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**Trauma Exposure and Transdiagnostic Distress: Examining Shared and PTSD-Specific
Associations**

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Abstract

Dimensional models of psychopathology suggest that the causes and consequences of psychopathology are attributable to a combination of syndrome specific and transdiagnostic features. There is considerable evidence that trauma exposure confers risk for a wide range of psychiatric conditions, yet no previous work has specifically examined the higher-order effects of trauma exposure within a structural model. We examined transdiagnostic and PTSD-specific associations with multiple forms of trauma exposure within a nation-wide sample (N = 1,649; 50% female) of military Veterans over-selected for posttraumatic stress disorder (PTSD). A higher-order Distress variable was estimated using PTSD, major depressive disorder (MDD), and generalized anxiety disorder (GAD) symptoms as indicators. A structural equation model spanning three measurement time points over an average of 3.85 years was then used to examine the unique roles of higher-order Distress and PTSD residual variance in accounting for the relations between trauma exposure and psychosocial impairment. Results suggest that the association between trauma exposure and PTSD symptoms is primarily mediated by higher-order Distress, but that PTSD severity does have a significant association with trauma exposure independent of Distress. Both higher-order Distress and PTSD-specific variance were necessary to account for the association between trauma exposure and future functional impairment. This work suggests there may be shared etiology linking cumulative trauma exposure and a range of internalizing symptoms. Continued application of higher-order dimensional models is needed to provide a more comprehensive understanding of the consequences of trauma exposure.

Keywords: PTSD, distress, hierarchical, Project VALOR, transdiagnostic

Trauma Exposure and Transdiagnostic Distress: Examining Shared and PTSD-Specific Associations

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5;* American Psychiatric Association, 2013) asserts that a specific subset of traumatic experiences (i.e., Criterion A) are *causally* linked to the development of a *distinct* set of reactions, namely, posttraumatic stress disorder (PTSD). Attempts to critically evaluate this core assumption of etiology, which demarcates PTSD as the signature trauma-related disorder, have resulted in significant controversy (Rosen & Lilienfeld, 2008). Trauma exposure confers risk for most forms of psychopathology and PTSD is highly comorbid with other conditions (Grant et al., 2008; McLaughlin et al., 2020; Rosen & Lilienfeld, 2008). Understanding the link between trauma exposure, PTSD, and other forms of psychiatric distress is critical to advancing the science of psychotraumatology.

Empirically-derived dimensional models of psychopathology attempt to model the natural structure of psychiatric distress based on the tendency of certain symptoms to co-occur. The higher-order structure of anxiety and depression symptoms has been recognized for decades (e.g., Krueger, 1999). Watson (2005) identified a dimension of “distress disorders” (p. 530) that primarily involve pervasive nonspecific negative emotionality, including PTSD, major depressive disorder (MDD), and generalized anxiety disorder (GAD). More recently, the Hierarchical Taxonomy of Psychopathology (HiTOP) workgroup proposed a similar higher-order structure that placed PTSD within a Distress factor including MDD and GAD symptoms, among others (Kotov et al., 2017). This nosology reflects considerable evidence of a higher-order dimension that captures shared variance across PTSD, depression, and anxiety symptoms (Forbes et al., 2011; Grant et al., 2008; Miller et al., 2008). Of note, PTSD may be best described

as existing between dimensions in that it also shares considerable variance with many other forms of psychopathology including externalizing and fear-related disorders (Forbes et al., 2021; Wolf et al., 2010). Current consensus, however, is that its strongest correlates are among the “distress disorders.”

Higher-order structural models allow for any cause or outcome of mental illness to be attributed to broad or specific psychiatric dimensions (Conway et al., 2019). From this perspective, trauma exposure may predict higher-order distress (thereby conferring risk for a range of clinical syndromes) and/or more specific lower-level psychiatric symptoms. The transdiagnostic associations of trauma exposure are in line with the former, as are observations that childhood maltreatment, and other environmental stressors (e.g., racial discrimination), are better predictors of transdiagnostic dimensions than particular diagnoses (Keyes et al., 2012; Rodriguez-Seijas et al., 2015). Importantly, there is also consistent evidence that transdiagnostic dimensions outperform diagnoses in the prediction of future health-related and functional outcomes (Eaton et al., 2013; Kim et al., 2021; South et al., 2011), as well as demonstrate stronger associations with genetic (Hawn, Zhao, et al., 2022; Waszczuk et al., 2020) and neurological (Kircanski et al., 2018; Reininghaus et al., 2019) indicators.

Thus, research suggests that trauma-related distress reflects a combination of transdiagnostic and diagnostic-specific distress that, when measured dimensionally, may provide greater predictive validity regarding clinically relevant outcomes, such as psychosocial functioning. However, the association between trauma exposure and higher-order distress has yet to be explored and the proportion of trauma-related symptoms and associated functional outcomes that can be accounted for by higher-order distress remains unknown. Improved understanding of the extent to which certain trauma-related outcomes are affected by broad

distress versus PTSD-specific content has the potential to improve the precision of theoretical models and refine treatment approaches. If the psychological and functional outcomes associated with trauma exposure were fully accounted for by transdiagnostic distress, it would suggest that transdiagnostic treatment approaches that specifically target symptomatology shared across disorders (e.g., Cognitive Behavior Therapy, Dialectical Behavioral Therapy) may be more effective at improving psychosocial functioning following trauma exposure.

Our first aim was to investigate the association between self-reported cumulative trauma exposure, transdiagnostic distress (representing the shared variance across self-reported PTSD, MDD, and GAD symptoms) and PTSD-specific variance. Our second aim was to contrast the effects of transdiagnostic distress and PTSD-specific variance on future psychosocial functioning. Given the transdiagnostic risk conferred by trauma exposure, and work highlighting the statistical advantages of dimensional phenotypes (Hawn, Wolf, et al., 2022; Wolf et al., 2018), we hypothesized that the effects of cumulative trauma on psychiatric symptoms and psychosocial functioning would be primarily mediated via transdiagnostic distress. We separately examined two measures of trauma exposure, a broad measure and a combat-specific measure, to test the boundaries of these associations and to gauge the robustness of observed effects.

