Is Posttraumatic Stress Disorder in a Class of Its Own? Longitudinal Comparison to Other Conditions Following Trauma and Life Stress Exposure

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Abstract

Objective: Posttraumatic stress disorder (PTSD) is defined by the assumption that qualifying traumatic events lead to a syndrome distinct from other internalizing disorders, while stressful life events play a prominent role in etiologic theories of major depressive disorder (MDD). We examined whether the environmental etiology of PTSD and MDD are distinct by evaluating the relative contributions of traumatic and stressful life events to both conditions. Harmful alcohol use and physical limitations served as noninternalizing comparators expected to show weaker associations with environmental factors.

Methods: Longitudinal cohort study of World Trade Center disaster responders who completed annual assessments of mental health and physical functioning from July 1, 2002, to December 31, 2020. Psychiatric diagnoses were ascertained in clinical interviews. Multivariate regression and multilevel modeling quantified the percentage of variance in psychopathology and physical limitations attributable to trauma versus life stress.

Results: 11,153 responders (mean age on September 11, 2001: 37.5 years; 91% male) completed 61,244 visits. The combined environmental effect of 9/11-trauma and life stress on PTSD and MDD was nearly identical (14.3% and 14.8% of between-person variability), but much weaker for alcohol use and physical limitations (0.8% and 9.1%). Life stress explained the most variance in all diagnoses and symptoms across longitudinal and cross-sectional analytic strategies.

Conclusions: In the longest study to date coexamining the environmental etiology of PTSD and MDD, trauma and life stress contributed to both conditions. Considering a spectrum of exposures from stressful life events to trauma and integrating knowledge across internalizing conditions may advance understanding and treatment of stress-related psychopathology.

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Controversy persists regarding the classification of posttraumatic stress disorder (PTSD) as etiologically distinct from internalizing disorders like depression and anxiety.[1,2] Many symptoms of PTSD overlap with these conditions,[3] but PTSD is distinguished primarily by the requirement for trauma exposure—Criterion A in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*).[4] Consequently, PTSD is defined by the notion that its symptoms are caused by a single qualifying traumatic event. The validity of this trauma-based distinction has been questioned for two reasons.

First, traumatic exposure contributes to many psychological and physical health outcomes, and its effects are not unique to PTSD.[5,6] The nonspecific role of trauma is reflected in *DSM*'s evolving conceptualization of PTSD, with the number of PTSD symptoms expanding from 12 in *DSM-III* to 20 in *DSM-5*. The price of this expansion is high heterogeneity of symptom presentations, which limits the utility of the PTSD diagnosis.[7,8]

Second, the distinction between trauma and stressful life events (SLEs) is a convention rather than an empirical conclusion.[9,10] This is apparent in *DSM*'s expansion of its definition of trauma to include indirect exposure. Importantly, SLEs are critical to the etiology of major depressive disorder (MDD).[11–13] The majority of individuals in clinical and community samples (50%–80%) experience an SLE just before MDD onset.[14] Accumulating evidence suggests that SLEs also play a major role in the development of PTSD symptoms, but

Clinical Points

- The environmental etiology of posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) may be similar, challenging their distinction in classification rubrics and suggesting that a dedicated class of "trauma- and stressor-related disorders" may create unnecessary silos.
- Helping trauma survivors address immediate life stress may reduce current and future symptoms of both PTSD and MDD.

this is not recognized in the *DSM* criteria.[9,15–19] Thus, the validity of the distinction between psychological sequelae of trauma versus life stress remains unresolved.

The present study examined critical assumptions about the nature of PTSD that have siloed the classification, research, and treatment of post-traumatic responses from internalizing conditions. We leveraged a sample of responders to the World Trade Center (WTC) disaster of September 11, 2001, to compare the relative contributions of SLEs and traumatic exposures to both PTSD and MDD. While MDD was chosen as a representative internalizing condition due to its strong connection with SLEs, we acknowledge that SLEs are implicated in the etiology of many psychiatric conditions.[5] Harmful alcohol use and physical limitations were considered as noninternalizing comparators to evaluate the specificity of environmental effects; these conditions were chosen given well-documented associations with trauma and SLE exposure.[20–23] Strengths of this study include systematic assessment of traumatic exposures, postdisaster life events, psychiatric diagnoses, and longitudinal data on relevant symptoms for up to 18 years post-9/11 (July 1, 2002–December 31, 2020).

