



Relationship Between Anxiety Sensitivity and Post-Traumatic Stress Symptoms in Trauma-Exposed Children and Adolescents: A Meta-Analysis

Henry Tak Shing Chiu¹ · Angel Hiu Tung Chan² · Richard Meiser-Stedman¹

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Abstract

Introduction Given the high rate of trauma exposure among children and adolescents, it is important to understand the risk factors for post-traumatic stress disorder (PTSD). Anxiety sensitivity has been implicated in multiple anxiety disorders, and an emerging evidence base has explored the relationship between this construct and PTSD. The present review investigated the size of the relationship between anxiety sensitivity and PTSD symptoms among children and adolescents exposed to trauma.

Method A systematic search on multiple electronic databases (MEDLINE, PsycINFO, CINAHL and PTSDpubs) returned a total of 2916 records, among which six ($n = 1331$) met study inclusion criteria and were included in our random effects meta-analysis.

Results Our results indicated a large effect size ($r = .56$, 95% CI = 0.47 – 0.64) for the relationship between anxiety sensitivity and PTSD symptoms; there was significant between-study heterogeneity.

Conclusion This supported current cognitive models of anxiety and PTSD. Clinical implications, strengths and limitations of the review were discussed.

Keywords Post-traumatic stress disorder · Anxiety sensitivity · Meta-analysis · Child psychology · Adolescent psychology

Introduction

The majority of children and adolescents are found to have experienced some form of trauma in their lives (i.e. between 56 and 84%; Copeland et al., 2007; Joseph et al., 2000; Karatzias et al., 2020; Landolt et al., 2013; McLaughlin et al., 2013). While many recover naturally from acute stress symptoms induced by trauma (Hiller et al., 2016), around 10–15% of youth go on to develop post-traumatic stress disorder (PTSD; Alisic et al., 2014; Bryant et al., 2007;

Copeland et al., 2007; Costello et al., 2002; Kassam-Adams & Winston, 2004; Ogle et al., 2013). According to Lewis et al. (2019) and Maercker et al. (2022), around 8% of children and adolescents have suffered from PTSD at some point in their life. PTSD is associated with a wide range of physical, emotional, social and developmental impacts on a young person (Fairbank & Fairbank, 2009). PTSD in the context of early life not only disrupts recently acquired developmental skills, but also impedes subsequent development of areas such as cognition, emotion regulation, social skills, perception of danger, self-concept and impulse control (Caffo et al., 2005; Davis & Siegel, 2000; Lubit et al., 2003; Pfefferbaum, 1997). These developmental delays are associated with significant impairment in social and academic functioning (Frieze et al., 2015; McLean et al., 2013). PTSD also increases the risk of developing other emotional and behavioral difficulties including depression, anxiety, substance abuse, conduct disorder, aggression, adjustment disorders and externalizing disorders (Bernhard et al., 2018;

✉ Henry Tak Shing Chiu
henrytschiu@gmail.com

¹ Department of Clinical Psychology and Psychological Therapies, Faculty of Medicine and Health Sciences, University of East Anglia, Norwich NR4 7TJ, UK

² Faculty of Education, Chinese University of Hong Kong, Sha Tin, Hongkong

Kerig et al., 2010; Shaw, 2000; Simmons & Suárez, 2016). If left unattended, these impacts are likely to persist into adulthood and later adulthood (Lupien et al., 2009; Ogle et al., 2013). Given such devastating and far-reaching consequences, it is important to understand the factors that predict the development of PTSD in order to prevent and mitigate it.

Predictors of PTSD in Children and Adolescents

A number of pre-trauma psychosocial risk factors, event-related risk factors and cognitive risk factors were found to predict PTSD symptoms in children and adolescent subsequent to trauma exposure. Psychosocial factors include prior life events, socioeconomic status, intelligence, self-esteem, social support and female gender (Allen et al., 2021; Cox et al., 2008; Trickey et al., 2012), while event-related factors include interpersonal versus non-interpersonal trauma, presence of deaths, injury severity, levels of pain, peritraumatic dissociation and perceived fear responses (Cox et al., 2008; Trickey et al., 2012; Vogt et al., 2007). These factors, however, tended to account for only small to medium effect sizes (Cox et al., 2008; Memarzia et al., 2021; Trickey et al., 2012). Conversely, cognitive factors such as trauma appraisals, data-driven processing (i.e. processing of sensory and perceptual information of the traumatic event instead of its meaning), nature of trauma memory, rumination and thought suppression (Brewin et al., 1996; Ehlers & Clark, 2000; Foa et al., 1989) were consistently shown to be strong predictors of PTSD with medium to large effect sizes (Ehlers et al., 2003; Gómez de La Cuesta et al., 2019; Meiser-Stedman et al., 2009; Memarzia et al., 2021; Stallard & Smith, 2007).