Methods

Participants and Procedure

We analyzed data from the Veterans After-Discharge Longitudinal Registry (VALOR) sample for this study. The Project VALOR sample was recruited from a national registry of Veterans of the Army and Marine Corps who had been deployed in support of Operation Enduring Freedom, Operation Iraqi Freedom, or Operation New Dawn and had undergone a

mental health evaluation at a Veterans Health Administration (VHA) facility between July 2008 and December 2009 (Rosen et al., 2012). PTSD was sampled at a ratio of 3:1 (probable PTSD: not probable PTSD). Women Veterans were oversampled at a ratio of 1:1 (female: male). The initial assessment (T1; N = 1,649), which included a questionnaire and a telephone-based clinical interview, was completed between 2009 and 2012. The second (T2; N = 1,345) and third (T3; N = 1,346) waves of data collection occurred from 2013 to 2014 and 2014 to 2015, respectively. The average length of time between T1 and T2 was 1,062 days ($SD = 263$ days). The average length of time between T2 and T3 was 343 days ($SD = 51$ days). Sample characteristics at T1 were as follows: *Sex* – 50% female; *Age* $M = 37.49$ ($SD = 9.9$); *Race* – 78% White, 16% African American; *Ethnicity* – 13% Latino; *Education* – 35% had an associates' degree or higher. The Institutional Review Board at the [blind for review] and the Human Research Protection Office of the U.S. Army Medical Research and Materiel Command approved all procedures.

Measures

Trauma Exposure (Measured at T1)

Life Events Checklist (LEC). The LEC (Gray et al., 2004) is a 17-item measure of lifetime trauma exposure history. Participants indicated whether they had ever experienced, witnessed, or learned about each of 17 different potentially traumatic events (e.g., natural disaster, fire or explosion, physical assault). For the current analyses, we used the total number of events participants identified as “happened to me” as an index of lifetime trauma exposure. The number of LEC events endorsed ranged from 0 to 17, with a median of six events endorsed.

Deployment Risk and Resiliency Inventory (DRRI). The DRRI (King et al., 2006; Vogt et al., 2008) measures psychosocial risk and resilience factors for military personnel. Only the combat exposure subscale ($\alpha = .91$) of the DRRI was used in this study. The combat exposure

subscale consists of 16 items measuring distinct potentially traumatic incidents (e.g., “I went on combat patrol missions”; “I personally witnessed someone from my unit, or an ally unit being seriously wounded or killed”). Participants responded using a scale from 1 (*Never*) to 5 (*Daily or almost daily*). Items were summed to generate a total score ($M = 32.83$, $SD = 12.8$; Median = 30).

Psychiatric Distress (Measured at T2)

PTSD Checklist for DSM-5 (PCL-5). We used the PCL-5 (Weathers et al., 2013) to assess PTSD symptom severity. The PCL-5 is a 20-item self-report measure of PTSD symptom severity during the past-month. Items are rated on a scale from 0 to 4 (0 = *not at all* to 4 = *extremely*) and correspond to each *DSM-5* symptom of PTSD. The PCL-5 has demonstrated good test-retest reliability, internal consistency, and construct validity in Veteran samples (Bovin et al., 2016). Subsets of PCL-5 item responses can be combined to generate severity scores for each of the four PTSD symptom clusters: Cluster B $\alpha = .92$; Cluster C $\alpha = .92$; Cluster D $\alpha = .91$; Cluster E $\alpha = .88$.

Patient Health Questionnaire (PHQ). The PHQ (Spitzer et al., 1999) was used to measure self-reported symptoms of MDD and GAD. Depression symptoms ($\alpha = .90$) were scored on a scale of 0 (*Not at all*) to 3 (*Nearly every day*). Generalized anxiety symptoms ($\alpha = .85$) were scored on a scale of 0 (*Not at all*) to 2 (*More than half the days*). Primary analyses of these scales utilized item-level responses.

Psychosocial Functioning (Measured at T3)

Inventory of Psychosocial Functioning (IPF). The IPF (Bovin et al., 2018) was used to measure psychosocial functioning symptoms. The IPF measures self-assessed functioning over the past 30 days in seven different life domains: romantic relationships, family, work,

friendships, parenting, educational experiences, self-care. All responses were scored on a scale of 0 (*Never*) to 6 (*Always*). Scale reliabilities ranged from $\alpha = .79$ (self-care) to $\alpha = .91$ (work). All subscale scores are calculated on a scale of 0 to 100 indicative of a percentage of the maximum possible score. Higher scores are indicative of worse functioning. The total IPF impairment score ($\alpha = .96$) was calculated by averaging across the seven subscales. Subscales were excluded from the total score calculation for individuals that identified a subscale as not applicable (e.g., not currently in a romantic relationship).

Data Analysis

All analyses were completed in *R* version 4.2.1 (R Core Team, 2022). Confirmatory Factor Analyses (CFA) and structural equation modeling (SEM) were completed with the *lavaan* package version 0.6-15 (Rosseel, 2012). Latent variable composite reliability (ω) was calculated using the *semTools* package version 0.5-6. Missing data were managed with pairwise deletion as direct ML estimation was not available. We evaluated goodness of fit for all models using standardized root mean square residual (SRMR), comparative fit index (CFI), and root mean square error of approximation (RMSEA). In line with the recommendations of Hu and Bentler (1999), acceptable model fit was defined by meeting at least two of the following three criteria: $SRMR \leq .08$, $CFI \geq .95$, $RMSEA \leq .06$.

