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Rates of post-traumatic stress disorder in trauma-exposed children and adolescents: meta-analysis

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Background

It is unclear how many children and adolescents develop post-traumatic stress disorder (PTSD) after trauma.

Aims

To determine the incidence of PTSD in trauma-exposed children and adolescents as assessed with wellestablished diagnostic interviews and to examine potential moderators of the estimate.

Method

A systematic literature search identified 72 peer-reviewed articles on 43 independent samples (n = 3563). Samples consisting only of participants seeking or receiving mental health treatment were excluded. Main analyses involved pooled incidence estimates and meta-analyses of variance. **Results**

The overall rate of PTSD was 15.9% (95% CI 11.5–21.5), which varied according to the type of trauma and gender. Least at risk were boys exposed to non-interpersonal trauma (8.4%, 95% CI 4.7–14.5), whereas girls exposed to interpersonal trauma showed the highest rate (32.9%, 95% CI 19.8–49.3). No significant difference was found for the choice of assessment interview or the informant of the assessment.

Conclusions

Research conducted with the best available assessment instruments shows that a significant minority of children and adolescents develop PTSD after trauma exposure, with those exposed to interpersonal trauma and girls at particular risk. The estimates provide a benchmark for DSM-5 and ICD-11. **Declaration of interest**

None.

It is unclear how many children and adolescents develop post-traumatic stress disorder (PTSD) after exposure to trauma. In a summary of the literature, one investigator noted that rates ranged from 0% to 100%.¹ A meta-analysis conducted in 1994 estimated that 36% of children exposed to trauma went on to develop PTSD;² however, the inclusion criteria and analytic strategy of this meta-analysis are unknown, and many new studies have since been conducted. The wide variability in estimates suggests that moderators have a role. For example, interpersonal trauma (e.g. assault, war) is thought to result in higher rates of PTSD than non-interpersonal trauma (e.g. accident, natural disaster).^{3,4} Evidence also suggests that girls are more likely to develop PTSD than boys,^{4,5} although this may be related to differences in type of exposure.⁶ Furthermore, studies show that parent–child agreement regarding PTSD symptoms is relatively poor,^{7,8} indicating a need to assess differences across informants. Finally, the specific instrument used to assess PTSD may have a role.⁹ We aimed first to establish an estimate of the risk of DSM-IV PTSD among children and adolescents exposed to trauma, based on well-established diagnostic interviews; second, to examine potential moderators (i.e. type of trauma, gender, informant and diagnostic measure) that could affect this estimate; and finally, if group differences were found, to report separate estimates of PTSD rates. A precise estimate of the proportion of children developing PTSD after traumatic exposure would allow for a better appraisal of the need for mental health resources, better allocation of these resources to those most in need and a better design of new studies in youth (e.g. with respect to power of the analyses). It would also provide a benchmark for evaluating the impact of the new DSM-5 criteria on rates of PTSD among traumatised children and adolescents.

Method

To obtain the most accurate PTSD rates we targeted studies that applied widely used and wellestablished diagnostic interviews for childhood PTSD according to DSM-IV criteria. Based on three reviews of PTSD measures for children and adolescents,^{5,10,11} we included the Clinician Administered PTSD Scale for Children and Adolescents (CAPS-CA), the Anxiety Disorders Interview Schedule for DSM-IV – Child version (ADIS-C), the Diagnostic Interview for Children and Adolescents – Revised (DICA-R), the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime version (K-SADS) and the Children's PTSD Inventory (CPTSDI).^{12–16}

Relevant studies were identified through systematic searches in four electronic databases – PubMed, EMBASE, PsycINFO and the Published International Literature on Traumatic Stress (PILOTS) – and reference lists of systematic reviews on child trauma.^{2,17–22} Electronic searches included the following keywords, with syntax adapted to the specified databases: ["Clinician-Administered" OR "CAPS-CA" OR "CAPS-C" OR "CAPS" OR "KSADS" OR ("Kiddie Schedule") OR "K-SADS" OR "KSADS" OR ("Kiddie SADS") OR "ADIS-C" OR "ADISC" OR ("Anxiety and Depression Interview Schedule") OR ("Diagnostic Interview for Children") OR "DICA" OR "DICA-R" OR "DICAR" OR "CPTSDI" OR ("PTSDI" AND Saigh) OR ("PTSD Inventory")] AND ["PTSD" OR ("Post-traumatic stress disorder") OR "Posttraumatic stress disorder")] AND [child OR children OR adolescents OR adolescent OR youth OR youths OR youngster OR youngsters OR toddler OR toddlers OR infant OR infants OR kid OR kids OR teen OR teens OR teenager OR teenagers OR preschooler OR preschoolers]. We restricted searches to empirical English-language papers with at least 10 participants (to avoid selection bias in case series), published in peer-reviewed journals between 1994 (when DSM-IV was published) and 1 October 2012. In addition the studies had to satisfy the following criteria:

