	.990	.034 [.020, .045]	22.517	17	.166
Factor variance	362.178*	306	.991	.992	.030 [.014, .042]	3.539	4	.472
Factor covariance	340.518	312	.995	.996	.021 [.000, .035]	4.730	6	.579
Factor mean	335.966	316	.997	.997	.018 [.000, .033]	3.089	4	.543

Note. df = Degrees of freedom; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = root mean square error of approximation; CI = confidence interval.

** $p < .01$,

* $p < .05$

[a] χ^2 difference test computed using the DIFFTEST option in Mplus.

[b] Fit statistics for the strict factorial invariance model are the same as the strong factorial/scalar invariance model but the model was compared to one that allowed residual variances to be freely estimated across groups, as indicated with results of the χ^2 difference test.

Table 5

Correlations between the factors of the dysphoria model.

	1.	2.	3.	4.
1. Re-experiencing	(.821)			
2. Avoidance	.881	(.608)		
3. Dysphoria	.878	.816	(.855)	
4. Hyperarousal	.762	.774	.921	(.635)

Note. Coefficients in parentheses along the diagonal are Cronbach's alpha coefficients.

All *p*s < .0001

Table 6

Results of Wald tests of parameter constraints for correlations between the factors of the dysphoria model with perceived threat in the ED and complete aspirin adherence one month after ACS evaluation.

	Correlation A	*r*	Correlation B	*r*	Wald test	*p*
Pairwise comparisons for correlations with perceived threat in the ED (correlation A vs. B)	Re-exp with perceived threat in the ED	.519***	Avoid with perceived threat in the ED	.366***	6.265	.012
	Re-exp with perceived threat in the ED	.519***	Dys with perceived threat in the ED	.505***	0.132	.716
	Re-exp with perceived threat in the ED	.519***	Hyper with perceived threat in the ED	.471***	0.739	.390
	Avoid with perceived threat in the ED	.366***	Dys with perceived threat in the ED	.505***	4.832	.028
	Avoid with perceived threat in the ED	.366***	Hyper with perceived threat in the ED	.471***	2.054	.152
	Dys with perceived threat in the ED	.505***	Hyper with perceived threat in the ED	.471***	0.606	.436
Pairwise comparisons for correlations with aspirin adherence (correlation A vs. B)	Re-exp with aspirin adherence	−.117	Avoid with aspirin adherence	−.310**	3.474	.062
	Re-exp with aspirin adherence	−.117	Dys with aspirin adherence	−.263**	3.083	.079
	Re-exp with aspirin adherence	−.117	Hyper with aspirin adherence	−.276**	2.209	.137
	Avoid with aspirin adherence	−.310**	Dys with aspirin adherence	−.263**	0.182	.669
	Avoid with aspirin adherence	−.310**	Hyper with aspirin adherence	−.276**	0.082	.775
	Dys with aspirin adherence	−.263**	Hyper with aspirin adherence	−.276**	0.025	.874

Note. ED = emergency department. ACS = acute coronary syndrome. Re-exp = re-experiencing symptoms. Avoid = avoidance symptoms. Dys = dysphoria symptoms. Hyper = hyperarousal symptoms.

*** $p < .0001$,

** $p < .01$