Cognitive Theories of PTSD

The cognitive model of psychopathology holds that it is not the events themselves, but the interpretation of events, that causes distress (Beck, 1972; Ellis, 1977). While originally used to formulate and treat depression, the model was later extended to the treatment of various anxiety disorders (Beck & Clark, 1997). Among cognitive theories in the PTSD literature (e.g. Brewin et al., 1996; Ehlers & Clark, 2000; Foa & Rothbaum, 2001), the Ehlers and Clark (2000)'s model is one of the most widely researched ones. According to the model, people with PTSD tend to process the traumatic event and its consequences in a way that produces a sense of ongoing threat. Two factors are proposed to play a role: First, memory of the trauma tends to be fragmented, sensory based, lacking in context, involuntarily triggered and possessing a here-and-now quality. Such disruption of autobiographical memory evokes a strong sense of current threat

to the person. Second, trauma and its sequelae (e.g. flashbacks, numbing and anger outbursts) tend to be appraised in negative, overgeneralizing or catastrophic ways. By way of illustration, one may endorse the beliefs “I attract disasters”, “the world is a dangerous place” and “the next disaster will strike soon” in relation to a traumatic event that has occurred. One may also have such beliefs as “I am going crazy”, “I am never going to recover” and “I have changed for the worst” in relation to one’s reactions to trauma. These maladaptive beliefs consequently fuel one’s sense of threat. In an attempt to contain the threat, one may engage in strategies such as safety-seeking behaviours, cognitive avoidance, rumination and thought suppression. However, despite their short-term benefits, these strategies tend to perpetuate and intensify anxiety in the long term, resulting in ongoing PTSD symptoms (Ehlers & Clark, 2000; Nolen-Hoeksema, 2004; Sibrava & Borkovec, 2006).

Anxiety Sensitivity and PTSD

A cognitive factor that may be conceptually associated with trauma appraisal is anxiety sensitivity. Anxiety sensitivity refers to the fear of anxiety and arousal-related sensations due to the belief that they have detrimental consequences for the individual (Reiss, 1985). According to the Anxiety Sensitivity Index (ASI; Reiss et al., 1986), these perceived consequences can be classified into physical domains (e.g. “When I notice my heart beating rapidly, I worry that I might be having a heart attack”), cognitive domains (e.g. “When I cannot keep my mind on a task, I worry that I might be going crazy”) and social domains (e.g. “Other people notice when I feel shaky”). An additional domain, unsteady concerns (e.g.

“It scares me when I can’t keep my mind on my schoolwork”), was found to be present among children and young people and was hence incorporated in the child adapted version of ASI, i.e. the Childhood Anxiety Sensitivity Index (CASI; Silverman et al., 1991).

As a cognitive construct, its association with panic disorder has been widely established (Donnell & McNally, 1990; Li & Zinbarg, 2007; McNally, 2002; Poletti et al., 2015). This is in line with cognitive models of panic disorder (e.g. Clark, 1986) which consider catastrophic misinterpretations of anxiety-induced bodily symptoms as the core maintaining factor of the disorder. In recent decades, research has begun to explore the role of anxiety sensitivity in other anxiety disorders such as PTSD (Asmundson & Stapleton, 2008; Marshall-Berenz et al., 2010; Olatunji & Wolitzky-Taylor, 2009; Taylor, 2003). A relationship between anxiety sensitivity and PTSD is postulated due to cognitive theories of PTSD regarding the impact of trauma appraisals (e.g. Ehlers & Clark, 2000) as well as high comorbidity rates between

PTSD and panic disorder (Leskin & Sheikh, 2002). Despite its potential significance, however, anxiety sensitivity is less researched than other cognitive factors such as trauma appraisals, rumination and thought suppression. To date this line of research has mostly focused on adults; less is known about how anxiety sensitivity might affect PTSD in the context of children and adolescents. Whilst individual studies (e.g. Hensley & Varela, 2008; Kadak et al., 2013) have reported correlated statistics around anxiety sensitivity and PTSD symptoms in this population, there are yet attempts to aggregate existing various findings around effect size. It remains unclear how strong a role anxiety sensitivity may play overall in contributing and maintaining PTSD symptoms in youths.

