

**Doctoral Thesis**

**Psychological predictors of post-traumatic stress disorder in children and  
adolescents**

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## **Abstract**

### **Background**

The experience of trauma in childhood, for a minority of individuals, can lead to chronic and distressing symptoms of post-traumatic stress disorder (PTSD) and other mental health difficulties. Cognitive models of PTSD demonstrate key factors involved in the development of symptoms, however, research evidence regarding the role of different pre- peri- and post-trauma predictors of PTSD in children and adolescents is limited and variable. Furthermore, there is scope to understand predictors of mental health outcomes other than PTSD. With the expected publication of ICD-11 in 2018, further research is also necessary to develop our understanding of the new diagnostic category of ‘Complex PTSD’ in children and adolescents.

### **Methods**

Firstly, a systematic literature review and meta-analysis was conducted, summarising the current evidence regarding the role of peritraumatic psychological risk factors in the development of PTSD. Secondly, empirical analysis of pre-existing data from a longitudinal study of children and adolescents experiencing a single-event trauma was conducted. Multiple linear regression models were used to assess four theory-derived predictive models of mental health outcomes (PTSD, CPTSD, depression and anxiety) of trauma in this sample.

### **Results**

Population estimates of effect size were moderate for peritraumatic subjective threat and fear as risk factors for PTSD. Effect size estimates for peritraumatic dissociation were small, and evidence for data-driven processing was limited. The empirical study

indicated that a cognitive model of predictors was most powerful in predicting the development of all four disorders following trauma, and psychosocial and objective event severity models were weak predictors of mental health outcomes.

### **Conclusions**

Cognitive processes occurring during and after trauma may be valuable markers of which individuals may be at risk of developing PTSD, CPTSD, depression or anxiety after trauma. Further research of multiple predictors and outcomes of trauma is required in children and adolescents, particularly related to CPTSD.

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**Chapter 1. Psychological peritraumatic risk factors for post-traumatic stress disorder in children and adolescents: A meta-analytic review**

(Written up to be published in Clinical Psychology Review. See Appendix A.1 for a summary of author guidelines for manuscript preparation)

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## 1.1 Abstract

There are currently few meta-analytic reviews of predictors of PTSD in children and adolescents. Existing reviews have incorporated a large majority of evidence related to pre- and post-trauma risk factors, with an identified paucity in the evidence-base regarding peritraumatic risk factors. This is despite previous reviews and theories of PTSD suggesting a significant role of peritraumatic experiences in the development of PTSD. The current review aimed to conduct a comprehensive systematic review and meta-analysis of studies exploring peritraumatic risk factors for PTSD in children and adolescents. Thirty-two studies were identified (total  $n=24,768$ ), and random effects meta-analyses were run, with meta-regressions to explore the moderating role of age, gender, trauma type and timing of assessment after trauma, upon the size of effect of predictive factors. Peritraumatic subjective threat and fear response, and data-driven processing yielded moderate to large estimates of population effect size, and peritraumatic dissociation yielded a small estimated population effect size. Estimates of heterogeneity were high in the main group of studies assessing perceived threat and fear ( $I^2 = 95\%$ ), but moderate and low within studies assessing dissociation and data-driven processing ( $I^2 = 57\%$  and  $0\%$ , respectively). Moderators of effect size, reasons for heterogeneity, limitations, clinical and research implications are discussed.

Keywords:

PTSD, children, adolescents, peritraumatic, risk factors, meta-analysis

## 1.2 Introduction

Our understanding, conceptualisation, and treatment strategies for post-trauma psychopathological reactions has evolved greatly over the past two decades, with particular development more recently in our insight into post-trauma reactions in children and adolescents. What was originally conceptualised as an adult disorder, post-traumatic stress disorder (PTSD) is now widely acknowledged as a similarly debilitating and distressing outcome of trauma for children and adolescents. The initial acknowledgement of the presentation of this disorder in children followed seminal research highlighting the importance of appropriate measures of PTSD symptomatology in children (Pynoos, et al., 1987; Terr, 1979; Yule & Williams, 1990), triggering shifts in research and clinical practice to consider and assess for the same set of symptoms as observed in adults. Clinically, the accurate identification of key psychological processes implicated in the development of PTSD, soon after trauma, is vital in recognising which children may go on to develop chronic symptoms of PTSD. Importantly, while a majority of children and young people will experience some kind of traumatic event in their young lives (an estimated 68%; Copeland, Keeler, Angold, & Costello, 2007), and acute symptoms of post-traumatic stress are common, only an estimated 8-10% of individuals develop chronic symptoms of PTSD (Alisic, et al., 2014; Bryant, Mayou, Wiggs, Ehlers, & Stores, 2004; Copeland, et al., 2007; Costello, Erkanli, Fairbank, & Angold, 2002; Kilpatrick, et al., 2013; Ogle, Rubin, Berntsen, & Siegler, 2013). There have been substantial research efforts to unpick the role of different psychosocial, trauma-related, and psychological factors, in order to identify key risk factors for the development of PTSD. However, evidence and conclusions drawn have been variable, and there have been very few reviews to summate and appraise this research.

