Building child trauma theory from longitudinal studies: A meta-analysis

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ABSTRACT

Many children are exposed to traumatic events, with potentially serious psychological and developmental consequences. Therefore, understanding the development of long-term posttraumatic stress in children is essential. We aimed to contribute to child trauma theory by focusing on theory use and theory validation in longitudinal studies. Forty studies measuring short-term predictors and long-term posttraumatic stress symptoms were identified and coded for theoretical grounding, sample characteristics, and correlational effect sizes. Explicit theoretical frameworks were present in a minority of the studies. Important predictors of long-term posttraumatic stress were symptoms of acute and short-term posttraumatic stress, depression, anxiety, and parental posttraumatic stress. Female gender, injury severity, duration of hospitalization, and elevated heart rate shortly after hospitalization yielded small effect sizes. Age, minority status, and socioeconomic status were not significantly related to long-term posttraumatic stress reactions. Since many other variables were not studied frequently enough to compute effect sizes, existing theoretical frameworks could only be partially confirmed or falsified. Child trauma theory-building can be facilitated by the development of encouraging journal policies, the use of comparable methods, and more intense collaboration.

INTRODUCTION

When children are confronted with trauma, caregivers and professionals search for ways to prevent or reduce long-term distress. Many children are exposed to traumatic events and their psychological and developmental consequences can be serious.[1] As the occurrence of severe distress after trauma appears to be a non-random phenomenon,[2] solid knowledge of risk factors, protective factors and the mechanisms by which they influence posttraumatic stress in children is needed. The purpose of this paper is to contribute to this knowledge base by focusing on theory use and theory validation in a meta-analytic approach.

Traumatic exposure involves a confrontation with actual or threatened death, serious injury, or other threat to physical integrity.[3] Examples include natural disasters, serious accidents, (mass) violence, and sudden loss of a loved one. Trauma exposure is fairly prevalent in children. Peacetime general population studies reported rates of exposure to any traumatic event from 14%[4] to more than 65%.[5,6] In addition, in a number of countries large populations, including children, are subjected to war.[7] Although it was previously thought that trauma caused only transient distress in children, it is now generally accepted that it can cause severe and long-term impairment.[8] The most studied psychological consequences of traumatic exposure in children are posttraumatic stress and its pathological extremity, posttraumatic stress disorder (PTSD[3]).

PTSD is characterized by overwhelming feelings of reexperiencing the traumatic event (e.g., nightmares and intrusive thoughts), by the avoidance of stimuli and emotional numbing (e.g., avoiding places related to the event and feeling detached from others), and by symptoms of hyperarousal (e.g., concentration difficulties and hypervigilance[3]). As was posited in the 1980s by Terr[e.g., 9] and confirmed many times since then, posttraumatic stress occurs not only in adults but also in children. Children can suffer from PTSD for many years[10] which affects their well-being and development in emotional, social, academic, as well as physical domains.[1,8,11,12]

Fortunately, most children who have been exposed to trauma do not develop PTSD. Estimations vary widely, but in a synthesis of 34 studies, 64% of the children who were exposed to trauma did not develop PTSD (Fletcher, 2003).[13] Nevertheless, about one in three children did, and several researchers suggest that subclinical levels of PTSD also cause severe impairment and distress.[14] Understanding the mechanisms underlying the considerable individual variability in psychological responses to trauma[2] is valuable both for the identification of children at risk for long-term distress and for the development of effective treatment programs. Which factors cause severe distress and which factors are levers that can be used to reduce symptoms and successfully strengthen children?

Theoretical views on posttraumatic stress in children

Although several theories have been proposed to explain the development of PTSD in adults (for an overview see Brewin & Holmes[15]) these cannot readily be applied to children. Children are thought to respond to traumatic events in a somewhat different way from how adults react (see e.g., Kenardy et al.[16]; Salmon & Bryant[17]). For example, children have a more limited knowledge base than adults. This may result in the failure to appraise an experience accurately, potentially influencing the memory of the experience and children's emotional response to it,[17] in a different way than it would influence adults. Also, children's ability to use various coping strategies to regulate emotion is likely to be influenced by their advances in development, such as their understanding of emotion (e.g., Gross & Thompson[18]; Salmon & Bryant[17]). In addition, young children appear to rely heavily on how their parents deal with stress.[19] Therefore, their adjustment to trauma is influenced differently by their environment than is adults' adjustment.

Although compared with adults little theory has been developed to understand childhood posttraumatic stress,[17] a number of conceptualizations have emerged. For example, La Greca, Silverman, Vernberg, & Prinstein[20] modeled responses to natural disaster. They identified exposure characteristics (e.g., life threat during the event and loss or disruption following the event), pre-existing child characteristics (e.g., gender, ethnicity, age), the post-disaster recovery environment (e.g., major life events, social support) and the coping skills of the child as important factors influencing children's posttraumatic reactions. Pynoos, Steinberg, and Piacentini[21] described a model that distinguishes between children's acute distress and longer-term adjustment after traumatic exposure. Short-term reactions are thought to be related to the experience of the trauma and influenced by a) proximal trauma reminders (e.g., media coverage), b) proximal secondary stresses (e.g., loss of resources), c) the ecology of the child (e.g., parental psychopathology), and d) child intrinsic factors (e.g., temperament). Children's ongoing adjustment is further related to e) ongoing reminders of the trauma, f) persistent secondary stressors, and g) related or sequential traumatization.

While the two models outlined above provide an overview of factors influencing posttraumatic stress in children, several authors have zoomed in on specific mechanisms. For example, Ehlers and colleagues[22] focused on cognitive processes and suggested that the Ehlers and Clark model[23] fits children. This model highlights the role of a) trauma memory deficits due to incomplete cognitive processing during the event and cognitive avoidance after the event, b) excessively negative appraisals of the event leading to a sense of current threat, and c) dysfunctional behaviors and cognitive strategies that are intended to control the perceived current threat but maintain the problem (e.g., thought suppression). Another illustration concerns child coping theory. While earlier coping theories dichotomized coping into a problem-focused approach, or primary coping on

the one hand, and emotion-focused, avoidant, or secondary coping on the other hand (see Compas et al.[24] for an overview), current theory assumes three or more clusters of strategies that are important in dealing with stress. For example, Ayers, Sandler, West, and Roosa[25] distinguished problem-focused, direct emotion-focused, distraction, avoidance, and support-seeking strategies. Researchers expect certain strategies to be more effective than others, depending on time point and context.[26] A final example of specific theories regards social processes. Scheeringa and Zeanah[19] have focused on parent-child interactions after trauma. They developed a theory of 'relational PTSD' and identified three dysfunctional interaction patterns between parents and young children after traumatic exposure that exacerbate children's symptoms (i.e., withdrawn, overprotective, and frightening patterns).

Several areas of trauma-focused theory that have been developed for adults, such as biological theories and theories invoking multiple representation structures (see e.g., Brewin & Holmes[15]), have not yet seen clear equivalents for children. Other, child-focused areas, such as those regarding social ecology,[e.g., 27] emotion regulation,[e.g., 18] and cognitive development (e.g., Piaget's and Neo-Piagetian cognitive stage theory, psychoanalytic theories of stages; see Miller[28] for an overview) have been developed for children in general but are rarely applied to the area of child traumatic stress.

In order to further child trauma theory and understand which factors influence post-traumatic stress and recovery, it is necessary to test current theories and build on them. Explicit theory building is an efficient method for field development in this regard.[29] When researchers explicitly describe the theoretical background of their work in the reports of their findings, this accelerates knowledge growth. Readers easily understand which theory is tested and which parts of it do or do not 'pass the test'. This stimulates focused new research that adds to these tests or develops alternatives. Therefore, for the development of a field, using theory explicitly to guide research efforts is more efficient than using it implicitly. The number of empirical studies in children who have been exposed to trauma is growing rapidly, which facilitates the task of testing current theories by synthesizing evidence.

