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Post-traumatic stress disorder research: a narrative review

Abstract

This review summarizes recent research publications (2019-2024) on the prevalence of post-traumatic stress disorder (PTSD), its negative effects, predictors/risk factors for the disorder, interventions and potential underlying biological mechanisms. The prevalence of PTSD has widely ranged from 10-70% for those experiencing a traumatic event and has most frequently involved samples that have experienced PTSD that was related to earthquakes (in Haiti, Japan, Nepal). The negative effects have included cognitive disability, sleep disorders, medical conditions/diseases, depression and mortality. The predictors/risk factors include female gender, worrying, rumination, anxiety, depression, neuroinflammation and stress from loss of friends, relatives, pets and/or property. The interventions have included reducing the stigma of PTSD, alternative therapies including yoga and mindfulness, cognitive behavioral therapy and medications. Potential underlying biological mechanisms include hypothalamic pituitary adrenal system dysfunction involving elevated cortisol during immediate stress and cortisol depletion during prolonged stress, connectivity dysfunction in the brain as well as white and grey matter loss and gene pathways. These have been noted in the "fear learning and memory network" of the prefrontal, amygdala, hippocampal and anterior cingulate cortex regions of the brain. Methodological limitations include the samples being primarily those with PTSD following earthquakes and the self-report data in cross-sectional studies that cannot suggest causality.

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Introduction

Post-traumatic stress disorder (PTSD) has been defined as a syndrome that derives from severe stress following a traumatic event that is typically prolonged.[1] The symptoms include re-experiencing the event, avoidance, negative alterations in cognition and mood and marked alterations in arousal and reactivity. Evidence-based therapies include cognitive behavioral therapy and medications including serotonin reuptake inhibitors.

Methodology

This narrative review includes summaries of 31 papers on PTSD in adults that were derived from a search on PubMed and PsycINFO entering the terms post-traumatic stress disorder and the years 2019-2024. Exclusion criteria for this review included papers on proposed protocols, case studies, and non-English language papers. The publications can be categorized as the prevalence of PTSD, negative effects of PTSD, predictors/risk factors, interventions and potential underlying biological mechanisms for PTSD. This review is accordingly divided into sections that correspond to those categories. Although some papers can be grouped in more than one category, 5 papers are included on the prevalence of PTSD, 4 papers on the negative effects of PTSD, 10 on predictors/risk factors for PTSD, 8 on interventions and 9 on potential underlying biological mechanisms for PTSD. These sections are followed by a discussion on methodological limitations of this literature.

Prevalence of PTSD

Reportedly as many as 90% of adults in the U.S. have experienced a traumatic event.[2] Among the most frequent traumatic events are death of a loved one, serious illness, car accident, severe injury, natural disaster, loss of home, domestic abuse, divorce, and job loss. Despite the high prevalence of traumatic events, reputedly only 7-10% develops PTSD (see Table 1).[2] The prevalence data in this recent literature have appeared primarily in studies on earthquakes in other countries including Japan, Nepal and Haiti where the prevalence has ranged from 10 to 25%. The sampling of earthquake survivors in the recent literature is a contrast to the military samples that appeared in earlier literature.

Table 1 Prevalence of PTSD (and first authors)

Prevalence	First authors
U.S.- 7-10%	Harnett
Japan earthquake and tsunami-10%	Inoue
Japan earthquake and tsunami- 25%	Li
Nepal earthquake-19%	Pandey
Haiti earthquake-25%	Cénat
Resident physicians-13%	Lo

A prevalence of 10% has been reported for data from the Japan Geological Evaluation Study on the 2011 Great East Japan Earthquake (N =580 adults greater than 65 years-old).[3] Not surprisingly, those who lost close friends, relatives and/or homes experienced twice the prevalence. Interestingly, a different research group reported a significantly greater prevalence of PTSD (25% versus 10%) in a larger sample following the same Great East Japan Earthquake and tsunami (N= 2965, mean age=73).[4] In this three-year follow-up study of that earthquake and tsunami, PTSD was also comorbid with depression. The rate of depression (33%) was greater in those experiencing PTSD (25%). The comorbidity of depression and PTSD led to high mortality rates. It's not clear whether the greater prevalence of PTSD in the latter sample relates to the comorbidity of PTSD and depression or whether it is related to the significantly larger sample size or some unmeasured factor.

