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The Latent Structure of Posttraumatic Stress Disorder: A Taxometric Investigation of Reactions to Extreme Stress

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Mental health professionals have debated whether posttraumatic stress disorder (PTSD) can be qualitatively distinguished from normal reactions to traumatic events. This debate has been fueled by indications that many trauma-exposed individuals evidence partial presentations of PTSD that are associated with significant impairment and help-seeking behavior. The authors examined the latent structure of PTSD in a large sample of male combat veterans. Three taxometric procedures—MAMBAC, MAXEIG, and L-Mode—were performed with 3 indicator sets drawn from a clinical interview and a self-report measure of PTSD. Results across procedures, consistency tests, and analysis of simulated comparison data all converged on a dimensional solution, suggesting that PTSD reflects the upper end of a stress–response continuum rather than a discrete clinical syndrome.

The addition of posttraumatic stress disorder (PTSD) to the psychiatric nosology in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–III*; 1980) reflected a recognition by the mental health community that individuals who are exposed to situations of extreme stress may react to these situations in maladaptive ways. Current formulations regard posttrauma reactions as maladaptive when they are marked by persistent reexperiencing of the traumatic event, avoidance of places or people associated with the event, emotional numbing, and hyperarousal (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed.; *DSM–IV;* American Psychiatric Association, 1994). What these formulations fail to address, however, is whether the symptoms of PTSD demarcate a homogeneous clinical entity that is distinguishable from normal reactions to extreme stress (Brett, 1996; O'Donohue & Elliott, 1992; Robins, 1990). Some have argued that the constellation of PTSD symptoms represents the extreme end of a normal distribution of stress reactions, a distribution that may only be divided into normal and pathological states by a somewhat arbitrary cut point (e.g., Davis, 1999; Robins, 1990). Others have instead asserted that maladaptive stress reactions can and should be distinguished from normal stress reactions and their associated sequelae (e.g., Belenky, 1987). This debate has been complicated by moral and political disagreement over the possible stigmatizing (Davis, 1999) versus legitimizing (Scott, 1990) effects of a diagnostic category that distinguishes between normal and pathological reactions to trauma (Marshall & Pierce, 2000).

A major problem contributing to the *threshold dilemma*, as it has been termed by Horowitz, Weiss, and Marmar (1987), lies in attempts to determine what constitutes a normal versus an abnormal reaction to the experience of a traumatic event (O'Donohue & Elliott, 1992; Robins, 1990; Schwartz, Eilenberg, & Thompson-Fullilove, 1995). Although PTSD is a psychological disorder and is thus presumed to reflect an abnormal or pathological response to extreme stress, research has revealed that it is *normal* to experience at least some symptoms of PTSD after traumatic exposure (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992; Steinglass & Gerrity, 1990). Studies have found very few trauma survivors to be fully asymptomatic even 1 year after their traumatic event, whether that event was rape (Veronen & Kilpatrick, 1983), torture (Ramsay, Gorst-Unsworth, & Turner, 1993), or a natural disaster (Steinglass & Gerrity, 1990). Although the PTSD symptom levels reported by trauma survivors are substantially elevated relative to those who have not been exposed to trauma, the number and intensity of the symptoms often remain below the diagnostic threshold for the disorder (Steinglass & Gerrity, 1990; Veronen & Kilpatrick, 1983). At the same time, these subsyndromal or partial presentations of PTSD are often associated with clinically significant psychosocial and occupational impairment (Kulka et al., 1990;

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Stein, Walker, Hazen, & Forde, 1997; Weiss et al., 1992) and with help-seeking behavior comparable to that of individuals who meet the full criteria for PTSD (Stein et al., 1997). These findings have raised questions about the taxonomic status and clinical importance of partial PTSD (Davidson & Foa, 1991; Rothbaum & Foa, 1993; Stein et al., 1997) and have led some researchers (e.g., Ramsay et al., 1993; Stein et al., 1997) to suggest that the boundary between normal stress reactions and the more extreme state of PTSD may not be as trenchant as previously thought.

Other research findings further challenge the status of PTSD as an entity that is distinct from normal stress reactions. For example, indications that the initial symptom picture of trauma survivors who develop chronic PTSD overlaps substantially with that of survivors who spontaneously recover (Rothbaum et al., 1992) seem inconsistent with the notion of a categorically discrete syndrome that can be qualitatively distinguished from normal stress states. Similar conclusions may be drawn from findings that certain symptoms of PTSD may persist even when other symptoms of the syndrome are reduced or eliminated (Neal, Hill, Hughes, Middleton, & Busuttil, 1995; Rothbaum et al., 1992). Such findings, suggest Stein et al. (1997), "should challenge our notions about where (and indeed, *whether*) dividing lines should be drawn" (p. 1119).

Our understanding of the boundary separating PTSD from normal stress reactions has fundamental implications for the ways in which we conceptualize, assess, and investigate the psychological consequences of trauma. Knowledge of the latent structure of PTSD should promote more accurate theoretical formulations about the nature and origins of this disorder (Flett, Vrendenburg, & Krames, 1997; Gangestad & Snyder, 1985; Haslam, 1997), maximize its predictive power by selecting the measurement approach that best matches its latent structure (Meehl, 1992; J. Ruscio & Ruscio, 2002), and identify research avenues and methodological designs that may be most appropriate for its future study (A. M. Ruscio, Borkovec, & Ruscio, 2001). Moreover, knowledge of latent structure may have important implications for the determination of treatment needs by clinicians, health insurers, and health policymakers, who, in turn, determine who may gain access to care (Stein et al., 1997).

The present study examined the latent structure of PTSD using the taxometric method, a family of statistical procedures designed to distinguish categorical from dimensional structure at the latent level. Multiple taxometric procedures were performed in a large sample of male combat veterans to determine whether PTSD denotes a cohesive syndrome that can be qualitatively differentiated from normal reactions to traumatic experiences.