Meta-analyses on predictors of posttraumatic stress in children

Two meta-analyses have examined predictors of posttraumatic stress in children to date.[30,31] Kahana et al.[31] looked into 26 studies in young people who had experienced accidental injuries (18 studies) or illness (8 studies). They found large to very large effect sizes for internalizing symptoms, depressive symptoms, symptoms of anxiety, dissociation, and acute stress disorder; small effects for socioeconomic status, social impairment and social support; and mixed results for age, gender, appraisal of trauma or illness severity, and life threat. Cox et al.[30] examined 14 articles on accidental injury in children (eight of which were

also included in the analysis by Kahana et al.). The strongest and most robust predictive factors accounted only for small to moderate effects. These factors were pretrauma psychopathology, female gender, life threat, and posttrauma parental distress.

These meta-analyses differ in their conclusions, implying that more research is necessary. They also have several limitations. First, they combined cross-sectional and longitudinal data. Cross-sectional estimates may provide misleading figures. For example, the appraisal of life threat may cause heightened posttraumatic stress scores but it is also possible that those children with higher stress scores are simply more prone to remembering life threat than children with lower stress scores. A second concern regards the meta-analysis performed by Kahana et al. which combined several effect sizes based on only two or three studies. Although it is true that two studies is the minimum for an average to be computed, these averages are heavily influenced by the few number of studies included, and they may be rather specific to these studies. Combined with the cross-sectional design, this may lead to over- or underestimation of effect sizes. Third, the findings of these meta-analyses are specific to the types of trauma studied (accidental injury and illness) and the research setting (hospital). Theory validation would profit from being tested across different types of trauma and different settings.[cf. 32]

Purpose of the present study

The purpose of this paper is to contribute to child trauma theory building by focusing on theory use and theory validation. For this purpose we have synthesized reports on longitudinal studies looking at recovery in children after a wide range of traumatic events. Our research questions were:

a) To what extent has longitudinal child trauma research been based on theoretical frameworks, and which theories are these?

b) To what extent have risk and protective factors in longitudinal studies been found to predict posttraumatic stress symptoms in children?

The answers will provide information on the validity of current theories, in whole or in part, and on gaps that should be addressed in future research.

METHOD

Retrieval and selection of studies

We targeted longitudinal studies depicting a natural process of recovery after trauma in children in order to shed light on relevant risk and protective factors. We defined this natural process as a situation in which some children and families will seek help and others will not,

as happens in 'normal' circumstances after trauma (samples should not be non-treatment seeking per se, but those studies that included the provision of an intervention were not selected).[cf. 33] Relevant studies were identified through systematic searches in electronic databases, reference lists (from literature reviews and from retrieved studies), and an issue-by-issue search of the *Journal of Traumatic Stress*, the *Journal of Child & Adolescent Trauma*, and the *Journal of the American Academy of Child and Adolescent Psychiatry*. The electronic databases consulted included PUBMED, EMBASE, PsychINFO, and PILOTS (a database for traumatic stress literature managed by the National Center for PTSD). Key words entered in the electronic databases were combinations of *posttraumatic stress*, *post-traumatic stress*, *traumatic event*, *traumatic experience*, and *traumatic exposure*; and *child, youth, young, youngster, kid, infant, toddler, preschooler, teen, teenager*, and *adolescent*. We restricted searches to empirical English-language papers published in peer-reviewed journals between 1980 (the year PTSD was first included in the DSM) and January 1st, 2010.

Papers were included in our database if they described a longitudinal study in children (i.e., child-related variables were measured at two or more time points) and satisfied the following criteria: 1) The study participants were all exposed to trauma as defined by the A1 criterion for PTSD in the DSM-IV, or separate data were shown for this subgroup; 2) The first measurement took place within three months after (the end of) the traumatic experience; 3) The last wave of the study took place at least three months after (the end of) the traumatic experience; 4) The study examined posttraumatic stress symptoms (combining at least reexperiencing and avoidance) in the participants at three or more months after (the end of) the traumatic experience; 5) The study participants were younger than 19 years old at the time of the PTSD/PTSS measurement; 6) The study did not have the evaluation of a psychological measure as its sole purpose; 7) The study participants were not recruited based on psychological characteristics (e.g., PTSD status) at either baseline or follow-up; and 8) The study did not include a psychological intervention (see Figure 1).

We chose the time frame of within three months (predictors) vs. three or more months (outcomes) posttrauma because we wanted to enhance the possibility that potential mediators in the relationship between trauma and posttraumatic stress were indeed related to the traumatic event and not to other (life) events. In addition, we assumed that longer-term posttraumatic stress reactions provide information about persistent problems while shorter-term posttraumatic stress reactions may remit spontaneously.[32] Measures taken at or after three months' time we refer to as 'at follow-up'.

Coding of studies

In addition to their publication details, the studies were coded according to use of theory, type of trauma, sample characteristics, posttraumatic stress measurement at follow-up, and predictors of long-term posttraumatic stress with their effect sizes. The studies were
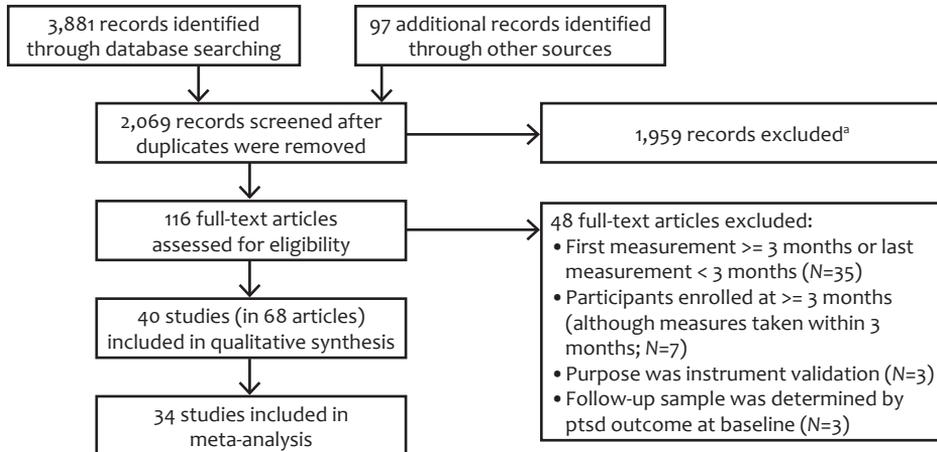
Figure 1 Flow-chart of the selection of studies. [a]Exclusion criteria at screening: not longitudinal; participants not all exposed to trauma or no separate data; no measurement of posttraumatic stress; participants were >= 19 years old and/or selected based on psychological characteristics; intervention study; psychometric study.

coded primarily by the first author. In addition to discussing any doubts that arose with the other authors we took several measures to ensure reliability. Before coding started, the coding scheme was tested and discussed with three researchers from University Medical Center Utrecht and a methodologist with expertise in meta-analyses on posttraumatic stress. Subsequently, the coding reliability of all variables except the associations was tested by independent coding by another researcher, from University Medical Center Utrecht. Each study had 26 or more coded variables, depending on the number of predictors and waves in the study. Reliability was calculated as a percentage of agreement between the two coders for eight studies (236 cells with 11 differences; 95% reliability). All effect size entries were verified by the third author. In addition, we contacted all authors of the original studies for correlational effect sizes (see below).

Use of theory

To gain an overview of the theoretical grounding of the studies we coded the extent to which they explicitly mentioned this grounding, and what the content was. Inspired by work from Lavee and Dollahite,[35] Pettigrew and McKechnie,[36] and Potter and Riddle,[37] we registered whether an aim was stated, whether theory was explicitly referred to in the introduction, and whether authors formulated hypotheses. We coded an aim of the study as present when words such as 'purpose', 'aim', 'objective', and 'this study sought to' were used (but not 'this study examined…' without further reference to a purpose). We coded theory as explicitly present as a basis for research when it was mentioned as

such in the article's title, in the introduction section of the abstract, or in the introduction. The theory had to be used to discuss a phenomenon and guide the research. The authors had to refer to it as theory or use key terms such as 'conceptualization', 'framework', 'grounded', 'underpinnings', or their variations. Hypotheses were coded as present when they were explicitly stated as such (e.g., with the words 'hypothesis', 'we expected'). If a study was reported in more than one eligible article, all of the articles were taken into account. In a later stage we also coded the discussion sections of the papers for explicit theory use, in order to get a complete view on explicit mentioning of theory.