Our investigation centered on two competing hypotheses. First, the conventional assumption that PTSD would be primarily explained by WTC trauma exposures and MDD by postdisaster SLEs as implied by *DSM-5* classification. Second, the alternative hypothesis that the contributions of SLEs and trauma to PTSD and MDD would be similar as implied by available research.[5,9,24] Additionally, we expected the effects of SLEs and trauma to be weaker for harmful alcohol use and physical limitations based on evidence that these links are relatively modest and often mediated by mental health conditions,[25,26] at-risk health behaviors,[27] coping strategies,[28,29] and physiological changes.[27]

METHODS

Participants and Procedure

Data were derived from the WTC-Health Program (WTC-HP), a consortium of 5 CDC-funded programs in New York and New Jersey that provide annual monitoring and treatment to responders with physical or mental health conditions related to WTC disaster exposure.[30] The WTC-HP began in July 2002, and enrollment remains open. The current study included data for the 11,153 responders monitored at the Stony Brook site of WTC-HP from 2002 to 2020. The original dataset is available from Roman Kotov. Participants completed monitoring visits that involved the same self-report questionnaires and interviews. Demographic information (date of birth, sex, race, etc.) was collected during enrollment in WTC-HP and at the first clinic visit via a structured medical interview and a patient information form (see Table 1). Participation in research is optional for WTC-HP enrollees, but most (>90%) provided written informed consent for their deidentified data to be used for research purposes. Demographic and occupational characteristics did not differ from those of the WTC-HP clinic enrollees as a whole.[30] All study procedures were reviewed and approved annually by the Institutional Review Board at Stony Brook University.

Table 1.

Demographic and Clinical Characteristics of WTC Responders

Characteristic	N	%/Mean (SD)
Type of responder	11,041	
Police		64%
Nontraditional		36%
Sex	11,153	
Male		91%
Veteran status	4,368	29.6%
Race/ethnicity	10,569	
White		84.6%
Black		5.9%
Other		3.2%
Hispanic		6.3%
Age on September 11, 2001	11,153	37.5 (8.4)
Clinical diagnosis	1,842	
PTSD only		3.3%
MDD only		2.3%
PTSD and MDD		4.9%
No PTSD or MDD		89.5%
Scales		
PCL	61,244	28.2 (13.5)
PHQ-9	58,734	3.9 (5.1)
AUDIT	49,116	3.1 (3.1)
PCS-12	45,835	43.6 (12.3)

Abbreviations: AUDIT = Alcohol Use Disorders Identification Test, MDD = major depressive disorder, PCL = PTSD Checklist for *DSM-IV*, PCS-12 = Physical Score of Short Form Survey-12, PHQ-9 = Patient Health Questionnaire-9, PTSD = posttraumatic stress disorder.

Measures

WTC exposure variables were evaluated and dichotomously coded (yes/no) using a clinical interview at the initial monitoring visit.[30] We focused on ten WTC

Table 2.

Percentages of Responders Who Experienced 9/11 World Trade Center Traumatic Exposures and Stressful Life Events Assessed Across 2002–2020

