Review article

Acute stress and subsequent health outcomes: A systematic review

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A R T I C L E   I N F O

A B S T R A C T

Objective: To systematically review the relationship between acute posttraumatic stress symptoms (< 1 month) and subsequent physical and mental health outcomes other than posttraumatic stress disorder (PTSD).

Methods: A systematic search of electronic databases (PubMed, PsycINFO, CINAHL, and Web of Science) was conducted to identify longitudinal studies examining the link between acute posttraumatic stress and physical and mental health. Inclusion criteria required assessment of acute posttraumatic stress (< 1 month post-event) and at least one follow-up assessment of a physical or mental health outcome (not PTSD).

Results: 1,051 articles were screened; 22 met inclusion criteria. Fourteen studies examined physical health outcomes and 12 examined non-PTSD mental health outcomes. Early psychological responses to trauma were associated with a variety of short- (< 1 year) and long- (≥ 1 year) term physical and mental health outcomes. Physical health outcomes included poor general physical health, increased pain and disability, lower quality of life, and higher risk of all-cause mortality. Significant psychological outcomes included more cumulative psychiatric disorders, depression, and anxiety. Significant psychosocial outcomes included increased family conflict.

Conclusions: Methodologically rigorous longitudinal studies support the utility of measuring acute psychological responses to traumatic events as they may be an important marker of preventable trauma-related morbidity and mortality that warrants long-term monitoring and/or early intervention.

1. Introduction

Traumatic events can trigger a variety of psychological and physiologic responses that may impact well-being over time. Decades of research has demonstrated that activation of various "fight-or-flight" responses to a perceived threat, including increased heart rate and blood preasure, activation of the Hypothalamic-Pitutitary-Adrenal (HPA) axis, increases in cortisol and other stress hormones, and altered lipid metabolism, can have significant, long-term impacts on health [1]. Single-event traumas (e.g., earthquakes) have also been associated with immediate, life-threatening stress responses, such as heart attacks, strokes, and elevated blood pressure [2–4]. In the aftermath of these traumas, high levels of psychological distress are common [5–8], and may signal a level of physiologic arousal that helps explain the link between trauma and long-term health problems.

Immediate posttraumatic stress often abates over time; nonetheless, many trauma survivors remain at increased risk for persistent distress symptoms and psychopathology [9, 10], including mental health problems such as posttraumatic stress disorder (PTSD) [11], depression, anxiety, and global distress [12, 13]. Trauma has also been associated with long-term physical health complications in both military and civilian populations [14, 15]. For example, archival analyses of both Civil War [16] and World War II [17] combat veterans indicated increased morbidity and mortality compared to soldiers reporting little or no combat exposure. Moreover, childhood trauma and distress have been linked with numerous lifetime physical and mental health complications [14]. Finding ways to identify trauma survivors at risk for long-term physical health problems is critical for administering early secondary prevention efforts. Identifying variability in acute posttraumatic stress responses (defined by DSM-IV criteria as presenting less than one month post-event), may be critical for understanding risk for long-term trauma-related health problems.

Acute stress disorder (ASD), conceptualized in the DSM-IV as an early indicator of subsequent PTSD, is characterized by an intense emotional reaction occurring within one month of trauma exposure [18]. Although ASD may predict subsequent PTSD in some people, its utility has been debated due to low sensitivity [19]. Many people with ASD later develop PTSD, yet less than half who are ultimately diagnosed with PTSD previously met ASD criteria. Although prior reviews have criticized the utility of using ASD to predict PTSD [20], a broader perspective that examines the relationships between acute posttraumatic responses to trauma in general (within the first month) and

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subsequent physical and mental health outcomes may better capture the predictive value of acute post-trauma assessments. Measuring acute stress symptoms (AS) is another way to capture the variability in post-event responses. Indeed, prior studies have linked both ASD and acute stress symptoms to increased incidence of cardiovascular ailments over time [15], increased pain after whiplash injury [21], rehospitalizations [22], and general psychiatric problems [19], among other conditions.