Current Review

The current review aimed to conduct a comprehensive search and analysis of the existing empirical studies on anxiety sensitivity and PTSD symptoms among trauma-exposed children and adolescents. To our knowledge, this constitutes the first meta-analysis in the area. Clarifying the relationship between anxiety sensitivity and post-traumatic stress symptoms (PTSS) would not only help enrich existing cognitive theories of PTSD, but also inform the prevention, management and treatment of the condition.

Method

Protocol and Registration

This review was registered with PROSPERO: International prospective register of systematic reviews (ID: CRD42022316095) on 16 March 2022 and reported with reference to the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) Statement (Moher et al., 2009).

Search Strategy

A systematic search was conducted by the primary author (HC) to identify all studies on anxiety sensitivity and PTSD among children and adolescents. Electronic databases included MEDLINE, PsycINFO, CINAHL and PTSD-pubs. Search terms summarized two key domains: anxiety sensitivity and PTSD OR post-traumatic stress OR post traumatic stress OR posttraumatic stress, and were run by “Abstract and Title”, keywords, and Medical Subject headings (MeSH). Initial searches were open to all ages rather than confined to children and adolescents. This ensured the inclusion of studies that assessed both adults and children

but reported on the groups separately. All searches were limited to human studies that were written in the English language and published from 1980 (when the Diagnostic and Statistical Manual of Mental Disorders <DSM> first defined PTSD) to November 2022.

Study Selection

Following the initial search and removal of duplicates, titles and abstracts were screened by the first author (HC) against defined inclusion and exclusion criteria. Among studies with relevant titles and abstracts, further full-text screening was conducted. A randomly selected 25% of the full-text articles were co-screened by a second reviewer (AC) to ensure adherence to the inclusion and exclusion criteria.

Studies were included in the review if they (1) were academic journal articles, doctoral or master’s theses/dissertations, (2) examined trauma-exposed children and adolescents under the age of 18 (in accordance with DSM-5 Criterion A, trauma exposure is defined as exposure to actual or threatened death, serious injury, or sexual violence), (3) adopted cross-sectional or prospective longitudinal designs, (4) assessed severity, symptoms or diagnosis of PTSD with a well-validated assessment measure and (5) contained a correlation analysis between anxiety sensitivity and PTSD, or any statistics that could be converted to correlation statistics (e.g. Cohen’s *d*).

On the other hand, studies were excluded from the review if they (1) were book chapters, clinical trials/treatment studies, reviews/meta-analyses, single case studies, qualitative studies or animal studies, (2) examined individuals over the age of 18, (3) used clinical or treatment-seeking samples of PTSD (due to difficulty in determining the predictive power of anxiety sensitivity with no comparison to individuals with lesser or no symptoms of PTSD), (4) used samples that were selected due to a mental health disorder (e.g. depression, anxiety) or neurodevelopmental condition (e.g. traumatic brain injury, learning disability) and (5) did not include a correlation analysis between anxiety sensitivity and PTSD or any statistics that could be converted to correlation statistics.

Data Extraction

The following data were extracted from each study: (1) article details (e.g. title, authors, year of publication), (2) study design (i.e. cross-sectional vs. prospective longitudinal), (3) demographic information (e.g. sample population description, age, gender), (4) weeks since trauma exposure, (5) types of trauma (i.e. interpersonal vs. non-interpersonal), (6) country (i.e. high income country vs. middle to low income country), (7) nature of PTSD measure (interview-based vs.

questionnaire) and (8) effect size and nature of effect size (e.g. raw correlation, Cohen's *d*, odd ratios).

In the interest of consistency, a number of rules governed the data extraction process. First, should PTSD be reported in both continuous measures (i.e. symptom severity) and dichotomous measures (i.e. diagnosis), effect sizes from continuous measures were prioritised as dichotomisation of data tends to underestimate effect size (Breh & Seidler, 2007). Additionally, for studies that presented multiple correlation statistics at different time points, the one derived at the earliest time point was selected for our analysis (i.e. cross-sectional data were prioritised over longitudinal data).