Following initial examination of bivariate associations, we estimated a CFA of the T2 PTSD, MDD, and GAD measures to confirm the previously established higher-order relationship between PTSD severity and associated mood and anxiety symptoms. The CFA was estimated using diagonally weighted least squares with robust standard errors and mean and variance adjusted test statistics (WLSMV). At the first level of the CFA, latent MDD and GAD severity scores were estimated using individual items from the PHQ, and PTSD symptom cluster scores

were estimated using the individual items on the PCL-5. The first item for each factor was used as the scaling variable. At the second level, PTSD severity was estimated using the latent PTSD symptom clusters as indicators. Cluster B was used as the scaling variable. At the third level, a transdiagnostic distress variable was estimated using latent PTSD, MDD, and GAD variables as indicators. MDD was used as the scaling variable. This third-order distress variable represents variance that is shared among the PTSD, MDD, and GAD constructs. Acceptable fit of the measurement model was confirmed and then examined within two separate SEMs spanning the three measurement time points. The first used the LEC as the index of trauma exposure. The second used the DRRI combat measure as the index of combat exposure.

The SEMs examined associations spanning two levels of the dimensional hierarchy. Cumulative trauma exposure reported at T1 was used to predict T2 Distress (level 3) and PTSD (level 2)¹, which, along with T1 trauma exposure, were each used to predict T3 psychosocial functioning (i.e., T2 latent Distress and PTSD were the mediators between T1 trauma exposure and T3 psychosocial functioning and a direct path between T1 trauma exposure and T3 psychosocial functioning was also modeled). Within the context of these path models, the factor loading of PTSD on Distress is interpreted as a regression coefficient with PTSD severity being regressed on transdiagnostic distress. Thus, the path models account for both the variance in PTSD that is shared with MDD and GAD as well as the variance that is unique to PTSD and not reflective of the broader distress dimension. Direct, indirect, and total effects on psychopathology symptoms (i.e., level 3 Distress, level 2 PTSD) and total functional impairment were then estimated. This approach allowed us to disentangle the relative magnitudes of the

¹ Direct associations with MDD and GAD latent variables were not included in these analyses as the distress latent variable is statistically dependent on the indicator variables. Including directional paths with the full measurement model (i.e., distress and all three indicators) results in collinearity.

associations between trauma exposure and higher-order distress versus PTSD-specific variance as well as to examine their unique associations with future psychosocial functioning.

Standard errors and 95% confidence intervals for all effects (i.e., direct, indirect, total) were estimated using 2,000 bootstrap draws. Statistical significance for all indirect effects were based on bias corrected 95% confidence intervals. All interpreted parameters are from the fully standardized model. All *R* syntax is provided in supplemental materials. The data are available by request to the senior author.

Results

Bivariate Correlations

We examined bivariate correlations among observed variables (see Table 1). All correlations were statistically significant. Combat and lifetime trauma exposure were associated with all measures of psychiatric distress, with LEC correlations ranging in magnitude from $r = .25$ (MDD) to $r = .32$ (PCL-5 Cluster B) and DRRI Combat correlations ranging from $r = .19$ (GAD) to $r = .30$ (PCL-5 Cluster B). In line with the hypothesized higher-order model, measures of PTSD, MDD, and GAD symptoms were highly correlated, with associations ranging from $r = .55$ (PCL-5 Cluster C and GAD) to $r = .81$ (MDD and GAD). All observed variables were positively associated with psychosocial impairment, ranging in magnitude from $r = .13$ (DRRI Combat) to $r = .62$ (PCL-5 Cluster D).

Measurement Model

The higher-order measurement model (see Figure 1) provided good fit to the data: $\chi^2(587) = 3,296.32, p = < .001$; robust CFI = .988; robust RMSEA = .062 (95% CI = .062-.064; SRMR = .053). Standardized factor loadings at the first level of the measurement model ranged from $\lambda = .80$ to $\lambda = .88$ on Cluster B, $\lambda = .92$ to $\lambda = .92$ on Cluster C, $\lambda = .56$ to $\lambda = .85$ on Cluster D, $\lambda =$

.62 to $\lambda = .82$ on Cluster E, $\lambda = .49$ to $\lambda = .82$ on MDD, and $\lambda = .58$ to $\lambda = .76$ on GAD.

Standardized factor loadings for the second-order PTSD variable ranged from $\lambda = .80$ to $\lambda = .97$.

Standardized factor loadings on the third-order Distress latent variable were $\lambda = .97$, $\lambda = .87$, and $\lambda = .94$ for MDD, PTSD, and GAD respectively. Latent variable reliabilities were acceptable across all levels of the measurement model with values ranging from $\omega = .85$ (GAD) to $\omega = .93$ (PTSD). The Distress latent variable had a reliability of $\omega = .90$.

Path Models

Fit statistics and standardized coefficients are provided in Table 2. Both path models had good fit. Scaled χ^2 difference tests suggested that direct effects did not differ across gender in either model: LEC model $\chi^2(6) = 8.01, p = .24$; $\chi^2(6) = 9.55, p = .145$. The path model depicting associations with lifetime trauma exposure as measured by the LEC is depicted in Figure 2. The path model depicting associations with cumulative combat exposure is depicted in Figure 3.

Effect of Trauma Exposure on PTSD and Higher-order Distress

There were significant ($p < .05$) direct effects from T1 trauma exposure to T2 Distress (LEC $\beta_1 = .28$; DRRI $\beta_1 = .22$). This represents the association between trauma exposure and variance shared by the PTSD, MDD, and GAD measures. There were also significant direct effects from T1 trauma exposure to T2 PTSD residual variance (LEC $\beta_2 = .10$; DRRI $\beta_2 = .11$). This represents the association between trauma exposure and variance in PTSD not accounted for by higher-order Distress.

The total effect of trauma exposure on the PTSD latent variable (LEC $\beta_{11} = .33$; DRRI $\beta_{11} = .29$) represents the bivariate association between trauma measures and reported PTSD symptoms. The indirect effect of trauma exposure on PTSD (LEC $\beta_7 = .24$; DRRI $\beta_7 = .18$) represents the relationship between trauma exposure and PTSD symptoms that is statistically

mediated by higher-order Distress. These results indicate that most of the association between trauma exposure and PTSD symptom severity is accounted for by variance that the PTSD latent variable with the MDD and GAD constructs. More specifically, 71% of the total effect of LEC on PTSD severity is mediated by Distress while 29% is independent of Distress, and 63% of the total effect of DRRI on PTSD severity is mediated by Distress while 37% is independent of Distress.