At present, the mechanisms that link trauma exposure to long-term mental and physical pathology require more investigation. Research conducted using animal models suggests that physiologic reactions to an acute psychological stressor may trigger cardiovascular responses that persist over time [23]. It is plausible – and likely – that heightened posttraumatic stress responses (e.g., ASD, AS) in humans in the early aftermath of an event are an outward manifestation of an internal cascade of stress-related physiologic and psychological processes. Given that early responses to trauma may be associated with deleterious outcomes, it may be useful to examine acute posttraumatic responses more broadly as a general marker of risk for trauma-related health problems.

Prior exhaustive reviews have synthesized the relationship between acute stress and PTSD [20]. Yet, given the strong theoretical and empirical evidence that physical and mental health outcomes may be associated with acute stress, a review that focuses on critical outcomes other than PTSD is warranted. Such a review has not yet been conducted, but could help explore the utility of measuring acute stress responses in the aftermath of trauma despite the imperfect predictive power of acute stress for PTSD. In this systematic review, we summarize the research examining the link between acute posttraumatic stress responses (i.e., early PTSD, acute stress symptoms, and ASD) assessed in the early aftermath of trauma and subsequent physical and mental health outcomes other than PTSD (prior reviews have synthesized and critiqued that relationship) [20,24]. Such a synthesis could identify response patterns of individuals at risk for trauma-related health problems and inform the development of early interventions to prevent trauma-related morbidity and mortality.

To present the most empirically supported evidence, we only evaluate data from longitudinal designs, as retrospective reports of psychological symptoms often have poor validity [25]. We reviewed prospective, longitudinal studies that assessed acute posttraumatic stress (i.e., acute stress symptoms, ASD or early-PTSD) within the first 4 weeks of exposure to trauma and included at least one follow-up assessment beyond the initial month post-event. As such, this review will examine whether measuring acute posttraumatic stress in the early aftermath of a traumatic event is useful for predicting physical and mental health outcomes beyond PTSD.

2. Methods

2.1. Eligibility criteria

Using PRISMA guidelines [26], we conducted a systematic review of the literature on non-PTSD outcomes associated with acute posttraumatic stress responses in the immediate aftermath of trauma. Our review is registered with PROSPERO as study number CRD42018092912. Eligible articles were written in English in peer-reviewed journals published through April 2018. Articles must have had prospective designs with at least two points of data collection: one within the initial month of the trauma and a second occurring more than one month post-trauma. Studies must have assessed exposure to a DSM-defined traumatic event. Acute assessments included any measure of acute stress symptoms, ASD, or early PTSD; outcomes included any physical ailment or non-PTSD psychological response. Studies that measured PTSD were included only if at least one additional outcome was assessed, but PTSD-related findings are not addressed herein. Intervention studies or those examining lab-induced stressors, chronic stressors, or other types of negative life events were not eligible. We had a number of exclusion

criteria: studies examining prevalence of disorders over time (e.g., percentage of participants meeting DSM criteria for AS and depression at baseline and follow-up) but not specifically the association between acute stress responses and outcomes were excluded. Studies using retrospective reports of acute stress were also excluded. Additionally, studies that examined only PTSD as an outcome, only included one assessment, or did not assess acute stress responses within the first month were excluded. Clinical trials and intervention studies were also excluded.

2.2. Information sources and searches

Pubmed, PsycINFO, Web of Science, CINAHL, and the Cochrane Library were systematically searched for relevant abstracts. The search strategy was designed to capture three primary criteria: 1) longitudinal design, 2) acute posttraumatic stress response, and 3) follow-up assessment of a psychosocial, physical, or psychological outcome. An example of a search criteria for PubMed is: ("Acute Psychological Stress" OR "Stress Disorders, Traumatic, Acute"[Mesh] OR "acute stress disorder" OR "acute stress disorders" OR "acute stress response" OR "acute stress responses" OR "acute stress symptom" OR "acute stress symptoms" OR "traumatic stress response" OR "traumatic stress responses" OR "psychological trauma" OR "psychological traumas") AND ("physical health" OR cardiovascular OR depression OR "immune system" OR immunity OR "immune response" OR anxiety OR panic OR distress OR pain OR "drug abuse" OR "alcohol abuse" OR "service utilization") AND ("Cohort Studies"[Mesh] OR "longitudinal"[tiab] OR Follow-up[tiab] OR Prospective[tiab] OR cohort[tiab]). (Note: "Mesh" stands for Medical Subject Heading, used to index all articles in PubMed; "tiab" indicates search criteria should be in title or abstract.) Authors of articles not available for download were contacted via email.