(a) the study participants were all exposed to trauma as defined by the A1 criterion for PTSD in DSM-IV, or separate data for this group were available;

(b) the study participants were less than 19 years old at the time of the PTSD measurement;

(c) the study participants did not represent a clinical sample with respect to mental health (e.g. psychiatric in-patients or a sample of children with post-traumatic stress symptoms seeking mental healthcare);

(d) the study protocol did not include a psychological or psychopharmacological intervention (i.e. potentially attracting participants with higher levels of distress);

(e) the study examined PTSD diagnosis at least 1 month after the trauma, according to DSM-IV criteria, with one of the five specified interviews or one of their revisions;

(f) the study did not have the psychometric evaluation of the diagnostic interview as its sole purpose;

(g) the article and/or the study author(s) provided enough information to derive the percentage of children who satisfied the criteria for PTSD diagnosis.

Screening and selection of studies (see Fig. 1) were conducted by E.A. and a trained research assistant, with differences and questions being resolved through consultation with at least one other member of the research team. We obtained full-text articles for all studies that were potentially relevant. In the few situations where eligibility remained unclear based on the article, we contacted the study authors for additional information.

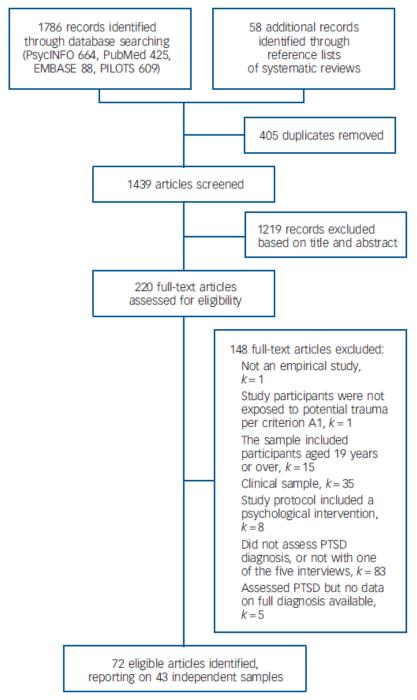


Fig. 1 Selection of samples (PTSD, post-traumatic stress disorder).

Coding of studies

Each study was coded based on consensus by at least three members of the research team. In addition to the publication details of each study, we extracted information on the sample, the nature of trauma exposure, the measurement of PTSD and the outcomes of the PTSD assessment (the coding manual is available from E.A.). Study authors were contacted to confirm codes and provide any coding information that was not included in the articles.

Sample characteristics

We recorded country of data collection, the number of children and adolescents who participated in the PTSD assessment, age of the sample (range, mean, standard deviation), the percentage of boys and any exclusion criteria that the authors applied.

Exposure characteristics

We noted a short description of the event and the type of exposure. The pre-specified types were disaster, war, terrorism, (injury due to) accident, (injury due to) violence, life-threatening disease, sudden death of a loved one, 'mixed' with violence, 'mixed' without violence. This variable was used to derive two categories: interpersonal trauma – war, terrorism, (injury due to) violence and 'mixed' with violence – and non-interpersonal trauma – disaster, (injury due to) accident, life-threatening disease, sudden death of a loved one and 'mixed' without violence.

PTSD measurement

We documented the number of times that PTSD was measured, the timing of these measurements (mean number of months post-trauma and range), the informant (child, parent, combination or other) and the clinical interview used.

Outcomes of the assessment

We recorded the number of children and adolescents with full PTSD according to DSM-IV and how many of them were boys. In cases of multiple measurements within one study, we recorded the information on the first eligible wave (i.e. at least 1 month post-trauma).