Type of trauma

The primary type of trauma was coded as disaster (e.g., hurricane, flood), accidents (e.g., road traffic accidents, accidents leading to burns), war/terrorism, violence (other than war or terrorism), illness (life-threatening condition or newly diagnosed chronic disease), injury (when accidents and violence were mixed), sudden loss of a loved one, and 'other'. We registered whether the event was a collective/community experience, such as war, or an individual experience, such as an accident.

Sample characteristics

We coded several sample characteristics. First, we registered the number of eligible participants. In some medical articles families that could not be reached or were not willing to participate were excluded from the number of eligible participants; we adjusted numbers in those cases.

Second, we coded numbers and demographics of the children who initially participated. Because authors had different approaches to reporting the demographics of their samples (e.g., reporting demographics of the initial participants, of the participants retained in one or more waves, or of the eligible participants while mentioning that there were no significant differences with the initial sample), we decided to code broad demographics. For age the age range of the initial participants (within 3 months post-trauma) was coded. When necessary, we estimated age from grade levels. We categorized gender distributions as less than 40% male, 40–60% male, or more than 60% male in the initial sample. A sample was registered as 'majority Caucasian' (or other ethnicity) when 60% or more of the sample consisted of this ethnicity, otherwise the sample was coded as 'mixed'. We briefly described the socioeconomic status of individuals in the sample (e.g., income, education, depending on the original authors' definitions). Information missing after the first measurement was searched for in cross-sectional papers on the study.

Third, we coded the largest number of participants included in the follow-up for one of the effect sizes studied (see below) to be able to estimate rates at which participants were retained in the studies. In a few cases none of the effect sizes were examined

univariately in the study so we registered the largest number of participants included in a multivariate analysis. When authors carried out more than one follow-up, we selected the first wave for which we had information on associations, as earlier waves had generally larger *N*s.

Finally, we coded whether a study reported a bias in age or gender distribution in the inclusion or retention of participants (differences with regard to other demographics were seldom reported). 'Certainly age/gender bias-free' were studies explicitly reporting so for both inclusion and retention in follow-up. 'Probably age/gender bias-free' were studies explicitly reporting so for inclusion or retention but not providing information on both. All other studies were coded as 'probable or certain age/gender bias'.

Posttraumatic stress measurement

With regard to posttraumatic stress measurement we coded the timing of the follow-up (in mean number of months), which instrument was used, and who the informant was (child, parent, or both). If both parent and child reports were available, we used child reports, because several authors have suggested that parents may underreport children's symptoms.[38] Next, if results were available for both a structured clinical interview and a self-report questionnaire, we included the results of the interviews because these are generally considered better instruments for measuring psychological symptoms.[39]

Predictors of posttraumatic stress

Because follow-up *N*s were generally small and the study settings varied widely, we adopted an approach that was more conservative with regard to selecting predictors of posttraumatic stress than that of Cox et al.[30] (2008) and Kahana et al.[31] We registered all potential predictors but coded the effect sizes only for those predictors reported in at least five independent studies. Because the focus of this synthesis was on relationships between variables and these were reported as correlations in most studies, we decided to use the (univariate) product moment correlation coefficient as the effect size for the meta-analyses. We contacted all authors with a shortlist of predictors that were studied sufficiently often and asked whether they could provide us with the correlations. We received effect size information for 18 out of 40 studies (45%).

If correlations were unavailable from the papers and from the authors, we estimated them based on other univariate statistics (means and standard deviations, *F*-test statistics, Chi-square statistics, and *p*-values) provided in the articles, according to guidelines by Lipsey and Wilson.[40] In cases of non-significant findings not further specified we chose one of two options to estimate the non-significant effect size as accurately as possible: either we imputed a correlation of zero (Lipsey & Wilson[40], p.70) or we imputed the correlations that would correspond to a *p*-value of .50.[41] The former was applied when we did not

have clear expectations with regard to the direction of the association based on the effect sizes already found (e.g., for age, gender, ethnicity). The latter was applied when we expected a direction of the association (e.g., for posttraumatic stress symptoms we would not expect a negative direction but a positive one, which was also confirmed by the effect sizes already coded). In most cases for one predictor (e.g., parental distress) a single effect size was available. In the few instances that two effect sizes were given (e.g., for maternal and paternal distress), we took the average to avoid dependencies in effect sizes.[40]

Analyses

Descriptive statistics were used to summarize the studies' characteristics with regard to theoretical bases, types of traumatic exposure, samples, and posttraumatic stress measurement. With regard to effect sizes, the general approach in meta-analyses is to combine effect sizes by weighing them based on the magnitude of samples, thereby taking into account that sampling error is smaller in larger samples. Because product-moment correlation coefficients have some undesirable statistical properties (a problematic standard error formulation),[42] we combined effect sizes using Fisher-Z transformations as recommended by Hedges and Olkin.[43] Effect sizes and weights were combined into a weighted mean effect size with corresponding confidence intervals (95%) by means of the SPSS macros provided by Lipsey and Wilson[40] and were transformed back to a product-moment correlation coefficient for ease of interpretation.

We expected both sampling error and between-study variance to play a role in the mean effect size estimates because of the variety in types of exposure, samples and methods in the studies. Therefore, a random-effects model was applied.[cf. 44] In random-effects models total error is comprised of both within-study variance (which can be derived from the confidence intervals) and between-study variance (labeled 'v'). If the 95% confidence interval did not include zero, the null hypothesis that the relationship between the specific predictor and PTSD symptoms was zero was rejected at the $p = .05$ level. If the relationship was significant, a higher weighted correlation would indicate a stronger association with long-term posttraumatic stress.

Publication bias (i.e., the fact that studies reporting significant effects get published more often than studies reporting smaller effect sizes) is less likely to occur in meta-analyses of predictors than in meta-analyses of treatment trials.[45] Nevertheless, we checked funnel plots with the transformed effect sizes on the X-axis and the corresponding sample weight on the Y-axis[cf. 44] and calculated a fail-safe N according to the formula provided by Orwin.[46] The fail-safe N was computed with a critical effect size of .10, which corresponds to the lower limit of a small effect.[47]

RESULTS

We retrieved 68 articles describing 40 independent studies (see Table 1). Four studies had been included by both Kahana et al.[31] and Cox et al.,[30] while seven were selected by one of them. We included 29 studies that had not been examined by Kahana et al. or Cox et al. Although we searched from 1980 onward, selected studies were published for the first time between 1992 and 2009, with modest peaks in 2003, 2006, and 2007 (five studies each). Most studies originated in the US (35%), followed by the UK (15%) and Australia (10%).

Theory use

Virtually all studies had a stated aim (see Table 2), varying from relatively broad objectives such as "Our purpose in this study was to quantify PTSD symptomatology after childhood traumatic brain injury and to identify predictors of PTSD symptomatology" (Max et al.[81], p.589) to rather specific purposes such as "Using the framework of La Greca and colleagues, our study sought to examine whether social support, discrimination, and coping predicted post-disaster mental health outcomes among youth survivors of Hurricane Katrina" (Pina et al.[127], p.565).

For slightly more than one-third of the studies (N = 14) theory was explicitly mentioned as a basis for research. Most often mentioned were 'overarching' models that included characteristics of the child, the stressor and the posttrauma environment (such as the model by La Greca et al.[20]). Biological theories regarding fear conditioning were referred to several times as well, followed by cognitive models (such as the model by Ehlers & Clark[23]; see Table 3). An example of explicit theoretical grounding was: "Guided by models of risk and resilience in the face of adversity and disasters, we examined the associations of previously unexamined emotional and social factors (i.e., fear reactivity, emotional regulatory abilities, and peer victimization) with PTSD symptoms" (La Greca et al.[20,77]; Luthar et al.[161]; Vernberg et al.[162]) by Terranova, Boxer, & Morris[123] (p.346). For 14 studies, the results were explicitly discussed in the light of theory, 11 of which also contained theory descriptions in the introduction section. The authors of three studies described theory in the discussion section only. Several studies implicitly mentioned theory without defining it as such, for example by mentioning theoretical papers only in the references. Only studies with explicit descriptions were taken into account in our coding.