D.R.G. performed the initial screening of article abstracts and read all articles whose abstracts met criteria. D.R.G. then conducted a forward and backward search of these articles (i.e., screened articles that were cited by or cited these articles), R.R.T. performed a follow-up screening to search for additional articles and verified D.R.G's initial search. Final inclusion eligibility decisions and data extraction were then carried out through consensual, iterative processes among the authors.

D.R.G. and E.A.H. read included articles and ascertained variables for extraction based on analyses of study methodologies, covariates, and independent and dependent variables reported. All authors reviewed the included articles to determine and verify the data to be extracted. Data extraction items included population, type of trauma experienced, number and timeframe of assessments, length of final follow-up, participation and retention rates, assessment instruments, number of participants, age of participants (range, mean, median), sample gender distribution, sampling strategy, location of assessment, country where study was conducted, assessment method, specific measure of acute stress used, whether AS/ASD was a significant predictor of outcomes, whether PTSD was also included as an outcome, whether a control group was included, and whether acute assessments were significant predictors in short- (< 12 months) or long- (≥1 year) term follow-up assessments or both.

3. Results

Database searches yielded a total of 1182 results: 517 from PubMed, 252 from Web of Science, 299 from PsycINFO, and 114 from CINAHL. After removing duplicates, 957 unique articles were available for eligibility screening. After screening abstracts, 88 articles were considered for inclusion. Of these, 66 were excluded either because they did not assess acute response within one month of the traumatic event or because an acute response was not examined as a predictor of subsequent non-PTSD outcomes (e.g., they assessed prevalence of a non-PTSD disorder at two time points, but did not use an acute assessment as a
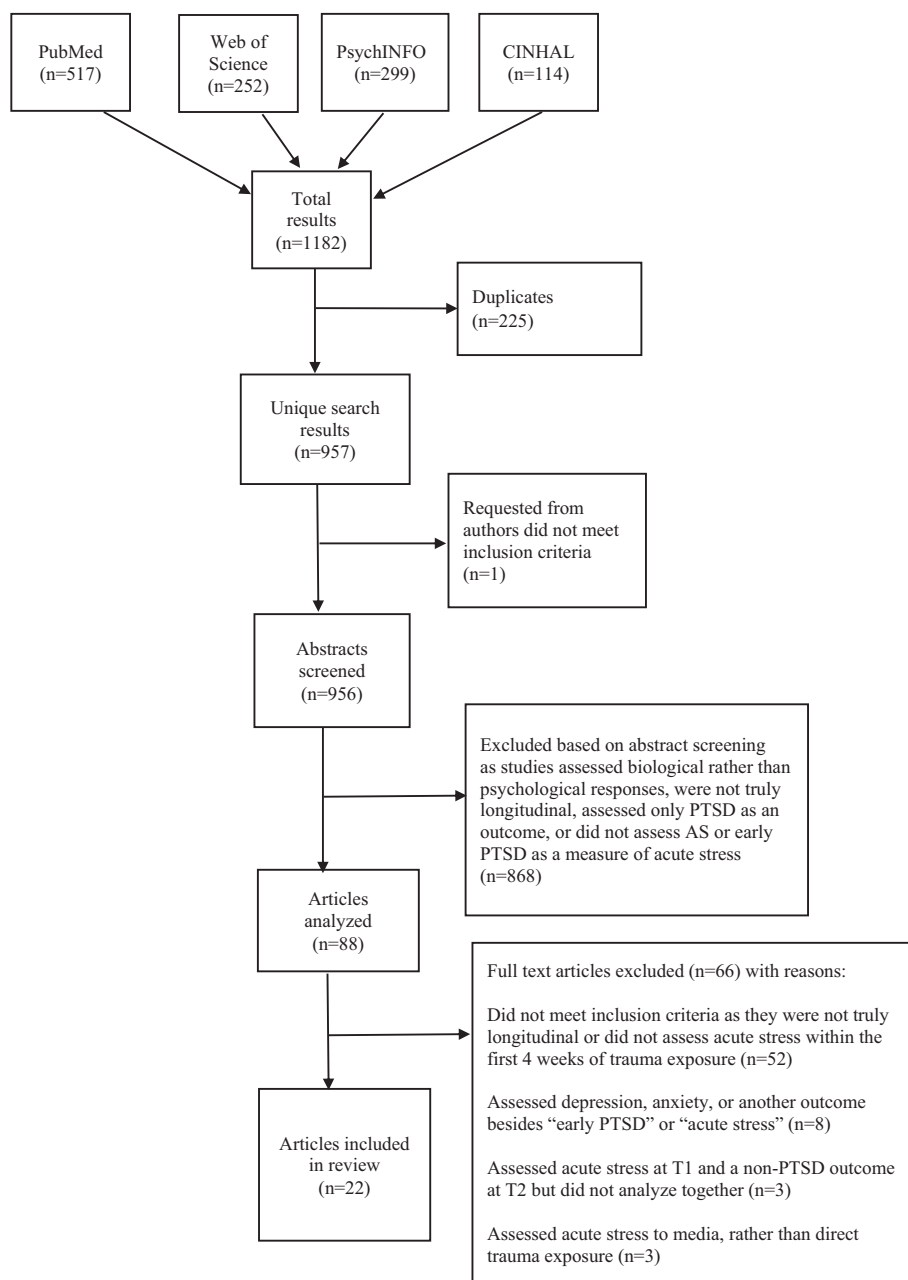
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│ (n=517)  │  │ Science  │  │ (n=299)  │  │ (n=114)  │
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Fig. 1. Flow chart of included studies. Note: some studies were excluded for multiple reasons.