Effect Size Calculation

Pearson's zero-order correlation coefficient (*r*), due to its wide usage and easy interpretability, was used as the primary estimate of effect size. For studies that reported *t*-tests, ANOVAs or odds ratios, *r* was derived using standardised procedures for transforming effect sizes (Borenstein et al., 2021; Cohen, 1988; Rosnow & Rosenthal, 1996). In accordance with Cohen (1988), a correlation coefficient of 0.1, 0.3 and 0.5 represented small effect, medium effect and large effect respectively. If a study reported more than one effect sizes that fulfilled our aforementioned prioritisation criteria (i.e. derived from continuous measures and at the earliest time point) – for example, when more than one PTSD scales were used – *r*'s were converted to Fisher's *z* to obtain a mean before being transformed back to *r* for analysis (Borenstein et al., 2021).

Quality Assessment

In line with recommended practice, a risk of bias assessment was conducted for all included studies to account for differences in methodological quality (Higgins & Altman, 2008). A quality assessment tool was developed for this analysis with reference to existing checklists such as the Quality Appraisal Checklist for Studies Reporting Correlations and Associations (NICE, 2012) and the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement (Von Elm et al., 2007). The tool consisted of four items concerning (1) sample representativeness (i.e. clear description of ≥ 3 of the following: location, gender, age and ethnicity), (2) appropriate sampling and recruitment (i.e. probability sampling vs. non-probability sampling), (3) non-response bias (i.e. response rate at least 40% or an analysis performed that showed no significant difference in relevant demographic characteristics between responders and non-responders) and (4) reliability of anxiety sensitivity measures (i.e. measure with internal consistency ≥ 0.7 as reported in the paper or measure that is

validated in other peer reviewed papers as having adequate internal consistency). Each item was rated on a “yes” (1) or “no” (0) scale, where a higher total score indicated a higher study quality / lower risk of bias.

To ensure process rigour, in addition to quality assessment by the main author (HC) using the above framework, all included studies were co-rated by a second-rater (AC). Inter-rater reliability was recorded and disagreements in quality rating were resolved by discussion.

Meta-Analytic Method

A meta-analysis was conducted using the R “metafor” (version 2.0.0) package (Viechtbauer & Cheung, 2010) in R (version 4.1.2). Random effects models, which did not assume any common or fixed parameters across studies, were employed to generalise findings beyond the included studies (Cuijpers, 2016; Hedges & Vevea, 1998). For meta-analysis of correlation coefficients, *metafor* undertakes a Fisher transformation of these statistics before the meta-analysis; back-transformed results are reported here. Heterogeneity of effect sizes were assessed using the *Q* statistic (where variation between studies is implied if *Q* is significant, $p < .05$; Higgins & Thompson, 2002) and the *I*² statistic (where *I*² values of 25%, 50% and 75% represent small, moderate and large degree of heterogeneity respectively; Higgins et al., 2003). As recommended by IntHout et al. (2016), 95% prediction intervals were reported alongside 95% confidence intervals to provide better estimates of effect sizes based on study heterogeneity.