A prevalence of 19% was noted for PTSD following the 2015 earthquake in Nepal.[5] The interviews that were conducted three years

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after the earthquake suggested a greater prevalence for those who experienced damage to their property. Females were noted to have a 1.6 greater prevalence than males and the rate was two times the 19% for those who were illiterate. In contrast, those who had social support had 60% lower odds of experiencing PTSD.

A higher rate of 25% was noted for the prevalence of PTSD following the 2010 earthquake in Haiti.[6] This rate was reliably based on a review and meta-analysis of as many as 28 studies. In addition to the high prevalence of PTSD, 33% experienced depression and 20% experienced anxiety. Again, females were more likely than males to experience PTSD (41% more likely) possibly because of comorbid depression that is more prevalent in females. Time was a moderator in reducing depression and anxiety, but not in reducing PTSD. This difference in duration of these disorders was uniquely reported in this study, highlighting the need for replication and research on comparisons of treatment responses in individuals with these different disorders and comorbidities.

The only other paper that could be found in the recent literature that focused on the prevalence of PTSD reported a rate of 13% for resident physicians at the University of British Columbia (N= 43).[7] The underlying risk factors were bullying, which was surprisingly a greater risk factor than violence, which was greater than medical error, which was, in turn, greater than the death of clients.

Negative effects of PTSD

Surprisingly, only a few publications were found that focused on negative effects of PTSD in this recent literature (see Table 2). They include research on cognitive disability, sleep disorders, medical conditions, depression and mortality.

Table 2 Negative effects of PTSD (and first authors)

Negative effects	First authors
Cognitive disability	Kuzono
Depression	Xiaoyu
Mortality	Xiaoyu
Sleep disorders	Maguire
Medical conditions	Lawrence

Cognitive disability has resulted from PTSD. Examples of cognitive disability include difficulty sustaining attention, poor performance on verbal learning and memory tasks, slow processing speed and impaired executive function. Cognitive disability was reported for a sample of those who experienced the 2011 Great East Japan Earthquake and tsunami.[8] Cognitive disability was associated with home loss, loss of friends and/or loss of pets. In a different sample from the same earthquake already described, a three-year follow-up assessment suggested that 33% experienced **depression**. **Mortality** was also associated with PTSD in this sample.

In a systematic review on 16 studies, **sleep disorders** were related to PTSD.[9] The sleep disorders were commonly insomnia and nightmares. Not surprisingly, those who had sleep disorders prior to the trauma were at greater risk for PTSD following the trauma.

In another review of the PTSD literature, several **medical conditions** were associated with PTSD.[10] These included cardiovascular, metabolic, autoimmune, and neurocognitive conditions as well as preterm birth. The authors attributed these to dysregulation of the hypothalamic pituitary adrenal axis, although the literature on cortisol has been mixed with some researchers reporting elevated cortisol typically associated with short-term PTSD and others noting low cortisol levels in those with prolonged PTSD.

Predictors/risk factors for PTSD

Several predictors/risk factors have been the focus of recent research on PTSD (see Table 3). They include female gender, metacognitive memory, worry, anxiety and depression, neuroinflammation, and loss of relatives, friends, and/or property during earthquakes. Some of these predictors/risk factors have also been considered negative effects or comorbidities of PTSD, e.g. anxiety and depression.

Table 3 Predictors/risk factors for PTSD (and first authors)

Predictors/risk factors	First authors
Female gender	Pandey, Cénat
Beliefs about traumatic memories	Brown, Clauss, Brown
Anxiety and depression	Lewis, Cénat
Neuroinflammation	Lee
Earthquakes	Pandey, Kuzono

At least two studies in this literature have reported greater prevalence of PTSD in **females** versus males. In each of these studies, already described, the samples have been exposed to earthquakes. After the 2016 earthquake in Nepal, the prevalence in females was 1.6 times greater than it was in males.[5] Following the 2010 earthquake in Haiti, women were 41% more likely to experience PTSD than men.[6] The greater prevalence of PTSD in women may relate to the comorbidity of depression as the prevalence of depression is typically greater in women (at a 2 to 1 ratio).