Additional flow chart text boxes:

Excluded based on abstract screening as studies assessed biological rather than psychological responses, were not truly longitudinal, assessed only PTSD as an outcome, or did not assess AS or early PTSD as a measure of acute stress (n=868)

Articles analyzed (n=88)

Full text articles excluded (n=66) with reasons:

Did not meet inclusion criteria as they were not truly longitudinal or did not assess acute stress within the first 4 weeks of trauma exposure (n=52)

Assessed depression, anxiety, or another outcome besides "early PTSD" or "acute stress" (n=8)

Assessed acute stress at T1 and a non-PTSD outcome at T2 but did not analyze together (n=3)

Assessed acute stress to media, rather than direct trauma exposure (n=3)

Articles included in review (n=22)

predictor). For example, some studies examined trajectories of distress responses, but did not consider how early responses predicted subsequent distress [27, 28], did not specifically report the relationship between early responses and non-PTSD outcomes, even if they were assessed and reported [29], or examined only PTSD as an outcome [30]. One article was unavailable through the University of California library and was requested from authors. It was excluded as it correlated initial symptom levels with subsequent outcomes at exactly the one-month mark, confounding the acute and subsequent response, and did not correlate initial outcomes with subsequent responses [31]. Eight articles were excluded because the measure of acute psychological distress was not specific to the indicated trauma (e.g., acute depression, rather than acute stress). Three articles were excluded because the indicated trauma did not meet the guidelines outlined in the DSM-V for a Criterion A trauma [15, 32, 33]. See Appendix A for a table summarizing included studies, the populations assessed, time frames of assessments, key measures, and key findings. See Fig. 1 for a diagram of inclusion

and exclusion decisions.

Twenty-two articles met inclusion criteria and were reviewed. Three articles reported findings from the same sample [22, 34, 35], thus the studies reviewed report data from 20 independent samples. The majority of studies ($n = 12$, 54.5%) were conducted with non-U.S. populations in Australia ($n = 5$, 18.2%) [19, 21, 36, 37, 38], Great Britain ($n = 1$, 4.5%) [39], Israel ($n = 2$, 9.1%) [40, 41]), Germany ($n = 1$, 4.5%) [42]; Switzerland ($n = 1$, 4.5%) [43], and Denmark ($n = 1$, 4.5%) [44]. One study was conducted in a non-Western country: Taiwan [45]. The remaining 10 studies were conducted in the United States (45.5%). No studies were conducted in non-industrialized nations.

