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REVIEW

Posttraumatic stress following spinal cord injury: a systematic review of risk and vulnerability factors

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Objectives: To summarise quantitatively the available evidence relating to pretraumatic, peritraumatic and posttraumatic characteristics that may increase or decrease the risk of developing posttraumatic stress disorder (PTSD) following spinal cord injury (SCI).

Study design: Systematic review.

Methods: Seventeen studies were identified from the PubMed, PsycInfo, Embase, Scopus, CINAHL, Web of Science and PILOTS databases. Effect size estimates (*r*) with associated 95% confidence intervals (CIs), *P*-values and fail-safe *N*s were calculated.

Results: Individual studies reported medium-to-large associations between factors that occurred before (psychiatric history $r=0.48$ (95% CI, 0.23–0.79) $P=0.01$) or at the time of injury (tetraplegia $r=-0.36$ (95% CI, −0.50 to −0.19) $P<0.01$). Postinjury factors had the strongest pooled effects: depressed mood ($r_w=0.64$, (95% CI, 0.54–0.72)), negative appraisals ($r_w=0.63$ (95% CI, 0.52–0.72)), distress ($r_w=0.57$ (95% CI, 0.50–0.62)), anxiety ($r_w=0.56$ (95% CI, 0.49–0.61)) and pain severity ($r_w=0.35$ (95% CI, 0.27–0.43)) were consistently related to worsening PTSD symptoms ($P<0.01$). Level of injury significantly correlated with current PTSD severity for veteran populations (Q_B (1) = 18.25, $P<0.001$), although this was based on limited data.

Conclusion: Combinations of peri- and post-injury factors appear to be influential in the development of PTSD among persons with SCI. Further studies are needed to extrapolate these findings to the broader spinal cord-injured population. More longitudinal research, driven by multicausal models of causation such as the diathesis-stress model, is also needed to determine the temporality of PTSD risk factors.

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INTRODUCTION

Traumatic stress reactions are common following an acquired spinal cord injury (SCI), with 40% of adults reporting an acute symptom pattern of avoidance, heightened (hyper) anxiety and intrusive trauma memories.[1] In total, 4% experience these symptoms beyond 1 month after SCI, warranting a diagnosis of posttraumatic stress disorder (PTSD).[2] Without treatment, the lifetime prevalence of PTSD for those with SCI remains high: up to 29% continue to report symptoms 30 years after injury.[1] Consistent with a diathesis-stress conceptualisation, a complex interplay of individual and contextual factors present before (pre), during (peri) or post-SCI appears to increase vulnerability to the development of PTSD.[1,3,4] Knowledge of these factors is important in order to accurately identify and support high-risk individuals. A better understanding of the factors that may exacerbate partial (subsyndromal or acute) PTSD can also help clinicians provide the necessary treatment before symptoms become chronic.

Female gender, in particular, may place injured adults at risk of developing PTSD.[4] Indeed, gender is a salient risk factor for other post-injury negative emotional responses, including depression,[5,6] despite a greater injury incidence and prevalence among males (80% males: 20% females).[7] However, gender may influence PTSD only to the extent that other sociocontextual factors are present. This includes lower levels of education and income and being divorced or widowed—although the identified connections between educational attainment, family stability and distress vary in magnitude.[2,8–11] The likelihood that an individual with SCI will develop PTSD after injury may also be determined by preexisting mental health problems (for example, depression, anxiety), negative coping styles and prior exposure to trauma.[2,5,8,12,13]

There are few data in relation to the role that peritrauma factors, namely SCI characteristics, have in PTSD aetiology and maintenance. Regardless of cause, a spinal cord lesion can be viewed as a traumatic event, with life-threatening SCI complications (for example, cardiovascular, respiratory) potentially reinforcing an elevated fear of death—considered a fundamental source of PTSD.[14] It is also postulated that individuals with higher level of injury experience more severe emotional reactions, resulting from a dependence on others for day-to-day tasks.[15,16] From a neurobiological perspective, sensory and motor pathways are compromised following tetraplegic injury which, in turn, may impede PTSD symptoms of sympathetic arousal (for example, hypervigilance, exaggerated startle response).[17] However, anecdotal clinical lines of evidence suggest that those with long-term paraplegia may struggle more so. This may relate to a sense that their degree of impairment, which commonly includes 'invisible'

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impairments such as chronic pain and incontinence, does not neatly fit the stereotypical view of disability.[18] Further support for these theories is, however, needed. In addition, veteran studies have demonstrated that intoxication or substance abuse at the time of injury can be detrimental to mental health outcomes.[17,19] The generalisability of these findings to the broader SCI population is, however, limited given that this subgroup may be at greater risk of mental illness due to their experience of prior trauma.[20,21]

Perhaps the most consistent evidence relates to the role of post-SCI complications and comorbidities and risk of PTSD. It is thought that chronic neuropathic or nociceptive pain and psychological distress are mutually maintaining.[22,23] Reactions of depression and PTSD also commonly co-occur.[5,6,24] Both depression and PTSD impede disability acceptance by reinforcing negative cognitions about the self (including 'self-blame') and the world.[12] Notably, this research is characterised by self-reported screening measures of trauma impact, which vary in the degree to which they map onto standardised diagnostic nomenclature (for example, Diagnostic and Statistical Manual of Psychiatric Disorder (DSM)). However, few SCI studies have empirically tested the impact of PTSD measurement type on risk factor estimates.

In summary, the burden of PTSD symptoms in those with SCI has been described. This provides an opportunity to quantitatively summarise the data, which, to date, have not occurred. Our objective was to evaluate a range of pre-, peri- and post-SCI factors that can potentially lead to PTSD by performing a systematic and meta-analytic review of the available research. A further aim was to examine the differential effects of sample (that is, military veterans) and measurement (that is, dichotomous, diagnostic measures versus continuous, symptom measures) characteristics on these findings. Research gaps in the current evidence base were also identified.[25]

METHODS

Literature search

A search of seven electronic databases (PubMed, PsycINFO, Web of Science, Scopus, Embase, CINAHL, PILOTS) was conducted for the period between January 1980 (to coincide with the inclusion of PTSD as a diagnostic category in the DSM-III) and December 2016. Search terms were tailored to each database and included a broad list of keywords and phrases related to SCI (for example, spine, spinal, spinal cord trauma, spinal cord fracture) and posttraumatic stress (for example, PTSD, traumatic neurosis, shell shock, war neurosis and combat stress) (see Appendix for logic grids with Boolean operators). A research librarian assisted with the development of the search terms to ensure their accuracy. The reference lists of all included studies were searched, as were book chapters[26] and relevant reviews,[1,27–33] in order to identify any research that may have been missed. A request for articles in press was additionally made through the American Psychological Association's Division 22 (Rehabilitation Psychology) Listserv. As a countercheck, international peer-reviewed journals targeted to SCI rehabilitation (*Archives of Physical Medicine and Rehabilitation*, *Disability and Rehabilitation, European Spine Journal, Journal of Spinal Cord Medicine*, *SCI Nursing, Spine, Spinal Cord, Rehabilitation Psychology, The Spine Journal, Topics in Spinal Cord Injury Rehabilitation*) were electronically searched using 'PTSD' and 'post-traumatic stress' as keywords.