Method

Data Source

Participants were 1,230 male combat veterans who received a psychological evaluation at the Behavioral Sciences Division of the Veterans Affairs Boston Healthcare System's National Center for PTSD during the period from 1985 to 2000. Demographic data for the sample are presented in Table 1. Participants were predominantly Caucasian and ranged in age from 22 to 85 years (*Mdn* = 48). About half of the participants were married or living with a romantic partner at the time of their evaluation. Although a majority of participants had received at least some college education, only one third were employed full-time; another third were unemployed, and many additional participants were retired or disabled. Over half of the participants had served in the Army, and another quarter had served in the Marines; the vast majority of the sample had served in the Vietnam theater. The severity of combat-related trauma to which participants were exposed, as measured by the Combat Exposure Scale (Keane et al., 1989), was comparable (*M* = 25.03, *SD* = 10.21) to the degree of combat exposure reported by services-seeking Vietnam-era veterans in previous research reports (*M* = 25.57, *SD* = 10.12; Keane et al., 1989). Participants reported a wide range of PTSD symptom severity at the time of assessment.

Table 1
Demographic Characteristics of the Sample

Variable	*n*	%
Education		
< high school degree	124	13
At least high school degree	200	20
At least some college	495	50
At least a bachelor's degree	165	17
Current employment status		
Full-time	336	34
Part-time	77	8
Retired or disabled	181	19
Unemployed	358	37
Other	24	3
Marital status		
Single or never married	155	16
Married, remarried, or living with partner	476	48
Separated or divorced	348	35
Widowed	11	1
Race		
White	828	84
Black	117	12
Hispanic	19	2
Other	25	3
Military branch		
Army	486	58
Air Force	57	7
Navy	92	11
Marines	230	28
Other	10	1
Service era		
Desert Storm	38	4
Vietnam	794	81
Korea	66	7
World War II	66	7
Other	22	2
Age		
Range	22–85	
M	49.97	
SD	9.81	
Combat Exposure Scale		
Range	0–41	
M	25.03	
SD	10.21	
Mississippi Scale for Combat-Related PTSD		
Range	44–170	
M	120.94	
SD	20.91	
Clinician-Administered PTSD Scale		
Range	0–129	
M	71.86	
SD	23.76	

Note. Demographic data were sporadically missing; all available data are reported. Column heads apply only to categorical variables in the table. PTSD = posttraumatic stress disorder.

To estimate the likely base rate of a putative PTSD category in the present sample, we examined the prevalence of PTSD diagnoses. Among the subset of participants ($n = 841$) who were administered the Clinician-Administered PTSD Scale (CAPS), 68% ($n = 575$) received a *DSM-IV* diagnosis of current PTSD by the symptom-calibrated scoring rule of the CAPS (Weathers, Keane, & Davidson, 2001; Weathers, Ruscio, & Keane, 1999). Comparison of scores on the Mississippi Scale for Combat-Related PTSD (Mississippi Scale) received by participants who were administered the CAPS ($M = 120.04$, $SD = 20.66$) and those who were not ($M = 122.73$, $SD = 21.32$) revealed a small difference in the severity of PTSD symptomatology reported by the two groups, $t(1061) = -1.98$, $p = .048$, $d = 0.13$. This suggested that the base rate of PTSD in the full sample was perhaps slightly higher than the rate of 68% estimated by the CAPS. This prevalence estimate was quite suitable for taxometric analyses, which are most powerful when the base rate of a construct is moderate (close to .50) rather than extreme (almost 0 or 1) in the sample under investigation.

Measures

Participants were administered two widely used measures of PTSD as part of a standard assessment battery. Ninety-three percent of the sample ($n = 1,142$) completed the Mississippi Scale (Keane, Caddell, & Taylor, 1988), a 35-item self-report inventory assessing the diagnostic criteria and associated symptoms of combat-related PTSD. Using a 5-point Likert scale, respondents are asked to indicate the extent to which each item is reflective of their experiences or symptoms. The Mississippi Scale is psychometrically strong and is one of the most commonly used measures of PTSD in veteran samples (Kulka et al., 1991; McFall, Smith, MacKay, & Tarver, 1990; Newman, Kaloupek, & Keane, 1996).

Sixty-eight percent of participants ($n = 841$) were interviewed using the CAPS (Blake et al., 1995; Weathers, Blake, et al., 2001), a semistructured clinical interview that assesses the *DSM–IV* criteria for PTSD (American Psychiatric Association, 1994). The interviewing clinician asks respondents to rate the frequency and intensity of each PTSD symptom on separate 5-point scales. These ratings can be combined to create both continuous PTSD severity scores and dichotomous diagnostic decisions. The CAPS has been shown to have excellent reliability and validity and has rapidly become one of the most popular diagnostic measures of PTSD in the field (Keane, Weathers, & Foa, 2000; Weathers, Keane, et al., 2001). Among participants in the present sample who completed the Mississippi Scale, 66% ($n = 753$) also completed the CAPS. These overlapping cases were included in analyses of both scales to maximize the sample size for all procedures.

Procedure

Meehl and his colleagues (e.g., Meehl, 1995; Waller & Meehl, 1998) have devised a methodological approach for exploring the latent structure of psychological constructs. This approach, called the taxometric method, consists of analytic procedures that search for relationships between variables that are uniquely indicative of latent classes, traditionally referred to as the *taxon* and *complement.* If these procedures were to detect a qualitative boundary between PTSD and milder stress reactions, the latent structure of PTSD would be referred to as *taxonic.* By contrast, if the method uncovered only quantitative differences between PTSD and milder reactions, with no evidence of underlying groups, the latent structure of PTSD would instead be regarded as *dimensional.*

Rather than using traditional null hypothesis significance tests, the taxometric method relies on the convergence of evidence from multiple, quasi-independent analytic procedures to corroborate one latent structure and refute another. Each procedure serves as a consistency test for the results provided by the others, with confidence in a structural solution increasing as each additional test is passed. To this end, the present study used three mathematically distinct taxometric procedures to evaluate the latent structure of PTSD: MAMBAC (mean above minus below a cut; Meehl & Yonce, 1994), MAXEIG (maximum eigenvalue; Waller & Meehl, 1998), and L-Mode (latent mode factor analysis; Waller & Meehl, 1998). MAMBAC has been implemented in several taxometric investigations (e.g., Haslam, 1997; J. Ruscio & Ruscio, 2000; Strong, Greene, Hoppe, Johnston, & Olesen, 1999; Waller, Putnam, & Carlson, 1996), whereas MAXEIG and L-Mode are newer procedures and have thus appeared in fewer studies. We briefly describe the rationale of these procedures within the context of the present PTSD investigation.

