

# Review

## STATISTICAL SUPPORT FOR SUBTYPES IN POSTTRAUMATIC STRESS DISORDER: THE HOW AND WHY OF SUBTYPE ANALYSIS

Constance J. Dalenberg, Ph.D.,<sup>1\*</sup> Dale Glaser, Ph.D.,<sup>1</sup> and Omar M. Alhassoon, Ph.D.<sup>1,2</sup>

*A number of researchers have argued for the existence of different subtypes of posttraumatic stress disorder (PTSD). In the current paper we present criteria by which to assess these putative subtypes, clarify potential pitfalls of the statistical methods employed to determine them, and propose alternative methods for such determinations. Specifically, three PTSD subtypes are examined: (1) complex PTSD, (2) externalizing/internalizing PTSD, and (3) dissociative/nondissociative PTSD. In addition, three criteria are proposed for subtype evaluation, these are the need for (1) reliability and clarity of definition, (2) distinctions between subtypes either structurally or by mechanism, and (3) clinical meaningfulness. Common statistical evidence for subtyping, such as statistical mean difference and cluster analysis, are presented and evaluated. Finally, more robust statistical methods are suggested for future research on PTSD subtyping. Depression and Anxiety 29:671–678, 2012. © 2012 Wiley Periodicals, Inc.*

**Key words:** *Dissociation; taxometrics; subtyping; diagnostic classification; latent class analysis*

### INTRODUCTION

A keyword search of the PsycINFO database for the words posttraumatic stress disorder or PTSD yields 49 citations in the 1980s, 5,146 cited publications in the 1990s, and 13,000 citations in the first decade of the 21st century. Empirically supported psychotherapies for the disorder have proliferated, with prolonged exposure<sup>[1]</sup> cognitive processing therapy<sup>[2]</sup> and eye movement reprocessing therapy<sup>[3]</sup> among the more prominent examples. With the massive rise in number of publications has come a recognition of the complexity of the dis-

order, with some researchers arguing for the value of identifying specific subtypes within PTSD. The distinctions that have garnered the most research support have been the complex PTSD subtype,<sup>[4,5]</sup> the externalizing–internalizing subtypes,<sup>[6,7]</sup> and the dissociative PTSD subtype.<sup>[8]</sup> The present paper will briefly present an argument for the minimum criteria necessary for justifying a subtype, will attempt to clarify and critique statistical analyses commonly applied in subtype analysis in the subtype examples above, and will present a proposal for the types statistical analyses that would be helpful in making a classification decision about the utility of a given PTSD subtype proposal, using dissociative PTSD as an example.

### SUBTYPES OF PTSD

**Complex PTSD.** Complex PTSD is perhaps the best known of the proposals for distinct syndromes within PTSD. Herman<sup>[4]</sup> originally proposed the hallmarks of complex PTSD by synthesizing the extant clinical literature. In the original proposal, Herman<sup>[9]</sup> proposed inclusion of the disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) under the rubric of “disorders of stress not otherwise specified.” The eventual compromise was the appearance of the list of complex symptoms under “associated

<sup>1</sup>Clinical Psychology Ph.D. Program, California School of Professional Psychology, San Diego, California

<sup>2</sup>Department of Psychiatry, University of California, San Diego, California

\*Correspondence to: Constance Dalenberg, California School of Professional Psychology, 10455 Pomerado Road, San Diego, CA 92121. E-mail: Cdalenberg2@alliant.edu

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features” of PTSD, noting the connection of these symptoms to repeated interpersonal trauma. These symptoms include dissociation, somatic complaints, hostility and social withdrawal, impaired affect modulation, feeling constantly threatened or permanently damaged, impaired relationships, self destructive and impulsive behavior, loss of previously sustained beliefs or change in previous personality characteristics, and feelings of ineffectiveness, shame, despair, or hopelessness. The most commonly used instrument, the Structured Interview for Disorders of Extreme Stress (SIDES,<sup>[10]</sup>) yields a total score for complex PTSD symptoms and subscores for Affective Dysregulation, Somatization, Alternations in Attention or Consciousness, Self-Perception, Relationships with Others, and Systems of Meaning.

**Externalizing/Internalizing PTSD.** In proposals for personality-based subtype of PTSD, several groups of researchers have defined three personality profiles within groups of individuals with PTSD symptoms.<sup>[6-7, 11]</sup> The “simple PTSD” cluster presented with low pathology and little psychiatric disturbance. The “internalizing” subtype was characterized by high negative emotionality, low positive emotionality, and mood symptoms such as depression, anxiety, social avoidance, and withdrawal. In contrast, the “externalizing” subtype showed high negative emotionality, low constraint, and problems in the areas of anger, antisocial behavior, substance misuse, and aggression.