Effects on Functional Impairment

T1 trauma exposure had no direct effect on T3 psychosocial functioning (LEC $\beta_5 = -.04$; DRRI $\beta_5 = -.04$) after accounting for the intermediate effects of Distress and PTSD symptom severity. There were significant direct effects from T2 Distress to T3 IPF (LEC $\beta_3 = .31$; DRRI $\beta_3 = .31$). This represents the association between higher-order Distress and future functional impairment while controlling for the effects of PTSD residual variance and trauma exposure. There were significant direct effects from T2 residual PTSD to T3 IPF (LEC $\beta_4 = .36$; DRRI $\beta_4 = .36$). This represents the association between PTSD-specific variance (i.e., content not shared with MDD and GAD measures) and future functional impairment after controlling for the effects of higher-order Distress and trauma exposure.

Indirect pathways from trauma exposure to impaired functioning were also examined. There were significant indirect effects through Distress (LEC $\beta_9 = .09$; DRRI $\beta_9 = .07$), representing the relationship between trauma exposure and future functional impairment that is mediated by Distress, independent of residual PTSD content. Variance shared among the PTSD, MDD, and GAD measures therefore accounts for roughly 40% of the total positive association between trauma exposure and future functional impairment (42% of the effect of LEC on impairment; 39% of the effect of DRRI on impairment). There were also significant indirect

effects through residual PTSD (LEC $\beta_8 = .04$; DRRI $\beta_8 = .04$). This represents the relationship between trauma exposure and future functional impairment that is mediated through PTSD severity and is independent of Distress (i.e., holding Distress constant in the model) and amounts to roughly 20% of the total positive association between trauma exposure and future functional impairment (17% of the effect of LEC on impairment; 22% of the effect of DRRI on impairment).² The serial mediation effect was also significant such that Distress mediated the relationship between trauma exposure and impairment by way of its association with PTSD severity (LEC $\beta_{10} = .08$; DRRI $\beta_{10} = .07$).

Parsing the Direct and Indirect Effects of Trauma-Exposure

Although most of the association between trauma exposure and PTSD severity was mediated by higher-order Distress, the observed direct effect raised questions regarding whether the PTSD latent variable was unique among the indicators of Distress in its retention of a direct association with trauma exposure. Additional post-hoc exploratory analyses were conducted to more thoroughly evaluate the extent to which each construct, across all levels of the hierarchy, was related to trauma exposure and determine the portion of that relationship that was independent of higher-order factors. Total, direct, and indirect effects of trauma exposure on each of the constructs within the hierarchy were estimated. As mentioned previously, the total effect for each construct represents the bivariate correlation with trauma exposure, the indirect effect represents the portion of that relationship that is mediated by all higher-order constructs, and the direct effect represents the association that is unique to that construct (e.g., MDD, GAD,

² Note that the proportions discussed in this section are in relation to the total *positive* effect (i.e., $\beta_8 + \beta_9 + \beta_{10}$) rather than the total effect (i.e., $\beta_5 + \beta_8 + \beta_9 + \beta_{10}$) of trauma exposure on functional impairment. We chose not to use the total effect as the denominator for these proportions as the small (non-significant) negative direct association would result in the cumulative indirect effects summing to more than 100% of the total effect.

PTSD; see Conway et al., 2021 *Tutorial 2* for tutorial of this method). The resulting values are provided in Table 3.

PTSD was unique among the modeled indicators of Distress in that it was the only indicator to retain a significant positive association with trauma exposure after accounting for higher-order Distress. For both MDD and GAD measures, net suppression effects were observed when regressed on trauma exposure alongside Distress. Therefore, unlike the PTSD construct, higher-order Distress fully accounted for the positive association between trauma exposure and MDD and GAD symptoms. We applied the same methodology to explore unique associations with trauma exposure among the first-order indicators of PTSD severity. At this level, all direct effects were small in magnitude with higher-order constructs (i.e., Distress, PTSD) mediating between 80% and 100% of the total associations with trauma exposure. Trauma exposure predicted intrusion symptoms (i.e., Cluster B), even when controlling for the effects of higher-order PTSD and Distress. For all other clusters, associations with trauma exposure were fully mediated by content shared among PTSD clusters and distress disorders. Suppression effects were observed for mood alterations (i.e., Cluster D), and direct effects on avoidance (i.e., Cluster C) and hyperarousal symptoms (i.e., Cluster E) were not statistically significant.

Discussion

Trauma exposure increases risk for a wide array of psychopathological distress, including internalizing and externalizing symptoms (Grant et al., 2008; Wolf et al., 2010). Empirically derived models of psychopathology offer a parsimonious means of accounting for this observation. From this perspective, psychopathology can be understood hierarchically with broader dimensions representing variance shared by more specific constructs. Such models suggest that trauma exposure may affect different “levels” of the hierarchy to varying degrees.

That trauma increases risk for a range of clinical syndromes suggests etiological effects on higher-order constructs. At the same time, the unique classification of PTSD among trauma- and stressor-related disorders implies an association between trauma exposure and PTSD symptoms that is distinct from other mood and anxiety disorders.

Using longitudinal data from a large sample of U.S. military Veterans, we examined measures of PTSD, MDD, and GAD to parse higher-order and PTSD-specific associations with two preceding measures of trauma exposure, a broad measure capturing lifetime trauma exposure and a specific measure of potentially traumatic combat exposure. No notable differences were observed across these measures of trauma exposure, nor were any gender differences in direct effects observed. Observed associations with trauma exposure were then extended to examine prospective relations with psychosocial impairment. Post-hoc analyses were conducted to examine all direct and indirect effects from trauma exposure.