A few studies have appeared in this literature on **beliefs about traumatic memories** and thought control strategies and their impact on PTSD symptoms. These data have been referred to as the meta-cognitive model or meta-memory. In the meta-cognitive model it is assumed that excessive worry, as in worry about worry, can lead to emotional problems. In a study on 412 earthquake survivors, for example, the risk factors were a greater number of meta-memory beliefs including greater worry as well as a history of mental health difficulties.[11]

Similar risk factors were noted in another study on trauma-exposed individuals (N= 237).[12] The predictors of PTSD were **worry**, internal threat and substance use. Attention control moderated (dampened) those risk factors. In a systematic review of 18 studies, similar predictors of PTSD symptoms severity were noted.[13] These were meta-cognitive beliefs, meta-memory beliefs, worry, thought suppression, experiential avoidance, and rumination.

Anxiety and depression were notable risk factors in a sample from the UK (N= 194, mean age =46).[14] The risk factors reported by these authors were greater anxiety, depression, and traumatic stress symptoms as well as lower income. In a study already described on survivors of the Earthquake in Haiti, depression was noted in one-third of the individuals sampled from 28 studies and anxiety in one-fifth of the individuals.[6] Anxiety and depression have been sometimes viewed as risk factors of PTSD and other times referred to as comorbidities.

In a review of the literature on **neuro-inflammation** in PTSD, increased levels of pro-inflammatory cytokine biomarkers were noted.[15] These included IL-1, IL-6, TNF-alpha and C-reactive protein. In this cross-sectional study, however, it is unclear whether these pro-inflammatory cytokines were prevalent before the trauma as in a genetic predisposition or whether they resulted from the trauma.

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As already noted, the prevalence of PTSD has been high especially after **earthquakes** which have accordingly been viewed as a risk factor. As already mentioned in the study on the 2015 earthquake in Nepal, damaged property was a risk factor for PTSD.[5] Also, as already described, the loss of close friends and relatives was a risk factor for those who experienced the great East Japan Earthquake in 2011, especially those who were greater than 65-years old.[3] The **losses of friends, family and property as well as pets** were listed as risk factors following the same earthquake by a different research group.[8]

Interventions for PTSD

Several interventions have appeared in this recent literature on PTSD. They include reducing the stigma of PTSD, guitar lessons, mindfulness, yoga, cognitive behavioral therapy, and medications (Table 4).

Table 4 Interventions for PTSD (and first authors)

Interventions	First authors
Reducing stigma	Cureus
Guitar lessons	Pezzin
Mindfulness sessions	Shapira
Vinyasa yoga	Cushing
Exercise	Reis
Medications	Coventry, Bertolini

Self-stigma or the internalization of negative societal views and stereotypes has been noted in 41% of a PTSD sample from the UK (N= 194, mean age = 46).[14] This prevalence, surprisingly, did not differ by gender, age, sexual or military trauma. A recent survey revealed that renaming PTSD "disorder" to "injury" would reduce the stigma.[16]

In this sample (N=1025) 48% experienced PTSD and 41% suggested they were experiencing self-stigma. Two-thirds agreed to a name change and more than 50% suggested that **reducing the stigma** would increase the occurrence of medical help-seeking behavior.

Guitar lessons have been an effective intervention for PTSD.[17] In this study, 43 individuals with PTSD were randomly assigned to receive six individual one hour guitar lessons and three group lessons. The PTSD symptoms decreased by 14% and the depression symptoms by 20% in the guitar lesson group. Although these results may be difficult to interpret, the randomization of this sample highlights the validity of the findings. Playing the guitar may be a form of stress-relieving music therapy and result in lower stress hormones, as has been noted in music therapy research.

In one of the few studies that could be found on veterans in this literature, those diagnosed with PTSD (N= 210) received **mindfulness sessions**.[18] This intervention resulted in a recalibration of cortisol levels, as well as a decrease in PTSD symptoms. In addition, a reduction occurred in pro-inflammatory cytokines that have been associated with PTSD including IL-6 and C-reactive protein.

In another study on PTSD in veterans that appeared in this recent literature, the veterans were engaged in Vinyasa Yoga which involves continuous movements.[19] In this protocol (N= 18), yoga was provided for 60 minutes per week for six weeks. At the end of this intervention PTSD was decreased in the three-symptom cluster of the Post-Traumatic Checklist – Military, including hyperarousal, re-experiencing, and avoidance.