MAMBAC creates a series of cuts along one PTSD indicator and examines differences in scores on a second PTSD indicator for cases falling above and below each cut. If the latent structure of PTSD is taxonic, a plot of these difference scores will be peaked, suggesting the presence of an optimal cutting score for distinguishing PTSD and non-PTSD cases. If, however, there are no underlying groups, the plot will take on a dish-shaped curve that is characteristic of dimensional latent structure.

MAXEIG is a multivariate generalization of the widely used MAXCOV (maximum covariance; Meehl & Yonce, 1996) procedure. Like MAXCOV, MAXEIG examines the degree of covariation between PTSD indicators (the outputs) along successive regions of another PTSD indicator (the input). If PTSD is taxonic, the covariation between indicators will reach a maximum within the region containing an equal mixture of PTSD and non-PTSD cases. If PTSD is instead dimensional, the covariation of indicators will be relatively constant across all regions of the input indicator. Whereas MAXCOV calculates the *covariance* between a *pair of indicators* within successive regions of the input, MAXEIG calculates the *eigenvalue* (the multivariate analogue of covariance) of the first principal factor of the *matrix of all remaining indicators* within successive regions of the input.

Finally, L-Mode works by factor analyzing all available PTSD indicators and examining the distribution of scores on the first principal factor obtained. If PTSD is taxonic, factor scores will be bimodally distributed; if PTSD is dimensional, factor scores will be unimodally distributed.

In the present study, MAMBAC, MAXEIG, and L-Mode were performed using all possible combinations of indicators drawn from the Mississippi Scale and the CAPS.[1] Each analysis provided a curve from which latent structure could be determined, as well as one or more estimates of the base rate of PTSD in the sample. These base-rate estimates served as a valuable consistency test. If mathematically diverse analyses—conducted with different sets of indicators—converged on a single PTSD base-rate estimate, this would suggest that there was, in fact, a latent PTSD taxon whose members could be reliably detected. By contrast, considerable variation among the estimates would suggest the absence of a coherent latent class. Thus, the degree of consistency among results yielded by different procedures, indicator sets, and analyses was used to gauge the reliability and likely validity of the structural solution.

Results

Selection and Construction of Indicators

Rather than using raw Mississippi Scale or CAPS items in taxometric analysis, we combined these items to create several

[1] For a given set of k indicators, different taxometric procedures permit different numbers of analyses. MAMBAC can be performed using all pairwise combinations of indicators bidirectionally in the roles of input and output, for a total of $k(k - 1)$ analyses. Alternately, if indicators do not vary sufficiently to serve as input indicators, each indicator can serve once as an output, with the sum of remaining indicators used as the input, yielding k analyses. MAXEIG can be run using each indicator once as the input, with all other indicators serving as output, for k analyses. L-Mode can be run once, using all available indicators. MAMBAC and MAXEIG yield one base-rate estimate per analysis, whereas L-Mode yields two estimates per analysis.

indicator sets. One reason for combining items was that both MAMBAC and MAXEIG require an input indicator (the *x*-axis variable above which curves are plotted) whose measurement scale contains a sufficient number of points to yield a stable and reliable taxometric curve. The larger the measurement scale of the input indicator, the more reliable the resulting curves will be and the greater the likelihood of validly interpreting these curves. A second reason for combining items was to create multiple indicator sets whose results could serve as consistency checks for one another. For example, indicators drawn from the two PTSD measures allowed the comparison of structural results yielded by self-report versus clinical interview data. Moreover, indicators created with differential emphasis on empirical versus theoretical construction approaches provided further opportunity to examine the convergence of taxometric results. For these reasons, three sets of indicators were created for the present study.

Mississippi paired indicators. The first set of indicators was constructed by summing select Mississippi Scale items in pairs. Whereas the 5-point response scale of an individual Mississippi item was too small to serve as an adequate input indicator, the sum of two such items (forming a 9-point scale) was sufficient for taxometric analysis. Thus, a set of items was chosen from the Mississippi Scale on the basis of two criteria: (a) high correlations with the total scale score, so as to identify items with the highest likely validity; and (b) substantially higher correlations between paired items than between unpaired items, so as to pair like items together while minimizing redundancy between indicators. These considerations led to the selection of 12 Mississippi items that were summed in pairs, yielding 6 indicators ranging in value from 2 to 10.[2] Complete data on these indicators were available for 1,111 cases.

To check that within-group (or *nuisance*) covariance was sufficiently low to permit valid interpretation of the taxometric curves, correlations among the six Mississippi paired-item indicators were calculated within groups of individuals likely to represent relatively pure taxon and complement groups: the upper and lower quartiles along the distribution of Mississippi Scale total scores (Golden & Meehl, 1979; Meehl & Golden, 1982). Nuisance correlations averaged just .09 within the groups, well within the tolerance limits of taxometric procedures (Meehl & Yonce, 1994, 1996). In the total sample (where strong, positive correlations are desirable), the six items correlated with one another at an average level of .38. These within-group and total-sample correlation values were substituted into a formula provided by Meehl and Yonce (1996, p. 1146) to obtain a rough estimate of the validity of the selected items—that is, their ability to separate the putative PTSD and non-PTSD groups. The average degree of separation achieved by the six pairs was 1.48 within-group standard deviation units (1.48 σ). Given a moderate taxon base rate, a sample size of 1,111 cases, and a separation of this magnitude, taxometric analyses typically have yielded clear and consistent results (see Meehl, 1995). Thus, the six pairs served as the first set of indicators for analysis.

Mississippi composite indicators. A second set of indicators was created on the basis of findings of prior factor analysis of the Mississippi Scale. In their confirmatory factor analysis of the Mississippi Scale using two large veteran subsamples, L. A. King and King (1994) found strong support for a hypothesized second-order model of PTSD, which consisted of a general PTSD factor subsuming four first-order factors. We therefore constructed four Mississippi composite indicators, each corresponding to one of these first-order factors: Reexperiencing and Situational Avoidance (11 items; $\alpha = .84$), Withdrawal and Numbing (11 items; $\alpha = .80$), Arousal and Lack of Behavioral or Emotional Control (8 items; $\alpha = .76$), and Self-Persecution (5 items; $\alpha = .71$). By including all of the Mississippi Scale items in combinations recommended by prior research with independent samples, the composite indicators enhanced coverage of the PTSD construct beyond that provided by the paired indicators and provided an added means of assessing convergence of, and thus confidence in, the structural findings. Complete data on the four composite indicators were available for 1,063 cases.