Exposure or event	N	%
9/11 WTC exposures[a]		
Dust cloud	11,012	18.1
Lost someone	10,459	65.3
Someone injured	10,348	54.0
Search and rescue	10,108	22.2
Collapse site	10,745	81.7
Human remains	8,771	65.8
Early arrival	11,012	71.4
Long work on-site	10,597	25.0
Slept on-site	10,602	14.5
Continuous work in September	11,155	22.9
Stressful life events (all years)[a]		
Job loss layoff, or substantial loss of income	49,940	12.2
Changed where you live	50,322	7.9
Break up with a spouse or partner	50,176	5.2
Break up with a best friend	50,291	3.6
Major car trouble	50,257	10.6
Robbery or house break in	50,381	0.9
Mugging or beaten up[b]	50,271	0.3
Bad debts or repossession	50,303	5.7
Serious illness	49,924	11.4
Injury	50,141	16.3
Arrests	50,264	0.7
Legal problems	50,252	3.6
Illness or injury of a household member	50,179	15.6
Death of a household member	50,264	4.1
Death among relatives or close friends	50,141	31.2
Pressure to provide shelter to someone you did not wish to house	50,207	4.0
Threatened or verbally abused by a household member	47,919	2.5
Physically assaulted or confronted by a household member[b]	47,968	0.9
Post-WTC stressful life event composite (all years)	**Total N**	**Mean (SD)**
	50,033	1.35 (1.61)

[a]All variables were dichotomized (1 = yes and 0 = no).
[b]Life events that may meet Criterion A for posttraumatic stress disorder in *DSM-IV* and were excluded from the stressful life event composite in sensitivity analyses.
Abbreviation: WTC = World Trade Center.

exposure variables (Table 2) that were associated with increased risk of PTSD and other health outcomes,[31–33] including being caught in the dust cloud; death of a colleague, family member, or friend on 9/11; knowing someone who was injured on 9/11; involved in search and rescue efforts; working primarily at or adjacent to the towers collapse site; exposure to human remains of 9/11 victims; early arrival (ie, on September 11 or 12, 2001); long work on-site (ie, top quartile of total hours worked in this sample); slept on-site any nights during September or October 2021; worked on-site every day from September 11 to September 30, 2001. The Disaster Supplement of the Diagnostic Interview Schedule[34] was used at each monitoring visit to assess whether 18 stressful life events occurred since the last visit (Table 2). Responses were summed to create a postdisaster SLEs composite score (range: 0–18). We chose this conservative operationalization of SLEs for two reasons. First, we conservatively corrected for the potential bias of representing SLEs as 18 distinct variables, because doing so would give SLEs an advantage over 10 WTC exposure variables in predictive power comparisons. Second, this cautious approach was aligned with our substantive interest in the *cumulative* burden of post-trauma life stress.[32,35,36]

Current (past month) PTSD and MDD diagnoses were ascertained using the Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders* (SCID), Fourth Edition. [37] The SCID was administered by trained master's-level interviewers who reviewed responders' medical and occupational and medical histories to facilitate rapport and enhance rating accuracy. PTSD symptoms were assessed in relation to traumatic WTC exposures (Criterion A). Inter-rater reliability for diagnoses in 55 independently rated audiotapes was very good ($\kappa \geq 0.82$).[31] Diagnostic

assessments were completed in the middle of the interval (December 29, 2011–October 26, 2016).

Self-report measures of emotional and physical health were administered at each monitoring visit. WTC-related post-traumatic stress symptom severity was measured using the 17-item PTSD Checklist (PCL) for *DSM-IV*.[38] PCL instructions were to rate symptoms in the past month "in relation to 9/11." Internal consistency of the PCL total score in the present sample was excellent (α = .96). Current *DSM-IV* depression symptoms (past 2 weeks) were measured with the 9-item Patient Health Questionnaire-9 (PHQ-9).[39] Internal consistency of the PHQ-9 total score was also excellent (α = .91). Harmful alcohol use in the past year was evaluated using the 10-item Alcohol Use Disorders Identification Test (AUDIT).[40] Internal consistency of the AUDIT total score was adequate (α = .71). The Short Form Survey-12 was administered to evaluate physical health outcomes (PCS-12).[41] The PCS-12 assesses general health, pain, mobility, and participation in activities in daily living and work. The scale is standardized to have mean of 50 and SD = 10 in the US general population, with higher scores reflecting better functioning.

Statistical Analyses

Cross-sectional analyses of environmental effects on self-report symptoms were conducted in SPSS 28.0 using multiple regression for continuous outcomes (2009–2020), and logistic regression was used for dichotomous diagnostic interview data (2011–2016). Longitudinal analyses were performed in SAS 9.4 using multilevel modeling and examined all data collected from 2002 to 2020.