Statistical analysis

The analyses were performed using Comprehensive Meta-Analysis, SPSS version 19.0 for Windows Vista and the macros provided by Wilson.23 We used random effects models to compute all pooled estimates based on the assumption that true effect sizes are likely to vary beyond subject-level sampling error.²⁴ The first stage of our data analysis involved determining an overall pooled incidence estimate of PSTD in children and adolescents who were exposed to a traumatic event, based on all included studies. Estimates of the proportion of traumatised youth with PTSD were transformed into logits for better estimation prior to the calculations and transformed back to proportions afterwards for ease of interpretation (Lipsey & Wilson: pp. 39–40).²⁴ When outcomes in individual studies equalled 0%, we added 0.5 to both cells (containing frequencies of events and non-events) before applying the logit transformation. To scan for possible outliers, we made a box plot. All observations more extreme than 1.5 times the interquartile range were marked as outliers. Following outlier detection we performed a sensitivity analysis to investigate the influence of the outliers. Next, tests of heterogeneity (Cochran's Q) were performed to determine whether differences in estimates across studies were greater than expected by chance. We also evaluated possible publication bias by inspection of a funnel plot. Because the Q-test was significant we subsequently evaluated the sources of variability in the pooled incidence estimate. We performed meta-analyses of variance (ANOVAs) for all moderators of interest (type of

trauma, gender, choice of interview, informant), calculating pooled estimates for all group levels. To test for significant differences in pooled incidence estimates between groups we used a Q-between test (Lipsey & Wilson: p. 136).²⁴ Whereas type of trauma, choice of interview and informant were between-sample moderators, gender was a within-sample moderator. Therefore, we created separate boys and girls 'samples' based on the numbers of boys and girls in each sample and the number of PTSD diagnoses for them.

Results

We retrieved 72 articles describing 43 independent samples, denoted as k (Fig. 1). In total they reported PTSD assessments for 3563 children and adolescents exposed to trauma as defined by criterion A1 of DSM-IV. All samples also met the DSM-5 exposure criterion. An overview of the samples and their references are given in online Table DS1. Child ages varied from 2 years to 18 years and approximately 57% of the children were boys (not reported for two samples). Most samples originated in the USA (k = 20; 47%), followed by the UK and Australia (both k = 5; 12%). Three samples came from non-Western countries (Afghanistan, China and South Africa). The children had been exposed to a variety of events, including motor vehicle accidents, the sudden loss of a parent, life-threatening illness, war experiences, domestic violence and child maltreatment. About half of the samples (k = 22; 51%) had been exposed to non-interpersonal trauma and the other half (k = 21; 49%) to interpersonal trauma or to a mix of both. The studies applied a range of exclusion criteria. Frequently excluded were participants with cognitive impairments (k = 25; 58%), insufficient language skills (k = 13; 30%), prior trauma (k = 9; 21%) and current or prior mental health problems (k = 9; 21%). The most commonly used instrument to assess PTSD was the CAPS-CA (k = 19; 44%) and the least used was the CPTSDI (k = 4; 9%). In the majority of the samples (k = 31; 72%) the children were the informants as opposed to the parents (k = 4; 9%). In 8 samples (19%) a combined reporting strategy was used. Reported rates of PTSD ranged from 0% to 89%.

Outlier analysis, sensitivity analyses and publication bias

One study was detected as an outlier on the box plot.²⁵ This study included a sample of youths highly exposed to interpersonal trauma. Sensitivity analysis revealed that without this study the pooled incidence estimate dropped from 16.9% (95% Cl 12.1–23.2) to 15.9% (95% Cl 11.5–21.5%). We performed the remainder of the analyses without this observation. We assessed possible publication bias (i.e. the preferential publication of striking findings, in this case high PTSD rates) by inspection of a funnel plot. Although the plot was asymmetrical, this asymmetry was not consistent with publication bias, as smaller studies tended to yield lower estimates of PTSD. Additional sensitivity analyses included assessment of the influence of each study on the overall estimates of PTSD rates by recalculating the pooled outcome proportions with one study removed and all others included. These analyses yielded PTSD estimates ranging from 15.1% (95% Cl 11.0–20.5) to 16.6% (95% Cl 12.1–22.4). Pooled incidence estimate For the overall sample (k = 42) we found that 15.9% (95% Cl 11.5–21.5) of the children and adolescents exposed to a traumatic event developed PTSD (Fig. 2). The Q-test for pooled estimates was significant (Q= 495.5, d.f.= 41; P50.001), indicating heterogeneity between studies. We therefore proceeded to conduct moderator analyses.