Hypotheses were stated in exactly half of the studies. An example was "We hypothesized that (1) heart rate assessed at emergency department triage would be related to later PTSD outcome in traumatically injured children and that (2) the relationship between heart rate and PTSD outcome would remain significant after controlling for child age, sex, and the presence of a severe injury".[67] Seven studies (18%) stated an aim, described a theory, and formulated hypotheses for the study in the introduction section.

Table 1 Studies included in the synthesis

#	Article(s) Country	Theoretical basis[a]			Traumatic event	Sample			Follow-up measuring posttraumatic stress after >= 3 months				
		A	T	H		Initial N	Age range	% males	Timing (months post-trauma)	Instrument[b]	Informant[c]	N	Predictors included in meta-analysis[d]
1	Ahmad,[48] Ahmad et al.,[49] Iraq	√			War: attack of Kurdish cities by Iraqi army	20	6–16	> 60%	4	PTSS-C[48]	C & P	18	G, A, P
2	Bronner et al.[50] The Netherlands	√		√	Injury/Illness: Pediatric Intensive Care (PICU) admission	36	8–17	40–60%	3	CRTI[51]	C	29	G, A, I
3	Bryant et al.[52-54] Australia	√	√	√	Injury: mainly falls and road traffic accidents	76	7–13	> 60%	6	UCLA PTSD Index for DSM-IV[55]	C	62	A, Ac, I, D, H
4	Di Gallo et al.[56] UK	√			Accidents: injury due to road traffic accidents	57	5–18	> 60%	3,1	PTSD-RI[57] and R-IES[58]	C	51	G, S, Ac
5	Dyb et al.[38] Norway	√			Accident: collective tramcar accident	16	7–12	< 40%	6	CPTS-RI[59]	C	16	G, A, S, P, Pa
6	Ehlers et al.,[22] Bryant et al.,[60] Dalgleish et al.[61] UK	√	√		Accidents: road traffic accidents	86	5–16	40–60%	3	adaptation of IES[60] and RI[57]	C, ((C&)P for young C	81	G, A, I
7	Karabekiroglu et al.[63] Turkey	√	√		Violence: murder at school	57	16–17	40–60%	5	CPTSD-RI[64]	C	35	G, A, Ac, Dp, Ax
8	Karakaya et al.[65] Turkey	√			Terrorism: bomb explosion nearby in Istanbul	132	12–14	> 60%	6	CPTSD-RI[64]	C	113	G

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Table 1 Continued

#	Article(s) Country	Theoretical basis[a]			Traumatic event	Sample			Follow-up measuring posttraumatic stress after >= 3 months				
		A	T	H		Initial N	Age range	% males	Timing (months post-trauma)	Instrument[b]	Informant[c]	N	Predictors included in meta-analysis[d]
9	Kassam-Adams et al.,[66,67] Dalgleish et al.[59] USA	✓		✓	Accidents: injury due to road traffic accidents	283	8–17	> 60%	6,4	CAPS-CA[68]	C	190	G, A, M, Ac, I, D, H
10	Kenardy et al.,[16] De Young et al.,[69] Olsson et al.,[70] Le Brocque[71] Australia	✓	✓	✓	Accidents: injuries due to accidents (falls, RTA, etc.)	255[e]	7–16	> 60%	6	CIES[72]	C	205	G, A, Ac, I, D, H
11	Kim et al.[73] Korea	✓			Accident: death of 2 mothers during fire escape drill at school	1,394	6–11	40–60%	6	CPTSD-RI[74]	C	335	G, A, Ac, P, Dp, Ax
12	Kuterovac-Jagodić[75] Croatia	✓	✓		War: war in Croatia	450	8–12	40–60%	17	QPTSR-C[76]	C	252	Ac
13	La Greca et al.[77] USA	✓	✓		Disaster: Hurricane Andrew	273	9–12	40–60%	3	PTSD-RI[64]	C	94	G, A, M
14	Landolt et al.[78,79] Switzerland	✓		✓	Accidents: injury due to road traffic accidents	78	6–14	40–60%	12	RI[59]	C	68	G, A, S, P, I, D, Pa
15	Mather et al.[80] Australia	✓			Accidents: road traffic accidents	43	6–15	40–60%	3	CPTS-RI[59]	C	32	G, A, P, Dp, Ax
16	Max et al.[81] USA	✓		✓	Injury: traumatic brain injury	50	6–14	> 60%	3	K-SADS[82] present episode + supplements	C & P	38	G, A, P, Dp, Ax

#	Study			Trauma	N	Age	%	Months	Measure	Informant	N	Symptoms
17	McDermott & Cvitanovich[83] Australia	✓		Accidents: motor vehicle accidents	53	8–13	40–60%	3	PTSD-RI[84]	C	26	I
18	Meiser-Stedman et al.,[85–88] Dalgleish et al.[61] UK	✓ ✓	✓	Accidents/violence: motor vehicle accidents or assault	106	10–16	>60%	6	ADIS-C[89]	C	68	G, A, M, S, Ac, I, D, Pa, Dp
19	Meiser-Stedman et al.[90] UK	✓		Accidents: motor vehicle accidents	114	2–10	40–60%	6	c: ADIS[89] p: PSSIOR YC[91,f]	C P	45 60	G, A, M, Ac, I, Pa, Dp
20	Mirza et al.[92] UK	✓		Accidents: road traffic accidents	125	8–16	>60%	6,2	FRI[93]	C	113	G, A, P
21	Nugent et al.[94–96] USA	✓	✓	Injury: mainly accidental and miscellaneous injury	82	8–17	>60%	6	CAPS-CA[68]	C	57	G, A, M, S, P, I, H, Pa
22	Ostrowski et al.[97,98] USA	✓	✓	Injury: mainly nonviolent injury	61	8–18	40–60%	7	CAPS-CA[68]	C	41	G, A, M, S, P, I, Pa, Dp
23	Pervanidou et al.[99,100] Greece	✓	✓	Accidents: injury due to motor vehicle accidents	60	7–18	>60%	6	K-SADS-PL[101]	C	48	G, A, M, I
24	Qouta et al.[102,103] Gaza	✓ ✓	✓	War: First Intifada	108	10–12	40–60%	36	PTSD-RI[59]	C	83	G, A
25	Rennick et al.[104,105] Canada	✓		Injury/illness: PICU admission	69	6–17	40–60%	6	Adaptation of IES[104]	C	60	
26	Rohrbach et al.[106] USA	✓	✓	Disaster: Hurricane Rita	602	15–16	<40%	7	PTSD-RI[55]	C	326	G, A, M, S

Table 5.1 continues on next page

Table 1 Continued

#	Article(s) Country	Theoretical basis[a]			Traumatic event	Sample			Follow-up measuring posttraumatic stress after >= 3 months				
		A	T	H		Initial N	Age range	% males	Timing (months post-trauma)	Instrument[b]	Informant[c]	N	Predictors included in meta-analysis[d]
27	Rusch et al.[107] USA	✓			Accidents: accidental mutilating facial/extremity injuries	57	3–12	>60%	12	Structured interview	C & P	57	G, A
28	Saxe et al.[108-110] USA	✓	✓	✓	Accidental and non-accidental injury	235	7–18	>60%	3	CPTSD-RI[68]	C	158	G, A, M, Ac, I[g], D, H, Pa, Dp, Ax
29	Schäfer et al.[111,112] Germany	✓			Accidents: road traffic accidents	76	8–18	40–60%	3,2	IES-R[113]	C	69	G, A, Ac, I, Dp, Ax
30	Schwarzwald et al.[114] Israel		✓	✓	War: missile attacks at Israel during Persian Gulf War	492	11–16	40–60%	12	CPTSD-RI[93]	C	329	G, A, Ac
31	Shaw et al.[115,116] USA	✓			Disaster: Hurricane Andrew	62	6–11	40–60%	7,4	PTSDRI[65]	C	47	
32	Shears et al.[117,118], Garralda et al.[119] UK	✓		✓	Illness: meningococcal disease	78	3–16	40–60%	4,1	IES[58]	C (P for young C)	52	G, A, M, S, I, D
33	Stoddard et al.[120] USA			✓	Accidents: burns	72	1–4	40–60%	4,5	CSDC-B[121]	P	11	
34	Sturms et al.[122] The Netherlands	✓			Accidents: injury due to road traffic accidents	79	8–15	40–60%	3	IES[58]	C	64	