**Dissociative/Nondissociative PTSD.** The relationship of dissociation to posttraumatic responses in general and PTSD in particular has deep historical routes. Descriptions of traumatic dissociation appeared in early work by Janet in the 1990s<sup>[12]</sup> and the Trauma Model of dissociation has dominated research on the dissociation construct.<sup>[13]</sup> Flashbacks, the hallmark symptom of PTSD, have often been referred to as “dissociative flashbacks” in the literature [e.g.,<sup>14, 15</sup>] and are cited as “dissociative reactions” in the proposed fifth edition of the DSM.<sup>[16]</sup> The controversy here thus is not about the existence of a relationship between dissociation and PTSD, which is largely acknowledged, but rather the exact role of the dissociative symptom. Carlson et al.<sup>[17]</sup> present statistical evidence against the hypotheses that dissociation is simply comorbid with PTSD, and clarify the remaining competing hypotheses of dissociation as a component of PTSD (with similar weight as other symptom clusters) versus a subtype of PTSD (more characteristic of a subgroup of survivors). The most central dissociative symptoms proposed for the dissociative subtype are dissociative flashbacks, depersonalization, and derealization.<sup>[18, 19]</sup>

#### MAKING THE CASE FOR A DISORDER SUBTYPE

We would argue here that a case for the viability of a subtype should rest on the following classes of supportive evidence. All three categories of evidence should be present to support the case for a subtype.

1. **Definitional requirement:** The criteria for the subtype or cutoff for the subtype on a given dimension should be clear and reliably measurable.
2. The subtypes should show either differing structure of PTSD *or* differing functional mechanism, or both.
  - **Structure requirement:** The two (or more) subtypes should differ in the basic structure of the disorder itself. For example, the dissociative subtype of PTSD should differ from the nondissociative subtype of PTSD on internal structure of the PTSD symptoms (i.e., base rate of symptoms and/or interrelationship among symptoms).
  - **Mechanism requirement:** The two (or more) subtypes should have different underlying biological mechanisms of action and/or should be differentiable on biologically based measures (e.g., physiological, neurochemical, or neuroanatomical).
3. **Meaningfulness requirement:** The distinction between subgroups should be clinically meaningful. This might be shown by (a) differing course of the disorder, (b) differing risk factors, (c) differing effective treatments, and/or (d) differing comorbidities.

#### JUSTIFICATION OF SUBTYPE REQUIREMENTS

PTSD is unique among Axis I disorders in its requirement of a precipitating traumatic event. A wide variety of disorders comorbid with PTSD may then ensue due to special circumstances in the traumatic event itself (e.g., depression after traumatic loss;<sup>[20]</sup> differing efforts to cope with the traumatic event (e.g., substance use;<sup>[21]</sup>), and differing vulnerabilities to the occurrence of traumatic events that are disorder-specific (e.g., traumatic reactions to hallucinations in psychosis;<sup>[22]</sup>). PTSD also may be more difficult to treat if it occurs in the context of other disorders, such as intellectual disability,<sup>[23]</sup> personality disorder,<sup>[24]</sup> or dissociative identity disorder.<sup>[25]</sup> Argument for subtyping based on other context disorders or personality differences might be made based on differing course of the disorder<sup>[26]</sup> or different patterns of comorbidities.<sup>[7, 27]</sup> Flood et al.,<sup>[28]</sup> for instance, implicitly using the meaningfulness criterion above, make the reasonable argument that differences between externalizing, internalizing, and simple PTSD groups on behavioral cause and all-cause (suicide, homicide) mortality justifies more routine subtyping based on this classification scheme. Others argue that subtype status is justified from the specific results of factor or cluster analyses,<sup>[29, 30]</sup> or that underlying dimensions that define the constructs are taxonic rather than continuous.<sup>[31]</sup>