Following the same procedure described above, we estimated nuisance correlations for the composite indicators to average .14, whereas the average manifest correlation in the total sample was computed to be .61. These values yielded an average estimated separation of 2.35 σ. Given the strength of these parameter estimates, the four composites served as a second set of indicators in taxometric analysis.

CAPS composite indicators. The first two indicator sets were drawn from a self-report measure of PTSD whose items include both core symptoms of the disorder and features commonly associated with the disorder in combat veterans. To ensure that the full symptom domain of PTSD was adequately represented and that the structural solution was consistent across multiple methods of assessment, a third indicator set was derived from diagnostic interview data provided by the CAPS. Although *DSM–IV* organizes the symptoms of PTSD into three clusters, factor analysis of the CAPS (D. W. King, Leskin, King, & Weathers, 1998) has revealed that scores on this scale are better represented by four distinct factors, with the behavioral avoidance and emotional numbing symptoms of Criterion C loading on separate factors. Thus, this indicator set was created by summing the frequency and intensity ratings for each CAPS item, then combining these items into four composite variables: Reexperiencing (5 items; $\alpha = .77$), Avoidance (2 items; $\alpha = .59$), Emotional Numbing (4 items; $\alpha = .73$), and Hyperarousal (5 items; $\alpha = .69$). Complete data on these four composite indicators were available for 762 cases.

Using the procedure outlined above, nuisance correlations for the CAPS composite indicators were estimated to average .10; the average manifest correlation in the total sample was computed to be .53. These values yielded an average estimated separation of 2.04 σ. Given their good validity and minimal nuisance covariance, these four composites served as the third and final set of taxometric indicators.[3]

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[2] Indicators contained the following item pairs: 4, 33; 9, 16; 10, 15; 13, 18; 17, 22; 27, 31.

[3] There are certain conditions under which it may be useful to include a mixture of self-report and interview items within the same indicator set—for example, in cases where a single measure would not adequately capture the full construct or where the use of multiple assessment methods would reduce high levels of estimated nuisance covariance to acceptable levels. Because each of the three indicator sets previously described provided good coverage of the PTSD construct and evidenced low rates of nuisance covariance, we did not mix items from different measures in the present study.

Simulated taxonic and dimensional data. Because it is impossible to anticipate how taxonic versus dimensional results will appear for all taxometric procedures under all conceivable parameter configurations, simulating comparison data that match the parameters of the sample under consideration can greatly facilitate the interpretation of taxometric results (A. M. Ruscio & Ruscio, in press). For this reason, as well as to provide readers with a point of comparison for less familiar taxometric procedures, we simulated taxonic and dimensional data sets with parameters comparable to our empirical data.[4] Both simulated data sets were of the approximate size ($n = 1{,}000$) of the Mississippi and CAPS samples and contained four continuous indicators whose average intercorrelation ($r = .52$) was comparable to that of all three empirical indicator sets. In the simulated taxonic data set, item covariances were achieved through taxonic mixture using a base rate of 68% (our best estimate of the diagnostic rate of PTSD) plus within-group correlations of .10 (to approximate the nuisance covariance in our samples). In the simulated dimensional data set, item covariances were achieved through loadings on a common factor. These two data sets were submitted to each taxometric procedure, and their results are presented alongside those of our empirical data.

MAMBAC Analyses

Each set of indicators was first analyzed using the MAMBAC procedure. MAMBAC was performed using all possible combinations of the four Mississippi composites and the four CAPS composites, with each indicator serving as the input in turn. Because the measurement scale of the six Mississippi item pairs was considerably smaller than those of the composite indicators, we analyzed the Mississippi item pairs by removing one pair at a time to serve as the output and using a combination of the remaining pairs as the input. All three indicator sets yielded dish-shaped MAMBAC curves similar to those obtained through analysis of the simulated dimensional data (see Figure 1).[5] In contrast, the simulated taxonic data yielded peaked MAMBAC curves.

For all three sets of empirical indicators, estimates of the PTSD base rate in the sample tended toward .50, a value typically obtained with dish-shaped curves. The same value was generated by the simulated dimensional data (see Table 2). Estimates for the simulated taxonic data, on the other hand, closely approximated the simulated PTSD base rate of .68. Taken together, these results provided clear support for the dimensionality of PTSD.

MAXEIG Analyses

Next, MAXEIG was performed with each of the indicator sets. Every indicator served as an input variable in turn, with all remaining indicators in the set serving as the output. Although eigenvalues were generally greater toward the left end of the MAXEIG plots for all three indicator sets (see Figure 2)—perhaps due to the negative skew of many indicators—the panel of curves did not reveal the marked, consistent peaks indicative of taxonic structure. The slight elevations that did emerge were incompatible with a taxon base rate of .68, which would be expected to produce MAXEIG curves peaked just to the left of center. In contrast, simulated taxonic data generated curves with sharp peaks in the expected region. Moreover, the *inchworm consistency test* (Waller & Meehl, 1998), in which the number of sliding windows along the input is gradually increased, further supported the dimensionality of PTSD. If an ambiguous peak at the end of a MAXEIG curve had resulted from taxonic latent structure, it would typically become more sharply defined as increasingly narrow windows honed in on the small latent class at the extreme end of the score distribution. By contrast, a peak resulting from dimensional latent structure would tend to disappear as the number of windows is increased. All apparent left-end peaks in the MAXEIG curves for the three indicator sets disappeared with increasing numbers of windows, as would be expected if PTSD were dimensional.

Comparison of taxon base-rate estimates revealed tremendous variability in the estimates derived from MAXEIG analyses within and across the indicator sets, as well as inconsistency with estimates obtained through MAMBAC analysis (see Table 2). These base-rate discrepancies were similar to those uncovered for the simulated dimensional data but differed from estimates generated by the simulated taxonic data, which were in reasonable agreement with the simulated taxon base rate. In sum, taxometric curves and base-rate estimates derived from the MAXEIG procedure unanimously pointed to a dimensional solution.

L-Mode Analyses

Each indicator set was submitted, in turn, to L-Mode analysis. All three sets of empirical indicators produced clearly unimodal L-Mode curves, as did the simulated dimensional data (see Figure 3). By contrast, the simulated taxonic data yielded a bimodal L-Mode curve.