Research efforts have explored pre-trauma factors (such as psychosocial and demographic participant characteristics), trauma-related factors (including trauma type,

injury severity and peri-trauma experiences), and post-trauma factors (such as cognitive processing of the trauma, social, and parental support). Evidence pertaining to pre-trauma and demographic factors as risk factors or predictors of PTSD development following trauma has been particularly mixed. For example, both younger age and older age have been suggested to be associated with increased likelihood of presenting with symptoms of PTSD following trauma, and the mixed evidence has led to suggestions that there could either be a non-linear relationship between age and PTSD risk, or that age does not have a significant role in predicting PTSD (Cox, Kenardy, & Hendrikz, 2008; Foy, Madvig, Pynoos, & Camilleri, 1996; Scheeringa, Wright, Hunt, & Zeanah, 2006; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Female gender has consistently been found to be a risk factor for PTSD, though a recent meta-analysis suggested an estimate of population effect size to be small ( $r=.15$ ; Trickey et al., 2012). Similarly, socioeconomic status was estimated to have a weak relationship with PTSD symptoms, but with a large range of effect sizes presented by studies for the strength of this relationship ( $r=.05 - .28$ , and a population estimate of  $.16$ ) (Trickey, et al., 2012). Experiencing previous trauma or stressful life events is generally conceptualised as a vulnerability factor for developing PTSD post-trauma, however, there has again been inconsistency in evidence, with some suggestion that experience of a similar prior trauma may protect against developing PTSD (Cox, et al., 2008; Keppel-Benson, Ollendick, & Benson, 2002). In summary, following the meta-analysis of available evidence of pre-trauma risk factors for PTSD in children and adolescents, it has been concluded that the strength of relationship, and so estimated predictive power of pre-trauma variables, except pre-trauma psychopathology, is small compared to trauma-related and post-trauma factors (Cox, et al., 2008; Trickey, et al., 2012).

Trauma-related factors, such as degree of exposure to aspects of events, whether death was caused by the trauma, and severity of injury to the child, have often been

conceptualised as factors which increase the likelihood of PTSD (Pine & Cohen, 2002). Subjective peritraumatic experiences are also deemed core factors in the development of PTSD. DSM-IV criteria for PTSD (Diagnostic and Statistical Manual for Mental Disorders 4<sup>th</sup> Edition; American Psychiatric Association, 2000) stipulated the experience of fear, horror, helplessness and/or perceived life threat as necessary characteristics of a trauma experience for a diagnosis of PTSD. However, these trauma characteristics are no longer noted as necessary for diagnosis in DSM-5 (American Psychiatric Association, 2013; Friedman, Resick, Bryant & Brewin, 2011), but they are still deemed to be key risk factors. Perceived life threat and strong peritraumatic emotions, such as extreme fear or panic, have been assessed in studies of both child and adult PTSD development, supporting the role of such factors in the development of PTSD. The strength of the effect of these peritraumatic subjective experiences has been reported to be moderate to large (population estimate of effect size  $r=0.36$  for perceived life threat), however, few studies were found to report on this relationship in the most recent review of predictors of PTSD in children and adolescents (Trickey, et al., 2012).