Resting the subtype conclusion *solely* on the fact that subgroups show differential treatment difficulties or treatment consequences (e.g., defining the subgroups by personality differences or comorbid disorder context) creates both statistical and practical challenges. First, the process would create an infinite number of subtypes of PTSD related differentially to an infinite number of comorbidities. Subtyping on the Big 5

personality dimensions,<sup>[32,33]</sup> for instance (high versus low, as in the internalizing–externalizing research), would create a possible 32 personality “subtypes” that could be applied to depression, anxiety disorder, or, in the current situation, PTSD. Therefore, such context studies, while greatly valuable to clinicians for their own sake, do not alone make a viable argument for subtype. Instead, they would serve to make the argument that individuals of many personality types and with many differing vulnerabilities may develop PTSD, and these context variables may affect prognosis and treatment. Second, it is unclear whether the use of the term “subtype” is useful if the two clusters of symptoms defining each subtype commonly occur together, as is the case with internalizing and externalizing symptoms.<sup>[27,34]</sup>

Central to the above argument is the tenet that subtypes of PTSD should have reference to a difference in the *central symptoms of PTSD* between or among types, rather than to clusters of comorbid symptoms that may accompany, aggravate, or ameliorate the disorder. The dissociative subtype, for instance, given the higher likelihood of comorbid alexithymia,<sup>[35]</sup> might include individuals who claim an absence of feeling during the event itself, thereby failing the current A2 criterion of fear, helplessness, or horror during trauma. The presence of the subtype might also change the relative base rate of the avoidance, hyperarousal, and intrusion symptoms, or change the relationship of the clusters themselves. These factors more clearly identify the group as a subtype of PTSD, in that PTSD itself differs across subtypes. The use of a subtype rubric might then also potentially improve diagnostic accuracy in some cases, for example, if a given subtype might be otherwise underdiagnosed due to lower base rate or lower report of a specific PTSD symptom.

Recent biological evidence has argued that dissociative and nondissociative PTSD might reflect different underlying processes. Lanius et al.,<sup>[8]</sup> for instance, present a persuasive argument for emotional overmodulation in dissociative PTSD, potentially mediated by prefrontal inhibition of limbic regions, with nondissociative PTSD representing emotional undermodulation of these same regions. Data presented by Felmingham et al.<sup>[36]</sup> also fits the corticolimbic theory. Using fMRI (functional magnetic resonance imaging), these researchers showed differences in prefrontal activation during fear processing, concluding that dissociation may be a regulatory process to cope with extreme arousal via inhibition of the limbic regions. These findings would satisfy the mechanism criterion above. Differential mechanism is accepted as an alternative to differential structure because a nonequivalent neurobiology of action might suggest eventual development of differing effective physiological or pharmacological treatments, even if these differing treatments are not yet available.

A potential modification of the mechanism criterion would be to allow the presence of differential antecedents to justify the two subtypes. Complex PTSD, for instance, is theoretically more likely to be related to interpersonal

trauma than is simple PTSD.<sup>[37]</sup> In the absence of a different pattern of symptoms or a different neurobiological substratum, however, it is not clear that a case has been made for a subtype of PTSD rather than a context in which a set of comorbid symptoms may also occur with PTSD.

The meaningfulness criterion is often required for justification of new diagnoses in DSM discussions. In general, the Scientific Review Group criteria for evidence of predictive validity of a subtype, which is one of the elements for justification of the need for diagnostic changes, includes evidence for diagnostic stability, course of illness, and response to treatment. Severity may be a fourth element here, with one subtype being a more severe form of the illness, but severity is generally labeled as a diagnosis modifier rather than as a subtype.

### COMMON STATISTICAL EVIDENCE FOR SUBTYPES

The mean statistical difference argument for subtypes is perhaps the most common justification given for the distinction between two purported subgroups within a diagnosis. Here the two groups are first distinguished (by an internalizing/externalizing scale, a dissociation scale, a complex PTSD scale, a personality disorder test, etc.); subsequently, it is shown that the two groups differ on other variables. If we were to create an antisocial personality subtype of PTSD, for instance, per Southwick, Yehuda and Giller’s suggestions,<sup>[24]</sup> then we would no doubt find that this subtype was distinctive on various criminal history and empathy variables. Similarly, the dissociative subtype is likely to have more somatic symptoms than the nondissociative group, given the relationship between dissociation and somatization.<sup>[38]</sup> Such analyses do not provide much distinctive information about dissociative PTSD, however, unless it is also shown that “dissociative PTSD” has some distinction from “PTSD plus comorbid dissociative symptoms.” Only under such circumstances is the meaningfulness requirement met. Thus, for example, interactional information (cf.<sup>[39]</sup>) might show that dissociation in the context of PTSD has a different pattern of comorbidity with other variables than dissociation or PTSD alone. The possibility of increased severity as an alternative hypothesis for the patterns found should also be addressed.<sup>[19]</sup> In the interactional model of testing, the main effect for severity can be differentiated from the interaction of severity with the alleged specifier for the subtype. In various regression model statistics, severity can be compared to the subtype specifier in terms of relative predictive power.