Eligibility criteria

Studies had to meet the following criteria for inclusion in this review: (1) participants with an acquired (traumatic or nontraumatic) SCI were assessed as a young child (that is, 8–11 years), adolescent (12–17 years) or adult (aged 18+ years); and (2) current PTSD symptomatology was assessed using a standardised self-report or clinician-based instrument. This included a broad group of participants: individuals experiencing acute stress symptoms and those reporting re-experiencing, avoidance and arousal symptoms consistent with established (DSM) PTSD criteria. (3) Studies also had to assess risk factors for PTSD as primary or secondary outcomes. This included pre-injury (for example, age, gender), injury-specific (for example, SCI level) and post-injury variables (that is, SCI sequelae). A risk factor was defined as any variable thought to contribute to symptom severity or diagnostic status.[31–33] (4) Studies also had to provide parametric data to enable the calculation of an effect size *r*. This included studies using a group design (which reported *t*-tests, chi-square χ^2 and exact *P*-values) or a correlational design (reporting Pearson's or point-biserial *r*). (5) Finally, studies had to be published in a journal in English.[34] To ensure generalisability of the findings, only PTSD correlates examined by two or more studies were considered.[35] Studies that examined multiple trauma groups or those that included individuals with spinal cord disorders of congenital or disease origin, without appropriate disaggregation within the results, were excluded. Additionally, studies that only provided data from multivariate analysis (for example, regression, factor analysis, structural equation modelling) were ineligible.[36]

Reliability of the article selection process was checked, with a second (DD) and third reviewer (SP) screening the titles and abstracts of 20 eligible articles randomly selected by the primary reviewer (KP). Inter-rater agreement was moderate ($\kappa = 0.60$ (95% confidence interval (CI), 0.17–0.97)). Discrepancies, which focussed on studies to exclude, were discussed and resolved by consensus.

Of the initial 1307 articles, 30 eligible studies were identified. These studies were further examined to ensure independence of the samples.[36] Lead authors of studies were contacted for clarification where necessary.[5,14,15] Sample overlap was identified in 20 studies led or co-authored by Boyer,[37,38] Chung[13,39,40] Kennedy,[41,42] Martz,[14,43,44] Livneh,[5,45] Nielsen[10,15] and Radnitz.[8,9,17,21,46,47] These studies were subsequently combined and treated as seven independent studies. Where a single study contributed multiple effect estimates for the same risk factor, data from the largest sample or most recently published article were used to ensure no duplication. This resulted in a final sample of 17 independent studies (see Figure 1).

Risk of bias

The methodological quality of included studies was assessed using a checklist modelled from existing rating tools.[48–51] Studies were rated on components that are considered critical to clinical research: statistical power (whether the study findings could be attributed to chance); internal validity (degree to which a study minimises bias in measurement and data collection) and external validity (extent to which the study findings can be generalised to the broader SCI population).[52] Two reviewers (KP and DD) were involved in the rating process, with each independently evaluating each study. Inter-rater reliability was high for all judgements (agreement = 94%).

Data collection and preparation

In line with evidence-based recommendations for the reporting of meta-analyses,[53] a data extraction sheet was developed to summarise key information from each study. Extracted data included the following: (1) sample demographics (for example, mean age, gender); (2) injury characteristics (for example, SCI severity, duration); (3) effect-size data (for example, means, s.d., correlations, *t*-tests, chi-square χ^2); (4) study characteristics (for example, PTSD measure, recruitment source); and (5) risk factors. Eighteen risk factors were identified and classified into one of the following three broad categories: pre-SCI (that is, gender, age at injury, relationship status, education, psychiatric history and previous trauma), peri-SCI (that is, lesion completeness, SCI level, age at injury, alcohol/substance use at injury and loss of consciousness); and post-SCI (that is, time since SCI, pain level, posttraumatic cognitions, comorbid depression, anxiety, distress and social support).

Data analysis

The correlation coefficient *r*, which quantifies the strength and direction of a relationship between two dichotomous or continuous variables, was selected as the primary effect size for review as it was the most commonly reported metric by eligible studies. Studies that did not directly report *r* provided test statistics (χ^2, *t* or F, exact *P*-value, frequency count), which can be converted.[54,55] To ensure that all effect sizes reflected the same relationship direction, lead authors were contacted to clarify the coding of their risk factors.[37,41,43]

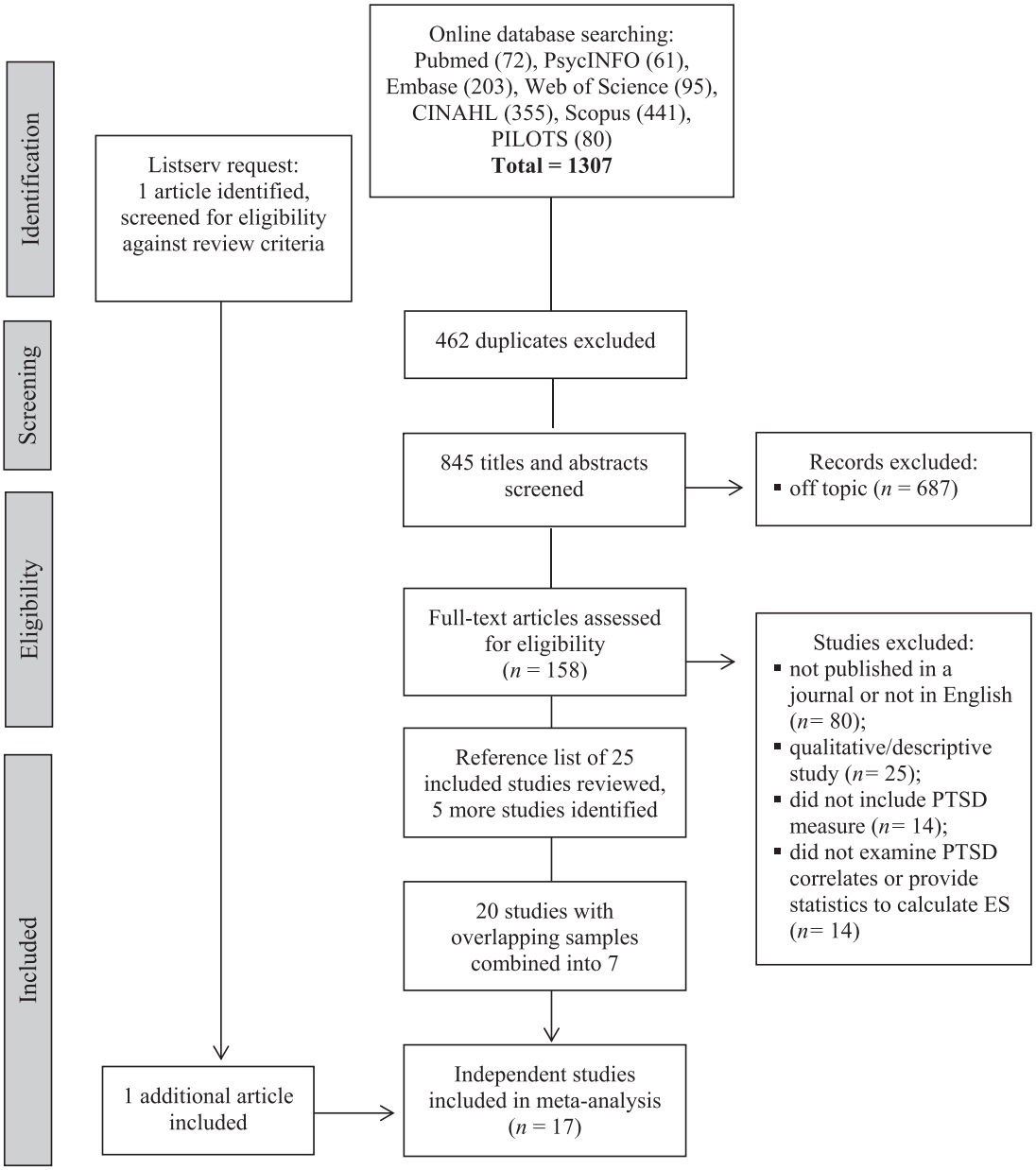

Figure 1 PRISMA flowchart of study selection process.