	Study	A	T	H	Type	N	Age	%	Months	Instrument	Informant[c]	n	Predictors
35	Terranova et al.[123] USA	✓	✓	✓	Disaster: Hurricane Katrina	177	11–12	40–60%	8	PTSD Checklist[124]	C	152	G, M, P, Ax
36	Thienkrua et al.[125] Thailand	✓			Disaster: Tsunami	371	7–14	40–60%	8,4	UCLA PTSD-RI[74]	C	119	
37	Weems et al.,[126] Pina et al.[127] USA	✓	✓	✓	Disaster: Hurricane Katrina	225	7–18	40–60%	6,5	PTSD checklist[124]	C	52	G, A, M, S,
38	Zatzick et al.,[128,129] Ghesquiere et al.[130] USA	✓		✓	Injury: accidental and violent injury	108	12–18	> 60%	5,1	PTSD-RI[74]	C	90	G, A, S, Ac, P, I, D, H, Pa, Dp
39	Zehnder et al.[26] Switzerland	✓	✓		Accidents/Illness: accidental injury / newly diagnosed chronic disease	161	6–15	> 60%	12	RI[59]	C	128	G, A, S
40	Zink & McCain[131] USA	✓			Accidents: injury due to motor vehicle accident	160	7–15	40–60%	6	DICA-R[132]	C & P	126	P

a Theoretical basis: A = aim explicitly mentioned in abstract/introduction, T = theoretical model(s) explicitly mentioned in abstract/introduction, H = hypotheses explicitly stated in abstract/introduction.

b Regards the instrument used for computing correlations in the present meta-analysis in case of more than one instrument.

c Informant: C = child-report, P = parent-report.

d Predictors: G = gender, A = age, M = minority status/ethnicity, S = SES, Ac = Acute stress symptoms (measured within 1 month posttrauma), P = posttraumatic stress symptoms (measured 1–3 months posttrauma), Dp = depressive symptoms, Ax = anxiety symptoms, I = injury severity, D = days in hospital, H = heart rate, Pa = parental acute or posttraumatic stress symptoms.

e Approximate number.

f Because measures differed according to age group and separate statistics were available, these groups were regarded as independent subsamples in the analyses. Correlations were available for the same predictors in both groups, except for depressive symptoms (only available for child-report).

g For injury severity, different measures (i.e. percentage of body surface burnt and injury severity score) were used for two independent subsamples (N = 51, N = 100); these were regarded as such in the analyses. For the other predictors, information available regarded the complete sample.

Study characteristics

The summary of study characteristics is shown in Table 2. Accidents were studied most frequently, followed by accidental injury/violent injury/life-threatening illness combinations, disaster, war or terrorism, other violence, and life-threatening illness alone. In total, 26 studies focused on individual experiences (all were carried out in hospital settings) and 14 studies examined collective experiences (all were carried out in community settings).

Studies targeted populations ranging in number from 18[38] to 1,456[73] children during their first wave. The total number of initial participants was 7,039. The mean response rate was 71% (for 30 studies reporting the number of eligible participants). Children's ages ranged from 1 to 18 years old, with children aged 8, 9, 10, 11, or 12 years old studied most often (>75% of studies for each age year), followed by children aged 7, 13, 14, 15, or 16 years old

Table 2 Synthesis of study characteristics

Characteristic	N (% / M / Mn / SD)		(Range)
Theory			
Aim	37 (93%)		
Explicit theory	14 (33%)		
Hypotheses	20 (50%)		
Settings			
Accidents	17 (43%)		
Traumatic injury	10 (25%)		
Disaster	6 (15%)		
War/terrorism	5 (13%)		
Other violence	1 (3%)		
Illness	1 (3%)		
Samples			
Eligible participants	≥ 8,922[a]		(18–1,456)
Included participants	7,039	(M = 176, Mn = 84, SD = 239)	(16–1,394)
Participation rate	M = 71%[b]	(SD = 21,1)	(31%–100%)
Participants in follow-up	4,000	(M = 100, Mn = 68, SD = 85)	(11–335)
Retention rate of eligible participants[c]	M = 49%	(SD = 19,4)	(11%–89%)
Retention rate of initial participants[d]	M = 71%	(SD = 21,2)	(15%–100%)
Timing of follow-up	M = 6,9 months	(SD = 5,65)	(3–36)
N ≥ 100 at follow-up	14 (35%)		
Certainly bias free	11 (28%)		
Probably bias free	14 (35%)		

Note: M = mean; Mn = median; SD = standard deviation; total N = 40.

[a] Unknown for N = 10; for these studies the number of initial participants was taken as an indication of minimum eligible N.

[b] Unknown for N = 10.

[c] Percentage of eligible participants retained in follow-up; unknown for N = 10.

[d] Percentage of initially included participants retained in follow-up.

(50–75% of studies). In most studies (58%) gender was fairly equally distributed; a number of studies were dominated by boys (38%). In 20 studies ethnicity or majority/minority status was described; of these studies, 12 (60%) had a predominantly Caucasian sample, one (3%) a predominantly Asian sample, one (3%) a predominantly Arab sample, and six (15%) had a mixed sample. The socioeconomic status of the children in the sample was described in some way in 18 studies (45%) and ranged from poor (e.g., poor neighborhoods, low

Table 3 Use of theory

Study (first author of first publication)	Theoretical models explicitly mentioned[a]
Bryant et al.	Cognitive models[23,133] Fear conditioning models[134]
Ehlers et al.[b]	Cognitive models[23]
Karabekiroglu et al.	Multivariate models[135,136]
Kenardy et al.[b]	Fear conditioning models[137,138] Models of symptom trajectories and resilience[32,139]
Kuterovac-Jagodić[b]	Model of characteristics of child, stressor, and posttrauma milieu[140]
La Greca et al.[b]	Model of characteristics of child, stressor, and postdisaster environment[e.g., 20,141]
Meiser-Stedman et al.[b]	Cognitive accounts in adults[e.g., 23,142] Conceptualizing pathology in younger populations with a different algorithm[e.g., 143,144]
Nugent et al.[b]	Three-factor model of PTSD[145] Three models on development of emotional numbing, regarding avoidance, depression, and fear conditioning[146] Biological theories of PTSD[e.g., 147 in adults, 148 in youth]
Qouta et al.[b]	Concept of flexible perception[149] Psychoanalytical ego theory regarding personality styles that affect behavior in stress situations[150] Coping/emotion theory[151,152]
Saxe et al.[b]	Proposal concerning overconsolidation of traumatic memory[153] Animal model of traumatic memory and PTSD[134,154]
Schwarzwald et al.[b]	Stress evaporation approach[e.g., 155 for adults, 156 for children] Residual stress approach[e.g., 157 for adults, 158 for children]
Terranova et al.[b]	Models regarding adjustment following disaster exposure and stressful environments[e.g., 20,77]
Weems et al.[b]	Framework first articulated by e.g., Green et al.[141] and La Greca and colleagues[e.g., 20] concerning aspects of traumatic exposure, pre-existing characteristics of the child, characteristics of the postdisaster recovery environment, and child psychological resources.
Zehnder et al.	Coping theory[159,160]

[a] When three or more references were given, we show two published examples.

[b] These studies also explicitly mentioned theory in the discussion or comments section of their paper(s). In addition, Ostrowski et al., Pervanidou et al., Rohrbach et al., and Zatzick et al., mentioned theory in the discussion or comments sections only. In total, 14 out of the 40 studies (35%) explicitly discussed their findings in the light of theory.

income) to mixed (e.g., families with incomes or education ranging from lower to upper class). Fourteen studies (35%) had a follow-up *N* >= 100 and four studies (10%) reached at least 70% of the initially eligible participants at follow-up. Eleven studies (28%) mentioned that there was no bias with regard to the gender and age distributions in inclusion and retention. The majority of the studies reported either did not provide information on inclusion and retention bias with regard to gender and/or age, or reported such a bias.

The initial follow-up took place up to 36 months after the event, with a mean of seven months. The UCLA Children's PTSD Reaction Index for DSM-IV[55] or one of its earlier versions (see Steinberg, Brymer, Decker, & Pynoos[74] for an overview) was used most frequently to measure associations with predictors (50% of studies), followed by the Children's Impact of Event Scale[163] or one of its earlier versions or adaptations (18% of studies). Note that a few studies also measured stress symptoms with parental interviews or child self-report questionnaires that we did not take into account because child measures or child interview scores were available respectively.