A second common method to establish the existence of subtypes is cluster analysis which has been used with groups of symptoms to define externalizing and internalizing subtypes.<sup>[34]</sup> The non-statistical reader should be aware, however, that cluster analysis always finds clusters. Successful groupings could be and are found if cluster analysis is done within PTSD on any other

matrix of symptoms with sufficient variability, such as multifactorial personality inventories<sup>[27]</sup> or personality disorder scales.<sup>[24]</sup> Another drawback of this method is that cluster analysis lacks well-established stopping rules or significance testing procedures.<sup>[40]</sup> In addition, the case order of the data has significant impact on cluster solutions. Blashfield's classic article on cluster definitions methods<sup>[41]</sup> stated, "users of cluster analysis are cautioned that there exist a wide variety of cluster analysis methods, that different methods can yield very different solutions, and that users should be careful to skeptically test the classifications generated by cluster analysis methods" (p. 377). Therefore, cluster analyses should be repeated on varying sets of data, and/or clusters should be defined using several of the dozens of available clustering methods.<sup>[42]</sup> In addition, caution should be exercised when determining the number of cluster using internal cross validation methods such as data splitting.<sup>[43]</sup> Although no uniform method of replicating number of clusters has been consensually adopted, comparable data using differing algorithms is useful. Further, follow-up analyses should be conducted to show distinctions between clusters on variables not used to determine the original clusters. Thus, it is important to validate the clusters with external criteria. Typically cluster analysis is used to establish the structure requirement by examining the interrelationship between symptoms, with follow-up comparisons of the clusters on other variables to satisfy the meaningfulness criterion.

Another set of tools used to establish the Structure requirement are the taxonic methods (e.g., Meehl's MAXCOV-HITMAX procedure;<sup>[44]</sup>) which are used for the more restricted goal of testing the hypothesis of an underlying dichotomy rather than a continuum for the concept in question. The basic conceptual argument here is that taxonic structure implies separate classes of participants. Unlike cluster analysis, taxonic methods are used with single measures of various constructs. Using large clinical and nonclinical samples, taxonic results have been found for dissociation within PTSD samples<sup>[34]</sup> and more general population samples.<sup>[45]</sup> Meehl's methods, however, require a rather subjective analysis of whether the resulting graph of the covariance function is sufficiently peaked (given the claim that taxonic variables will yield a peaked curve and dimensional variables will yield a flat curve). Unfortunately, peaks in covariance curves appear to be dependent on item/symptom frequency (how frequently the indicator symptom occurs) even in the absence of any latent taxon.<sup>[46]</sup> Thus, for example, adding a group of Dissociative Identity Disorder (DID) patients to a normal college sample might artificially create a taxonic "look" to the data by noninclusion of the moderately symptomatic group that might be contained in a large clinical sample. Miller<sup>[46]</sup> suggests that evidence for taxonicity should come not from the peakedness of the covariance curve, but from the reliability of the estimates of base rates of the subtypes across samples and measures. Great care

should also be taken in sample and indicator selection in taxon analyses. As Lenzenweger<sup>[47]</sup> argued, "Simply put, a taxometric investigation that is based on a sample that contained 100 persons diagnosed with XYZ disorder and 500 normal university students is not likely to tell us much about the latent structure of XYZ disorder" (p. 19).

Finite mixture modeling (latent class analysis and latent profile analysis [LPA]) is the more recent alternative to cluster and taxonic methods, and has enjoyed a recent surge in use in the diagnostic literature.<sup>[7,9,48]</sup> Unlike cluster analytical methods, factor mixture modeling (FMM) and latent class analysis use iterative procedures such as estimation maximization to produce maximum likelihood estimates of parameters.<sup>[49]</sup> However, latent class models have to be identifiable in order for these estimation-maximization procedures to work. Several methods are available to assess the identifiability of a model.<sup>[49]</sup> In addition to assessing model identifiability, a number of methods also exist to test model fit; something that cluster analysis lacks.<sup>[50]</sup> Thus, researchers are able to statistically examine whether, for example, a two-class model is a better fit for the data compared to a three-class model. Although articles that use these methods tend to be of high quality, there are limitations to this approach that are not addressed by all researchers. First, fit of a latent class model to a given set of data does not mean that a variable is not dimensional.<sup>[51,52]</sup> A dimensional variable which has been sampled at the extremes (as when large college samples are augmented by hospitalized or extremely symptomatic samples) can easily produce a fit to a latent class model. Second, latent class analysis requires that the observed variables are uncorrelated within the classes. This assumption is often not addressed, and would be presumptively violated when multiple symptoms known to commonly covary are used in the analyses (cf.<sup>[53]</sup>). This is a challenging assumption to deal with given the real world of highly comorbid symptomatology, but can be creatively approached by combining like items into a smaller number of independent predictors. Given that the presence of residual intercorrelations within classes may indicate further subclasses, or may indicate poor choice in manifest variables, these intercorrelations should be tested in latent class analyses (LCA).