Effect sizes were interpreted as per Cohen's guidelines,[56] with correlations of 0.10, 0.30 and 0.50 representing small, medium and large associations, respectively.

The calculation of effect sizes was a multistage process. First, an individual *r* was obtained for each relationship between a standardised measure of PTSD and risk factor. Second, if a study provided multiple-effect sizes for a single risk factor (for example, several PTSD measures), an average *r* was calculated. This involved transforming individual *r*s to Fisher's *Z* (so that a normal distribution could be provided), computing the average Fisher's *Z* and then back-transforming this average to *r*.[36] This ensured that each study only contributed a single-effect estimate to the pooled *r* for any given risk factor.[36] Third, effect sizes from different studies that assessed the same risk factor were grouped by PTSD measure and then pooled. The only exception to this was social support, where individual effect estimates were considered for this multidimensional risk factor. Similarly, effect estimates for studies that examined acute versus chronic PTSD were separately examined. As the reliability of individual effect sizes is somewhat dependent on their underlying sample size (that is, effect sizes from larger samples embody less sampling error and, therefore, are more precise in their estimate), study *r*s were weighted by their inverse variance before being pooled (mean r_w).[36] Weighting was based on a random-effects model.[57]

To determine the accuracy of individual and weighted effect sizes, 95% CIs and exact *P*-values were calculated. Effect sizes were considered statistically significant if the CI did not include the value of zero and the associated *P*-value was >0.05.[58] In addition, fail-safe Ns (N_{fs}) were calculated to address a potential validity threat—publication bias.[59] N_{fs} represents the hypothetical number of unpublished or unidentified studies reporting no effect (that is, no relationship) required to render a calculated effect size as meaningless (that is, $r<0.1$). The higher the N_{fs} value, the more confidence we can have in the result. A conservative approach was adopted in this meta-analysis, with an N_{fs} value needing to exceed the number of studies contributing to an effect-size estimate (that is, $N_{fs}>N_{studies}$) to be considered robust.

The I^2 statistic was calculated to assess the degree of consistency in pooled effect-size estimates.[60] The value of I^2 represents the percentage of between-studies variance that can be accounted for by actual differences as opposed to

chance. I^2 values >50% suggest moderate-to-substantial heterogeneity across individual effect-size estimates, potentially due to methodological variation in addition to clinical heterogeneity that characterises research with medical populations.[25] Importantly, calculation of the I^2 statistic is not dependent on the number of studies included in a meta-analysis.[60]

The moderating effects of sample (veterans versus civilians) and measurement characteristics (self-report versus clinician rating) were additionally evaluated for those risk factors associated with statistical heterogeneity ($I^2 \geqslant 50\%$).[60] Specifically, effect estimates for subsets of studies that investigated the same risk factor (for example, SCI level) were combined, once again using Fisher's Z_r transformation and back transformation. Group mean differences were then examined using the *Q*-test of homogeneity and a mixed-effects model (which assumes some within-group variation of true effect estimates).[57] These analyses were conducted with the Comprehensive Meta-Analysis Software (Version 3, 2014 Biostat, Englewood, NJ, USA).

In combination, these statistics were used to assess the clinical relevance of each potential PTSD risk factor for individuals with an acquired SCI. Specifically, a risk factor was considered an important predictor of PTSD

Table 1 Study characteristics ($N_{studies} = 17$)

Lead author	*Country*	*N (male: female)*	*Mean age (s.d.)*	*Mean time since injury (s.d.)*	*Injury cause*	*Recruitment source*	*PTSD measure*
Agar[12]	UK	50 (43:7)	38.9 (13.42)	3–24 months post	Traumatic	Inpatient	PDS; IES
Boyer[37,38]	USA	21 (9:12)	—	5.9 years (4.4)	Traumatic and non-traumatic	Inpatient and outpatient	PDS; CPSS
Chung[13,39,40]	Greece	62 (43:19)	45.08 (13.97)	1.7 years (3.46)	Traumatic and non-traumatic	Inpatient	PCL
Hatcher[4]	UK	102 (83:19)	46.66 (10.98)	15.2 years (11.76)	non-traumatic	Inpatient and outpatient	IES
Kennedy[1,41,42]	UK	85 (68:17)	32.6 (—)	6–24 weeks post	Traumatic	Inpatient	IES
Krause[65]	USA	927 (662:265)	—	22.5 years (10.7)	Traumatic	Outpatient	PPTSD-R
Livneh[5,45]	USA	95 (68:27)	47.5 (17.61)	7.5 years (6.8)	Traumatic and non-traumatic	Outpatient	PPTSD-R
Martz[14,43,44]	USA	312 (271:42)	50.7 (14.8)	14.1 years(13.0)	Traumatic and non-traumatic	Outpatient	PPTSD-R
Migliorini[6]	Australia	443 (346:97)	51.78 (14.44)	19.2years (13.27)	non-traumatic	Outpatient and community	IES-R
Moodley[61]	South Africa	112 (72:40)	29.54 (9.65)	Up to 1 month post	Traumatic	Inpatient	IES-R; PDS
Mona[63]	USA	195 (109:86)	37.17 (6.74)	12.2 years (6.43)	Traumatic	Outpatient	PDS
Nielsen[10,15]	Denmark	69 (—) 168 (125:43)	48 (16) 42.7 (12.5)	0.2 years (0.18) 14.0 years (10.1)	Traumatic and non-traumatic	Inpatient, outpatient and community	HTQ
Otis[3]	Canada	71 (56:15)	41.06 (12.27)	12.1 years (11.52)	Traumatic	Outpatient	SCID
Radnitz[8,17,21,46,47] Danner[9]	USA	125 (124:1)	49.51 (13.8)	18.8 years(13.11)	Traumatic	Inpatient, outpatient	IES; CAPS; SCID
Schonenberg[24]	Germany	102 (86:16)	41.31 (12.6)	3.7 years (1.79)	Traumatic	Outpatient	IES-R
Ullrich[64]	USA	87 (86:1)	27.2 (6.9)	—	Traumatic	Inpatient	PC-PTSD
Warren[11]	USA	23 (13:10)	34.5 (14.4)	14.3 days (18.4)	Traumatic	Inpatient	PC-PTSD

Measure abbreviations: CAPS Clinician Administered PTSD Scale; CPSS Child PTSD Symptom Scale; IES/IES-R Impact of Events Scale (Revised); HTQ Harvard Trauma Questionnaire; PC-PTSD Primary Care PTSD screen; PDS Posttraumatic Diagnostic Scale; PCL Posttraumatic Stress Disorder Checklist; PPTSD-R Purdue Posttraumatic Stress Disorder-Revised; SCID Structured Clinical Interview for DSM-IV/DSM-III.