In a review of nine studies on veterans, **exercise** was offered to reduce PTSD symptoms.[20] Six single-arm studies were offered on yoga and three randomized controlled trials on yoga, aerobics or resistance training. Medium size effects were noted for these studies, suggesting a reduction in PTSD symptoms.

In a systematic review and meta- analysis on **psychological and pharmacological interventions** for PTSD, 18 randomized controls were included (N= 933 participants, mean age = 43).[21] The follow-up assessment occurred 12 weeks after the interventions. The psychological interventions were more effective than the pharmacological therapies in terms of reducing PTSD and depression symptoms. There was also less attrition in the psychological intervention versus the antipsychotic medication groups. The most effective psychological Interventions included cognitive restructuring (helping change unhelpful thought patterns) and imaginal exposure (imagining anxiety-provoking thoughts, images or narratives). The authors suggested that most of the studies were limited by the samples involving individuals who had self-reported PTSD rather than clinically diagnosed PTSD.

Pharmacological intervention studies have been surprisingly scarce in this recent literature on PTSD. In a review paper entitled, "Early pharmacological interventions for prevention of PTSD in individuals experiencing acute traumatic stress symptoms", 8 studies were reviewed (N=779).[22] Although the authors mentioned that a couple researchers reported that escitalopram and hydrocortisone decreased PTSD symptoms, they suggested that the data were inconclusive.

Potential underlying biological mechanisms for PTSD

Various biological mechanisms have been implicated as potentially underlying PTSD. They include dysregulation of the hypothalamic pituitary adrenal system, inflammatory processes, connectivity problems in the amygdala and hippocampal regions, a reduction of white matter and gray matter in various regions of the brain and gene pathways.

In a review on the **hypothalamus** and PTSD, the traumas included sexual assault, war, natural disasters, burns and car accidents.[23] The hypothalamus and HPA axis are linked to the prefrontal cortex and limbic structures, especially the amygdala and hippocampus. This system was not only involved in initiating a stress response but also by secreting growth hormone, prolactin, and oxytocin (positive acting hormones) and dopamine (an activating neurotransmitter) (Table 5).

Table 5 Potential underlying biological mechanisms for PTSD (and first authors)

Mechanisms	First authors
Hypothalamic pituitary-adrenal axis dysregulation	Lawrence, Raise-Abdullahi
Inflammatory process	Maguire, Womersley
Prefrontal cortex, hippocampus and amygdala dysfunction	Harnett, Kunimatsu
Decreased white matter and gray matter volume	Doherty
Cortisol-related regulation genes	Castro-Vale
Immune system and oxidative stress genes	Katrinli
Neuroinflammatory genes	Zass
Linoleic acid metabolism	Crombach

In a study entitled "PTSD associated **hypothalamic–pituitary–adrenal (HPA) axis dysregulation** and physical illnesses", the stress response was immediately mediated by the sympathetic adrenal medulla through norepinephrine and epinephrine and by

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a slower response by the hypothalamic pituitary adrenal system through cortisol.[10] HPA axis activity has been implicated along with **inflammatory processes** and trophic factor regulation (involving the control of the survival and death of cells) in a review of 16 studies on PTSD.[9]

In another systematic review of longitudinal studies, inflammation and endocrine measures were also the most commonly assessed molecular markers.[24] These studies involved samples of military veterans, individuals admitted to the hospital for traumatic stress and females exposed to interpersonal violence or rape. The review highlighted the processes related to inflammation, stress responding and learning and memory.

In a paper entitled "Post-traumatic stress disorder-related neuroimaging abnormalities in brain function, structure and biochemistry", the fear learning and memory network centered on the **prefrontal cortex, hippocampus and amygdala** was dysfunctional.[2] In a similar study entitled "Anomalous gray matter structural network in recent onset PTSD", regional connections involving the fear-processing and re-experiential processing cortex were noted to maintain PTSD symptoms.[25]

In an fMRI study (N=25 with PTSD and N=25 with trauma), the fMRIs showed compromised **white matter integrity** in tracts connecting limbic structures including the amygdala to the frontal regions including the anterior cingulate cortex which, in turn, impaired threat/ fear processing.[26] **Decreased gray matter volume** was also noted in the hippocampus, amygdala and anterior cingulate cortex.