3.1. Characteristics of the samples

Sample sizes varied greatly. The smallest included 56 participants and the largest 1166; the mean was 384.6 ($SD = 399.4$) and the median was 198. Studies tended to include more males than females or be

comprised entirely of male participants: 69.1% of studies had more men than women, with 58% of the sample, on average, being male. Eleven studies reported some degree of racial/ethnic breakdown of the sample: six (27.3%) reported whether participants were White/European decent or other, and five (22.7%) reported more specific racial/ethnic breakdown of the sample. Of the data available, those of White/European decent comprised the majority of participants (mean percentage = 65.4%; median percentage = 73.2%; range 4%–100%). Five studies reported the percentage of those of African descent ($M = 37.6\%$, $SD = 32.9$) and three reported percentage of Hispanics in the sample ($M = 12.0$, $SD = 15.7$). One study reported the percentage of Asian Americans (5%) and one study was conducted on a Taiwanese population [45]. The mean age of participants was 37.4 ($SD = 11.6$). Three studies examined adolescent or child populations [46, 47, 48].

3.2. Methodology

3.2.1. Sampling strategies

The majority of studies ($n = 19$, 86.4%) used convenience samples from medical clinics or hospital settings. Clinical samples included assault survivors [39], emergency department (ED) patients [45, 46], those recently admitted to a hospital for treatment of burns [49], whiplash [21, 38, 44], myocardial infarction [41], or general traumatic injury [19, 50], and intensive care unit (ICU) patients [21, 34, 35]. Several studies examined the parents of hospitalized children, such as children who suffered acute burns [48]. One study used a convenience sample of a specific population: Israeli male veterans [40].

More stringent methodologies based on random sampling strategies were infrequently used. Schweininger et al. [37] randomly selected from admissions to 4 major trauma hospitals; Zatzick et al. [47] sampled adolescent survivors of intentional and unintentional injuries using random number assignment; and Bryant et al. [19] randomly selected from admissions to five Level 1 Trauma Centers.

Assessment strategies varied; multiple methods were sometimes used. In-person interviews were included in assessment protocols by 68.2% ($n = 15$) of studies. Three studies (13.6%) implemented surveys, questionnaires, or other forms of self-report. Several used a verbally-administered questionnaire (either in person or via phone; [22, 34, 35, 46]), one used in-person interviews, phone calls, and written surveys [37], and one examined death records to assess 15-year all-cause mortality [41]. Three studies did not report the method of assessment [38, 40, 49]. While self-report methods were common, a number of studies incorporated objective methods of assessment. These included biological response markers (serum leptin and cortisol) [45], number of hospitalizations, ED visits, and outpatient visits [22], re-injury due to violence [46] and death records to assess 15-year all-cause mortality [41].

3.2.2. Number and timing of follow-up assessments

See Table I for complete data on number and timing of baseline (acute response) and follow-up assessments and retention rates (if provided). Ten studies examined physical health outcomes, eight examined mental health outcomes, and four examined both. Mean number of assessments was 2.8 ($SD = 0.9$; Range 2–5), most studies reported either 2 ($n = 10$; 45.5%) or 3 ($n = 8$, 36.4%) assessments. Median time between baseline and final follow-up was 12.0 months ($M = 27.6$; $SD = 60.0$; range = 1–240). A control or comparison group of participants who did not meet criteria for ASD or report substantial acute stress symptoms was utilized by 5 (22.7%) studies [19, 22, 34, 35]; most studies compared symptoms within participants along a continuum (i.e., symptoms scores rather than clinical diagnoses).

3.3. Acute stress and health outcomes

Twenty studies (90.9%) found ASD or early PTSD predictive of later adverse outcomes; two studies did not find this effect [46, 49]. Ten

studies measured posttraumatic stress along with non-PTS outcomes at later time points in the study. Five assessed whether the impact of AS on longitudinal outcomes was independent of the association between AS and PTSD [36, 37, 40, 43, 45]; all found that the associations between AS and non-PTS outcomes were independent of comorbidity between PTS and non-PTS mental and physical health outcomes, including self-reported pain [36, 43], anxiety [37], depression [37], and general physical health [40].