Base-rate estimates can be derived in two ways from L-Mode plots (Waller & Meehl, 1998). First, the location of each mode may be used to generate an estimate, and these two values are averaged to provide a more stable value. Second, the proportion of the sample that is classified as taxonic by the L-Mode procedure can serve as another estimate of taxon size. Both approaches, when applied to the L-Mode curves generated by the empirical indicators and the simulated dimensional data, yielded taxon base-rate estimates of about .50, a value typical of unimodal distributions. Although these values were consistent with one another and with base-rate estimates derived from MAMBAC, they were inconsistent with estimates derived from MAXEIG and with the .68 rate of PTSD diagnosis in the sample. In contrast, the bimodal L-Mode curve generated by the simulated taxonic data set yielded base-rate estimates that closely matched the simulated rate of PTSD in the sample. Thus, the unimodal L-Mode curves yielded by the PTSD

[4] Because sampling error has a negligible influence on multivariate taxometric procedures with samples this large, we simulated only one data set for each latent structure. These two data sets were comparable to our empirical data with one main exception: Whereas the empirical indicators were somewhat negatively skewed and distributed across a limited number of values, simulated indicators were normally distributed across a fully continuous range of values. The simulated data sets were therefore not expected to produce results identical to those of our empirical data but were instead viewed as a useful point of comparison to help interpret the taxometric curves and base-rate estimates yielded by the empirical data.

[5] Interpretations were based on the full panels of curves, which are available on request.

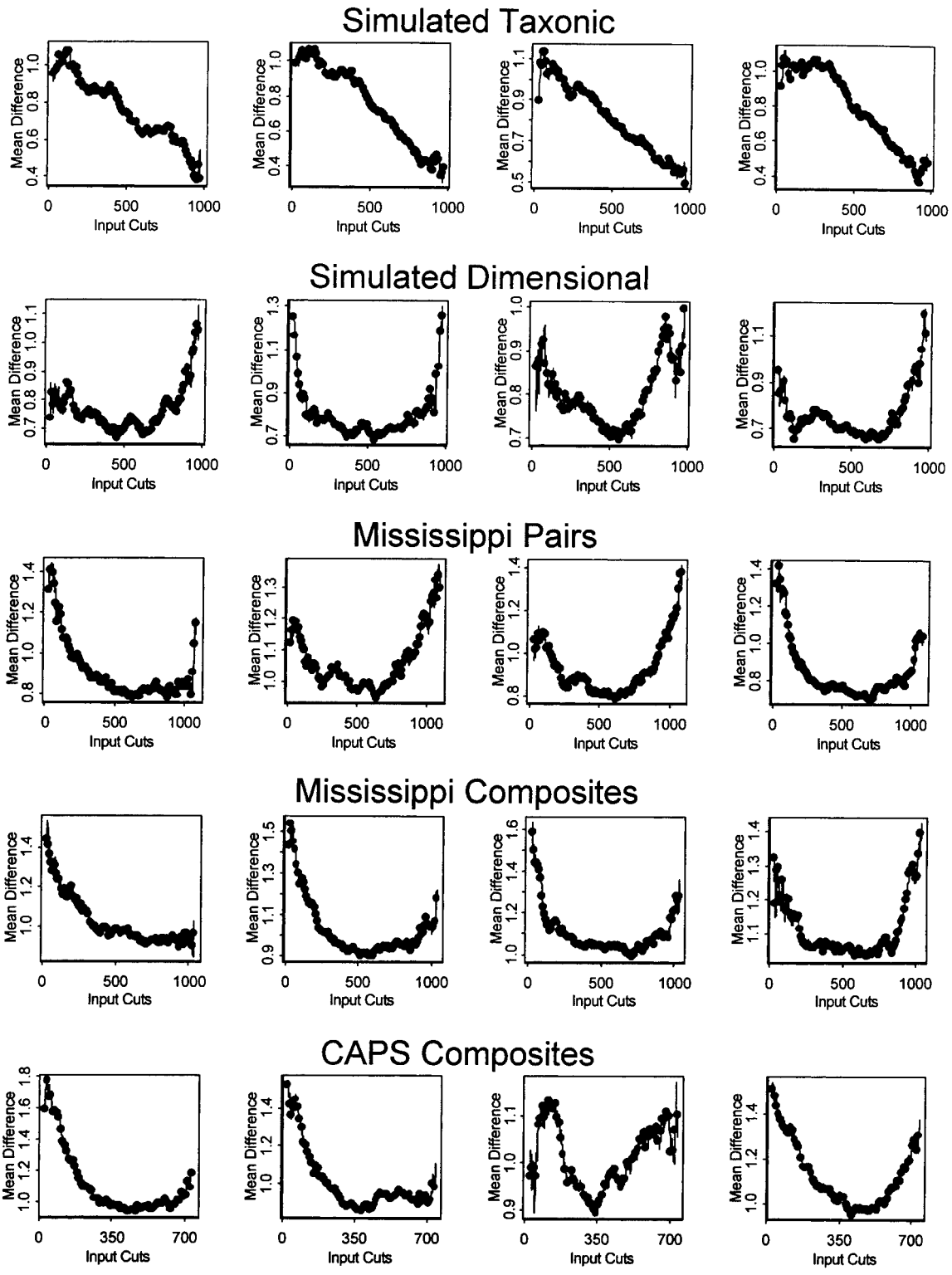

Figure 1. Mean above minus below a cut (MAMBAC) curves for the simulated taxonic and dimensional data sets plus each of the three empirical indicator sets (Mississippi Scale for Combat-Related PTSD pairs, Mississippi composites, and Clinician-Administered PTSD Scale [CAPS] composites, respectively). For each curve, cases were sorted by their scores on the input indicator, then cuts were made between each case in the data set beginning and ending 25 cases from the extremes. The *x*-axis reflects these input cuts, whereas the *y*-axis represents the difference between the mean scores of individuals falling above and below each cut. To conserve space, only the first four MAMBAC curves in each series are presented here.