For cross-sectional multiple regression models, data were stratified by year to ensure independence of observations (as monitoring visits were at least 12 months apart). To obtain accurate R^2 estimates, we required ≥1,000 observations,[42] which were available in 2009 through 2020. This 12-year range therefore provided 12 internal replications for each of the 4 symptom outcomes (PCL, PHQ-9, AUDIT, and PCS-12), resulting in a total of 48 hierarchical regression models. In each model, the continuous outcome was regressed on the 10 WTC-related trauma exposures entered simultaneously in step 1, and the SLEs composite for the interval just before the visit was entered in step 2. This ordering of variables reflects the temporal sequence of environmental factors.*

Longitudinal analyses consisted of multilevel models† with 2 levels (observations nested within individuals) using the restricted maximum likelihood estimator in SAS, a full information maximum likelihood approach to missing data that uses all available information.[43] For each of the 4 symptom outcomes, we specified 3 separate models in the following order. In the first model, only year since 9/11 was the predictor to control for trends in outcomes over time. In the second model, 10 WTC exposures were added as predictors. In the third model, the SLEs composite was added. In all models, the intercept was a random effect, and predictors were fixed effects. Decrease in variance of the random intercept relative to the first model indicates the proportion of between-person variance in the outcome explained by the predictors across visits.[44]

Environmental effects on diagnoses were tested in the subset with diagnostic interview data. Two logistic regression models were estimated with current diagnoses of PTSD (N = 1,837) and MDD (N = 1,842) as the binary outcome variables. These models were conducted in 3 steps. Years since 9/11 were entered to control for time trends in step 1. The ten WTC-related trauma exposures were entered in step 2. The SLEs composite for the interval preceding diagnosis were entered in step 3. Nagelkerke R^2 values indicated the percentage of variability in the outcomes explained by SLEs and WTC trauma. To guard against inflated pseudo-R^2 values and potentially misleading conclusions, we also ensured that both diagnostic variables met sample size guidelines for logistic regression[45] (N = 700 given 12 independent variables).

Lastly, we assessed the robustness of our findings through sensitivity analyses for cross-sectional hierarchical and logistic regression models. In this separate set of analyses, we excluded 2 stressful life events that were likely to meet Criterion A for PTSD (ie, mugging or beaten up and physically assaulted or confronted by a household member) to minimize conceptual overlap between SLEs and Criterion A events.

RESULTS

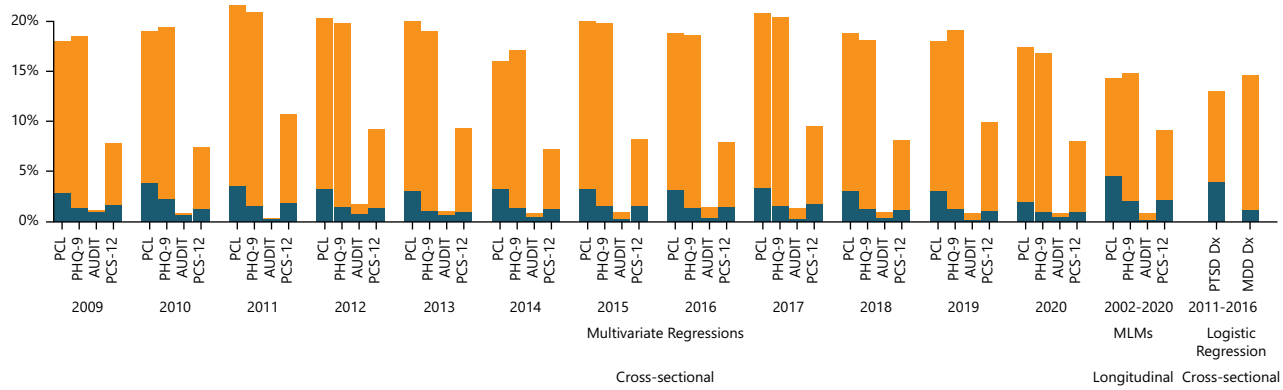
The majority of the sample was male (91%) and white (80.1%) law enforcement workers (64%; see Table 1). The average age on 9/11 was 37.5 years (SD = 8.4), and 29.6% reported a history of military service. Aggregate descriptive statistics for each self-report outcome variable (PCL, PHQ-9, AUDIT, PSC-12) using all available data (2002–2020) are presented in Table 1. Over 10% were diagnosed with current PTSD or MDD via the SCID, nearly half of which had both PTSD and MDD. Other participant characteristics have been reported elsewhere (eg, marital and employment status).[30,31]