A further peritraumatic experience which has received increasing attention in PTSD research is dissociation. This phenomenon refers to when an individual enters a state of emotional numbness, derealisation, or depersonalisation during or shortly after a trauma, and is thought to be a risk factor for developing PTSD (Breh & Seidler, 2007). It has been conceptualised as a neurophysiological attempt to conserve resources during heightened threat by shutting down responsiveness, which has unfortunate detrimental consequences (Saxe, et al., 2005). Dissociation at the time of the trauma is thought to increase feelings of helplessness and disrupt the normal processing of an event. As a result, memories of the event are stored in a fragmented and poorly integrated manner, leading to the increased likelihood of flashbacks and intrusive thoughts (Ehlers & Clark, 2000). Dissociation has mostly been investigated in the context of the DSM-IV acute

stress disorder (ASD) diagnosis. A review of findings related to dissociation and PTSD across the lifespan concluded that it was not an optimal predictor of PTSD (Bryant, 2007). Cognitive models of PTSD in children and adults elucidate how subjective peritraumatic experiences, such as perceived threat, data-driven processing (feeling muddled or confused), feelings of panic and fear play a role in the development of PTSD. These theoretical models outline how a number of cognitive processes including, how trauma memories are formed at the time of the trauma, how the trauma event is appraised, and the use of maladaptive thinking styles post-trauma, lead to the development and maintenance of PTSD symptoms (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989).

Trickey et al.'s recent meta-analytic review of predictors of PTSD in children and adolescents demonstrated that the vast majority of studies explored the role of pre-trauma and demographic factors, and post-trauma factors; only nine effect sizes related to peritraumatic factors were drawn from 62 identified studies of PTSD predictors in children and adolescents. Post-trauma factors included factors related to how the individual appraised and coped with the trauma (blaming others, thought suppression, and distraction), comorbid psychological problems, parental poor mental health, poor family functioning, further life events, media exposure, and poor social support. Estimates of population level effect sizes for these post-trauma factors ranged from .1 (media exposure) to .69 (thought suppression), with the cognitive factors demonstrating the greatest effect sizes.

### **1.2.1 Purpose of the current review**

This review aimed to conduct a comprehensive search and collation of empirical research shedding light on the role of peritraumatic psychological processes in the development of PTSD in children and adolescents. This aimed to provide an update and

development of previous reviews, with a specific focus on peritraumatic risk factors. Allowing comparison between different peritraumatic psychological processes, and identifying which have stronger or weaker relationships with PTSD symptoms, will have important implications in developing the theoretical understanding of the disorder in this population, and directing future research and clinical practice. Previous reviews suggest that there have been relatively few studies of peritraumatic processes in children and adolescents. However, cognitive theories of PTSD place certain peritraumatic processes as central in the development of PTSD, and diagnostic manuals have previously stated that a diagnosis of PTSD depends upon experiencing key thoughts and feelings during or immediately after a trauma. This review also aimed to shed light on the measurement and reporting of peritraumatic processes in previous research, so as to inform methodological practices in future research.

## **1.3 Method**

### **1.3.1 Search strategy**

An initial search of the leading psychological and medical literature databases was conducted, including PubMed (Medline), PsycInfo and the National Centre for PTSD research's Published International Literature on Traumatic Stress (PILOTS) database. Reference sections of included studies and existing meta-analyses of predictors of PTSD were also studied to identify any possible relevant studies. The search dated from 1980 (when PTSD was first defined as a diagnosis in the Diagnostic and Statistical Manual of Mental Disorders, 3<sup>rd</sup> Edition (DSM-III); American Psychiatric Association, 1980) to December 2017. The search terms were developed by reviewing other meta-analyses and review articles, and were refined for the purposes of identifying broadly applicable studies initially. The search terms were 'PTSD OR

posttraumatic stress disorder OR post-traumatic stress disorder OR posttraumatic stress OR post-traumatic stress' AND 'peri-traum\* OR peritraum\* OR during' AND 'dissociat\* OR fear OR helpless\* OR horror OR confus\* OR threat OR defeat OR perceive OR perception OR panic OR emotion\* OR distress\* OR data-driven OR data driven OR cognit\* OR process'. There was an acknowledged difficulty in identifying studies of peritraumatic factors, as they are often not defined as such, and so search terms were broadened to include all terms for the possible risk factors as well as 'peritraumatic' or 'during'. Initial searches were open to all ages, and then child and adolescent studies were identified by screening within this, as some studies may have assessed adults and children and reported on the groups separately. All searches were run by searching 'full text', however, initial search results suggested that some databases may not have access to search full texts, and as risk factors, particularly peritraumatic factors, may not be mentioned in titles or abstracts, a fourth database search was run using PsycArticles which successfully searched article full texts. A Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow chart (Figure one) illustrates the review search, screening, and inclusion and exclusion processes.