Table 2
Estimates of the Base Rate of PTSD for Each Indicator Set and Taxometric Procedure

Indicator set/Data	Taxometric procedure		
	MAMBAC	MAXEIG	L-Mode
Mississippi pairs			
No. of estimates	6[a]	6[b]	2
Range	.43–.57	.09–.99	.49, .51
M	.49	.69	.50
SD	.06	.44	
Mississippi composites			
No. of estimates	12	4	2
Range	.47–.65	.05–.99	.47, .49
M	.55	.75	.48
SD	.06	.47	
CAPS composites			
No. of estimates	12	4	2
Range	.48–.62	.87–.94	.47, .51
M	.55	.91	.49
SD	.04	.03	
Simulated taxonic data			
No. of estimates	12	4	2
Range	.62–.91	.70–.82	.67, .67
M	.70	.76	.67
SD	.09	.06	
Simulated dimensional data			
No. of estimates	12	4	2
Range	.41–.59	.14–.58	.50, .50
M	.50	.44	.50
SD	.05	.20	

Note. No standard deviations are listed for L-Mode because only two values contributed to the means; they are listed in the Range rows under L-Mode. PTSD = posttraumatic stress disorder; MAMBAC = mean above minus below a cut; MAXEIG = maximum eigenvalue; L-Mode = latent mode factor analysis; Mississippi = Mississippi Scale for Combat-Related PTSD; CAPS = Clinician-Administered PTSD Scale.
[a] MAMBAC was conducted once using each indicator in turn as the output, with the sum of all remaining indicators as the input. [b] MAXEIG was conducted using each indicator in turn as the input; hence, there were more curves generated for this indicator set than the others.

data, paired with additional evidence of base-rate discrepancies, provided further support for a dimensional solution.

Discussion

Questions have been raised about the nature of PTSD since the addition of this disabling psychological condition to the diagnostic nomenclature. The present study addressed one such question: whether PTSD is a dimensional condition varying only quantitatively from milder reactions to traumatic experiences, or whether it is a discrete syndrome qualitatively different from normative stress reactions. We sought to answer this question by evaluating the latent structure of PTSD in a large sample of veterans exposed to the extreme stress of combat. Three mathematically distinct taxometric procedures, each performed multiple times using three different sets of valid PTSD indicators, yielded curves that were consistent with a dimensional latent structure. Estimates of the base rate of PTSD provided by each analysis also failed to cohere around a consistent value, further corroborating the absence of a latent PTSD taxon. These results were highly similar to those of data simulated to match the parameters of the veteran sample through an underlying dimension. In contrast, when analyses were performed using data simulated to match our sample parameters through the mixture of two latent taxa, results were clearly indicative of latent groups. These findings suggest that our empirical data were sufficiently valid to provide a meaningful test of latent structure, yielding compelling evidence for a dimensional latent structure of PTSD.

The present findings raise implications for the theoretical conceptualization and explication of posttrauma reactions. First, theorists have suggested that whereas taxonic latent structure is suggestive of all-or-none hereditary or environmental causes, dimensional structure is more consistent with graded and additive etiological models (see Haslam, 1997). The etiological implications of the dimensional solution uncovered here are therefore consistent with many contemporary theories of PTSD, which hypothesize that the simple experience of a traumatic stressor—although essential for the onset of PTSD—does not by itself determine who will or will not develop the disorder. Instead, the present findings suggest that graded factors (e.g., severity of the traumatic stressor, number of additional stressful life events, amount of postwar social support, and strength of personal characteristics such as hardiness; L. A. King, King, Fairbank, Keane, & Adams, 1998) operate alone or in tandem to produce a particular level of symptom severity after traumatic exposure. Second, whereas contemporary theoretical formulations of PTSD emphasize causal and vulnerability factors associated with the disorder as it is presently classified, the finding that PTSD is continuous with milder stress reactions suggests the need for theories that extend beyond predictors of the presence or absence of the disorder. Instead, it may be more fruitful to consider aspects of the traumatic experience, the posttrauma environment, and the trauma-exposed individual that are associated with varying levels of symptom severity and chronicity after traumatic exposure.

The present dimensional findings may at first appear inconsistent with studies by Yehuda and colleagues (see Yehuda, 1998; Yehuda & McFarlane, 1995) which suggest that the pathology of PTSD may be distinct from that of the normal stress response. These studies have revealed significantly lower levels of cortisol among trauma survivors diagnosed with PTSD relative to survivors without PTSD, despite the elevations in cortisol typically associated with stress (but see Lemieux & Coe, 1995, for opposing findings). Although a thorough review and critique of this literature is beyond the scope of the present article, it is important to note that past neuroendocrine findings are as consistent with a dimensional model of PTSD as they are with a categorical model. Whereas some might suggest that the relationship between cortisol and PTSD resembles a step function, with different cortisol levels across PTSD and non-PTSD groups separated by a dramatic drop in cortisol at the diagnostic boundary, it is equally possible that cortisol levels share a linear relationship with PTSD severity, with cortisol steadily decreasing as symptom severity increases. Unfortunately, because neuroendocrinology studies of PTSD have relied almost exclusively on comparative methodological designs, it is presently difficult to determine which of these functions—and its associated latent model of PTSD—best accounts for the relationship between cortisol and PTSD. Future studies are needed to systematically chart cortisol levels across the entire spectrum of symptom severity for varying levels of stress exposure to flesh out the full shape of the cortisol-symptom function and determine