DTCD	Data		OFO	~
PTSD	Rate	and	95%	CI

Study

0100)	100 110	co arra .			
	%	95	% CI		
Abroad at al (2000)44	9.1	4.1	18.8		
Ahmad <i>et al</i> (2000) ⁴⁴ Bal <i>et al</i> (2004) ⁴⁵	50.0	40.3	59.7		
Bayarri Fernandez	50.0	40.5	57.7		
et al (2011) ⁴⁶	6.3	3.3	11.6		
Bosquet Enlow et al	0.5	3.5	11.0	■	
(2010) ⁴⁷	17.0	10.8	25.7		
Bui et al (2010) ^{48,4}	4.9	2.1	11.2		
Catani <i>et al</i> (2009) ⁵⁰	4.7	30.4	66.4		
Chemtob & Carlson	40.1	30.4	00.4		
(2004) ⁵¹	41.7	24.1	61.7		
Daud et al (2005) ^{52,53}	48.9	34.8	63.2		
Daviss et al (2000) ⁵⁴	12.5	5.7	25.2		
Dolohopty of al	12.0	5.7	20.2	-	
Delahanty et al (2005) ^{55–58}	6.6	2.8	14.9		
Erickson <i>et al</i> (2008) ⁵⁹	13.8	5.3	31.5		
Iselin et al (2010) ^{60,61}	4.3	2.2	8.5		
Ji et al (2010) ⁶²	5.3	3.4	8.2	12 1 1	
	5.5	0.4	0.2		
Jones-Alexander et al (2005) ⁶³	23.8	10.3	46.0		
Kenardy <i>et al</i> (2006) ⁶⁴	23.0	10.5	40.0		
(partly ^{65–68})	0.7	0.1	4.9		
(partiy at al (2004)64	0.7	0.1	4.9		
Kenardy et al (2006) ⁶⁴	2.2	0.6	8.5		
(partly) Kolko (1998) ^{69,70}	23.3	15.7	33.2		
Landolt et al (1998) ⁷¹	52.2	32.5	71.2		
	18.6	9.6	33.0		
Landolt <i>et al</i> (2009) ⁷² Lemos-Miller	10.0	9.0	33.0		
	35.6	26.4	45.9		
& Kearney (2006) ³⁷	33.0	20.4	40.7		
Linning & Keamey (2004) ⁷³	67.3	53.9	78.3		
(2004)	07.5	55.7	10.5		
MacMillan et al (2009) ^{74,75}	26.2	16.9	38.1		
Mather et al (2003) ⁷⁶	41.9	28.2	56.9		
	2.8	1.1	7.3		
Max et al (2011) ⁷⁷ Maisor Stadman	2.0	1.1	1.5		
Meiser-Stedman et al (2005) ^{78–82}	12.5	6.4	23.1		
Meiser-Stedman	12.0	0.4	23.1		
et al (2008a) ⁷	1.7	0.2	10.9		
Meiser-Stedman	1.7	0.2	10.7		
et al (2008b) ⁷	18.8	10.1	32.3		
	10.0	10.1	32.3		
Melhem <i>et al</i> (2007) ^{83–85}	8.8	5.5	13.9		
Nixon <i>et al</i> (2010) ⁸⁶	4.7	1.8	11.7		
	4.7	1.0	11.7		
Ostrowski et al (2007) ^{87,88}	2.0	0.3	12.9		
Ostrowski et al (2011) ⁸⁹	0.5	0.0	7.5	_	
Pervanidou et al (2007) ⁹⁰⁻	92 40 4	28.5	53.5		
Pfeffer <i>et al</i> (2007) ⁹³	28.9	17.6	43.6		
Pine et al (2005) ^{94–96}	55.2	46.1	64.0		
	5.3	0.7	29.4		
Rees et al (2004)97 Responsed al (2012) ³⁶	12.5	4.8	28.9		
Rosner et al (2012) ³⁶	29.0	19.1	41.4		
Samuelson et al (2010) ⁹⁸ Schäfer et al (2006) ⁹⁹	0.7	0.0	10.4		
Stallard at al (2006)	35.3	27.2	44.4		
Stallard et al (1998) ^{100,101}	35.3 22.0	12.6	35.5		
Suliman et al (2005) ³⁸	35.3	12.6	35.5 59.6		
Vanderbilt et al (2008) ¹⁰²	30.3)6 E 4				
Winston et al (2003) 103-10	5.4	3.0	9.5		
Overall/Pooled	15.9	11.5	21.5		
				0% 50% 100%)
Fig 2 Forest plat of po			+	licardar (DTCD) ratas in in	

Fig 2 Forest plot of post-traumatic stress disorder (PTSD) rates in individual samples (for full references, see online data supplement).