Transdiagnostic and PTSD-specific Effects of Trauma Exposure

Trauma exposure assessed at T1 was positively associated with both higher-order Distress and PTSD-specific variance at T2. Results demonstrated that a large proportion of the total effect of trauma exposure on PTSD severity was mediated by Distress, suggesting that much of the trauma-related distress that is characteristic of PTSD is shared across disorders. This pattern of results was consistent across trauma types. Of note, the PTSD latent variable was unique in that it was the only indicator of Distress that retained a significant association with trauma exposure after accounting for the effects of higher-order Distress. These results provide a parsimonious account of trauma exposure's transdiagnostic effects. Trauma exposure likely increases risk for all "distress disorders" that were examined by increasing levels of non-specific negative emotionality. At the same time, trauma exposure predicted increases in PTSD-specific

symptoms. These findings underscore the need for incorporating measures of higher-order psychopathology into trauma research and practice, while also maintaining support for the unique association between trauma exposure and PTSD symptoms. Specifically, evidence of PTSD-specific effects provides some support for the distinct classification of trauma and stressor related disorders in the *DSM-5* and the utility of retaining Criterion A as a diagnostic criterion.

Both the higher-order Distress variable and PTSD-specific variance uniquely predicted impaired psychosocial functioning. Distress alone mediated roughly 40% of the total association between trauma exposure and functional impairment. The remaining 60% of the total effect required PTSD-specific effects to be accounted for, either alone ($\approx 20\%$) or serially in combination with Distress ($\approx 40\%$). It is no surprise that individuals with a broader range of emotional dysfunction are more likely to experience impairments in daily living. These results suggest that accounting for higher-order distress may be important for understanding the etiological effects of trauma exposure on daily functioning, but PTSD-specific measures do offer incremental utility beyond distress alone. Assessing severity of both higher-order Distress and PTSD-specific symptoms has the potential to identify individuals with the greatest risk for functional impairment for intervention and resource allocation.

Recognition of the hierarchical effects of trauma exposure can inform ongoing controversies regarding PTSD and its associations with other constructs. Consistent with the suggestions of previous work (Marshall et al., 2019), most of trauma's adverse effects, both with regard to clinical symptomology and functional impairment, were mediated by non-specific negative emotionality (i.e., distress). The established multifinality of trauma exposure (i.e., trauma exposure results in many different outcomes across individuals) and comorbidities of PTSD are a natural result of such a condition. Heterogeneous responses are inevitable to the

extent that higher-order distress predicts a broad range of specific phenotypic expressions. Nevertheless, there are distinct features of the PTSD construct that are uniquely associated with trauma exposure, and importantly, that specific content provides incremental value beyond transdiagnostic distress alone in predicting functional impairment. Therefore, PTSD is a necessary but not sufficient construct with regard to trauma-related outcomes in that it captures unique characteristics related to trauma exposure that are not captured by higher-order distress, however it is not the “end all-be all” of trauma-related psychopathology and, in fact, may be best considered in conjunction with other measures that capture broad trauma-related distress to maximize both research and clinical outcomes.

Previous criticisms of the PTSD construct emphasize the need to understand what separates PTSD from other related disorders (e.g., MDD) and have highlighted failures to identify biologically-based indicators unique to PTSD (Rosen & Lilienfeld, 2008). Our findings are consistent with recent recommendations (Hawn, Wolf, et al., 2022; Waszczuk et al., 2020) which, building off alternative dimensional research frameworks such as HiTOP (Kotov et al., 2017) and the Research Domain Criteria (RDoC; Cuthbert & Insel, 2013), suggest that the use of dimensional models allowing for the parsing of disorder-specific and broader dimensional variance can optimize our ability to detect trauma-related biomarkers.

Measurement Implications

These results have important measurement implications. Much of the research examining the psychobiological sequelae of trauma exposure has focused primarily on individuals with PTSD (O'Donnell et al., 2004; Olff et al., 2005). Much of the association between trauma exposure and PTSD exists at the level of distress. Only about a third of the total effect that trauma exposure had on PTSD symptoms was independent of non-specific emotional distress.

Specific examinations of mechanisms linking trauma exposure and PTSD symptoms that fail to account for higher-order content will not be able to distinguish between effects that are specific to PTSD and those that reflect a broader association between trauma exposure and distress.

Understanding the specific mechanisms linking trauma exposure to psychiatric problems requires a broader assessment approach capable of distinguishing PTSD specific and higher-order correlates.

These findings are also informative as they relate to the distinct diagnostic definitions of PTSD found within the *DSM-5* and the 11th edition of the International Classification of Diseases (*ICD-11*, World Health Organization, 2018). There is considerable overlap between the *DSM-5*'s definition of PTSD and disorders of distress (Byllesby et al., 2017; Forbes et al., 2011; Marshall et al., 2019). Much of that overlap can likely be attributed to Cluster D symptoms (Simms et al., 2002), but features of Clusters E (e.g., sleep disruption, difficulty concentrating) are also diagnostic among distress disorders. The *ICD-11* definition attempts to limit that overlap by excluding all non-specific indicators and constraining the PTSD construct to only the most distinct trauma-related symptoms (Brewin et al., 2009). However, the non-specific higher-order content is necessary to account for the breadth of social and behavioral distress associated with trauma exposure. Our results therefore suggest that although the *ICD-11*'s approach may be effective at capturing a greater proportion of PTSD-specific associations, if used in isolation, it may underestimate the magnitude of associations with trauma-related criteria.

Limitations and Future Directions

Our work has many strengths, including a longitudinal design, which supports the validity of the mediation models by establishing the temporal direction of associations. However, our results should be considered in light of some limitations. All measures were limited to self-

report symptom inventories. Although reliance on self-report measures is common, the shared method variance may have inflated the size of the Distress variable. Future work should incorporate multimethod assessments to control for such method-related variance. Future work should also expand these analyses to examine other higher-order constructs (e.g., fear symptoms, externalizing symptoms). Trauma exposure has been associated with nearly all forms of psychiatric distress, only a small subset of which were modeled here. Although structural models frequently model PTSD among the “distress disorders,” item-level analyses have shown that its content is strongly related to that of fear disorders (e.g., specific phobias) (Forbes et al., 2021). There is also considerable evidence that trauma exposure is strongly related to externalizing symptoms (Miller, 2003; Miller et al., 2003). Additional models incorporating these higher-order constructs will further clarify the breadth of trauma-related sequelae and the distinguishing features of the PTSD construct. It should also be acknowledged that we have no means of controlling for time since trauma exposure in these analyses, and it is possible that symptom chronicity may be important for understanding associations with functional impairment. Finally, although the study was able to test temporal directions of association, we were unable to examine causal associations, given that third variables could confound the associations of interest.