Results

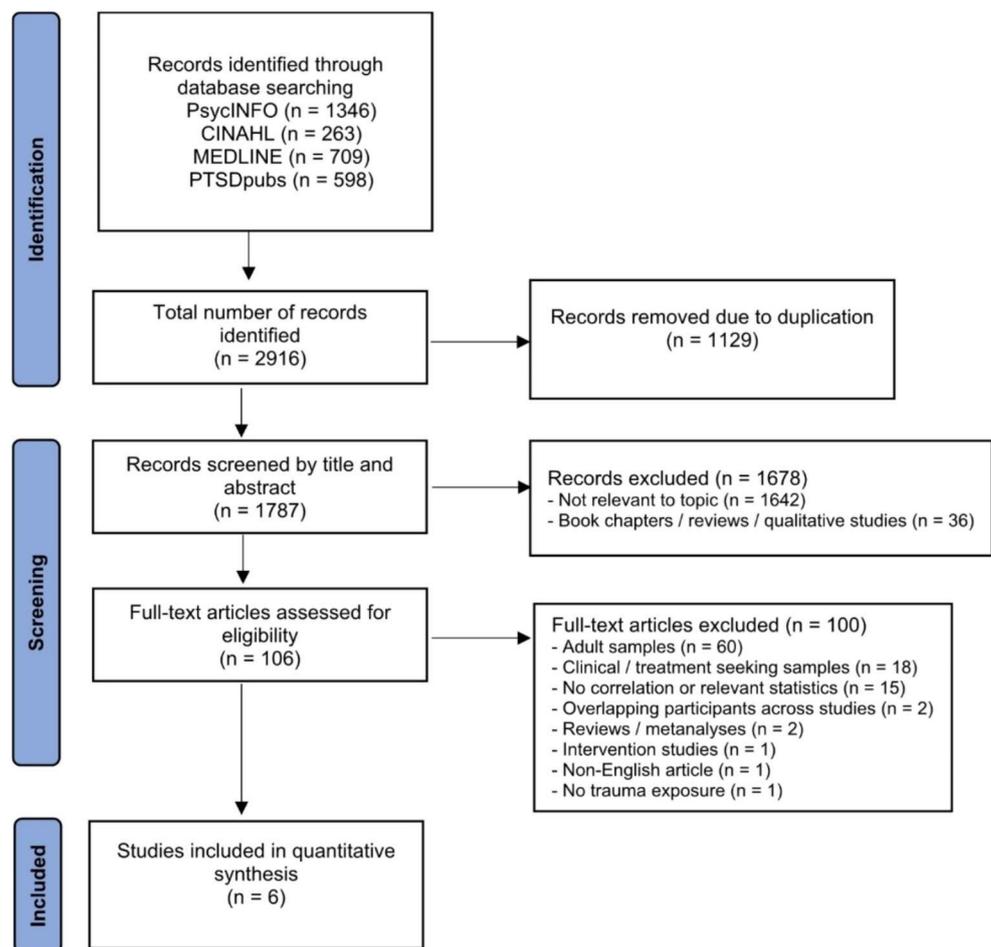
Search Outcomes

The search strategy produced 2916 references. After removing duplicates, 1787 records were screened based on titles and abstracts. The process resulted in the exclusion of 1678 records that were deemed irrelevant. Full-text screening were then conducted with the remaining 106 studies. Six published papers met the inclusion criteria for this systematic review (see Fig. 1). Based on the co-screening results of 25% of full-text articles, the interrater reliability of the full-text screening process was 100%.

Study Characteristics

Table 1 shows the characteristics of the six studies that were included in this review, and Table 2 summarises the key descriptive statistics for these studies. Published between the years 2007 and 2015 across the United States ($k = 2$), the

Fig. 1 PRISMA flow chart



United Kingdom ($k = 1$), the Netherlands ($k = 1$) and Turkey ($k = 2$), the included studies examined a total of 1331 adolescents with non-interpersonal trauma (e.g. earthquakes; $k = 3$) and mixed interpersonal and non-interpersonal trauma ($k = 3$). Participants had a mean age of 14.7 and 48% were females ($n = 639$). In terms of ethnic diversity, two studies were majority White, two studies were moderately diverse (with participants belonging to African American, Caucasian, Hispanic / Latino and Asian / Pacific Islander ethnic groups) and two studies did not comment specifically on the ethnicity of their participants. While most studies took place after the acute stress period (i.e. one month post-trauma; $k = 4$), two studies conducted their data within the acute stress period. All studies were cross-sectional in nature and employed well-validated measures of anxiety sensitivity (i.e. Child Anxiety Sensitivity Index) and PTSD (i.e. Child Posttraumatic Stress Reaction Index, Child PTSD Symptom Scale and Child Revised Impact of Events Scale).

The details of each study are outlined as follows, organized in alphabetical order. Leen-Feldner et al. (2008) examined the relationship between anxiety sensitivity and PTSD in US adolescents aged 10 to 17, who had been exposed to

traumatic events such as natural disasters or witnessing significant injury/death. The findings, based on questionnaires conducted an average of 37 months after the traumatic events, suggested that global anxiety sensitivity and its sub-factors correlated positively with post-traumatic stress levels. Meiser-Stedman et al. (2007) focused on acute stress disorder in youths aged 10 to 16 who attended an emergency department in London after an assault or motor vehicle accident. Within 2 to 4 weeks of the trauma, structured interviews and questionnaires revealed a connection between anxiety sensitivity and diagnosis of acute stress disorder. Verdujin et al. (2015) studied children aged 8 to 17 in the Netherlands and Belgium, who had visited an emergency department following a traumatic event. Questionnaires were conducted 3 weeks after trauma. Results suggested that PTSD symptoms were correlated with anxiety sensitivity, emotional reasoning and subjective trauma severity. Anxiety sensitivity remained significantly correlated with PTSD symptoms 2.5 months after trauma. In Turkey, Kılıç et al. (2008) assessed the role of anxiety sensitivity as a vulnerability factor in the development of PTSD in children aged 8 to 17, five years after experiencing a major earthquake.