*We acknowledge that a potential consequence of limiting our definition of trauma to a single event is that some unmeasured trauma exposures may be mediated by SLEs. Thus, while the total effect of WTC exposures on symptoms was captured in block 1 (whether mediated by SLEs or direct), any trauma exposures that are not in block 1 and mediated by SLEs would count toward block 2 (SLEs) but would belong to block 1 (trauma exposure).

†To ensure multilevel modeling was the best approach, we tested the following 4 assumptions: (1) the explanatory variable is related linearly to the response; (2) the errors have constant variance over time; (3) the errors are independent of fitted values; (4) the errors are normally distributed.

Figure 1.

Total Percentage of Variance Accounted for in Psychiatric and Physical Outcomes by WTC Trauma and Stressful Life Events Across Cross-Sectional and Longitudinal Analytic Strategies[a]



[a]Blue is variance explained by 10 separate WTC trauma exposures. Orange is the additional variance explained by a composite of stressful life events. Linear regressions were stratified by year, 2009–2020. Multilevel model results show the percentage of between-person variability in outcomes explained in aggregate across 2002–2020, controlling for the assessment year. Logistic regression results present the percentage of variance accounted for in MDD and PTSD diagnoses collected between 2011 and 2016, controlling for the assessment year.

Abbreviations: AUDIT = Alcohol Use Disorders Identification Test, Dx = diagnosis, MDD = major depressive disorder, MLM = multilevel model, PCL = PTSD Checklist for *DSM-IV*, PCS-12 = Physical Score of Short Form Survey-12, PHQ-9 = Patient Health Questionnaire-9, PTSD = posttraumatic stress disorder.

Environmental Effects on Symptoms in Cross Section

Contributions of trauma exposures and SLEs to symptoms in each year (2009–2020) were calculated using linear regression. Results are depicted in Figure 1 and are remarkably consistent over the years (details given in Tables 3 and 4). The total R^2 statistics showed that WTC exposures and SLEs jointly accounted for the same moderate amount of variance in PTSD (across years mean $R^2 = 19\%$; all $P < .001$) and MDD (mean $R^2 = 19\%$; all $P < .001$) but contributed far less to harmful alcohol use (mean $R^2 = 1\%$; all $P < .033$) and physical limitations (mean $R^2 = 8.8\%$; all $P < .001$). The SLEs composite was the main predictor of both PTSD and MDD symptoms (mean $R^2 = 16\%$ and 17.6%), whereas WTC exposures accounted for a small percentage of variance (mean $R^2 = 3.1\%$ and 1.4%). The same pattern was observed for harmful alcohol use and physical impairment, with greater contribution from the SLEs composite (mean $R^2 = 0.6\%$ and 7.5%) than WTC exposures (mean $R^2 = 0.4\%$ and 1.3%), although all effects were smaller.

Environmental Effects on Symptoms Across Study Interval

Multilevel modeling allowed us to examine environmental effects on symptoms across 18 years (see Figure 1; Table 3). Together, WTC exposures and SLEs accounted for similar amounts of variance in PTSD and MDD symptoms (14.3% and 14.8%), but much less variance in hazardous alcohol use and physical limitations (0.8% and 9.1%). Across outcomes, SLEs were the main predictors, explaining 2.1–7.0 times more between-person variance in symptoms than exposures.