3.3.1. Physical health outcomes

3.3.1.1. General self-reported physical health. Four studies examined the link between acute stress and subsequent self-reported physical health [40, 44, 47, 49]. Acute stress was associated with neck disability, reduced working ability, and lower self-reported general health in whiplash patients one year post-accident [44]. Acute stress also predicted both short- and long-term general self-rated physical health in samples of veterans [40] and injured adolescents [47]. However, in a study of hospitalized burn patients, in-hospital early PTSD symptoms were not associated with self-reported health at follow-up [48].

3.3.1.2. Pain. Five studies assessed the link between acute stress and physical pain: two reported significant associations at short-term follow-ups [21, 43], one did so at long-term follow-ups [44], and two studies reported significant associations at both short- and long-term follow-ups [36, 47]. In a sample of injury patients, Liedl et al. [36] examined the ASD subscales individually, finding significant associations between early re-experiencing and arousal symptoms and subsequent pain; in full path analysis, re-experiencing and arousal predicted pain at 3 months. Baseline re-experiencing, avoidance, and arousal were also associated with pain at 12 months in bivariate models.

3.3.1.3. Other physical health outcomes. Several studies examined acute stress as a predictor of more specific physical health outcomes. Acute intrusive and avoidance symptoms were associated with poor self-reported quality of well-being 12 and 18 months after major trauma [52]. Acute stress responses also predicted objective measures of health such as short- and long-term healthcare service utilization in ICU patients [22] and quality of life in patients following hospitalization for injury [47] and major trauma [52]. In a study measuring physiologic stress markers, ASD following the Taiwanese earthquake was associated with increased Leptin levels 18 months post-event, but not with increased cortisol [45]. Additionally, although in-hospital dissociative symptoms predicted 15-year all-cause mortality in a sample of MI patients, other acute stress symptoms (arousal, avoidance, intrusion) did not [41].

3.3.2. Mental health and psychosocial outcomes

3.3.2.1. Global distress. Two studies assessed early trauma-related symptoms as a predictor of global distress. Acute stress responses predicted global distress less than one year after patients experienced general injury [50]. More severe acute PTSD symptoms were also associated with worse mental health as measured by the SF-36 in a sample of whiplash injury patients after one year follow-up [44].

3.3.2.2. Depression. One study found a significant short-term (< 1 year) association between acute stress and later depression [51]. Acute stress was also associated with the development of major depressive disorder one year later [19], and with depressive symptoms reported three and twelve months later in another study [35].

3.3.2.3. Anxiety and phobia. Two studies found that acute stress symptoms predicted later anxiety: one at 3-month [37] and one at 12-month [19] follow-ups. Bryant and colleagues [19] found that ASD significantly predicted the development of multiple anxiety disorders at one-year follow-up, including panic disorder, agoraphobia, social

phobia, specific phobia, obsessive compulsive disorder, and generalized anxiety disorder.

3.3.2.4. Cumulative psychiatric disorders. Three studies assessed a composite sum of psychiatric problems as the longitudinal outcome. Two studies found associations between acute stress and cumulative psychiatric problems at 12 months [19, 44], and one found associations at 2, 5, and 12 months [47]. Two studies of traumatic injury patients suggested that an ASD diagnosis or early PTSD significantly predicted a broad spectrum of psychiatric problems after one year [19, 47], although the majority of those with ASD did not go on to develop any disorder [19]. Greater acute stress symptoms were also associated with poorer mental health (as measured by the SF-36) after one year in a sample of whiplash patients as well [44].

3.3.2.5. Additional outcomes. We identified a number of additional psychosocial outcomes associated with acute stress. At one year, early PTSD was associated with more unhealthy alcohol use [34] and positively predicted the development of substance abuse disorder [19]. However, in other studies, AS was not associated with subsequent reduction in respondents' ability to work after accounting for baseline pain intensity [44] or return to work [50] in long term follow-ups, nor did it predict violent reinjury in a sample of injured adolescents [46]. Higher dissociative symptoms (a component of DSM-IV ASD criteria) in parents but not in the child after the child was hospitalized for burns were associated with increased parent-child conflict two months later [48].