Conclusions

The psychological effects of trauma exposure are far reaching. Although most individuals exposed to trauma do not experience persistent adverse psychiatric effects, those that do are highly heterogeneous in psychiatric presentation. Our findings offer evidence for the value of examining the etiological effects of trauma exposure through the lens of a higher-order structural model. Results suggest that although much of the effect of trauma exposure can be accounted for by higher-order distress, PTSD severity, as measured by the PCL-5, does retain unique

associations with trauma exposure. This pattern of effects informs the overall pattern of trauma-related comorbidity and highlights the need to understand trauma-related outcomes on both broad and specific components of psychopathology.

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (Fifth Edition). American Psychiatric Association.
- Bovin, M. J., Black, S. K., Rodriguez, P., Lunney, C. A., Kleiman, S. E., Weathers, F. W., Schnurr, P. P., Spira, J., Keane, T. M., & Marx, B. P. (2018). Development and validation of a measure of PTSD-related psychosocial functional impairment: The Inventory of Psychosocial Functioning. *Psychological Services, 15*(2), 216–229.
- Bovin, M. J., Marx, B. P., Weathers, F. W., Gallagher, M. W., Rodriguez, P., Schnurr, P. P., & Keane, T. M. (2016). Psychometric properties of the PTSD Checklist for Diagnostic and Statistical Manual of Mental Disorders–Fifth Edition (PCL-5) in veterans. *Psychological Assessment, 28*(11), 1379–1391.
- Brewin, C. R., Lanius, R. A., Novac, A., Schnyder, U., & Galea, S. (2009). Reformulating PTSD for DSM-V: Life after Criterion A. *Journal of Traumatic Stress, 22*(5), 366–373.
- Byllesby, B. M., Elhai, J. D., Tamburrino, M., Fine, T. H., Cohen, G., Sampson, L., Shirley, E., Chan, P. K., Liberzon, I., Galea, S., & Calabrese, J. R. (2017). General distress is more important than PTSD's cognition and mood alterations factor in accounting for PTSD and depression's comorbidity. *Journal of Affective Disorders, 211*, 118–123.
- Conway, C. C., Forbes, M. K., Forbush, K. T., Fried, E. I., Hallquist, M. N., Kotov, R., Mullins-Sweatt, S. N., Shackman, A. J., Skodol, A. E., South, S. C., Sunderland, M., Waszczuk, M. A., Zald, D. H., Afzali, M. H., Bornovalova, M. A., Carragher, N., Docherty, A. R., Jonas, K. G., Krueger, R. F., ... Eaton, N. R. (2019). A hierarchical taxonomy of psychopathology can transform mental health research. *Perspectives on Psychological Science, 14*(3), 419–436.

- Conway, C. C., Forbes, M. K., & South, S. C. (2021). A hierarchical taxonomy of psychopathology (HiTOP) primer for mental health researchers. *Clinical Psychological Science*. <https://doi.org/10.1177/21677026211017834>
- Cuthbert, B. N., & Insel, T. R. (2013). Toward the future of psychiatric diagnosis: The seven pillars of RDoC. *BMC Medicine*, *11*(1), 126.
- Eaton, N. R., Krueger, R. F., Keyes, K. M., Wall, M., Hasin, D. S., Markon, K. E., Skodol, A. E., & Grant, B. F. (2013). The Structure and Predictive Validity of the Internalizing Disorders. *Journal of Abnormal Psychology*, *122*(1), 86–92.
- Forbes, D., Lockwood, E., Elhai, J. D., Creamer, M., O'Donnell, M., Bryant, R., McFarlane, A., & Silove, D. (2011). An examination of the structure of posttraumatic stress disorder in relation to the anxiety and depressive disorders. *Journal of Affective Disorders*, *132*(1), 165–172.
- Forbes, M. K., Sunderland, M., Rapee, R. M., Batterham, P. J., Calexar, A. L., Carragher, N., Ruggero, C., Zimmerman, M., Baillie, A. J., Lynch, S. J., Mewton, L., Slade, T., & Krueger, R. F. (2021). A detailed hierarchical model of psychopathology: From individual symptoms up to the general factor of psychopathology. *Clinical Psychological Science*, *9*(2), 139–168.
- Grant, D. M., Beck, J. G., Marques, L., Palyo, S. A., & Clapp, J. D. (2008). The structure of distress following trauma: Posttraumatic stress disorder, major depressive disorder, and generalized anxiety disorder. *Journal of Abnormal Psychology*, *117*, 662–672.
- Gray, M. J., Litz, B. T., Hsu, J. L., & Lombardo, T. W. (2004). Psychometric properties of the life events checklist. *Assessment*, *11*(4), 330–341.