### **1.3.2 Inclusion and exclusion criteria**

To be considered for inclusion, studies had to present data on predictive or risk factors for PTSD, with clearly defined assessment of one or more psychological peritraumatic risk factor(s) in child and adolescent samples. Peritraumatic psychological processes were defined as a cognitive or emotional process or experience which occurred during or immediately after the trauma. Studies were required to use a validated and reliable measure of PTSD which considered the diagnostic criteria for

PTSD: symptoms of intrusion, avoidance and hyperarousal. One study was included which did not use a previously validated measure of PTSD, but utilised a psychiatric interview conducted by a qualified psychiatrist based on a checklist of ICD-10 symptoms for PTSD (World Health Organisation, 1992). This was deemed to have sufficient reliability and eligibility for the review. The assessment of PTSD in this study not being based on a validated measure with published reliability psychometrics was reflected in the study quality assessment score.

Clinical and community samples were included, as long as clinical samples did not just present data on participants with PTSD alone (some comparison group or inclusion of individuals without PTSD was required to calculate effect sizes of risk factors for PTSD). Studies were excluded if participants were recruited primarily due to a specific comorbid disorder or presentation, or had a traumatic brain injury, in order to ensure that the effect sizes estimated were related to PTSD, not any other psychopathological process, or affected by cognitive impairment from brain injury impeding memory of peritraumatic experiences. Study methodology was considered, with studies excluded if peritraumatic factors were assessed more than two years post trauma, in line with previous reviews and evidence to suggest that reporting of peritraumatic experiences is not stable over time due to forgetting (Candel & Merckelbach, 2004; Cox, et al., 2008). This criterion was intentionally set with a broad time period to allow for the inclusion of studies initially, and it was planned that consideration would be given regarding the time between trauma and assessment of peritraumatic factors as part of the quality assessment of studies. Studies were also excluded if when the trauma occurred, or the time since the trauma, were not clearly stipulated, or 'lifetime' trauma was assessed. PTSD must have been assessed after one month or more following trauma (in line with DSM-5 criteria); studies of acute stress reactions or acute stress disorder were excluded. All academic journal articles,

dissertation papers, longitudinal, follow-up or cross-sectional studies were considered. Single case studies, studies presenting qualitative data alone, and clinical treatment trials were excluded. See the PRISMA flow diagram (Figure 1) demonstrating the study selection, exclusion, and inclusion processes.