4. Discussion

This systematic review provides substantial evidence that acute stress responses may help identify individuals at risk for subsequent trauma-related health problems. Importantly, our search criteria focused on the most rigorous studies available, increasing the likelihood that our review summarizes the most valid and unbiased data available. All studies reviewed were longitudinal, many had relatively high retention rates, and all measured acute stress responses within the one-month time frame required for an ASD diagnosis, minimizing concerns about retrospective recall bias in early response assessments. Two studies were outliers in that they followed participants more than a few years [40, 41], highlighting both the potential for long-term health problems and an important area for improvement in the extant literature: studies that follow people for many years could help strengthen our understanding of the long-term implications of acute stress symptoms. Although most studies used clinical samples (e.g., ED patients) with some form of physical injury that could be confounded with physical health outcomes, the large majority of studies minimized the likelihood of confounding by statistically adjusting for injury severity.

Depression and depressive symptomatology was the most frequently examined outcome; high acute stress responses uniformly predicted higher risk for subsequent depression or depressive symptomatology, particularly in short-term (< 1 year) follow-ups. Although PTSD is more commonly the target of early post-trauma interventions, these findings suggest acute stress assessments may prove useful for identifying individuals at risk for subsequent depression or depressive symptomatology as well [53]. PTSD and depression are frequently co-morbid, particularly in the acute [54] and short-term aftermath of a trauma [55], but they are distinguishable in terms of chronic responses [55], and treatment recommendations. Thus, depression and PTSD and their related symptomatology can be targeted for interventions both as disorders that are frequently comorbid and as separate disorders, given that many people who develop post-trauma depression do not develop PTSD and vice versa [19, 55], despite shared vulnerabilities [56]. Moreover, depression exhibits robust and reciprocal relationships with several physical health problems [57-59], illustrating the importance of addressing trauma-related depression to improve overall health.

Furthermore, physical health problems were linked with acute posttraumatic stress responses in a number of studies in both short-term and longer-term follow-ups. Indeed, general physical health, all-cause mortality, and pain, and healthcare service utilization (a proxy for physical impairment) emerged as significant long-term outcomes predicted by acute stress response. This is important considering the robust links between trauma exposure and physical health problems such as injury, pain, and cardiovascular problems [14, 60] and increased utilization of general and psychiatric medical services by trauma survivors [61].

The long-term health implications of acute stress are especially important to consider given that stress-related processes are tied to the leading cause to death and disability in the world – cardiovascular disorders (CVD). It is well known that chronic and subacute stress are linked to CVD [62], however acute stress may also trigger acute cardiovascular events in vulnerable individuals through several physiologic pathways [3, 63]. Recent evidence suggests that acute psychological stress may trigger cardiac endocannabinoid activity, lipogenesis and inflammation, autonomic/endothelial dysfunction, arrhythmogenicity, thrombosis, and more [23, 62, 63]. Together, these early physiologic processes may contribute to the subsequent development of atherosclerosis, cardiac steatosis, heart failure, and arrhythmia, all of which are known risk factors for serious cardiovascular events. If acute stress portends potential risk for these pathophysiologic changes, it may facilitate identification of targets for early interventions to prevent trauma-related CVD and its morbidity/mortality. Given the critical role of stress physiology and inflammation in chronic disease [64, 65], such early interventions could potentially have important health implications for beyond CVD prevention.

5. Contributions and limitations

This review used rigorous inclusion/exclusion criteria to identify the most methodologically sound studies available that inform our understanding of long-term correlates of acute stress response to trauma. Although we sought to synthesize the current knowledge on the link between acute stress and both physical and mental health outcomes, we did not examine effect sizes due to wide variability in both assessment tools and outcome measures. For example, the variability in the physical health outcomes examined limits the ability to draw clear conclusions regarding specific physical health correlates of acute stress. Nonetheless, the pattern across many studies suggests that acute stress/early PTSD is associated with various physical health-related outcomes over time.

Bias in reporting likely occurred; studies with null effects are often not published, although several of the articles we reviewed did report null findings. Most studies reviewed were of high quality, given the search criteria, although many studies assessed very specific samples (e.g., veterans, injured individuals), which may limit generalizability. Although we searched several primary databases, it is possible that some studies were inadvertently not included. Moreover, while clinical trials are generally registered in order to address such biases, our study focused on examining the evidence for a longitudinal association between acute stress and subsequent health, not interventions to treat acute stress, thereby excluding such studies. Most studies used self-report measures, to the exclusion of objective health assessments, and are thus subject to reporting bias. Studies also used a mix of diagnoses and symptomology to measure outcomes, which could limit comparability across studies. Some researchers agree that using symptoms rather than formal diagnosis may be beneficial to capture dimensionality in responses [66], yet this can limit estimates of diagnostic rates in a population. Finally, the findings reported were primarily from clinical samples in Western populations; cultural differences likely exist to some degree, although similar longitudinal post-trauma symptom course has been indicated in cross-cultural work on terrorism survivors [67].