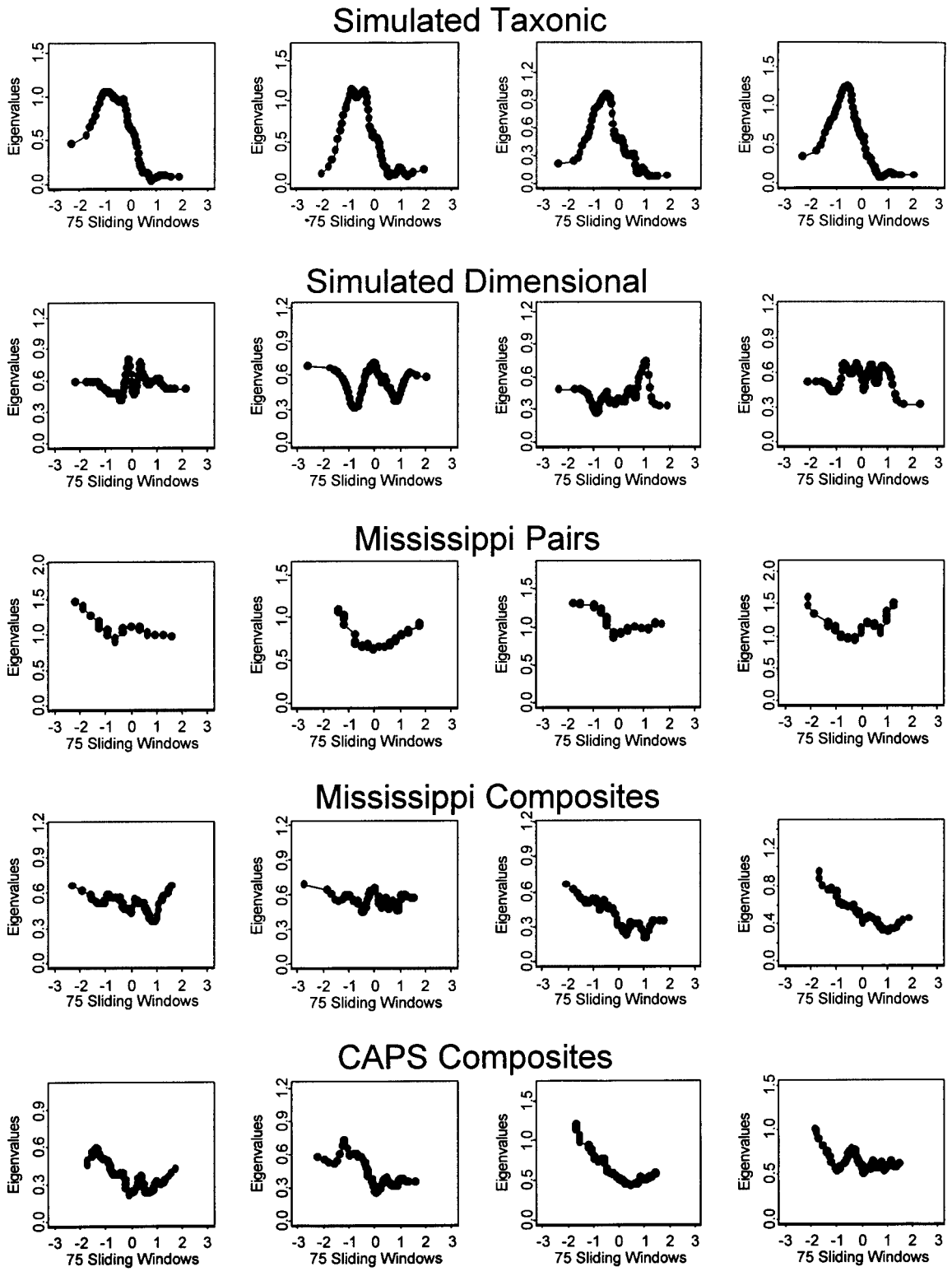

Figure 2. Maximum eigenvalue (MAXEIG) curves for the simulated taxonic and dimensional data sets plus each of the three empirical indicator sets (Mississippi Scale for Combat-Related PTSD pairs, Mississippi composites, and Clinician-Administered PTSD Scale [CAPS] composites, respectively). For each curve, cases were sorted by their scores on the input indicator and then grouped into partially overlapping windows (subsamples) along regions of the standardized input indicator. The *x*-axis represents these windows, whereas the *y*-axis plots eigenvalues to represent the degree of interindicator association within each window. For the Mississippi pairs, only the first four of the six MAXEIG curves are presented here to conserve space.

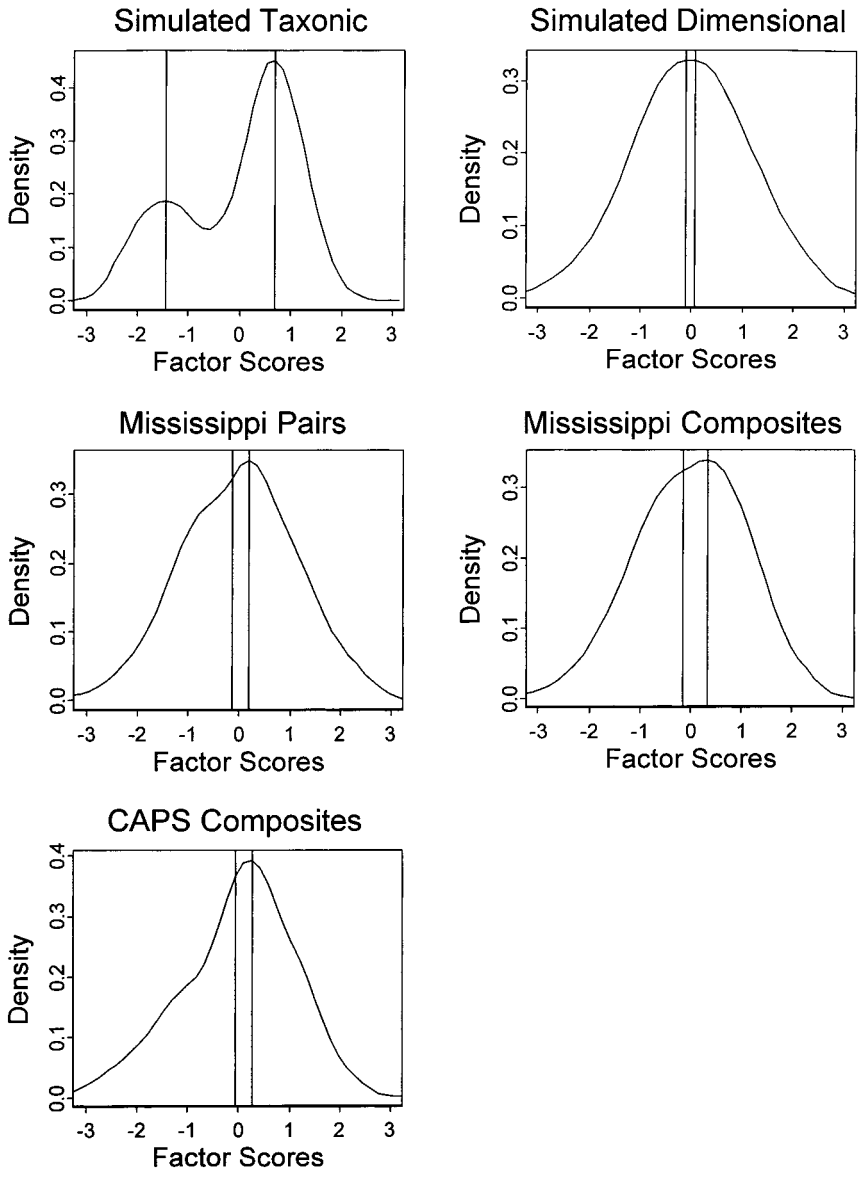

Figure 3. Latent mode (L-Mode) factor analysis curves for the simulated taxonic and dimensional data sets plus each of the three empirical indicator sets (Mississippi Scale for Combat-Related PTSD pairs, Mississippi composites, and Clinician-Administered PTSD Scale [CAPS] composites, respectively). Each curve displays, in the form of a density plot, a frequency distribution of standardized scores on the first factor underlying the indicator set.