Environmental Effects on Diagnoses

Effects of WTC exposures and SLEs on current PTSD and MDD diagnoses were examined using logistic regression (see Figure 1; Table 4). Jointly, WTC exposures and SLEs predicted PTSD and MDD similarly (Nagelkerke $R^2 = 13.0\%$ and 14.7%, above and beyond time). For both disorders, SLEs generated the most predictive power (Nagelkerke $R^2 = 9.1\%$ and 13.6%).

Sensitivity Analysis

Sensitivity analyses for cross-sectional hierarchical and logistic regression models confirmed the robust effects of SLEs on current PTSD and MDD diagnoses and self-reported symptoms. Effect sizes for SLEs were nearly identical to our initial set of analyses, which included 2 stressful life events that may meet Criterion A for PTSD (ie, mugging or being beaten up and physically assaulted or confronted by a household member), with variance explained shifting no more than 0.40%.

DISCUSSION

This study investigated the nosological question of whether PTSD falls in a distinct class of disorders defined by traumatic exposure. We compared contributions from WTC traumatic exposures and SLEs to PTSD, depression, harmful alcohol use, and physical

Table 3.

Multilevel Modeling and Multiple Regression Results Showing the Percentage of Variance Accounted for in Psychiatric and Physical Functioning Measures by WTC Trauma and Stressful Life Event Variables Assessed Yearly Between 2002 and 2020

Year of visit	Predictor	PCL	PHQ-9	AUDIT	PCS-12
Multilevel models		Percent between-person variance explained			
2002–2020	10 WTC Exposures	4.6	2.0	0.1	2.1
	WTC Exposures + SLEs	14.3	14.8	0.8	9.1%
Multiple regression models		R^2 (%)			
2009	10 WTC Exposures	2.8	1.3	0.9	1.6
	WTC Exposures + SLEs	17.4	18.1	1.1	9.8
2010	10 WTC Exposures	3.8	2.2	0.6	1.2
	WTC Exposures + SLEs	19.0	19.4	0.8	7.4
2011	10 WTC Exposures	3.5	1.5	0.2	1.8
	WTC Exposures + SLEs	21.6	20.9	0.4	10.7
2012	10 WTC Exposures	3.2	1.4	0.7	1.3
	WTC Exposures + SLEs	20.3	19.8	1.8	9.2
2013	10 WTC Exposures	3.0	1.0	0.6	0.9
	WTC Exposures + SLEs	20.0	19.1	1.0	9.3
2014	10 WTC Exposures	3.2	1.3	0.4	1.2
	WTC Exposures + SLEs	16.1	17.1	0.8	7.2
2015	10 WTC Exposures	3.2	1.5	0.2	1.5
	WTC Exposures + SLEs	20.1	19.8	0.9	8.3
2016	10 WTC Exposures	3.1	1.3	0.3	1.4
	WTC Exposures + SLEs	18.8	18.6	1.5	8.0
2017	10 WTC Exposures	3.3	1.5	0.2	1.7
	WTC Exposures + SLEs	20.8	20.4	1.4	9.5
2018	10 WTC Exposures	3.0	1.2	0.3	1.1
	WTC Exposures + SLEs	18.9	18.1	0.9	8.1
2019	10 WTC Exposures	3.0	1.2	0.1	1.0
	WTC Exposures + SLEs	18.0	19.2	0.9	9.9
2020	10 WTC Exposures	1.9	0.9	0.4	0.8
	WTC Exposures + SLEs	17.4	16.8	0.8	8.0

Abbreviations: AUDIT = Alcohol Use Disorders Identification Test, PCL = PTSD Checklist for *DSM-IV*, PCS-12 = Physical Score of Short Form Survey-12, PHQ9 = Patient Health Questionnaire-9, SLEs = stressful life events, WTC = World Trade Center.