Given that most of the studies in our review were clinical in nature,

rather than epidemiological, it was impossible to compare rates of physical and mental health after these traumatic events with those in a non-trauma exposed sample. While this is a limitation of the extant research, we believe that conducting more post-trauma studies that assess acute stress in the immediate aftermath of an event using representative samples is a promising area of future research. Indeed, after Hurricane Katrina, epidemiological studies that compared pre- and post-rates of mental illness found marked increase in disorders post-hurricane [68, 69], although these studies did not include acute assessments that followed people over time. The studies that have assessed acute stress within the DSM-time frame (< 4 weeks post-event) using representative samples included individuals with both direct- and media-based trauma exposure and were thus not included in these analyses [15, 32, 33]. Prospective representative studies of trauma that start with assessments taken prior to the event, assess survivors in the immediate aftermath of the trauma, and follow people over time could help clarify these relationships.

As no studies examined in this review include pre-event measures of mental health, it is possible that in some instances acute stress responses are preceded by psychopathology or distress symptoms present *before* trauma exposure; no studies in this review assessed pre-event physical health either. This means that any significant associations found between acute stress and subsequent outcomes could be spuriously linked to pre-event mental or physical health. The limited research that has included pre-event assessments of mental or physical health in studies of acute stress responses to trauma has found pre-event symptoms to predict post-event responses [32, 70]. However, representative national studies have concurrently found that acute stress responses predict mental and physical maladies over time, even after statistically controlling for pre-event measures of mental and physical health [15, 32, 71] suggesting a relationship between acute stress response and subsequent pathology independent of pre-event health. Only truly prospective, longitudinal research with representative samples can help tease these associations apart. However, to our knowledge, studies of this kind have all included individuals exposed to the event both directly and via the media. We suggest that, despite the logistical difficulties incorporating pre-trauma assessments of mental and physical health, this is a critical area for future research.

Nonetheless, we maintain that results from this review suggest that assessing acute stress responses to traumatic events can provide important insights into who is at risk for long-term health problems. Rather than separately screening for depression, anxiety, poor health, and so on, a short measure of acute stress during disaster triage or early contact with medical professionals could yield important insights as to who to target for longer follow-up and intervention over time, regardless of pre-event health status.

6. Conclusions and recommendations

Although acute stress has poor sensitivity for predicting subsequent PTSD [20], our data suggest that, when incorporating a wider range of outcomes, assessments of acute stress following traumatic events may indeed identify those more likely to experience long-term mental and physical health problems in the years following trauma exposure. Although many who are initially distressed recover over time [72], information on those at-risk for subsequent problems can help clinicians, hospital staff, emergency workers, and aid workers assess survivor's immediate needs and target them for early interventions to prevent long-term problems with necessary follow-up assessment and efficacious care, as needed. For example, "screen-and-treat" post-disaster interventions, the most empirically supported model, involve acute assessments of survivors [73]; those with higher initial symptomatology, who are more likely to experience longer-term problems, are then specifically targeted with monitoring and prevention efforts. Such methods could translate to other settings, such as ED intake [74], as discussed above.

This review suggests future research should include assessments of pre-event mental/physical health, acute posttraumatic stress responses, and non-PTSD trauma-related outcomes in longitudinal studies seeking to understand the health implications of trauma exposure. When the data permits, early acute stress assessments should also be examined as predictors of a variety of health outcomes over time; such analyses were not always conducted even when data was available. Doing so would improve our ability to understand who is at risk for long-term trauma-related health problems, support development of early interventions to decrease trauma-related morbidity and mortality, and thereby improve health and well-being of trauma survivors.

Acknowledgements

Conflict of interest

The authors have no competing interests to report.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2018.05.017.

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