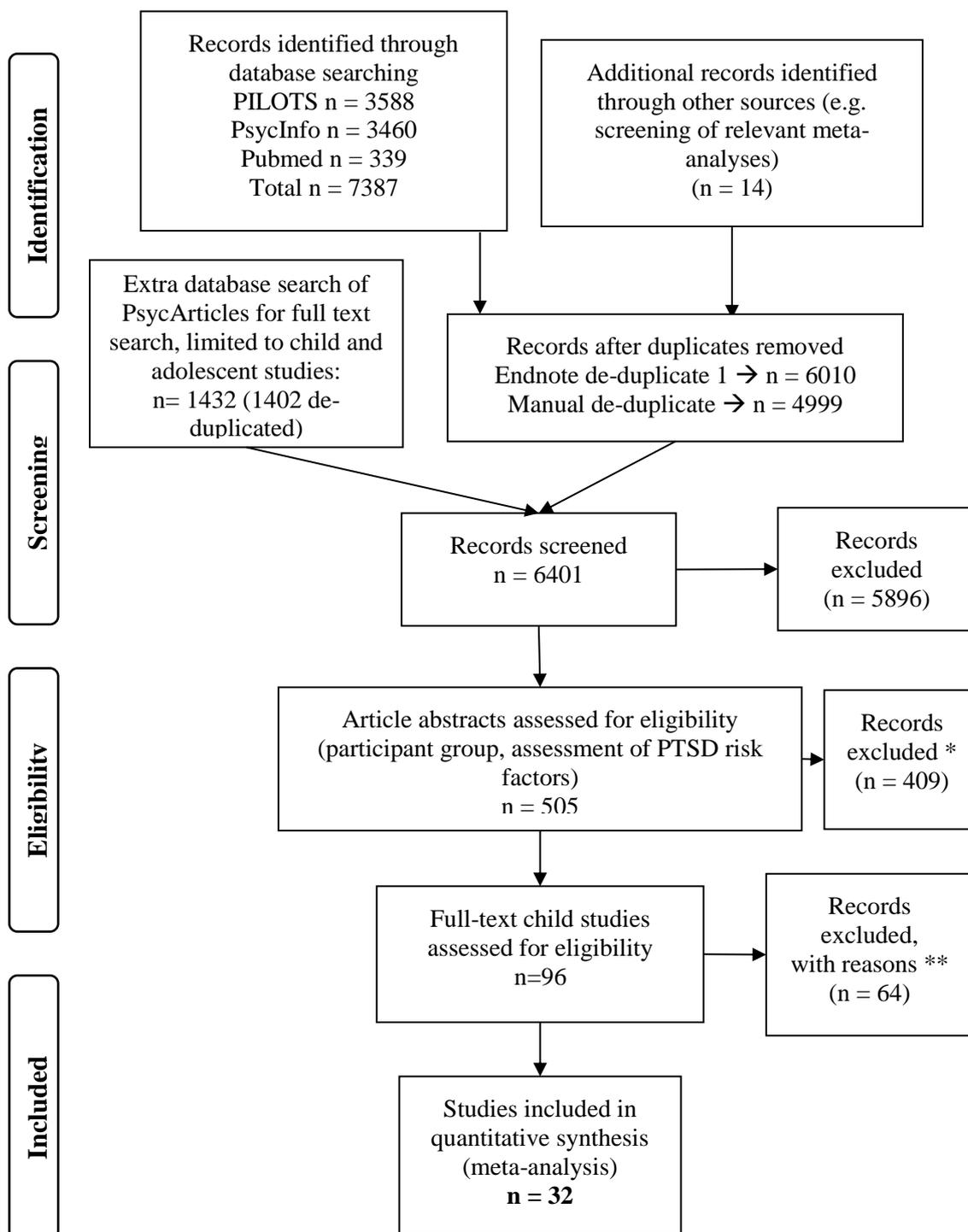


Figure 1. PRISMA flow chart. \*Articles excluded from initial screening of abstracts if it was not a child or adolescent sample, or it was deemed unlikely that predictors of PTSD were assessed within the study, or another eligibility criterion was clearly ruled out from information given in the abstract. \*\*Reasons for exclusion from screening of full-texts were: peritraumatic risk factors not assessed (n=31); peritraumatic experiences were assessed but within other assessment of non-peritraumatic factors (n=4); time since trauma not reported or ‘lifetime’ trauma assessed (n=7); no access to the article (authors contacted where possible; n=7); the study reported effect sizes from a sample presented by another included study (n=6); peritraumatic factors were assessed but the statistics reported were not transformable into a r correlation coefficient (n=6); peritraumatic factors were assessed by parent report and not child self-report (n=1); child and adult study sample (n=1); PTSD/Acute Stress was assessed within 1 month after trauma (n=1).

### **1.3.3 Data extraction**

Data extraction forms were used to record the following data from each study: (a) article details (for example, type of publication, journal), (b) study design, (c) demographic information (sample population description, age mean and range, percentage of the sample female), (d) type and detail of index trauma experienced, (e) the time between the trauma and when participants were assessed for peritraumatic factors and PTSD, (f) for longitudinal studies, time of follow-up assessments, (g) details of how PTSD was assessed, (h) details how peritraumatic factors were assessed, and (i) result statistics reported (effect sizes if reported, or alternative necessary statistics required to compute effect sizes).

A number of rules were adhered to clarify uncertainty in the data extraction process. If information was given on both lifetime and current PTSD, effect sizes for current PTSD were used. If PTSD was identified by using both continuous measures (symptom severity) and dichotomous measures (diagnosis), effect sizes from continuous measures were prioritised to avoid underestimation of effect size due to dichotomisation of data (Breh & Seidler, 2007). For longitudinal studies with multiple points of assessment of PTSD, for consistency, effect sizes were derived from the time point nearest to the traumatic event, as long as it was more than one month after the event. Methods are available to consider effect sizes from multiple time points, and account for the correlations between them, in meta-analyses of longitudinal studies (Ishak, Platt, Joseph, Hanley & Caro, 2007; Musekiwa, Manda, Mwambi & Chen, 2016). However, as this meta-analysis included cross-sectional studies also, it was decided that the most appropriate and consistent method for the current review would be the selection of one effect size from each study from the nearest time point, along with the analysis of ‘time since trauma’ as a moderator of effect size.