Table 1 Characteristics of studies included in the meta-analyses

Article	Trauma type	Recruitment method	Time since trauma	Sample size	Mean Age (SD)	Age range	Female (%)	Race / Ethnicity	Country of study	PTSD measure
Hensley & Varela, 2008	Hurricane	School	5–8 months	302	12.41 (0.94)	10–15	61%	46% AA, 37% Ca, 8% HL, 6% API, 2% NR	US	CPTS-RI
Kadak et al., 2013	Earthquake	School	6 months	738	16.22 (0.88)	13–17	45%	NR	Turkey	CPTS-RI
Kiliç et al., 2008	Earthquake	CS	5 years	81	11.2 (2.2)	8–15	51%	NR	Turkey	CPTS-RI
Leen-Feldner et al., 2008	Mixed	CS	Mixed (Mean: 37 months)	68	14.74 (2.49)	10–17	63%	90% Ca, 6% HL, 4% Asian	US	CPSS
Meiser-Stedman et al., 2007	Assault or MVA	ED	2–4 weeks	93	13.9 (1.9)	10–16	35.5%	56% Black, 33% Ca, 11% Other	UK	CRIES
Verduijn et al., 2015	Mixed	ED	3 weeks	49	13.8 (2.2)	8–17	35%	100% Ca	Netherlands	CPSS

Notes: AA = African American; API = Asian / Pacific Islander; Ca = Caucasian; CPSS = Child PTSD Symptom Scale; CPTS-RI = Child Posttraumatic Stress Reaction Index; CRIES = Child Revised Impact of Events Scale; CS = community survey; ED = emergency department; HL = Hispanic / Latino; MVA = motor vehicle accident; NR = not reported. All studies used the Childhood Anxiety Sensitivity Index (CASI) as the measure of AS.

Their findings, based on questionnaires, indicated that children with more severe PTSD symptoms had higher anxiety sensitivity scores. Hensley & Varela, (2008) explored the relationships between trait anxiety, anxiety sensitivity, PTSD symptoms and somatic complaints in US children aged 10 to 15. Questionnaires were conducted 5 to 8 months after Hurricane Katrina. Their results indicated that anxiety sensitivity accounted for a significant variance in PTSD symptoms, more so than exposure and trait anxiety. Finally, Kadak et al. (2013) investigated whether anxiety sensitivity and metacognitions predicted PTSD symptoms in Turkish high school students aged 13 to 17, 6 months after Van earthquake. Results from their questionnaires revealed that anxiety sensitivity was a significant contributor to the development of PTSD symptoms among these adolescents. Collectively, these studies underscore the significant role that anxiety sensitivity plays in influencing PTSD symptoms in children and adolescents following traumatic events.

Meta-Analyses

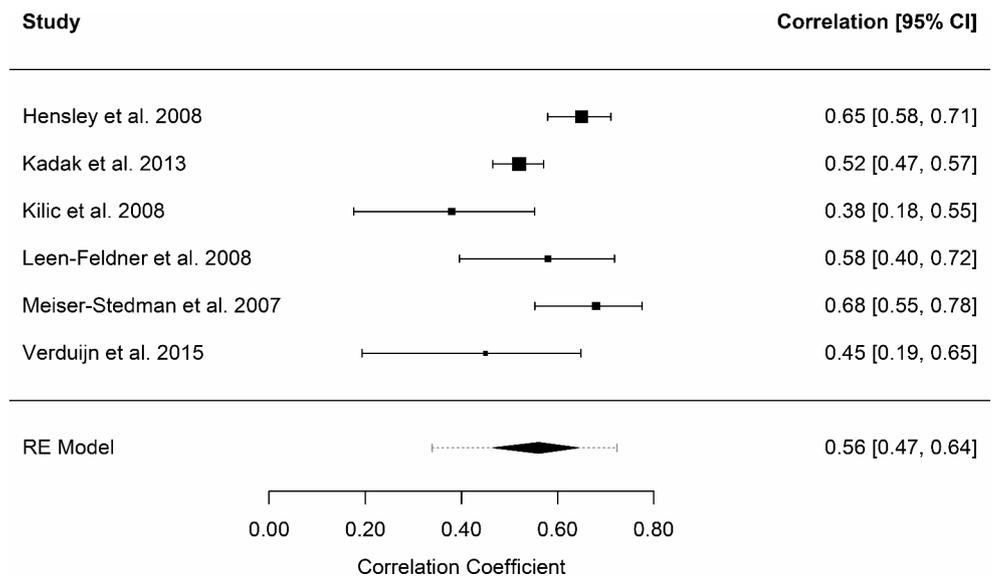
A random-effects meta-analysis of six independent effect sizes indicated a large effect size for the relationship between anxiety sensitivity and PTSS, $r = .56$, 95% CI = 0.47, 0.64. The 95% prediction interval was 0.34, 0.72. Estimates of heterogeneity suggested significant variance across the studies, $Q = 17.2$, $df = 5$, $p < .01$, $I^2 = 72.7\%$. A forest plot of all effect sizes and confidence intervals from each individual study is shown in Fig. 2. With fewer than 10 studies included in the present meta-analysis, tests for funnel plot asymmetry (i.e. publication bias) were not carried out due to insufficient power to distinguish chance from real asymmetry (Borenstein et al., 2009).