Predictors of long-term posttraumatic stress in children

We initially registered more than 50 different potential predictors of posttraumatic stress at follow-up. Variables ranged from very general (e.g., prior psychopathology, life events since trauma, intellectual capacity, social support) to very specific (e.g., number of X-ray pictures taken while in hospital, parental history of trauma, whether a child was immobilized in the ambulance). For twelve predictors we had sufficient information (i.e., data from at least five independent studies) to analyze the effect sizes. These were gender, age, minority status, socioeconomic status, injury severity, duration of hospital stay, heart rate shortly after hospital admission, acute stress symptoms (<1 month posttrauma), short-term stress symptoms (1–3 months posttrauma), symptoms of depression, anxiety symptoms, and parental posttraumatic stress symptoms (0–3 months posttrauma; see also Table 1). For both minority status and gender we performed analyses with point biserial correlations. We distinguished between acute and short-term posttraumatic stress symptoms for the children, parallel to Acute Stress Disorder and Acute Posttraumatic Stress Disorder conceptualizations.[3] This was not possible for parental posttraumatic stress because of a smaller number of relevant studies.

For six studies we could obtain only multivariate statistics. Therefore, the total number of studies that contributed to the meta-analysis was 34. Funnel plots did not show highly skewed distributions of effect sizes. For the interpretation of the effect sizes, we applied Cohen's criteria, which define a correlational effect of .10 as a small effect, .30 as a medium effect, and .50 as a large effect.[47] Table 4 shows, for each predictor, the number of individuals involved, the number of studies included, the weighted effect size *r*, the lower and upper limit of the 95% confidence interval, the between-studies variance (*v*), and the fail-safe *N*.

Table 4 Meta-analysis of predictors of posttraumatic stress symptoms in children

Predictor	N	K	Weighted r	p	Weighted CI Lower limit	Weighted CI Upper limit	v	Fail-safe N
Gender	3,195	31	.13	<.01	.08	.17	.01	8.4
Age	2,940	29	-.01	.78	-.07	.05	.02	
Minority status	1,308	13	.09	.20	-.05	.23	.05	
Socioeconomic status	888	11	-.07	.10	-.16	.02	.01	
Injury severity	1,381	18	.09	.02	.01	.16	.01	
Days in hospital	889	8	.18	.02	.03	.33	.04	6.5
Heart rate	658	6	.18	<.01	.08	.27	.00	4.6
Acute stress symptoms[a]	1,857	14	.51	<.01	.43	.59	.03	57.7
Posttraumatic stress symptoms[b]	1,196	12	.56	<.01	.44	.66	.06	55.3
Depressive symptoms	813	9	.48	<.01	.32	.61	.06	33.8
Anxiety symptoms	745	6	.44	<.01	.31	.57	.03	20.7
Parental acute/posttraumatic stress symptoms	515	9	.34	<.01	.24	.43	.01	21.4

Note: N = number of children included in effect size; K = number of samples; Weighted r = weighted correlation according to random-effects model; CI = 95% confidence interval; v = between-study variance.

[a] Measured within one month posttrauma.

[b] Measured one to three months posttrauma.

Five out of the twelve predictors significantly and moderately/strongly related to long-term posttraumatic stress reactions in children: acute stress symptoms (0–1 month posttrauma; weighted r = .51), short-term posttraumatic stress symptoms (1–3 month posttrauma; weighted r = .56), parental posttraumatic stress symptoms (weighted r = .34), depressive symptoms (weighted r = .48), and anxiety (weighted r = .44). This implies that the greater a child's acute or short-term stress symptoms are, the greater the long-term posttraumatic stress symptoms will be, and that this relationship is strong. Likewise, but to a somewhat lesser extent, children's anxiety, depressive symptoms and their parents' symptoms predict subsequent posttraumatic distress. These findings are fairly robust; 20 to 57 studies reporting a correlation of zero would be needed to make the weighted effect size drop to the lower bound of a small effect (r = .10).

Four predictors yielded significant but small mean effect sizes: gender (weighted r = .13), injury severity (weighted r = .09), hospital stay in days (weighted r = .18), and heart rate shortly after admission to the hospital (weighted r = .18). On average, girls, more severely injured children, children who were hospitalized for a longer period, and children with a higher initial posttraumatic heart rate demonstrated more posttraumatic stress reactions in the long run. Four to eight zero-effect studies would be needed to lower the mean effect sizes for gender, hospital stay and heart rate to .10; conclusions about these predictors are less firm than those about the moderate/strong predictors. Note that for injury severity, the mean effect size was already smaller than the criterion for the fail-safe N, and therefore the fail-safe N was not computed.

Three predictors were non-significantly related to long-term posttraumatic stress reactions in children: age, minority status, and socioeconomic status. Their weighted effect sizes were -.01, .09, and -.07 respectively.

DISCUSSION

The purpose of this paper is to contribute to child trauma theory building by focusing on theory use in longitudinal studies and on theory validation based on the findings of these studies. We retrieved 40 studies published in the last 30 years that examined predictors (within three months posttrauma) of long-term posttraumatic stress (at three or more months posttrauma) in children. We summarized their use of theory, study characteristics, and the correlational effect sizes for 12 predictors. One of the main findings was that explicit theoretical frameworks were present in a minority of the studies only. When theory was explicitly referred to, general risk factor models, biological theories, and cognitive models were most present. The most notable predictors of long-term posttraumatic stress were symptoms of acute and short-term posttraumatic stress, depression, anxiety, and parental posttraumatic stress. Female gender, injury severity, duration of hospitalization, and heart rate shortly after admission to the hospital accounted for small effects. Age,

minority status, and socioeconomic status were not significantly related to long-term posttraumatic stress reactions in children.

Strengths and limitations

The present paper adds to the literature in several ways. With regard to theory use, we are unaware of any other systematic review of theoretical grounding of child trauma studies. This is new to the field. With regard to theory validation, the present analysis considerably broadens earlier work. The meta-analyses by Kahana et al.[31] and Cox et al.[30] included studies on injury and illness only and all samples were hospital-based. The present analysis took into account the complete range of traumatic events as defined by the DSM-IV as well as samples approached in a variety of settings. Also, the earlier meta-analyses mixed cross-sectional and longitudinal findings, which may lead to over- or underestimation of the effect sizes, while we selected longitudinal studies only. They included smaller numbers of studies in their analyses and reported contradictory findings. Our analysis sheds new light on these issues and also included new predictor variables. Finally, this meta-analysis adds to the literature by identifying gaps in research with regard to testing child trauma theory.

Meanwhile, several limitations of this study should be kept in mind. First, longitudinal child trauma studies are still relatively rare. Our sum Ns were rather small for some predictors. The scarcity of studies compelled us to use simple univariate statistics since similar approaches in multivariate analyses are not yet commonplace. Although univariate relationships are important building blocks for more complicated models, these relationships are obviously simplifications of complex, multivariate processes.

A second limitation regards conclusions about causality. Even though focusing exclusively on longitudinal findings brings us a step closer to knowledge about factors causing or moderating long-term distress after trauma than including cross-sectional findings would do, it does not allow any firm conclusions as we did not systematically manipulate these factors in experiments, or examine studies that did so. For ethical reasons this is possibly the closest we can get since we do not want to purposely traumatize children (although a few authors were 'lucky' enough to study child traumatic stress in a natural experiment when disaster struck after they had examined various psychological characteristics of the children in their study).

Another limitation of the current study relates to the decisions we made. We may have influenced the results by our definition of theory use, the choice of correlations as the effect size, the preferences for the informants of symptoms, the decision to contact authors, and the requirement that predictors were measured shortly after exposure, to name a few reasons. We expect that our conclusions about the medium and strong effects will not be affected by these choices, but they could affect the less-robust results and confirmation by other systematic reviews will be necessary.