impairment. This yielded 3 main findings. First, the combined effect of environmental factors—exposures and SLEs—on PTSD and MDD was nearly identical. Second, this effect was notably weaker for harmful alcohol use and physical impairment. Third, SLEs contributed much more than WTC exposures to both PTSD and MDD, despite our conservative design. Trauma exposure was represented by 10 variables vs 1 for SLEs and focused PTSD assessment on the WTC disaster, giving traumatic exposure maximal advantage. Results were consistent across multiple cross sections and longitudinally for symptoms and diagnoses. These internal replications strengthened our confidence in the current findings, while the large sample size enabled us to assess even small effects with high precision.[46] Our results appear inconsistent with the conventional assumptions that traumatic exposures contribute to PTSD and SLEs to MDD as implied by their classification in contemporary nosologies. Instead, findings highlight a nosological similarity between PTSD and MDD. If replicated for other traumas, it may be that the environmental etiology is qualitatively similar and largely shared across PTSD, MDD, and likely other internalizing disorders. Our findings connect with the prior literature in 3 respects.

First, our results are consistent with evidence that trauma contributes to internalizing conditions as well as PTSD.[6,24,47] Trauma exposure frequently results in features observed in depression (eg, sense of defeat, reduced self-efficacy, chronic stress reactivity, rumination, and social detachment),[24,48] calling into question the privileged relationship between trauma and PTSD in dominant

Table 4.

Logistic Regression Results Showing the Percentage of Variance in PTSD and MDD Diagnoses (2011–2016) Explained by the Number of Years Since 9/11, Trauma, and Stressful Life Events

	Nagelkerke R^2	
Predictor	PTSD Dx	MDD Dx
Years since 9/11	0.9%	1.7%
10 WTC exposures	3.9%	1.1%
Stressful life events	9.1%	13.6%
Total	13.9%	16.4%

Abbreviations: Dx = diagnosis, MDD = major depressive disorder, PTSD = posttraumatic stress disorder, WTC = World Trade Center.

nosologies. Recognition of post-traumatic reactions other than anxiety also supports the field's effort to broaden the narrow conceptualization of PTSD as a fear disorder using extinction paradigms.[49,50] The limitations of short-term exposure-based monotherapies for PTSD (ie, high dropout rates and inadequate response),[49,51,52] which do not consistently outperform non-trauma-focused interventions,[53] underscore this shift. In contrast, contributions from the environmental stressors to harmful alcohol use were very weak, perhaps due to an indirect influence. Trauma survivors might drink to mitigate distress,[54] and PTSD-related increases in externalizing features like behavioral disconstraint may also result in risk of harmful alcohol use.[55,56] Associations between environmental stressors and physical health were also more modest relative to PTSD and MDD, consistent with prior research showing that these links are often mediated by mental health conditions, at-risk health behaviors (eg, poor diet and insomnia), and physiological/neurobiological changes.[23,26,27] However, the combined effects of environmental exposures on physical health align with existing evidence of a dose-response relationship, with the cumulative burden of environmental stress likely contributing to allostatic load and subsequent health complications.[21,23,57]

Second, our findings agree with prior evidence that non-Criterion A life events (eg, relationship dissolution) can result in the same rates of PTSD as qualifying trauma (eg, injury due to a serious accident).[9,15,58] For instance, prolonged exposure to chronic interpersonal stressors and/or systemic oppression may impair stress regulation and coping capacities, leading to the emergence of PTSD symptoms.[59] Criterion A suffers from other problems, including poor agreement between assessors and participants on the "worst event"[60] and inadequate attention to both contextual and individual differences factors that shape the severity of events.[61] Moreover, some life events that do not qualify as Criterion A (eg, homelessness) can have greater psychological ramifications than qualifying exposures (eg, witnessing a car crash). Recasting traumatic events along a continuum with SLEs may address these problems.[62] One strategy for conceptualizing this dimension is in terms of event severity, such as its contextual threat (ie, objective negative impact).[63] A dimensional approach can help connect disparate literatures on trauma and life stress and on PTSD and internalizing conditions. It could also promote comprehensive assessment of environmental contributors to psychopathology through the option to add a stress specifier to various psychiatric disorders.[50,62]