whether the biological substrates of PTSD differ qualitatively or quantitatively from those of the normal stress response.

In addition to these theoretical implications, results of the present study further suggest that assessment instruments designed to differentiate individuals with and without PTSD may be less useful and less powerful than those that measure the severity with which PTSD symptoms are experienced. If PTSD is indeed dimensional, attempts to divide this dimension into PTSD and non-PTSD groups will only introduce needless error into measurement of the construct. By contrast, the use of assessment devices that yield continuous symptom severity scores would provide greater sensitivity to, and finer discrimination among, different symptom presentations along the full stress–response continuum. The use of continuous PTSD measures would also increase the statistical power of investigations in which these measures are used, thereby enhancing the ability to predict symptom patterns, symptom course, and response to treatment at varying levels of the severity continuum.

Finally, the present findings raise several important implications related to health care policy and mental health services for trauma-exposed individuals. Within the U.S. Department of Veterans Affairs, eligibility for veterans benefits and disability compensation, as well as coverage of mental health treatment, hinges on the diagnosis of a psychiatric disorder that can be linked to events

experienced during military service. The present findings underscore the need to determine which trauma-exposed individuals are most likely to require and benefit from clinical intervention and to identify both the criteria and the cutoff with which this decision can most validly be made (Widiger & Clark, 2000). Although the point of demarcation currently in use (the PTSD diagnosis) may turn out to represent the best cutoff for this purpose, past studies revealing considerable symptom severity and help-seeking behavior among individuals with partial PTSD suggest that current diagnostic criteria for PTSD may not constitute the most appropriate threshold for conferring or withholding benefits. Clearly, more work is needed to establish an optimal cutoff for determining veterans' eligibility for disability benefits.

In addition, it is important for research to examine the utility of using PTSD symptom severity as the primary criterion for disability decisions. It may be that other psychological or behavioral factors (e.g., suicidality, pronounced avoidance, severe interpersonal difficulties, self-medication through substance abuse), when aggregated, are more strongly predictive of long-term suffering, disability, or impairment and are therefore more defensible criteria for determining who will receive services and support. Finally, it may be that individuals falling at different levels of the stress–response continuum will respond differently to various interventions or will require differing degrees of therapeutic attention. Research is needed to ensure appropriate responding to the needs of different trauma survivors, as well as to maximize the efficiency of our treatments.

The present study had several significant strengths that promote confidence in the validity of its conclusions. The clinical sample was characterized by various features amenable to taxometric analysis, including a large sample size, a mixture of cases reflecting the full range of PTSD symptom severity, and a moderate-to-high (but not extreme) base rate of diagnosed PTSD. Moreover, the combat veteran composition of the sample represented an added strength of the study. Combat exposure encompasses an array of severe traumatic stressors that have long been associated with chronic PTSD. Thus, this veteran population provided favorable conditions for the detection of a PTSD taxon, and failure to find such a taxon argues against its existence. Furthermore, because combat veterans are by far the most widely studied population in the trauma literature, the present study has the potential to inform a large network of researchers and practitioners and to help direct future work in this prolific research area.

Although these qualities of the sample enhanced the validity and value of the present study, the restriction of the sample to male participants and to relatively homogeneous stressors related to military experience may limit the generalizability of our results. It is noteworthy, however, that early terms such as *battle fatigue, shell shock,* and *combat neurosis*—used to describe extreme psychological reactions of soldiers to war—were abandoned in favor of the more inclusive term of *PTSD* in recognition that the symptoms experienced by soldiers were essentially the same as those of men, women, and children exposed to physical and sexual assault, serious accidents, natural disasters, and other civilian traumas (see Herman, 1992; Keane & Barlow, 2001). The discovery of fundamental similarities in the reactions of human beings to extreme stress suggests that the structural nature of PTSD may well be comparable across sexes, age groups, and different traumatic stressors. However, this is clearly an empirical question, and further research is needed to replicate the present findings in other trauma-exposed populations.

Another important strength of the present investigation was its use of data collected by means of two distinct methods: clients' ratings of their own symptoms on a self-report questionnaire; and clinicians' ratings of symptom frequency and intensity made during a lengthy, structured clinical interview with the client. Both PTSD measures are widely used, have been validated on combat veteran samples, and have excellent psychometric properties. Multiple indicator sets were drawn from these measures, each having acceptably high validity and low estimated nuisance covariance and reflecting the full range of difficulties—cognitive, physiological, affective, behavioral, and interpersonal—that are characteristic of PTSD. These indicator sets yielded consistent results across three taxometric procedures and multiple base-rate comparisons, results that were further supported by comparisons with corresponding simulated data. The convergence of findings across different reporting methods, measures, symptom clusters, taxometric procedures, and supplemental consistency tests provides strong evidence for the dimensional solution uncovered here (Meehl, 1995, 1999). Future research might seek to replicate the present findings using still other indicators of PTSD, including physiological recordings (e.g., elevated heart rate in response to trauma-related stimuli, exaggerated startle reflex) and collateral ratings made by family members of the trauma survivor.

The dimensional structure of PTSD has potentially important implications for our understanding of the human response to situations of extreme stress. Reconceptualizing PTSD within the broadened framework of a response continuum will likely promote more effective assessment and treatment of trauma survivors, more valid procedures to identify survivors in greatest need of professional attention, and more rapid accumulation of knowledge about the full spectrum of psychological and behavioral reactions to the experience of traumatic events.

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