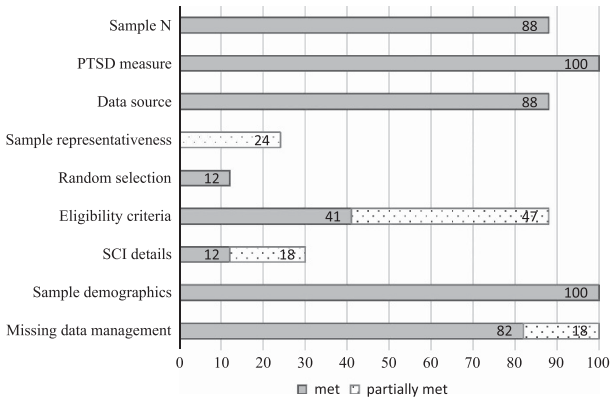

Figure 2 Risk of bias assessment.

symptomatology if it was associated with a moderate r ($r>0.10$) that was statistically significant (95% CIs≠0, $P<0.05$) and an N_{fs} score that was greater than the number of studies, which contributed to the pooled effect size.

RESULTS

Study characteristics

Data from 17 studies were included in this meta-analysis. This included several studies from the United States ($N_{studies}=8$) and Europe ($N_{studies}=6$), with single studies from Australia, Canada and South Africa also contributing (see Table 1). Most utilised a cross-sectional survey design, with Moodley and Pillay[61] repeating PTSD measurements over a 4-week inpatient timeframe. Despite the broad publication range (1998–2016), the majority of PTSD measures corresponded with DSM-IV criteria: the two exceptions being the Impact of Event Scale and Clinician Administered PTSD Scale (CAPS) for DSM-III-R. All studies utilised self-report screening tools of PTSD severity. Single studies supplemented this information with full DSM criteria based on the CAPS or Structured Clinical Interview for DSM Axis I Disorders,[2,9,18] although they reported findings as a continuous measure of overall PTSD symptom severity rather than as a dichotomous diagnosis.

Risk of bias assessment

As seen in Figure 2, most studies provided sufficient information relating to study design and conduct to detect potential sources of bias. Aside from two pilot studies,[11,37] most were also sufficiently powered ($N=26$, power at 0.80, $\alpha=0.05$, $r=0.50$;[62]). The requirement that included studies utilise robust PTSD tools and primary data sources also minimised measurement error. However, the majority relied on a single recruitment source (for example, single rehabilitation unit), as opposed to broad recruitment strategies (for example, electronic advertising, postal mail and so on), which may limit the degree to which the findings can be generalised to the broader SCI population. Chung *et al.*[13,39,40] and Mona *et al.*[63] incorporated some form of random selection for their recruitment (for example, computer-generated sampling). Inclusion and exclusion criteria were routinely reported, with potential participants with preexisting medical conditions (for example, stroke, psychosis) or associated head trauma excluded. Critical sample character parameters (that is, age, gender) were provided by all studies, although few referred to international standards (ASIA Impairment Scale, Frankel Scale) for the neurological classification of SCI. Finally, missing data were at least partially explained by all studies (that is, by providing $N_{participants}$ per measure or N incomplete data), thereby minimising the risk of potential attrition bias.

Participant characteristics

The total pooled sample comprised 2980 individuals with an acquired SCI (see Table 2), most assessed as adults. Boyer's family study[37,38] exclusively focussed on paediatric SCI. The male-to-female ratio of 3:1 is consistent with reported SCI prevalence data,[7] with individuals primarily injured in motor vehicle crashes. Eleven studies exclusively recruited those with a traumatic injury onset with three independent studies (Radnitz *et al.*[8,9,17,21,46,47]; Martz *et al.*[14,43]; Ullrich *et al.*[64]) examining PTSD associations in veteran groups. Two studies screened for PTSD in the acute phase of a SCI trauma.[11,61]

Effect-size estimates

Individual and pooled *r*s for 18 PTSD risk factors are listed in Table 3 and rank ordered by size. The weighted effect estimates for five factors, all post-injury variables, can be considered clinically important in accordance with the following criteria adopted for this review (that is, $r>0.10$; $N_{fs}>N$; CIs≠0, $P<0.05$): depression comorbidity, posttraumatic cognitions, psychological distress, anxiety, and pain level.

Pre-trauma factors

Single studies identified a strong correlation between prior history variables and PTSD symptomatology. The experience of psychiatric illness (defined as a single-item question on history of clinically diagnosed affective, anxiety, substance abuse or psychotic disorders) was a positive risk factor for patients reporting distress in the acute trauma setting.[11] In the longer term, those who had experienced an adverse life event[2] prior to injury were at greater risk of developing PTSD (Table 3). However, pooled estimates for these variables did not reach significance. A fairly consistent pattern of gendered differences in symptom severity was reported, with women reporting more severe symptomatology, both in the acute and chronic stages of SCI.[3,12,41] Nonsignificant associations were found for the remaining sociodemographic variables.

Peri-trauma factors

Pooled data revealed a small-to-medium association between SCI-related factors and PTSD aetiology, although the direction of effects varied (Table 3). Adults with a complete lesion reported significantly more symptoms of hyperarousal, flashbacks and avoidance.[2,12,41] Although Nielsen *et al.*[10] reported a similar finding among their sample of inpatients, they identified injury completeness as a key variable, which mitigated the development of PTSD in a larger-scale study.[15] Other sociomedical indices—alcohol or substance use at the time of injury, age at injury, loss of consciousness and injury type—did not correlate significantly with PTSD, although these findings were based on limited data.[2,11,15,17] Notably, SCI type was associated with a broad range of effect-size estimates: adults with paraplegia reported heightened distress ($r=-0.37$ (95% CI, −0.51 to −0.21), $P<0.01$),[8] whereas a higher percentage of individuals with tetraplegia reported a moderate level of acute stress symptoms.[61] In addition, avoidance symptoms were common among children and adolescents with tetraplegic injuries ($r=0.51$, (95% CI, 0.10–0.77), $P=0.02$),[37] although this finding may be confounded by behavioural changes associated with reduced physical capabilities in the younger cohort.[37,38]

Table 2 Sample characteristics ($N_{participants} = 2980$)

Variable	$N_{studies}$	$N_{participants}$ *(%)*	*Mean (s.d.)*	*Range*
Sample size	17	2980 (100)	175.3 (221.1)	21–927
Age at study recruitment (years)	15	2032 (68)	41.0 (7.7)	27–52
Age at the time of injury (years)	9	2037 (68)	29.6 (8.4)	12–39
Time since injury (years)	11	2646 (89)	12.8 (6.4)	0.02–22.5
Education				
Years	6	937 (31)	11.5 (2.7)	6–13.6
Gender				
Male	17	2270 (76)		
Female	17	709 (24)		
Marital status				
Single/widowed	11	810 (27)		
Married/partnered	11	765 (26)		
Employment status				
Employed	3	144 (5)		
Unemployed	3	164 (6)		
Type/level of SCI				
Tetraplegia	14	899 (30)		
Paraplegia	14	727 (24)		
Completeness of injury				
Complete	12	878 (29)		
Incomplete	12	841 (28)		
Nature of injury				
Traumatic	13	2427 (81)		
Nontraumatic	2	62 (2)		
Injury cause				
Motor vehicle accident	15	1167 (39)		
Fall	13	367 (12)		
Sports related	10	249 (8)		
Violent	10	279 (9)		
Other	14	294 (10)		

Abbreviation: SCI, spinal cord injury.
$N_{studies}$ = number of studies providing data; $N_{participants}$ = number of participants providing this data.