Among the effect sizes of the six included studies, two involved the relationship between acute PTSD (i.e. PTSS reported within one month of trauma exposure) and anxiety sensitivity. To explore whether acute PTSD might have affected our results, a random-effects meta-analysis was conducted with the remaining four effect sizes. This produced a similarly large effect size, $r = .55$, 95% CI = 0.44 – 0.64 with the 95% prediction interval being 0.31, 0.72; the significant heterogeneity across studies persisted, $Q = 12.5$, $df = 3$, $p < .01$, $I^2 = 77.6\%$. An additional, unplanned sensitivity analysis was undertaken to consider whether the decision to exclude treatment-seeking samples had an excessively strong impact. Reviewing studies that had been previously been excluded led to only one additional sample (Viana et al., 2018) being added to the meta-analysis. The addition of this extra sample did not significantly shift the above findings ($r = .56$, 95% CI = 0.47, 0.63; 95% prediction interval, 0.36, 0.71; $Q = 17.3$, $df = 6$, $p < .01$, $I^2 = 66.7\%$; for

Table 2 Descriptive statistics for included studies

Article	Mean PTSS score (SD)	Percentage of participants that exceed clinical cut-off for PTSD	Mean CASI score (SD)	Correlation coefficient of PTSS and AS
Hensley & Varela, 2008	NR	71%	NR	0.65
Kadak et al., 2013	NR	40.7%	NR	0.52
Kılıç et al., 2008	NR	90%	32.4 (7.3)	0.38
Leen-Feldner et al., 2008	4.1 (6.5)	NR	28.2 (5.9)	0.58
Meiser-Stedman et al., 2007	NR	19.4%	NR	0.68
Verduijn et al., 2015	6.9 (5.3)	NR	23.1 (2.5)	0.45

Notes: AS=Anxiety Sensitivity; CASI=Childhood Anxiety Sensitivity Index; CPSS=Child PTSD Symptom Scale; CPTS-RI=Child Post-traumatic Stress Reaction Index; CRIES=Child Revised Impact of Events Scale; NR=not reported; PTSD=Post-Traumatic Stress Disorder; PTSS=Post-Traumatic Stress Symptoms; SD=Standard Deviation

Fig. 2 Forest plot

revised forest plot see Supplementary Fig. 1, and Table S3 for revised study characteristics).

Quality Assessment

All six studies were rated against our quality assessment framework, in which higher scores translated to higher study quality (lower risk of bias). Out of a total score of 4, three studies were rated as 2, two studies rated as 3 and one study rated as 4. Inter-rater reliability was calculated for all six study ratings and an agreement rate of 95.8% was found (intra-class correlation coefficient=0.88).

Discussion

This review sought to examine the strength of the relationship between anxiety sensitivity and PTSD symptoms among trauma-exposed children and adolescents. The current findings indicated a strong positive relationship between anxiety sensitivity and PTSD symptoms in this

population. Based on the data of six empirical studies published between 2007 and 2015 ($n=1331$), a large effect size ($r=.56$) was generated. The effect size remained large after excluding studies on acute PTSD ($r=.55$). This was comparable to the findings of Gómez de La Cuesta et al. (2019) which conducted a meta-analysis of the relationship between PTSD and various trauma appraisals.

The current findings are consistent with current cognitive models of PTSD. According to Ehlers and Clark (2000), PTSD could be conceptualised as a sense of current threat maintained by (1) trauma memory and (2) maladaptive appraisals of trauma and its sequelae. Anxiety sensitivity may reflect a pre-trauma vulnerability factor that serves to intensify these trauma-related maladaptive appraisals, especially those in relation to anxiety sensations. This consequently exacerbates one's sense of current threat and PTSD symptoms.