Fourth, we did not include studies measuring outcomes other than posttraumatic stress, such as generalized anxiety disorder, major depression, and posttraumatic growth. Anxiety and depression have overlapping symptoms with posttraumatic stress disorder but also have distinctive symptoms that we could not account for. The mechanisms related to positive outcomes (e.g., posttraumatic growth, resilience) are probably different from those connected to posttraumatic stress.[32] Therefore, generalizations of our findings should not be made to psychological consequences other than posttraumatic stress symptoms.

Theory use

"Few people other than theorists ever get excited about theories" (Toracco[164], p.114). In the empirical child trauma field we may indeed suffer from a lack of theorists: only a minority of the longitudinal studies included was driven by explicit theoretical considerations. Although the relative youth of the child trauma field[165] could be a reason, Toracco suggests that the predominance of non-theoretical research is a more general phenomenon. Indeed, it exists in established fields as well. For example, Hawley and Geske[166] found only 42% of 95 articles on family therapy research that referred to theory explicitly.

Since a lack of theory-centeredness is not the privilege of young fields, child trauma theory and empirical research run the risk of remaining somewhat separate entities, at least at an explicit level. This is a serious shortcoming since theory serves a variety of purposes directly related to empirical research and interventions, such as interpreting new research data, responding to new problems, evaluating solutions, discerning priorities, interpreting old data in new ways, and identifying new research directions.[167] Or, as Lewin[168] succinctly put it: "Nothing is as practical as a good theory" (p.129). Theory moves a field forward, expecially when it is made explicit.[cf. 29]

Currently, theory building is not optimally stimulated in the scientific community. For example, some journal policies communicate non-theoretical interests. Several high-impact journals, in (general and psychiatric) medicine, prefer short introductions. This encourages authors to point only to the relevance of the topic, some earlier research, and the purpose of the present study (see e.g., Thienkrua et al.[125]). More generally, empirical papers tend to emphasize highly sophisticated methods instead of comprehensive theoretical grounding, while both should be considered important. Finally, variables tend to be studied because they are easily available instead of being chosen based on theory. Focusing on specific variables to confirm or falsify theory would further our knowledge faster.

Although they were a minority, several authors in our study based their work on theory, predominantly on general risk factor models,[e.g., 20] biological theory,[e.g., 137] and cognitive theory.[e.g., 23] It is striking that these longitudinal studies borrowed far more from general trauma theory than from child (i.e., developmental) theory. Developmental theories (see Miller[28] for an overview), were absent. This is in line with Pat-Horenczyk, Rabinowitz, Rice,

and Tucker-Levin[169] who argued that "most current conceptualizations of childhood PTSD are some distance away from a genuinely developmental approach" (p.62). The theoretical grounding the authors used had its reverberations on the variables tested in the studies.

Theory validation

Based on our meta-analysis, conclusions can be drawn regarding the validity of parts of the models mentioned in the introduction. General risk models[e.g., 20,21] describe characteristics of the stressor, the child, and the child's environment as influencing children's posttrauma adjustment. Our findings suggest that certain child demographics (age, minority status, socioeconomic status) need to be emphasized less as important risk factors. They may, however, play a moderating role that we were not able to examine in the present analyses. In addition, although we did not find an association with the number of children's posttraumatic stress symptoms, age (as an index for development) could still be related to the quality of these responses, as has been suggested by Salmon and Bryant.[17] For example, children may show the same number of symptoms but with different patterns than adults, or children show other symptoms not included in standard PTSD instruments, such as regression and separation anxiety (see e.g., Kaminer, Seedat, and Stein[170]). Child characteristics other than demographics, such as academic skills and pre-trauma anxiety, should be studied more frequently in order to draw robust conclusions. The same applies to life threat during the event, loss/disruption following the event (stressor characteristics), major life events and social support (characteristics of the environment), and coping, the other variables put forward in the models. In contrast, in line with Pynoos' model, acute stress symptoms and biological reactions (increased heart rate) appear to be related to later stress symptoms. In sum, the overarching models could be partially confirmed. Some elements can be removed and most elements remain to be tested.

One of the more specific theories we described regarded the cognitive model by Ehlers and Clark[23] that focuses on trauma memory deficits, appraisals, and PTSD-maintaining behaviors and cognitive strategies. Although, for example, the appraisal of life threat may play an important role in the development and maintenance of posttraumatic stress in children,[e.g., 22] we were unable to quantify this relationship. Coping models applied to the field of traumatic stress[e.g., 26] also await testing in future meta-analyses. Only a few studies have examined the effects of coping. It would be valuable to discover which coping styles are related to increased or decreased levels of posttraumatic stress.

Even though the relational PTSD model formulated by Scheeringa and Zeanah[19] was never explicitly described as a basis for research in our set of studies, our findings suggest that this model merits future attention. The specific mechanisms by which parental stress symptoms influence children's post-trauma adjustment (such as overprotectiveness vs. being unavailable) could not be tested in the present study but parental distress was

found to be a significant predictor of children's distress. Our findings indicate that this relationship exists not only with very young children, as has been described in research by Scheeringa and Zeanah, but also in older age groups.

Biological theories were only briefly mentioned in the introduction to this paper because they have not yet focused on children to a significant extent. However, they were quite prevalent in the studies included in the meta-analysis. Fear conditioning models posit that exposure to a traumatic event leads to a strong fear reaction which becomes conditioned to many stimuli associated with the traumatic event. Stress hormones "released at the time of the trauma, marked by an increased heart rate, are thought to contribute to fear conditioning and overconsolidation of trauma memories" (O'Donnell et al.[137], p.256). The current analysis appears to confirm these propositions, as an increased heart rate was found to predict subsequent stress symptoms.

In summary, many proposed relationships await testing. Although both Pat-Horenczyk et al.[169] and Layne et al.[32] posited that the era of "studying shopping lists" of variables has ended, this is not because of any overwhelming evidence regarding the variables on these shopping lists. We agree with these authors, however, that more advanced methods to study trajectories of symptoms, such as growth mixture models, are now available and should be used. Meanwhile, even if few predictors were analyzed in the present study, it has shown that future theoretical models need to focus on factors proximal to the trauma (e.g., initial physiological arousal, acute stress reactions, parental distress) compared to factors distal to trauma (e.g., demographics), as has been posited by Ozer et al.[2]

Our findings provide new insights to the results of earlier meta-analyses by Cox et al.[30] and Kahana et al.[31] (see Table 5). We converted the findings of Kahana et al. (Cohen's *d*) to *r* and applied Cohen's rule of .10 being a small effect[47] as a rule of thumb to identify differences. The two earlier analyses showed highly variable findings for gender (ranging from .04 to .22) and age (ranging from -.48 to .04), in considerably smaller number of studies (maximum 15 vs. 31 and 29 in our study). We found a small effect for gender and no effect for age. We found no effect for socioeconomic status based on 11 studies where Kahana et al. reported a small effect based on three studies. Kahana et al. reported variable effect size ranges for depressive symptoms (.47 to .62; 3 studies) and anxiety symptoms (.41 to .70; 4 studies). We found the effect sizes to be on the lower side of these ranges (.48 and .44 respectively, for 9 and 6 studies), which may have to do with the difference between longitudinal and cross-sectional measurements. The findings regarding injury severity, acute and short-term posttraumatic stress, and parental distress were similar, whereas our findings with regard to minority status, duration of hospitalization, and heart rate were new.

Although the included studies varied widely in samples and methodology, the research base was dominated by studies carried out in medical settings, studies in children aged 8 to 12 years old, and studies with relatively small follow-up samples. It would be valuable to

Table 5 Comparison of the results of meta-analyses on predictors of posttraumatic stress in children

Predictor	Alisic et al.		Cox et al.[a]		Kahana et al.[b]	
	K	Weighted r	K	Weighted r	K	Weighted r
Gender[c]	31	.13	8	.18 (1st)	15	.09 (inj)[d]
			5	.22 (f-u)	6	.04 (ill)[d]
Age	29	-.01	7	-.04 (1st)	15	.04 (inj)
			3	-.12 (f-u)	7	-.48 (ill)
Minority status[e]	13	.09				
Socioeconomic status	11	-.07			3	-.22 (inj)
Injury severity	18	.09	5	.09 (1st)		
			4	.08 (f-u)		
Days in hospital	8	.18				
Heart rate	6	.18				
Acute/posttraumatic stress symptoms	14	.51 (acute)			2	.43 – .56 (inj)
	12	.56 (pts)				
Depressive symptoms	9	.48			3	.47 – .62 (inj)
Anxiety symptoms	6	.44			4	.41 – .70 (inj)
Parental acute/posttraumatic distress	9	.34	5	.29 (1st)		
			3	.41 (f-u)		

Note: K = number of samples.