Post-trauma factors

Medium-to-large effect-size estimates were noted by several studies that evaluated the cross-sectional association between PTSD and SCI sequelae. Indices of psychological adjustment were strongly related: those who experienced negative emotions and cognitions related to their SCI (that is, a reflection of lower levels of acceptance/adjustment) reported severe PTSD symptoms (Table 3). Although depression, as a construct, was characterised by between-study heterogeneity, potentially reflecting its multidimensional nature (that is, as a feature of general psychological distress[6] or a case-finding instrument[65]), the pooled weighted effect estimate ($r > 0.50$) was highly significant given the large N involved. Those reporting daily or severe neuropathic pain also experienced intense psychological distress.[2,43] There was also a trend for those in the acute stages of SCI rehabilitation to report subtle PTSD symptoms, although this finding was characterised by publication bias.

Seven independent studies examined the relative contribution of perceived and actual support (Table 3). Although medium-to-large effect estimates ($r > 0.30$; Table 3) for perceived satisfaction were noted—those who expressed greater satisfaction with the emotional and instrumental support provided by close family and friends endorsed fewer PTSD symptoms—this finding was primarily based on single studies. Similarly, Chung *et al.*[13] identified a strong relationship between the ability to carry out social functions and hyperarousal ($r = 0.49$ (95% CI, 0.27–0.66) $P < 0.01$), re-experiencing ($r = 0.44$ (95% CI, 0.21–0.62) $P < 0.01$) and avoidance ($r = 0.41$ (95% CI, 0.18–0.60) $P < 0.01$) symptoms. In comparison, well-being was not significantly associated with the more support that was received[2,3,9,12] nor attempts to actively seek support.[45] In combination, these findings highlight the empirical distinctness of perceived social support from social network size.

Moderator analyses

Analysis by measure and sample type was conducted for the following three risk factors: SCI severity, age at injury and SCI level. This yielded significant results for one risk factor only: veterans with paraplegia were at risk for PTSD (Q_B (1) = 18.25, $P < 0.01$; veterans $r = -0.37$ (95% CI, −0.57 to −0.21) $P < 0.01$; civilians $r = 0.10$ (95% CI, −0.04 to 0.24) $P = 0.15$). However, given that this subgroup analysis was based on a limited dataset[9,17,43], any between-studies variance identified is likely to have poor precision.[57] Group equivalence could not be compared for depression, as a risk factor, as measurement and sample characteristics did not vary between studies.

DISCUSSION

This meta-analytic review examined potential risk factors associated with PTSD development following an acquired SCI. The combined findings help refine current understanding of the aetiology of PTSD following SCI, with postinjury adjustment issues likely to increase an individual's vulnerability to distress. The summary evidence also helps to profile types of SCI studies examining PTSD and aspects needing further enquiry.

In contrast to previous reviews of trauma-exposed adults, pooled effect estimates suggest that preexisting socioenvironmental factors—namely, prior trauma exposure,[28,66] higher education[31] and spousal support[67]—did not significantly influence risk of PTSD development, at least for the SCI sample examined in this review. Notably, the number of studies involved in these analyses was limited in quantity, underlining the need for further research before drawing any firm conclusions on pretrauma risk factors. This includes the potential contribution of age: the younger the age of trauma exposure, the more potent its effect on future PTSD psychopathology.[68] The impact of trauma severity (that is, trauma uniqueness, stressfulness) and type on PTSD development also requires further examination.[17,31,33] Within the civilian SCI population, however, these relationships have not been identified. To help inform a diathesis-stress formulation of PTSD, future SCI studies might also consider examining concurrent influences of biological diatheses. Emerging evidence suggest that cognitive impairment, in particular, can impede an individual's ability to process and manage a traumatic experience such as SCI.[62,69] Notably, loss or impairment of consciousness at the time of injury, which may effect subsequent cognitive and psychological functioning,[70] was not consistently associated with PTSD symptom severity in this review.

Table 3 Risk factors for post-traumatic stress listed by individual study

Pre-injury	*Lead author*	*SCI stage*	*Measure*	$N_{studies}$	$N_{participants}$	r	r_w	*95% CI*		P	N_{fs}	I^2
								Lower	*Upper*			
Psychiatric history	Warren (2016)	Acute	PC-PTSD	1	23	0.48[a]		0.23	0.79	0.00	4	
	Agar (2006)[b]	Chronic	PDS	1	50	0.23		–0.06	0.47	0.12	1	
	Otis (2012)	Chronic	SCID	1	71	0.05		–0.19	0.28	0.68	0	
	Agar (2006)[b]	Chronic	IES	1	50	0.03		–0.25	0.31	0.84	0	
			Total	3	144		0.18	–0.06	0.39	0.13	2	44.72
Previous trauma (no. of)	Otis (2012)	Chronic	SCID	1	71	0.29[a]		0.06	0.49	0.01	2	
	Chung (2006)	Chronic	PCL	1	62	0.17		–0.08	0.40	0.19	1	
	Livneh (2011)	Chronic	PPTSD-R	1	95	0.08		–0.12	0.28	0.44	0	
			Total	3	228		0.17	0.04	0.29	0.01	2	0.00
Gender (female)	Agar (2006),[b] Hatcher (2009), Kennedy (2001)	Chronic	IES	3	237		0.26[a]	0.13	0.37	0.00	5	0.00
	Moodley (2013)	Acute	PDS	1	107	0.21		0.02	0.38	0.03	1	
	Otis (2012)	Chronic	SCID	1	71	0.15		–0.09	0.37	0.21	1	
	Warren (2015)	Acute	PC-PTSD	1	23	0.14		–0.29	0.52	0.54	0	
	Agar (2006),[b] Mona (2000)	Chronic	PDS	2	245		0.10	–0.03	0.22	0.13	0	0.00
	Livneh (2014), Martz (2005)	Chronic	PPTSD-R	2	407		0.08	–0.02	0.18	0.04	0	0.00
	Nielsen (2003b)	Chronic	HTQ	1	168	0.07		–0.08	0.22	0.37	0	
			Total	10	1213		0.14	0.09	0.19	0.00	4	0.00
Relationship status (married)	Warren (2016)	Acute	PC-PTSD	1	23	0.29		–0.13	0.62	0.17	2	
	Nielsen (2003b)	Chronic	HTQ	1	168	0.19		–0.04	–0.33	0.01	1	
	Danner (2000)[b]	Chronic	CAPS (current)	1	96	0.14		–0.10	0.30	0.33	0	
	Danner (2000)[b]	Chronic	IES	1	96	0.04		–0.16	0.24	0.70	0	
	Otis (2012)	Chronic	SCID	1	71	0.02		–0.21	0.25	0.87	0	
			Total	4	358		0.13	0.03	0.23	0.01	1	0.00
Education level	Livneh (2014)	Chronic	PPSTD-R	1	95	0.18		–0.02	0.37	0.08	1	
	Warren (2016)	Acute	PC-PTSD	1	23	0.12		–0.30	0.50	0.59	0	
	Danner (2000)[b]	Chronic	CAPS (current)	1	96	0.11		–0.09	0.30	0.29	0	
	Danner (2000)[b]	Chronic	IES	1	96	0.07		–0.13	0.27	0.50	0	
	Otis (2012)	Chronic	SCID	1	71	0.01		–0.14	0.33	0.40	0	
	Nielsen (2003b)	Chronic	HTQ	1	168	0.07		–0.08	0.22	0.37	0	
			Total	5	453		0.10	0.01	0.19	0.03	0	0.00
Age (current)	Hatcher (2009), Kennedy (2001)	Chronic	IES	2	393		–0.18	–0.38	0.04	0.12	2	57.68
	Livneh (2014)	Chronic	PPTSD-R	1	95	–0.18		–0.37	0.02	0.08	1	
	Chung (2006)	Chronic	PCL	1	62	0.09		–0.22	0.39	0.49	0	
	Warren (2015)	Acute	PC-PTSD	1	23	0.09		–0.33	0.48	0.69	0	
	Nielson (2003b)	Chronic	HTQ	1	168	–0.06		–0.21	0.09	0.44	0	
			Total	6	536		–0.10	–0.21	0.01	0.07	0	29.55
Peri-injury												
SCI severity (completeness)	Otis (2012)	Chronic	SCID	1	71	0.29[a]		0.06	0.49	0.01	2	
	Agar (2006),[b] Kennedy (2001)	Chronic	IES	1	135	0.28[a]		0.00	0.53	0.05	2	
	Nielsen (2003b)	Chronic	HTQ	1	168	–0.17		–0.31	–0.02	0.03	1	
	Agar (2006),[b] Mona (2000)	Chronic	PDS	1	245	0.16		–0.13	0.42	0.28	1	
			Total	5	569		0.12	–0.06	0.29	0.21	1	77.39
Alcohol/substance use	Warren (2016)	Acute	PC-PTSD	1	24	0.08		–0.33	0.47	0.71	0	
	Radnitz (1998)[b]	Chronic	IES	1	125	0.07		–0.11	0.24	0.44	0	
	Radnitz (1998)[b]	Chronic	CAPS (current)	1	125	0.02		–0.16	0.19	0.83	0	
			Total	2	149		0.05	–0.21	0.10	0.52	1	0.00