#### **1.3.4 Grouping of peritraumatic factors**

We explored the peritraumatic experiences assessed within each study, and how they were assessed, i.e. the vocabulary and content of items used to measure each peritraumatic factor, to inform how the effect sizes from each study were grouped to reflect certain peritraumatic factors. A number of studies used measures of risk factors which assessed a range of different psychological, cognitive, and emotional experiences at the time of trauma; labelling the overall peritraumatic experience as ‘peritraumatic distress’, ‘peritraumatic reaction’, or ‘A2 criteria’, and reported one effect size from this overall peritraumatic experience measure. The assessment of ‘A2’ criteria included

measures of the PTSD DSM-IV criteria of experiencing fear, horror, helplessness, or perceived life threat during or immediately after the trauma. This informed our grouping of all effect sizes measuring ‘subjective threat’ or ‘A2’ criteria; including these overall measures of peritraumatic reactions of fear, helplessness, horror, and perceived life threat. A second group of effect sizes focussed on peritraumatic dissociation, which was measured very specifically within the studies included. A third group of effect sizes reflected the assessment of data-driven processing (feeling confused or muddled during the trauma), which again was very specifically measured within the studies included. A final group focussed on the effect sizes related to ‘pure’ perceived life threat, assessed specifically (most with one single item) and without mention of any other peritraumatic experience within the measure. Any peritraumatic factors which had been assessed by only one study were not included in the analysis; the only exclusion of an effect size was related to ‘feeling sick’ during the trauma, which was assessed by one study (Holmes, Creswell, & O'Connor, 2007), and was not assessed by any other studies and not deemed suitable to be grouped amongst other peritraumatic factors.

### **1.3.5 Calculating effect sizes**

Pearson’s correlation coefficient,  $r$ , was used as the effect size in this current meta-analysis; most studies reported this statistic in their analysis of risk factors of PTSD, and those which reported  $\beta$ , t-tests, ANOVAs, or odds ratios, ‘ $r$ ’ was computed following standardised calculations for transforming effect sizes (Borenstein, Hedges, Higgins & Rothstein, 2009; Cohen, 1965; Rosnow & Rosenthal, 1996; Peterson & Brown, 2006). Pearson’s correlation coefficient is also readily interpretable, lending the results to easy application of conclusions to the population. The general rule of thumb as applied in considering a ‘small’ effect to be represented by ‘ $r$ ’ of approximately .1; a

medium effect to be approximately  $r=.3$ ; and a large effect to be indicated by approximately  $r=.5$  or higher (Cohen, 1988). If a study reported multiple effect sizes for one peritraumatic factor, for example, perceived life threat to self and perceived life threat to others was assessed separately (whereas other studies assessed this within one measure and reported one effect size),  $r$ 's were converted to Fisher's  $z$ , a mean was calculated and then the  $z$  was transformed back to  $r$  to be included in the meta-analysis (Borenstein, 2009). This method was applied if more than one peritraumatic factor was reported from one study which was relevant to the grouping of factors, for example an effect size was reported for helplessness and an effect size was reported for fear; for both to be considered within the meta-analysis of overall peritraumatic 'A2' response, a Fisher's  $z$  transformed mean  $r$  was calculated.

Where a particular peritraumatic risk factor was reported as having a non-significant effect on PTSD and no statistic for this result was provided, the effect size was assumed as being 0, in line with recommendation from Rosenthal (1991). This method of including something to represent non-significant findings avoids possible bias resulting from excluding non-significant results (Pigott, 2009).