LCA or LPA have the added advantage of comparing models via an array of information criterion statistics such as the Akaike's Information Criteria (AIC), Bayesian Information Criteria (BIC), the Likelihood Ratio Chi-square, and other fit statistics.<sup>[54-56]</sup> Moreover, software such as Mplus<sup>[57]</sup> include the Lo-Mendell-Rubin likelihood ratio test of model fit as well as a bootstrapping option to compare the  $k$ -th class model to the  $k - 1$  mixture model.<sup>[58,59]</sup> Although there is a rich history of methods for determining the obtained number of clusters (cf.<sup>[60,61]</sup>), there is neither history of using the Akaike's or Bayesian Information Criteria for ascertaining model fit nor comparing more or less constrained models in cluster analysis, which might contribute to

inconsistencies in findings when the cluster technique is used.

Like taxonic and cluster methods, a disadvantage of LCA is that base rates of the latent classes (sometimes produced by differing exclusion criteria) will strongly affect results. Also, like taxonic or cluster methods, the resulting LCA classes should be validated with some of the outcome variables. Such validation is most compelling if it shows not only difference (outcome 1 is associated with Class A and not Class B), but also reverse association<sup>[62]</sup> (association between outcome variables changes markedly or reverses direction across classes). Thus, the type of analysis and validation would potentially speak to both the structure and mechanism requirement, as well as, to the meaningfulness requirement.

Finally, it is also certainly the case that using a variable dimensionally versus dichotomously might yield differing or even opposite results. For example, Sorensen et al.<sup>[63]</sup> evaluated the relative merits of a dimensional versus subtype approach to paranoia. He found that when paranoia was measured dimensionally, those with more paranoid symptomatology were lower on other measures of current functioning, a finding inconsistent with other literature. However, when the same group was reclassified into subtypes, with the paranoid subtype requiring the *predominance* of paranoid symptoms, the paranoid subtype had *higher* current functioning than other groups, a finding consistent with other literature. A given variable also may be distributed continuously rather than dichotomously (as a taxon), and yet may change in its relationship to a variety of negative outcome variables at a particularly cutpoint. Thus, the normal distribution of a variable, as opposed to the often-skewed distribution characteristic of a taxonic variable measured continuously, does not negate the possibility that the measure can be used to define a subtype.

#### FURTHER STATISTICAL ANALYSIS OF SUBTYPE QUESTIONS

**Definitional Requirement.** Interestingly, it is most appropriate statistically to establish or reaffirm the cutoff for a subtype as the last step, rather than simply the first step, of the development of criteria for a subtype. Once it is shown that group with likely differing base rates of the subtypes do differ on the structure of PTSD, and that these broadly defined subtypes do have clinical meaning, the cutpoints in which these changes occur can then be established. The use of taxonic analysis or receiver operating characteristic (ROC) curves is valuable, but in a slightly differing manner than has been typically presented. Ginzburg et al.,<sup>[64]</sup> for instance, use ROC with dissociation as the predictor and PTSD as the criteria to establish the subtype cutoff, but this would give us the level of dissociation that predicts membership in the PTSD group, not the level of dissociation within the PTSD group that creates the set of risks specific to dissociative PTSD that we are concerned about

(which arguably could be higher or lower than the cutoff predictive of PTSD presence).

Researchers studying the “factors” of dissociation commonly find strong intercorrelations among the factors, with cross-loadings for the commonly cited dissociative symptoms—depersonalization/derealization, amnesia, identity fragmentation, and absorption [cf.<sup>[13]</sup>]. Numbing also is often considered a dissociative symptom<sup>[65]</sup> (perhaps as a variant of derealization), but is not well represented on the most commonly used dissociation measure, the Dissociative Experiences Scale. Foa et al.<sup>[66]</sup> suggested that the numbing symptoms are of relatively greater importance than other PTSD symptoms in distinguishing those with and without PTSD.