Table 3 (Continued)

Peri-injury												
Age (at injury)	Hatcher (2009)	Chronic	IES	1	102	0.18		–0.02	0.36	0.01	1	
	Otis (2012)	Chronic	SCID	1	71	0.08		–0.16	0.31	0.51	0	
	Mona (2000)	Chronic	PDS	1	195	–0.07		–0.21	0.07	0.33	0	
			Total	3	368		0.05	–0.11	0.20	0.54	2	54.35
Loss of conciousness	Otis (2012)	Chronic	SCID	1	71	–0.18		–0.39	0.06	0.13	1	
	Warren	Acute	PC-PTSD	1	24	–0.13		–0.49	0.28	0.56	0	
	Radnitz (1998)[b]	Chronic	IES	1	125	0.11		–0.07	0.28	0.22	0	
	Radnitz (1998)[b]	Chronic	CAPS (current)	1	125	0.09		–0.09	0.26	0.32	0	
	Nielsen (2003b)	Chronic	HTQ	1	168	–0.07		–0.21	0.08	0.37	0	
			Total	4			–0.04	–0.17	0.08	0.49	2	26.58
SCI level												
	Radnitz (1988)[b]	Chronic	CAPS (current)	1	125	–0.36[a]		–0.50	–0.19	0.00	3	
	Radnitz (1998),[b] Hatcher (2009)	Chronic	IES	2	227		–0.29[a]	–0.45	–0.10	0.00	4	
	Moodley (2013)	Acute	PDS	1	107	0.29[a]		0.11	0.45	0.00	2	
	Mona (2000), Boyer (2000)	Chronic	PDS	2	215		0.22	–0.11	0.50	0.18	2	47.98
	Otis (2012)	Chronic	SCID	1	71	0.14		–0.10	0.36	0.25	0	
	Nielsen (2003a)	Chronic	HTQ	1	69	0.10		–0.20	0.39	0.52	0	
	Chung (2006)	Chronic	PCL	1	62	–0.02		–0.26	0.24	0.92	0	
			Total	7	756		–0.04	–0.14	0.22	0.66	4	82.42
Post-injury												
Depression	Livneh (2011)	Chronic	PPTSD-R/CES-D	1	95	0.76[a]		0.66	0.83	0.00	7	
	Krause (2010)	Chronic	PPTSD-R/PHQ	1	927	0.74[a]		0.71	0.77	0.00	6	
	Schonenberg (2012)	Chronic	IES-R/CES-D	1	102	0.60[a]		0.46	0.77	0.00	5	
	Migliorini (2008)	Chronic	IES-R/DASS21	1	443	0.58[a]		0.52	0.63	0.00	5	
	Kennedy (2001)	Chronic	IES/BDI	1	85	0.57[a]		0.41	0.70	0.00	5	
	Chung (2006)	Chronic	PCL/GHQ	1	62	0.49[a]		0.27	0.66	0.00	4	
			Total	6	1714		0.64 [a]	0.54	0.72	0.00	32	87.02
Posttraumatic cognitions	Agar (2006)[b]	Chronic	PDS/PTCI	1	50	0.65[a]		0.45	0.79	0.00	6	
	Hatcher (2009), Agar (2006)[b]	Chronic	IES/PTCI	2	152	0.59[a]		0.42	0.72	0.00	10	45.18
			Total	2	152		0.63 [a]	0.52	0.72	0.00	11	0.00
Psychological distress	Nielsen (2003a)	Chronic	HTQ/MEDS	1	69	0.53[a]		0.34	0.68	0.00	4	
	Migliorini (2008)	Chronic	IES/DASS21	1	443	0.57[a]		0.50	0.63	0.00	5	
			Total	2	512		0.57 [a]	0.50	0.62	0.00	10	0.00
Anxiety	Kennedy (2001)	Chronic	IES/STAI	1	85	0.53[a]		0.36	0.67	0.00	4	
	Migliorini (2008)	Chronic	IES-R/DASS21	1	443	0.55[a]		0.49	0.62	0.00	5	
	Chung (2006)	Chronic	PCL/GHQ	1	62	0.51[a]		0.29	0.67	0.00	4	
			Total	3	590		0.56 [a]	0.49	0.61	0.00	14	0.00
Pain (pain severity)	Martz (2005)	Chronic	PPTSD-R/MPQ	1	312	0.38[a]		0.28	0.47	0.00	3	
	Otis (2012)	Chronic	SCID	1	71	0.38[a]		0.16	0.56	0.00	3	
	Warren (2016), Ullrich (2013)	Acute	PC-PTSD	2	110	0.23[a]		0.05	0.41	0.00	3	0.00
			Total	4	493		0.35 [a]	0.27	0.43	0.00	10	0.00
Time since SCI	Otis (2012)	Chronic	SCID	1	71	–0.08		–0.31	0.16	0.51	0	
	Radnitz (1998)[b]	Chronic	CAPS (current)	1	125	–0.08		–0.25	0.09	0.37	0	
	Radnitz (1998),[b] Hatcher (2009)	Chronic	IES	2	227		–0.22	–0.35	–0.07	0.00	2	0.00
	Livneh (2011)	Chronic	PPTSD-R	1	95	–0.21		–0.35	–0.01	0.04	1	
	Mona (2000)	Chronic	PDS	1	195	–0.01		–0.15	0.13	0.89	0	
	Chung (2006)	Chronic	PCL	1	62	–0.01		–0.26	0.24	0.92	0	
	Nielsen (2003)	Chronic	HTQ	1	168	–0.16		–0.30	–0.01	0.04	1	
			Total	7	716		–0.12	–0.19	–0.05	0.00	1	0.29

Table 3 (Continued)