- Hawn, S. E., Wolf, E. J., Neale, Z., & Miller, M. W. (2022). Conceptualizing traumatic stress and the structure of posttraumatic psychopathology through the lenses of RDoC and HiTOP. *Clinical Psychology Review*.
- Hawn, S. E., Zhao, X., Sullivan, D. R., Logue, M., Fein-Schaffer, D., Milberg, W., McGlinchey, R., Miller, M. W., & Wolf, E. J. (2022). For whom the bell tolls: Psychopathological and neurobiological correlates of a DNA methylation index of time-to-death. *Translational Psychiatry, 12*(1).
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal, 6*(1), 1–55.
- Keyes, K. M., Eaton, N. R., Krueger, R. F., McLaughlin, K. A., Wall, M. M., Grant, B. F., & Hasin, D. S. (2012). Childhood maltreatment and the structure of common psychiatric disorders. *The British Journal of Psychiatry, 200*(2), 107–115.
- Kim, H., Turiano, N. A., Forbes, M. K., Kotov, R., Krueger, R. F., Eaton, N. R., & Workgroup, H. U. (2021). Internalizing psychopathology and all-cause mortality: A comparison of transdiagnostic vs. diagnosis-based risk prediction. *World Psychiatry, 20*(2), 276–282.
- King, L. A., King, D. W., Vogt, D. S., Knight, J., & Samper, R. E. (2006). Deployment risk and resilience inventory: A collection of measures for studying deployment-related experiences of military personnel and veterans. *Military Psychology, 18*(2), 89–120.
- Kircanski, K., White, L. K., Tseng, W.-L., Wiggins, J. L., Frank, H. R., Sequeira, S., Zhang, S., Abend, R., Towbin, K. E., Stringaris, A., Pine, D. S., Leibenluft, E., & Brotman, M. A. (2018). A latent variable approach to differentiating neural mechanisms of irritability and anxiety in youth. *JAMA Psychiatry, 75*(6), 631–639.

- Kotov, R., Waszczuk, M. A., Krueger, R. F., Forbes, M. K., Watson, D., Clark, L. A., Achenbach, T. M., Althoff, R. R., Ivanova, M. Y., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Moffitt, T. E., Eaton, N. R., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., ... Zimmerman, M. (2017). The hierarchical taxonomy of psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology, 126*(4), 454–477.
- Krueger, R. F. (1999). The Structure of Common Mental Disorders. *Archives of General Psychiatry, 56*(10), 921–926.
- Marshall, G. N., Jaycox, L. H., Engel, C. C., Richardson, A. S., Dutra, S. J., Keane, T. M., Rosen, R. C., & Marx, B. P. (2019). PTSD symptoms are differentially associated with general distress and physiological arousal: Implications for the conceptualization and measurement of PTSD. *Journal of Anxiety Disorders, 62*, 26–34.
- McLaughlin, K. A., Colich, N. L., Rodman, A. M., & Weissman, D. G. (2020). Mechanisms linking childhood trauma exposure and psychopathology: A transdiagnostic model of risk and resilience. *BMC Medicine, 18*(1), 96.
- Miller, M. W. (2003). Personality and the Etiology and Expression of PTSD: A Three-Factor Model Perspective. *Clinical Psychology: Science and Practice, 10*(4), 373–393.
- Miller, M. W., Fogler, J. M., Wolf, E. J., Kaloupek, D. G., & Keane, T. M. (2008). The Internalizing and Externalizing Structure of Psychiatric Comorbidity in Combat Veterans. *Journal of Traumatic Stress, 21*(1), 58–65.
- Miller, M. W., Greif, J. L., & Smith, A. A. (2003). Multidimensional Personality Questionnaire profiles of veterans with traumatic combat exposure: Externalizing and internalizing subtypes. *Psychological Assessment, 15*(2), 205–215.

- O'Donnell, T., Hegadoren, K. M., & Coupland, N. C. (2004). Noradrenergic mechanisms in the pathophysiology of post-traumatic stress disorder. *Neuropsychobiology*, *50*(4), 273–283.
- Olf, M., Langeland, W., & Gersons, B. P. R. (2005). The psychobiology of PTSD: Coping with trauma. *Psychoneuroendocrinology*, *30*(10), 974–982.
- R Core Team. (2022). *R: A language and environment for statistical computing* (4.2.1) [Computer software]. R Foundation for Statistical Computing. <https://www.R-project.org/>
- Reininghaus, U., Böhnke, J. R., Chavez-Baldini, U., Gibbons, R., Ivleva, E., Clementz, B. A., Pearlson, G. D., Keshavan, M. S., Sweeney, J. A., & Tamminga, C. A. (2019). Transdiagnostic dimensions of psychosis in the Bipolar-Schizophrenia Network on Intermediate Phenotypes (B-SNIP). *World Psychiatry*, *18*(1), 67–76.
- Rodriguez-Seijas, C., Stohl, M., Hasin, D. S., & Eaton, N. R. (2015). Transdiagnostic factors and mediation of the relationship between perceived racial discrimination and mental disorders. *JAMA Psychiatry*, *72*(7), 706–713.
- Rosen, G. M., & Lilienfeld, S. O. (2008). Posttraumatic stress disorder: An empirical evaluation of core assumptions. *Clinical Psychology Review*, *28*(5), 837–868.
- Rosen, R. C., Marx, B. P., Masarejian, N. N., Holowka, D. W., Gates, M. A., Sleeper, L. A., Vasterling, J. J., Kang, H. K., & Keane, T. M. (2012). Project VALOR: Design and methods of a longitudinal registry of post-traumatic stress disorder (PTSD) in combat-exposed Veterans in the Afghanistan and Iraqi military theaters of operations. *International Journal of Methods in Psychiatric Research*, *21*(1), 5–16.
- Rosseel, Y. (2012). lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, *48*(2).

- Simms, L. J., Watson, D., & Doebbellling, B. N. (2002). Confirmatory factor analyses of posttraumatic stress symptoms in deployed and nondeployed veterans of the Gulf War. *Journal of Abnormal Psychology, 111*, 637–647.
- South, S. C., Krueger, R. F., & Iacono, W. G. (2011). Understanding general and specific connections between psychopathology and marital distress: A model based approach. *Journal of Abnormal Psychology, 120*, 935–947.
- Spitzer, R. L., Kroenke, K., Williams, J. B. W., & the Patient Health Questionnaire Primary Care Study Group. (1999). Validation and utility of a self-report version of PRIME-MD: The PHQ primary care study. *JAMA, 282*(18), 1737–1744.
- Vogt, D. S., Proctor, S. P., King, D. W., King, L. A., & Vasterling, J. J. (2008). Validation of scales from the deployment risk and resilience inventory in a sample of Operation Iraqi Freedom veterans. *Assessment, 15*(4), 391–403.
- Waszczuk, M. A., Eaton, N. R., Krueger, R. F., Shackman, A. J., Waldman, I. D., Zald, D. H., Lahey, B. B., Patrick, C. J., Conway, C. C., Ormel, J., Hyman, S. E., Fried, E. I., Forbes, M. K., Docherty, A. R., Althoff, R. R., Bach, B., Chmielewski, M., DeYoung, C. G., Forbush, K. T., ... Kotov, R. (2020). Redefining phenotypes to advance psychiatric genetics: Implications from hierarchical taxonomy of psychopathology. *Journal of Abnormal Psychology, 129*, 143–161.
- Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology, 114*(4), 522–536.
- Wolf, E. J., Miller, M. W., Krueger, R. F., Lyons, M. J., Tsuang, M. T., & Koenen, K. C. (2010). Posttraumatic stress disorder and the genetic structure of comorbidity. *Journal of Abnormal Psychology, 119*(2), 320–330.