Third, postdisaster SLEs' major role in PTSD aligns with observations in various traumatized populations.[9,16,32,64] That the SLE index remained more predictive than WTC trauma variables suggests that PTSD symptoms were likely exacerbated or prolonged by subsequent SLEs.[65] An immediate clinical implication is that mental health providers of trauma survivors should consider the cumulative effects of post-trauma life stress. Addressing the complex relationship between life stress and various health outcomes requires tailoring interventions to individual needs and emphasizing skills for coping with ongoing adversity, including problem-solving, present-focused self-reflection, emotional/arousal regulation, and interpersonal effectiveness.[48] Addressing emotional suppression may also mitigate chronic stress and adverse health consequences, especially for individuals who do not present with significant emotional distress.[57] Such approaches for improving adaptive responses to stress are integral to a range of evidence-based cognitive-behavioral, psychodynamic, and emotion-focused therapies.

The study had several limitations. First, the majority of the sample was white male police officers. Our findings are consistent with research in other populations,[15,66] especially military veterans,[19] but replication in more diverse samples is needed. Second, the assessment of traumatic exposures was limited to the WTC disaster so subsequent traumas could not be considered. Analysis of a common trauma allowed us to control for time since WTC exposure and is aligned with PTSD assessment, as it focused on the events of 9/11. Generalizability to other trauma types is important to examine. Third, the relative potency of WTC exposures vs SLEs may be confounded by the time elapsed since the disaster. SLEs were assessed at each follow-up visit, resulting in a measure of stress that was more proximal and dynamic. We did not observe a change in the predictive power of WTC exposures from 2009 to 2020, but they may have been more consequential in the first few years after 9/11. Studies that monitored symptoms shortly after 9/11/2001 are in a better position to evaluate this question. Moreover, it may be that grouping all "stressor-related" conditions is nosologically inaccurate. Acute stressor-related disorders with 1- to 6-month windows could be conceived of as "purer" trauma/stressor-related

conditions as conceptualized in early formations of PTSD ("shell shock"), while chronic stressor-related disorders may increasingly resemble other disorders like depression. Fourth, PTSD measures reflected *DSM-IV* criteria, excluding *DSM-5* features corresponding to distress and externalizing. While maintaining a consistent definition of PTSD enabled direct comparisons across all years, determining which definition provides optimal trauma-related specificity remains challenging, particularly when considering alternative diagnostic frameworks like the International Classification of Diseases.[67] Fifth, some SLEs may have met Criterion A (eg, injury, death of a family member, and threatened by a household member) but were not excluded in our sensitivity analysis because we lacked sufficient information to categorize them as Criterion A events. Relatedly, some SLEs might have been related to 9/11 (eg, death of a family member), making it difficult to distinguish between SLEs and trauma and to decipher whether a SLE might qualify as an index trauma. Lastly, effect sizes may be exaggerated due to possible confounding of self-report measures. For instance, someone suffering from major depression might perceive and recall both past and present events in a way that leads them to report more SLEs and PTSD symptoms.

CONCLUSIONS

Consistent with Dohrenwend's [5] comprehensive stress continuum framework, our findings demonstrate the value of considering both event-specific trauma and ongoing life stress in the study of how stressors contribute to physical and psychiatric outcomes. If our results are replicated for other index traumas, it may be that embedding etiology in the diagnostic criteria for PTSD creates an unnecessary silo that hinders a transdiagnostic view of the impact of trauma and life stress. Appending a stress-specifier to various psychiatric disorders rather than creating a dedicated disorder class would support a nuanced understanding of the links between a spectrum of stressful events and a variety of psychopathology symptoms.[50,62] Considering the influence of adverse life events beyond the confines of PTSD diagnostic criteria is aligned with our finding that environmental stress (Criterion A in combination with non-Criterion A events) was equally related to MDD and PTSD and with the evolving trend toward precision medicine in psychiatry. Precision medicine takes a personalized and transdiagnostic approach to both assessment and treatment by considering individual differences in symptomatology and treatment response.[68] More work is needed to clarify the nosological implications herein in light of strong evidence that PTSD symptoms overlap with various transdiagnostic domains (eg, externalizing and thought disorder)[50,69] and that traumatic events are likewise related to symptoms outside of PTSD and internalizing.[5]

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