Moderator analyses

We conducted meta-ANOVAs to test differences in the pooled incidence estimate based on type of trauma, gender, choice of diagnostic interview and informant of the assessment (Table 1). The PTSD rate following non-interpersonal trauma was 9.7% (95% CI 6.1–15.2), whereas following interpersonal trauma it was 25.2% (95% CI 16.8–35.8), a significant difference (P = 0.002). Boys developed significantly less PTSD (11.1%, 95% CI 7.0–17.1) than girls (20.8%, 95% CI 13.6–30.5; P = 0.04). Table 1 also shows the PTSD rates for type of trauma by gender. Boys exposed to non-interpersonal trauma showed the lowest rates of PTSD (8.4%, 95% CI 4.7–14.5) whereas girls exposed to interpersonal trauma showed the highest rates (32.9%, 95% CI 19.8–49.3); see Fig. 3 for forest plots of the subgroups. There was no significant difference in PTSD rates related to the informant or to the diagnostic interview used for the assessment.

Predictor	Rate % (95% Cl)		Between-group homogeneity		
		k ^a	Q	d.f.	Р
Type of trauma					
Non-interpersonal	9.7 (6.1–15.2)	22	9.66	1	0.002
Interpersonal	25.2 (16.8–35.8)	20			
Gender					
Boys	11.1 (7.0–17.1)	30	4.11	1	0.043
Girls	20.8 (13.6-30.5)	31			
Type of trauma by gender ^b					
Boys – non-interpersonal	8.4 (4.7–14.5)	18	13.03	3	0.005
Boys – interpersonal	16.8 (8.8–29.6)	12			
Girls – non-interpersonal	13.3 (7.4–22.9)	18			
Girls – interpersonal	32.9 (19.8–49.3)	13			
Choice of diagnostic interview					
ADIS-C	9.8 (4.1–21.4)	8	8.17	4	0.086
CAPS-CA	12.1 (7.2–19.7)	18			
DICA-R	19.0 (7.6–40.0)	5			
K-SADS	22.6 (11.4–39.8)	8			
CPTSDI	51.4 (17.4–84.1)	2			
Informant					
Child	17.3 (11.8–24.7)	30	3.53	2	0.171
Parent	5.1 (1.3–17.4)	4			
Combined	13.4 (5.7–28.4)	7			

Table 1 Meta-analyses of variance with post-traumatic stress disorder rate as outcome variable

ADIS-C, Anxiety Disorders Interview Schedule for DSM-IV – Child version; CAPS-CA, Clinician Administered PTSD Scale for Children and Adolescents; CPTSDI, Children's PTSD Inventory; DICA-R, Diagnostic Interview for Children and Adolescents – Revised; K-SADS, Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime version.

a. Number of samples.

b. Post hoc analyses involving the samples for which we had separate information on boys and girls (k = 31) showed a significant difference between non-interpersonal and

interpersonal trauma in the girls' samples (Q= 5.31, d.f. = 1, P = 0.021) and a trend in the boys' samples (Q= 2.77, d.f. = 1, P = 0.096). There was no significant difference between

boys and girls for the non-interpersonal trauma samples (Q= 1.23, d.f. = 1, P = 0.268) and a trend for the interpersonal trauma samples (Q= 3.06, d.f. = 1, P = 0.080).

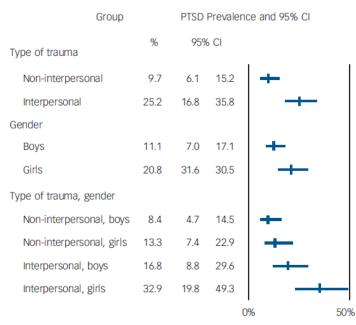


Fig 3 Forest plot of subgroups (PTSD, post-traumatic stress disorder).

Discussion

There has been substantial uncertainty regarding the incidence of PTSD in children and adolescents exposed to trauma. Our meta-analysis summarises the evidence collected with well-established diagnostic interviews. Our findings indicate that, overall, approximately one in six children and adolescents (16%) developed PTSD after exposure to a DSM-IV criterion A1 or DSM-5 trauma. There was considerable variation in this rate based on the type of trauma: approximately one in ten developed PTSD after non-interpersonal trauma, whereas one in four developed PTSD after interpersonal trauma. Variation was also related to gender, with girls being at higher risk than boys. The overall rate of 16% is lower than the estimate of 36% reported in a previous review.² However, given that the book chapter reporting the estimate did not describe the method used, it is difficult to compare the two findings. The sizeable difference may be related to assessment methods: whereas we focused on diagnoses made through clinical interviews, it appears that the previous review also included rates based on scores above cut-off on self-report questionnaires. The latter have been shown to overestimate PTSD in adults.⁹ Nevertheless, 16% represents a significant minority of children and indicates that the full burden of trauma, including other mental health consequences such as generalised anxiety disorder, depression and separation anxiety disorder,²⁶ is substantial.