In a review on fMRI findings in PTSD, **alterations in the anterior cingulate cortex, amygdala**, **hippocampus and insula** were reported.[27] Abnormalities were noted in fear-learning and reactions to threat. The brain networks regulating autobiographical memory, retrieval and self-thought, salience detection and autonomic responses or attention and emotional control were affected by these alterations.

Gene pathways have also been explored as underlying biological mechanisms for PTSD, although only 19 studies on genes were found in the PTSD literature from the last five years. These genes included cortisol-related regulation genes, genes involved in the immune system and oxidative stress, inflammation-associated genes and linoleic acid pathway genes.

In a review of the literature on **cortisol-related regulation genes**, the authors suggested that 30 to 46% of the variance in PTSD was accounted for by genetic factors.[28] They then discussed the stress related disorder of the glucocorticoid receptor. Acknowledging that PTSD is polygenetic, their meta-analysis suggested that at least two genes were associated with the underlying glucocorticoid processes involved in the maintenance of PTSD.

A meta-analysis on PTSD symptom severity in three military cohorts has implicated changes in **genes involved in the immune system and oxidative stress**.[29] In this study, 429 participants from three male military cohorts were included in the analysis. This analysis identified PTSD-associated changes in four genes involved in oxidative stress and the immune system. **Neuro-inflammatory genes** have also been associated with PTSD, suggesting the comorbidity of inflammation and PTSD.[30]

In a study involving 191 military returning from a war zone, **linoleic acid pathway genes** (associated with memory and immune related processes) were associated with PTSD symptoms.[31] Those with PTSD-improving symptoms were matched with those who had PTSD-worsening symptoms. The linoleic acid metabolism pathway was significantly related to post-deployment PTSD symptoms.

These gene pathway studies are extremely complex, and the diversity of genes the researchers have assessed suggests that the selection of gene pathways may be arbitrary and relate to the researchers' interests. In at least one of the warzone-PTSD related gene studies, assessments revealed some 343 genes, highlighting the complexity of this genetic research.[32]

Methodological limitations of this literature

This recent literature on post-traumatic stress disorder has several methodological limitations that relate to the sampling, variable selection and data analytic methods used by the different research groups. These limitations are highlighted by the relative absence of meta-analyses in this literature.

The samples have been primarily survivors of earthquakes in Haiti, Nepal and Japan which limits the generalizability of the data regarding victims of other traumas and other cultures. Presumably there would be differences across traumas and cultural differences, but comparisons have not been made even between the differential effects of these three earthquakes. Age differences would also be expected in the severity of PTSD, but age effects were typically not reported, and the earthquake survivors were older adults. Although female gender was a notable risk factor in two studies, gender differences were infrequently reported.

The severity of PTSD would also be expected to have differential effects. However, the research reviewed here did not measure the severity of PTSD or compare those with short-term post-traumatic stress with those with longer term post-traumatic stress disorder. And the research on negative effects of PTSD was limited to four studies including one study on cognitive disability, one on sleep disorders, one on diseases and one on depression and mortality.

Most of the studies have been cross-sectional rather than longitudinal. As a result, the directionality of the risk factors and negative effects could not be determined. The negative effects and risk factors have been very similar. The same variables, for example, cognitive dysfunction and depression have been considered as negative effects of PTSD by some investigators and risk factors by others. However, only a few research groups have reported that the negative effects and risk factors were reciprocal.

Although many of the studies in this recent literature on PTSD have involved interventions, they have not been the typical cognitive behavioral therapy or pharmacological interventions. They have involved integrative therapies like mindfulness and yoga or atypical interventions like guitar lessons. The research on potential underlying biological mechanisms has typically focused on connectivity dysfunction and gray and white matter loss in similar regions of the brain including the amygdala and the hippocampus, but most of the mechanism papers are reviews rather than empirical studies.

Conclusion

Despite these methodological limitations, this literature has highlighted the prevalence of PTSD especially among survivors of earthquakes. The prevalence of PTSD and its negative effects have highlighted the need for further intervention research, especially on other traumas that have led to PTSD and on therapies that have been frequently used for PTSD. The studies on predictors/risk factors would seemingly help identify those who may need therapy.

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However, intervention data are needed to inform clinicians on potential treatments for those who have PTSD. Further research is also needed to specify the relative significance of the predictors/risk factor variables for identifying those who need intervention and the specific intervention techniques that are effective in reducing PTSD.

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Conflicts of interest

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