Risk factor		*Lead author (date)*	*SCI stage*	*Measure(s)*	$N_{studies}$	$N_{participants}$	r	r_w	*95% CI*		P	N_{fs}	I^2
									Lower	*Upper*			
Social support	Satisfaction	Nielson (2003b)	Chronic	HTQ/CSS	1	168	–0.48[a]		–0.59	–0.35	0.00	4	
		Agar (2006)	Chronic	PDS/PSS	1	50	–0.39[a]		–0.60	–0.12	0.01	3	
		Agar (2006)	Chronic	IES/PSS	1	50	–0.36[a]		–0.58	–0.09	0.01	3	
	Social dysfunction	Chung (2006)	Chronic	PCL/GHQ	1	62	0.45[a]		0.23	0.63	0.00	4	
	Perceived support (quantity)	Otis (2012)	Chronic	PDEQ/PNSBS	1	71	–0.24		–0.45	0.00	0.04	1	
		Hatcher (2009)	Chronic	IES/PSS	2	102		–0.21	–0.33	–0.08	0.00	2	0.00
		Danner (2000)											
		Danner (2000)	Chronic	CAPS/PSS	1	124	–0.15					1	
		Agar (2006)	Chronic	PDS/SSQ	1	50	–0.04		–0.31	0.25	0.81	0	
		Agar (2006)	Chronic	IES/SSQ	1	50	–0.02		–0.26	0.29	0.91	0	
	Seeking support	Livneh (2011)	Chronic	PPTSD-R/COPE	1	95	–0.12		–0.31	0.08	0.25	0	

Note: $N_{studies}$ = number of studies providing data; $N_{participants}$ = number of participants providing this data; r_w = weighted mean correlation; 95% CI = confidence interval with lower (L) and upper (U) limits; *Nfs* = Fail-safe *N*; I_2 = heterogeneity index.
Measure abbreviations: BDI Beck Depression Inventory; CES-D; CSS Crisis Support Scale; COPE, COPE Inventory; DASS-21 DASS21, Depression Anxiety Stress Scales-21 item; GHQ General Health Questionnaire; MPQ McGill Pain Questionnaire; MEDS, Medical-Based Emotional Distress; PHQ Patient Health Questionnaire; PNSBS Perceived Negative Spouse Behaviors Scale; PSS Perceived Social Support Scale; PTCI Post-traumatic cognitions inventory; SSQ Short Form Social Support Questionnaire; STAI StateTrait Anxiety Inventory.
[a]significant effect size: $r>0.10$, CI≠0, *Nfs*> $N_{studies}$, $P<0.05$, *Nfs*>*N*studies, [b]studies providing more than one effect per factor were averaged for overall effect size calculation.

Although a sizeable group of SCI studies have examined the contribution of post-SCI factors in increasing or decreasing the risk of developing PTSD, there remains little available data relating to the core trauma that may trigger PTSD. Recent studies suggest that factors related to the acute hospital experience (for example, exposure to an intensive care unit, sedation level, intubation and reintubation and length of stay) contribute to a unique constellation of PTSD symptoms, including sleep problems.[71–73] Notably, the finding that psychological risk factors (that is, negative affect, general distress, depression and anxiety) were critical to PTSD was consistent across the examined studies. Depression and anxiety are known to mediate stress reactions in the trauma population.[74,75] However, it remains unclear whether these mental health problems were preexisting or co-occurring in response to SCI as contributing studies did not routinely examine pre-morbid psychiatric illness. Symptom overlap between PTSD and depression also warrant consideration, with arousal (for example, sleep problems) and avoidance (for example, loss of interest in usual activities) being characteristic of both.[76] Moreover, cognitive responses to trauma such as dissociation reactions, self-blame and negative cognitions of the world, although well established in the general PTSD literature,[77] require further investigation with the SCI cohort.

That PTSD severity is enhanced by pain is consistent with current theory that pain sensations serve as a constant reminder of trauma, triggering PTSD re-experiencing symptoms.[7] It is thought that pain contributes to the development and maintenance of PTSD.[78] The importance of familial relationships in maintaining mental health was also highlighted in this review.[15] Balanced and nurturing personal relationships are particularly important for young injured persons as they help to foster encouragement, expectations and independence, helping to buffer distress associated with SCI as a traumatic event.[37,38] In comparison, family instability (that is, history of mental health) may increase the risk of PTSD.[37,38] Notably, SCI studies have not routinely considered how differences in the structure and function of perceived supports (for example, informational, tangible or emotional supports) may have different mental health implications.

Clinical implications

The combined findings, which suggest that PTSD following SCI involves a complex mix of life experiences, inherited mental health risks and coping capabilities, underscore the importance of efficacious screening tools targeted to this patient cohort. One promising tool is the 19-item Spinal Cord Injury—Quality of Life Psychological Trauma item bank.[79] Another emerging measure is the DSM-5 Cross-Cutting Symptoms Measure, designed to assess the presence and severity of co-occurring psychological disorders or symptoms,[80] although the feasibility of this DSM-5 measure in SCI practice and research is still to be determined.

Multi-domain interventions in the acute stages of SCI rehabilitation can prevent subsequent psychopathology for individuals identified as having high-stress diathesis.[81] This might include cognitive-behavioural programmes,[82,83] which have shown promise as an approach for managing adjustment problems, such as depression and anxiety. Psychoeducation is another critical therapy component for individuals at risk of developing PTSD. Even basic information about trauma-related symptoms can help to reduce misconceptions of mental illness and influence psychological recovery in a positive way.[84] This informational support can be supplemented with social support networking skills. This includes capitalising on the quality of support within existing social networks in addition to fostering new connections for those who are socially isolated.[2,65,85] Rehabilitation strategies that reinforce injury health behaviours, including coping self-statements and proactive strategies for managing chronic pain, can also help protect against trauma-induced psychopathology.[2]

Study limitations

The present findings need to be considered in the context of a number of important limitations. First, the search criteria may have failed to capture all relevant studies. In an attempt to minimise this limitation, multiple search strategies (for example, electronic database searches, manual search of reference lists of reviews, contacting experts in the topic) were adopted. Further, N_{fs} statistics were calculated. It is acknowledged, however, that this statistic does not fully alleviate the problem of publication bias.[59]

Second, the tendency for SCI studies to use overlapping data (for example, the same original patient sample, or secondary analyses of data that cover the same outcomes or overlapping subsets of them) presents statistical difficulties for meta-analysis methods.[36] Importantly, through a careful search of similar studies, data independence was ensured. Although this resulted in a reduced sample, none of the observations overlapped, and no single study provided a disproportionate amount of data to the calculation of a pooled effect size *r*. To ensure transparency of data, it is nonetheless important for SCI substudies to cite any primary publication.