- Wolf, E. J., Miller, M. W., Sullivan, D. R., Amstadter, A. B., Mitchell, K. S., Goldberg, J., & Magruder, K. M. (2018). A classical twin study of PTSD symptoms and resilience: Evidence for a single spectrum of vulnerability to traumatic stress. *Depression and Anxiety, 35*(2), 132–139.
- World Health Organization. (2018). *International classification of diseases for mortality and morbidity statistics* (11th Revision).

Table 1*Observed Variable Bivariate Correlations*

Measure (Time)	1	2	3	4	5	6	7	8	9
1. LEC (T1)	1,646								
2. DRRI Combat (T1)	.32	1,467							
3. PCL-5 Cluster B (T2)	.32	.30	1,337						
4. PCL-5 Cluster C (T2)	.28	.23	.76	1,345					
5. PCL-5 Cluster D (T2)	.29	.22	.75	.70	1,319				
6. PCL-5 Cluster E (T2)	.30	.29	.80	.68	.80	1,316			
7. PHQ MDD (T2)	.25	.20	.67	.57	.76	.73	1,298		
8. PHQ GAD (T2)	.25	.19	.65	.55	.66	.72	.81	1,322	
9. IPF (T3)	.16	.13	.49	.45	.62	.53	.58	.52	1,345

Note. PCL-5 cluster scores represent arithmetic mean of cluster. Diagonal provides sample size for each variable. All correlations are statistically significant at $p < .01$.

Table 2*Structural Equation Model Results*

Trauma Exposure Measure		LEC	DRRI
Model Fit			
χ^2 (df)		4160.75* (655)	4140.94* (655)
CFI		.989	.989
RMSEA [95% CI]		.057 [.055, .059]	.057 [.055, .059]
SRMR		.051	.052
Direct Effects			
	#	β [95% CI]	β [95% CI]
a1	1	.28* [.22, .34]	.22* [.16, .28]
a2	2	.10* [.06, .13]	.11* [.07, .14]
b1	3	.31* [.18, .45]	.31* [.17, .45]
b2	4	.36* [.22, .49]	.36* [.22, .50]
c	5	-.04 [-.09, .00]	-.04 [-.10, .01]
d	6	.84* [.82, .87]	.85* [.82, .87]
Indirect Effects			
Trauma → Distress → PTSD	7	.24* [.19, .28]	.18* [.13, .24]
Trauma → PTSD → IPF	8	.04* [.02, .05]	.04* [.02, .06]
Trauma → Distress → IPF	9	.09* [.05, .13]	.07* [.03, .10]
Trauma → Distress → PTSD → IPF	10	.08* [.05, .12]	.07* [.04, .10]
Total Effects			
Trauma → PTSD	11	.33* [.28, .38]	.29* [.24, .35]
Trauma → IPF	12	.16* [.11, .22]	.13* [.07, .19]

Note. Direct effect labels correspond to those used in Figures 2 and 3. All reported effects and 95% confidence intervals are from the fully standardized model. Statistical significance is based on unstandardized bias corrected 95% CI.
* $p < .05$

Table 3*Direct, Indirect, and Total Effects of Trauma Exposure*

	LEC			DRRI		
	Direct (%)	Indirect (%)	Total	Direct (%)	Indirect (%)	Total
Distress	.33* (100)		.33*	.28* (100)		.28*
PTSD	.10* (29)	.24* (71)	.33*	.11* (37)	.18* (63)	.29*
MDD	-.10* (0)	.36* (100)	.26*	-.09* (0)	.30* (100)	.21*
GAD	-.06* (0)	.33* (100)	.27*	-.08* (0)	.28* (100)	.21*
Cluster B	.04* (12)	.29* (88)	.33*	.06* (20)	.24* (80)	.31*
Cluster C	.04 (12)	.26* (88)	.30*	.02 (7)	.23* (93)	.25*
Cluster D	-.05* (0)	.34* (100)	.29*	-.12* (0)	.33* (100)	.21*
Cluster E	-.02 (0)	.33* (100)	.31*	.03 (10)	.27* (90)	.30*

Note. All reported effects and 95% confidence intervals are from the fully standardized model. Statistical significance is based on unstandardized bias corrected 95% CI. Where negative direct effects were observed, indirect effect was identified as accounting for 100% of the total positive effect.

* $p < .05$

Figure 1*Third-Order Measurement Model*

[Figure 1 here.]

Note. All depicted loadings are from the fully standardized model. Dotted lines identify the scaling variable (i.e., loading was constrained to 1.00 in unstandardized model). Omega value identifies the latent variable's composite reliability.

Figure 2

SEM Depicting Relations Between LEC, Distress, PTSD, and Psychosocial Functioning

[Figure 2 here.]

Note. The measurement model depicted at Time 2 is the same model represented by Figure 1, its depiction was simplified here for the sake of clarity. All depicted effects are from the fully standardized model.

* $p < .05$

Figure 3

SEM Depicting Relations Between DRRI, Distress, PTSD, and Psychosocial Functioning

[Figure 3 here.]

Note. The measurement model depicted at Time 2 is the same model represented by Figure 1, its depiction was simplified here for the sake of clarity. All depicted effects are from the fully standardized model.

* $p < .05$