Given the strong intercorrelations cited above between types of dissociation, it may prove unimportant that those studying dissociative PTSD have thus far used somewhat distinct definitions. Some use global dissociation measures such as the Dissociative Experiences Scale<sup>[31]</sup> or the Multiscale Dissociation Inventory.<sup>[61]</sup> Others use a more defined subset of symptoms, such as elevated flashbacks, depersonalization, and derealization;<sup>[18]</sup> depersonalization, derealization, and numbing;<sup>[8]</sup> or elevated depersonalization and derealization only.<sup>[19]</sup> Each of these correlated symptoms sets might be useful in establishing the existence of a set of classes. However, it is clear that consensus of the criteria for the presence of dissociative PTSD is necessary for certainty as to the comparability of study results.

**Structure Requirement.** In addition to use of LCA mentioned above, FMM has recently been applied to PTSD.<sup>[67]</sup> FMM combines factor analytic and latent class techniques to model population heterogeneity within a factor analytic model. In other words, FMM searches for classes in which the basic factor structure fits differently (e.g., high avoidance and high intrusion do or do not correlate strongly). Classes identified by FMM may be qualitatively different (i.e., taxonic) or may be defined by a quantitative difference (e.g., when a given level of severity is reached).

Once tentative indicators allowing subtype identification are found, multigroup structural equation modeling (SEM) also would be valuable. Multigroup SEM is frequently used to test invariance of models, that is, the assumption that some model of PTSD symptoms will be similar across races, genders, or measures. Here, the researcher would be looking for non-invariance (or variance), not invariance, in the models (although the tests used would be the same). The variance could be in the measurement model (which indicator variables fell on which factors with what loadings within classes) or in the structural model (how are the latent variables related to each other within classes). Partial invariance testing, when some indicators pass the invariance test and some do not, also is easily tested.<sup>[68]</sup>

**Meaningfulness Requirement.** The underlying argument for subtype rests on the assumption or belief that the identification of the subtypes will in some way aid the diagnostic or treatment process. It has been argued, for

instance, that dissociative individuals might have lower degree of treatment success after PTSD treatment or less pronounced negative slope in symptoms or time.<sup>[13]</sup> High comorbidity of dissociation with other disorders has been shown, including in studies of the dissociative subtype.<sup>[31]</sup> The former should be shown in interactional designs, while the latter could be tested through comparison of binomial probabilities (for nominal indicators), independent tests of correlations (for continuous indicators), or through structural invariance tests within an SEM design.

Finally, we would argue that the meaningfulness test should also include a test of the alternative hypothesis that the observed categories differ in PTSD severity only, a hypothesis tested in more recent dissociative subtype analyses.<sup>[18,19]</sup> Severity may be extremely important within a given diagnosis, and it is possible that severity could function in a manner consistent with a subtype (i.e., if at a given level of severity relationship between indicators change and comorbidities becomes more common). However, the issue to be tested in the dissociative case, for instance, is whether dissociation is nothing more than a stand-in for severity of diagnosis. In that case, there would be nothing unique about the dissociative symptom as defining the subtype. Analyses that cross severity of PTSD with severity of the subtype indicators (e.g., dissociation) could compare simple effects for dissociation within more severe PTSD to simple effects for dissociation within less severe PTSD, with a prediction of an interaction.<sup>[39]</sup> Subtype hypotheses would predict unique elevations in symptoms for the dissociative PTSD cell, producing incremental significance for the interaction over and above variance for the main effect of level of severity of PTSD and the main effect of level of dissociation. Such interactions could be studied both dichotomously, based on cutoffs, or continuously, using centered interaction variables within a regression.<sup>[69]</sup>

## CONCLUSION

The increasing complexity of our understanding PTSD and the variety of symptom presentation has yielded evidence consistent with numerous proposals for subtypes of the disorder. However, it is in the interest of diagnostic parsimony and precision to constrain our tendencies to subdivide the PTSD diagnosis into subtypes that do not serve the purpose of more accurate identification and more successful prediction of prognosis. By restricting the term subtype to those subgroups which have shown clear definitions, mechanism or structural variance, and evidence of meaningfulness (including differentiation from the alternative hypothesis of classes based on severity alone), we believe that focus can be centered on those distinctions between classes that have greatest clinical significance.

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