Third, the majority of the data were self-reports, which are prone to recall, confounding and sample selection biases.[86] Notably, individual effect sizes did not markedly differ across individual PTSD measures. This included similar effect estimates produced with the original and revised Impact of Event Scale, despite the former's two symptom (that is, intrusion, avoidance) cluster conceptualisation of PTSD, which no longer matches DSM diagnostic criteria.[76] Nonetheless, the current findings need to be interpreted with caution given the reliance on earlier DSM versions. The current DSM-5 incorporates a four-symptom model that considers alterations in mood and cognitions (for example, negative beliefs, negative emotional state) and may enable more accurate diagnosis of PTSD in the SCI cohort.[87] Indeed, cognitive and affective symptoms are just as critical to the experience of PTSD, following SCI, as the experience of physiological (arousal) symptoms.[12]

Fourth, as the majority of studies were conducted in Western countries, the extrapolation of findings may be limited. Although the diagnosis of PTSD is cross-culturally valid, it is acknowledged in the DSM-5 that expression of PTSD and symptom comorbidities may differ across cultures.[88] Fifth, the cross-sectional and retrospective nature means that the examined variables were, for the most part, assessed at a single time point and may be confounded by prior symptoms and functioning.[89] There is also evidence that traumatic symptoms are dynamic and can vary in intensity over time.[2,90] Indeed, recent data suggest that demographic (for example, less years of education, not partnered) and injury variables (for example, lesion completeness) correctly predict the likelihood of developing a mental disorder up to 6 months after discharge.[2] In order to determine the temporality between PTSD risk factors for those with SCI, longitudinal studies driven by articulated biopsychosocial models of causation—such as the diathesis-stress model—are needed. Attempts to minimise potential sources of bias in sampling, selection, measurement and data analysis will also help to ensure that the results are generalisable and translational.[90]

CONCLUSION

This meta-analytic review exposed significant relationships between several postinjury risk factors and PTSD symptoms following SCI. The suggestion is that attention needs to be given to the role of personal variables in the identification and management of posttraumatic stress symptoms in this cohort. Issues of further study were also identified, including the need to examine sociocontextual and biological factors that may contribute to high-stress diathesis.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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APPENDIX

Logic grids and boolean operators for electronic database searches

	PTSD	SCI
CINAHL	Stress Disorders, Post-Traumatic[mh] OR PTSD[tw] OR Post-Traumatic stress disorder[tw] OR Post-traumatic stress[tw] OR Post-traumatic stress[tw] OR post-traumatic psychos*[tw] OR post-traumatic syndrome[tw] OR post-traumatic neuros*[tw] OR post-traumatic psychos*[tw] OR post-traumatic syndrome[tw] OR post-traumatic neuros*[tw] OR traumatic stress[tiab] OR war neuros*[tiab] OR combat stress*[tiab] OR combat neuros*[tiab] OR shell shock [tiab]	Spinal cord injuries [mh:noexp] OR spinal injuries[mh] OR spinal injur*[tw] OR spine injur*[tw] OR spinal cord injur*[tw] OR spinal cord trauma*[tw] OR spinal trauma*[tw] OR spine trauma*[tw] OR spinal fracture*[tw] OR spine fracture* [tw] OR spinal cord fracture*[tw] OR tetrapleg*[tiab] OR quadripleg*[tiab] OR parapleg*[tiab]
EMBASE	MH 'Stress disorders, post-traumatic' OR TX 'stress disorders, post-traumatic' OR TX PTSD OR TX 'post-traumatic stress' OR TX 'post-traumatic stress' OR TI 'traumatic stress' OR AB 'traumatic stress' OR TX 'post-traumatic psychos*' OR TX 'post-traumatic syndrome' OR TX 'post-traumatic neuros' OR TX 'post--traumatic psychos' OR TX 'post-traumatic syndrome' OR TX 'post-traumatic neuros*' OR TI 'war neuros*' OR AB 'war neuros*' OR TI 'combat stress' OR AB 'combat stress' OR TI 'combat neuros*' OR AB 'combat neuros*' OR TI 'shell shock' OR AB 'shell shock'	MH 'spinal cord injuries' OR MH 'spinal injuries' OR TX 'spinal cord injur*' OR TX 'spinal fracture*' OR TX 'spine injur*' OR TX 'spinal cord trauma*' OR TX 'spinal trauma*' OR TX 'spine trauma' OR TX 'spine fracture*' OR TX 'spinal cord fracture*' TI parapleg* OR AB parapleg* OR TI quadripleg* OR AB quadripleg* OR TI tetrapleg* OR AB tetrapleg*
PSYCINFO	(post-traumatic stress disorder or traumatic neurosis or stress reactions).sh. or PTSD.tw. or post-traumatic stress.tw. or post-traumatic stress.tw. or post-traumatic stress.tw. or post-traumatic psychos*.tw. or post-traumatic syndrome.tw. or post-traumatic neuros*.tw. or post-traumatic psychos*.tw. or post-traumatic syndrome.tw. or post-traumatic neuros*.tw. or traumatic stress.ti,ab. or war neuros*.ti,ab. or combat stress.ti,ab. or combat neuros*.ti,ab. or shell shock.ti,ab.	spinal cord injuries.sh OR spinal cord injur*.mp OR spine injur*.mp OR spinal injur*.mp OR spinal cord trauma*.mp OR spinal trauma*.mp OR spine trauma*.mp OR spinal fracture*.ti,ab OR spine fracture*.ti,ab OR spinal cord fracture*.tw OR parapleg*.ti,ab OR quadripleg*.ti,ab OR tetrapleg*.ti,ab
PUBMED	'post-traumatic stress disorder'/syn OR 'post-traumatic stress' OR 'post-traumatic stress' OR 'combat neurosis' OR 'combat neuroses' OR 'shell shock' OR PTSD	'spinal cord injury'/syn OR 'spine injury'/syn OR 'spine injury' OR 'spine injuries' OR quadripleg*:ab,ti OR 'tetrapleg*':ab,ti OR parapleg*:ab,ti 'spinal cord traumas':ab,ti OR 'spinal traumas':ti,ab 'spine traumas':ti,ab OR 'spinal cord fracture':ti,ab OR 'spinal cord fractures':ti,ab OR 'spinal fracture':ti,ab OR 'spinal fractures':ti,ab OR 'spine fracture':ti,ab OR 'spine fractures':ti,ab
SCOPUS	((ALL ('post-traumatic stress disorder' OR 'post-traumatic stress' OR 'post--traumatic stress' OR 'post-traumatic stress') OR TITLE-ABS-KEY('traumatic stress' OR 'war neuros*' OR 'combat stress' OR 'combat neuros*' OR 'shell shock' OR 'post-traumatic psychos*' OR 'post-traumatic syndrome' OR 'post--traumatic neuros' OR 'post-traumatic psychos' OR 'post-traumatic syndrome' OR 'post-traumatic neuros*' OR 'combat stress' OR 'traumatic neurosis' OR 'PTSD')))	TITLE-ABS-KEY ('spinal cord injuries' OR 'spinal cord injur*' OR 'spine injur*' OR 'spinal injur*'OR 'spinal cord trauma*' OR 'spinal fracture*' OR 'spinal cord trauma*' OR 'spinal trauma*' OR 'spine trauma' OR 'spine fracture*' OR 'spinal cord fracture*' OR 'parapleg*' OR quadripleg* OR tetrapleg*)
WEB OF SCIENCE	TS = ('post-traumatic stress disorder' OR 'combat stress' OR 'traumatic neurosis' OR 'PTSD' OR 'post-traumatic stress' OR 'post-traumatic stress' OR 'post-traumatic stress' OR 'traumatic stress' OR 'war neuros*' OR 'combat stress' OR 'combat neuros*' OR 'shell shock' OR 'post-traumatic psychos*' OR 'post-traumatic syndrome' OR 'post-traumatic neuros' OR 'post-traumatic psychos' OR 'post-traumatic syndrome' OR 'post-traumatic neuros*')	TS = ('spinal cord injuries' OR 'spinal cord injur' OR 'spine injur*' OR 'spinal injur*'OR 'spinal cord trauma*' OR 'spinal trauma*' OR 'spine trauma' OR 'spinal fracture*' OR 'spine fracture*' OR 'spinal cord fracture*' OR 'parapleg*' OR quadripleg* OR tetrapleg*)