Consistent with findings in the adult literature,^{27,28} the most prominent moderator of PTSD rates was the type of trauma. Interpersonal trauma may lead to higher rates of PTSD because it is more often chronic, erodes social support (in cases where the perpetrator is a family member), leads to more selfblame or other maladaptive cognitions,⁶ represents a 'betrayal' of trust,²⁹ or more clearly 'shatters assumptions' about the world in ways that affect daily functioning.³⁰ Our findings suggest that screening and treatment resources will in particular need to be allocated to children exposed to interpersonal trauma. In addition, the time lag between exposure and assessment (without structured psychological care in between) was often large for these samples, underlining a need for early detection of both exposure and mental health problems. Especially in childhood and adolescence, when the risk of a cascade of disruptions in development is high,^{4,31-33} timely intervention is essential. Girls were more likely than boys to develop PTSD following trauma exposure. This might be partially due to their greater exposure to interpersonal trauma.²⁷ However, researchers have generally noted that although differences in rates of interpersonal trauma contribute to different rates of PTSD, they do not fully explain the trend.⁶ This finding is consistent with our observations. Future research should explore explanations for these differences. These explanations might include boys' higher likelihood of engaging in externalising rather than internalising behaviours following trauma;⁶ girls' higher rates of internalising disorders in general (both before and after trauma);³⁴ girls' stronger self-blaming or threat appraisal;⁶ girls' experience of peritraumatic dissociation or girls' increased hypothalamic–pituitary–adrenal (HPA) axis dysregulation.²⁷ When more studies in different age ranges are available it will be interesting to test whether gender differences arise around pre-adolescence, given that gender differences in other internalizing disorders tend to surface around this time.³⁴

Study limitations

The limitations of our study need to be considered. First, because of our decision to include only studies applying well-established interviews, children traumatised by war or disaster were underrepresented. Our findings will need to be compared with the best available assessments in these samples in the future. Second, although we have used the term 'incidence' for the proportion of children who developed PTSD after a traumatic event, it could be argued that we have measured point prevalence in a specific group;³⁵ most primary studies were cross-sectional in nature and did not assess whether children had recovered from PTSD between exposure and assessment. Third, it was not possible to examine time since trauma as a potential moderator. Many studies, in particular those after interpersonal trauma, reported a wide variability in the timing of the assessments,^{36–38} and using the sample mean in this case would be vulnerable to aggregation bias.³⁹ Visual inspection of the few studies with multiple time points indicated a slight decrease of PTSD rates over time. Fourth, although we applied quite strict inclusion and exclusion criteria, some potential forms of bias or confounding (e.g. with the chronic nature of certain types of trauma and the way they are detected) could not be ruled out and will need to be taken into account in the future. In particular, more than half of the samples excluded participants with cognitive limitations and about one in four samples excluded children and adolescents with a current or prior mental health diagnosis (ranging from depression to psychosis), medication or trauma history. Given the known vulnerability of these children,^{40,41} the included studies may have underestimated the true PTSD incidence rates following exposure.

Implications

With the release of DSM-5 and the imminent release of ICD-11, this study may serve as a benchmark for forthcoming research on childhood PTSD. At least one study has found that, compared with DSM-IV, the DSM-5 criteria may lead to similar or slightly lower PTSD rates in adults,⁴² but we have yet to determine what this will mean for child populations. In particular, earlier criticisms of the diagnosis related to its lack of child-centredness,⁴³ and led to the inclusion of the new subtype 'pre-school PTSD', which may yield larger detection rates. For example, Meiser-Stedman et al reported an almost six-fold increase (from 1.7% according to DSM-IV to 10.0% according to an algorithm similar to 'pre-school PTSD') for a sample of children 2–6 years old exposed to motor vehicle accidents.⁷ The ICD has been seen as more childfriendly but has served less often as a basis for child assessment in studies (hence our focus on DSM). It will be important for future research to determine whether the DSM-5 and the ICD-11 criteria align well with children's functional outcomes. Until then, our findings provide critical information regarding expected rates of PTSD among trauma-exposed children and adolescents.

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