[a] Cox et al.[30] summarized effect sizes for initial (1st) and follow-up (f-u) measurements separately.

[b] Kahana et al.[31] summarized effect sizes for studies regarding injury (inj) and illness (ill) separately. We converted Kahana et al.'s findings to r for this comparison.

[c] A positive correlation corresponds with female gender.

[d] This appears to be a correlation with full PTSD in a dichotomous way. Correlations with 'partial PTSD' are higher: .50 and .88 for studies examining injury and illness respectively.

[e] A positive correlation corresponds with having a minority status.

truly cover a range of settings and ages, in order to understand similarities and differences between them. This would enable a more thorough understanding of whether a general trauma theory is indeed possible, whether specific theories for specific types of trauma are necessary, or whether a modular theory (e.g., with a common 'core' and theoretical modules specific to the type of trauma that is addressed) would fit best.

In addition, it was striking that risk factors such as stress symptoms were studied far more often than protective factors. A quick search in the PILOTS database for all types of empirical papers on *children* and *risk factor* or *protective factor* yields about four times more publications on risk factors than on protective factors. Identifying protective factors is important for the development of programs to prevent and treat long-term posttraumatic stress in children. [cf. 171] Although the numbers are still small, recent publications indicate that the field is slowly but surely moving toward balanced attention given to both negative and positive aspects of traumatic stress in children (see e.g., Kilmer & Gil-Rivas[172]; Layne et al.[32]).

Practical implications

The findings of this meta-analysis have several implications for clinical practice. First, it will not be possible to easily identify children at risk for long-term distress based on a few demographics and exposure criteria. Earlier *psychological* symptoms provide the best indicators when predicting posttraumatic stress symptoms. Differences between the predictive strength of stress symptoms measured within one month (r = .51) and one to three months (r = .56) are small, which may encourage clinicians to measure this factor as early as possible to identify children at risk. This is, however, not in line with the guidelines of the United Kingdom's National Institute for Health and Clinical Excellence,[34] which recommend 'watchful waiting'. Early screening for stress symptoms could enable early interventions. However, early interventions such as debriefing are generally not recommended, although they have rarely been studied in children.[e.g., 173] In our view, there may be good alternative early interventions that do not follow the debriefing format. For example, when a child has been identified as being at risk, an intervention focused on activating social support and/or preparations to engage the child in therapy could be started. The effects of these interventions would obviously have to be studied.

Although early stress symptoms are the best indication of later distress compared to the other predictors we studied, they only account for 26–31% of the variance in long-term stress symptoms. Therefore, other predictors should also be taken into account when screening children. Symptoms of anxiety and depression are found to be informative, but it is quite possible that these do not explain substantial additional variance since there are overlapping symptoms. A more separate, important indication is parents' posttraumatic stress. In addition, in medical settings, injury severity, length of hospital stay (or one of both as they will probably overlap) and heart rate will add to the accuracy of a screening tool.

A screening tool that has been developed for the medical setting and that includes some of these variables is the STEPP (Screening Tool for Early Predictors of PTSD).[174] Several of its variables, such as separation from parents during an incident and injury or death of someone else in an accident could not be confirmed by the present analysis, but Winston et al.[174] showed the overall performance of the measure to be good. Developing similar tools for other settings will be valuable.

Finally, if the factors that are found to be moderate/strong predictors are also actually causal influences on long-term posttraumatic stress, this has implications for treatment. The factors found to be most strongly related to posttraumatic stress are potentially modifiable. In line with clinical impressions, it appears to be highly important to include parents in treatment.[39] Interventions for traumatized children that focus on parents or parenting have rarely been studied[175] and deserve more attention. There is a trend towards looking more specifically at the family and social environment of children and possible interventions, which will contribute to further theory building.

Theory building

There are four important trends that will be significant to building child trauma theory in the next few years. First, there is a trend toward looking beyond PTSD, as was mentioned above. Authors increasingly look at positive outcomes, such as resilience and posttraumatic growth and to broader constructs, such as self-regulation (see e.g., Brom, Pat-Horenczyk, & Ford[176]; Kilmer & Gil-Rivas[172]). This enables an approach that is more wellness-oriented[177] instead of pathology-oriented.

Second, authors are increasingly looking beyond single predictors.[169] As Layne and colleagues[32] (p.15) put it: "because people's risk factors and coping resources seldom operate or travel in isolation, the practice of examining risk and beneficial factors one at a time both decontextualizes the object of study and misses the broader point". While at this moment decontextualization is necessary for comparisons, as in the present analysis, in the future we may be able to compare more complex sets of factors.

Third, related to the tendency to look at aggregates of factors is a development toward looking beyond linear relationships in psychology. Non-linear dynamical systems theory[178] proposes that certain changes are far from gradual. When the challenges to a current steady state are too great to assimilate, change is characterized by sudden disturbance and increased variability in the 'systems' behavior before reorganization (Hayes et al.[176], p.716). Within this increased variability, several individual trajectories of adaptation[32] can be predicted. A first application of the study of individual trajectories after trauma in children was carried out in one of the studies in our synthesis.[71]

Fourth, there is a more general tendency to look beyond the individual. In child trauma literature, the family has been getting more attention. This applies not only to the role of parents[169] but also to the influence of and on siblings.[e.g., 180] In addition, although it is not yet prominent in trauma theory, network theory has gained attention. (Social) network theory is related to dynamical systems and chaos theory, and focuses on systems of interacting components. For example, Christakis and Fowler[181] studied the person-to-person spread of obesity and concluded that obesity travels through social ties. Similarly, posttraumatic reactions may develop in families and communities.

One important tendency is lacking in these trends: a trend towards a more developmentally oriented trauma theory is not yet apparent (with the exception of models regarding very young children, see e.g., Scheeringa and Zeanah[19]). As has been posited by Salmon and Bryant[17] (see the introduction), there is a need to understand the impact of children's developmental stages on their trajectories of recovery after trauma. Although the present analyses did not show a univariate correlation with the level of posttraumatic stress symptoms, the mechanisms of trauma processing in children are thought to be qualitatively different from those of adults,[17] and child-specific surrounding factors, such as parents,[19]

turn out to play an important role. Therefore, the development of child trauma theory should be facilitated.

Facilitating child trauma theory building

In our view, child trauma theory building could be facilitated in three areas: publication policy, methodology, and collaboration. Above, we described that the scientific community is not optimizing its research potential for theory building. Journal policies could encourage the building of theory more strongly. Several journals require authors to provide key points: 'What is already known' and 'What this study adds'; a simple solution would be to have authors show in addition 'How this study builds theory'. Beyond the policies of the top journals, funding organizations could require theory building. There is a growing trend towards funding those projects that have clear practical implications, but theory building could equally be stimulated.

In order to enhance theory building, theory not only has to be conceived and described. It must also be tested, in a thorough manner. The current study showed that more and more comparable longitudinal research is needed. Several interesting factors, such as coping, social support, and the appraisal of life threat, have not yet been studied extensively enough. There is a great deal of fragmentation in constructs examined and measures used. More diversity is needed, since the current longitudinal database is dominated by medical studies. In order to generate theoretical frameworks that apply to children who are confronted with any trauma, develop theories for specific settings, or construct a modular theory (see above), it would be valuable to focus on children in non-hospital settings as well.

Finally, collaboration is a necessary foundation for building child trauma theory. As the models by Pynoos et al.[21] and Layne et al.[32] show, there are many domains and disciplines to cover, and especially exploring the possibility of integrating theories would be valuable. Child trauma theory would benefit from more thorough collaboration between scholars. The National Child Traumatic Stress Network in the United States is a fine example of such collaboration, but this endeavor would need to be more international. One way to build more collaboration would be to have theory discussion meetings at international trauma conferences, rather than one-way presentations. Collaboration would make it possible to influence journal policies, to exchange ideas, and to reach larger samples.

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