Moreover, anxiety sensitivity could influence PTSD through increasing the use of maladaptive cognitive and behavioural strategies. Given their aversion to anxiety, adolescents with high anxiety sensitivity may be more likely

to employ strategies such as avoidance, thought suppression, selective attention to threat cues and safety behaviours (Ehlers & Clark, 2000). In particular, Wilson and Hayward (2006) found that anxiety sensitivity tends to precede and aggravate avoidance behaviours. While these strategies may bring relief in the short term, they tend to perpetuate anxiety and PTSD in the long term through various mechanisms (Ehlers & Clark, 2000; Beck & Haigh, 2014; Wells & Leahy, 1998).

It is also possible for anxiety sensitivity to contribute to PTSD symptoms indirectly through panic attacks. As outlined above, anxiety sensitivity is a strong predictor of panic disorder (Donnell & McNally, 1990; Li & Zinbarg, 2007; McNally, 2002; Poletti et al., 2015) as in line with cognitive models of panic disorder (e.g. Clark, 1986). Both peritraumatic and posttraumatic panic attacks were found to predict subsequent development of PTSD (Boscarino & Adams, 2009; Nixon & Bryant, 2003). In other words, anxiety sensitivity may predispose one to experience panic attacks during and after trauma, which in turn maintain and intensify symptoms of PTSD such as avoidance and hypervigilance.

The fact that there were only six studies in our meta-analysis appeared to suggest a gap in the literature around anxiety sensitivity and PTSD in youth; more research is clearly warranted. That said, the present findings may have some preliminary implications for the assessment, prevention and treatment of PTSD among children and adolescents. Given the strong association between anxiety sensitivity and PTSD both during and beyond the acute one-month period, post-trauma anxiety sensitivity level could potentially inform the screening of at-risk youths at various time points. For those assessed to have high risks of developing PTSD, brief intervention targeting anxiety sensitivity (e.g. psychoeducation on anxiety and its function) might prove to be useful. Furthermore, given the malleable nature of the anxiety sensitivity construct (Keough & Schmidt, 2012; Smits et al., 2008; Vujanovic et al., 2012), anxiety sensitivity could potentially be a target for treatment for those diagnosed with PTSD. Specifically, alongside psychoeducation, intervention such as interoceptive exposure could be considered due to its direct impact on anxiety sensitivity (Smits et al., 2008). Other interventions such as mindful awareness of bodily sensations and behavioural experiments around the effects of anxiety could also be beneficial, although more research would be needed to examine their roles in anxiety sensitivity (Keough & Schmidt, 2012; Smits et al., 2008).

Strengths, Limitations and Future Research Directions

The present review was strengthened by the pre-registration of its protocol, homogeneity in anxiety sensitivity measure

(i.e. CASI) and large sample size of certain studies (e.g. Kadak et al., 2013). There was also considerable ethnic diversity across study samples (comprising Asian, Caucasian and Black children and adolescents) which increased the generalisability of our findings.

The review is however not without its limitations. A major limitation is the small number of studies included. This made it difficult to detect publication bias or to conduct moderator analyses around factors such as demographics, trauma types (interpersonal trauma versus non-interpersonal trauma), PTSD measures (interview versus self-reported questionnaire) and country of study (high income country versus low to mid income country). Second, most of our studies were conducted in developed countries. This limits the generalisability of our findings to developing countries where trauma exposure could potentially be higher. Third, as our data were cross-sectional in nature, it is difficult to draw definitive conclusions around causality. It may be that experiences of trauma affect anxiety sensitivity as much as the other way round (although in either case, anxiety sensitivity remains a potentially important target for treatment given its malleable nature). Lastly, as the included studies focused predominantly on PTSD in the context of single traumatic events, it remains uncertain whether the present findings could be generalised to those with experiences of complex trauma such as childhood abuse.

To fill the above knowledge gaps, more research is required. Future studies could employ prospective longitudinal designs to complement existing cross-sectional findings. They could also recruit youths with complex trauma histories (e.g. abuse, maltreatment). As the literature around the topic becomes larger, moderator analyses would be possible in future systematic reviews and meta-analyses. This may help create a more nuanced understanding of when and under what circumstances anxiety sensitivity influences PTSD and thereby constitutes an effective intervention target.

Conclusion

The current review indicated a large effect size for the relationship between anxiety sensitivity and PTSD symptoms among children and adolescents exposed to trauma. However, this was based on a limited number of studies, and more research is clearly warranted.

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Declarations

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