### **1.3.6 Quality assessment and risk of bias**

Assessment of study quality and risk of bias is an established and recommended practice within meta-analysis of intervention or clinical treatment randomised controlled trials, in order to account for variation in methodological quality in studies included (Higgins & Altman, 2008; Higgins, et al., 2011). Studies with poor quality design, such as flaws in the design, recruitment, analysis method or detail in the reporting of results, can lead to increased risk of bias, such as under or overestimation in the results and reduced accuracy of the conclusions reported. Many quality assessment frameworks and

a well-established Cochrane risk of bias tool are available to guide researchers in the consideration of these factors when meta-analysing RCTs (Higgins, et al., 2011), however, to date there are no published risk of bias or quality assessment frameworks for meta-analyses of non-treatment studies assessing predictors of a disorder using correlation coefficients. Therefore, methodological rigor was considered within the development of inclusion and exclusion criteria, and a quality assessment framework to provide a score reflecting study quality and risk of bias was developed for the current study. The NICE Quality Assessment Checklist for Studies reporting Correlations and Associations (2012) and the NIH Quality Assessment Tool for Observational Cohort and Cross-section Studies (National Heart Lung and Blood Institute, 2014) were used to inform the development of this tool. The assessment framework developed included seven items considering: how well the study population was defined; if appropriate random sampling or other appropriate recruitment method was utilised; if non-response rate was reported, was minimal or accounted for (for example, differences between responders and non-responders were nonsignificant); if loss to follow-up was minimal in longitudinal studies; how reliable the measurement of PTSD and peritraumatic factors were; and how soon after the trauma peritraumatic factors were assessed. Each item was given a score of 0-2, with 0 indicating low quality, and 2 indicating high quality and thus low risk of bias. Scores were summed and converted to a percentage; studies scoring more than 70% were deemed high quality (with low risk of bias), studies with scores of 50-70% were deemed medium quality (capturing the median score of 58%), and studies with scores below 50% were deemed low quality. The researcher completed quality ratings for all studies, and a second rater was instructed to use the quality assessment framework to score a random selection of 20% (n=7) of included studies. Inter-rater reliability of the scale was assessed by calculating a kappa score of agreement between the raters' scores on each item for the seven double-rated studies.

Within the analysis method, further consideration of risk of bias within the results was also planned; calculation of a ‘fail-safe n’ would indicate the validity and generalisability of the results by calculating the number of non-significant or conflicting evidence required to significantly challenge the overall conclusion of the meta-analysis (Oswald & Plonsky, 2010; Rosenthal, 1991). Furthermore, consideration of any evidence of publication bias was also planned by generating funnel plots to visually represent the data, with observation of asymmetry in plots and by generating Kendall’s tau tests of asymmetry to indicate possible publication bias, and the ‘trim-and-fill’ method to indicate whether the study sample is missing weaker studies (Duval & Tweedie, 2000).

### **1.3.7 Meta-analytic method**

The meta-analysis to examine the relationship between peritraumatic psychological experiences and PTSD symptoms was conducted via user interface software (MAVIS version 1.1.3 (Hamilton, 2017) and OpenMetaAnalyst (Wallace, et al., 2012)) which run the meta-analyses using R (version 3.4.3) and the ‘metafor’ (version 2.0.0) package (Viechtbauer, 2010). Random effects models with restricted maximum likelihood estimators of between study variance were used. This approach is deemed most suitable for meta-analyses of studies with variable sample parameters, for studies in mental health research, and where it is hoped to achieve generalisability of findings beyond the samples included (Cuijpers, 2016; Hedges & Vevea, 1998; Viechtbauer, 2010). Random effects models allow for true effect sizes to differ between studies, and the studies included are treated as random samples of all possible studies that may meet the inclusion criteria. A large amount of variation between studies in terms of study method, trauma and participant characteristics was estimated in the

current review, and yet it was intended that conclusions drawn may be suitably applicable to the wider population and not just to the samples included in the review. Therefore, random effects models are reported in all analyses. Extracted  $r$  values entered into the software were transformed into Fisher's  $z$  for the analysis, and transformed back to  $r$  correlation coefficients for reporting of results and interpretation.

Heterogeneity of effect sizes was estimated by calculating a  $Q$  statistic, whereby if  $Q$  is significant ( $p < .05$ ) true effect size variation is implicated, and the amount of this variation was estimated by  $I^2$ . Higgins et al. (2003) suggest that an  $I^2$  value of 25% represents a small degree of heterogeneity, 50% is moderate, and 75% represents a large degree of heterogeneity.

**Meta-regression analyses of moderators.** Meta-regression was used to explore how certain characteristics of the studies or samples were related to variation seen in the effect sizes reported by the studies. This analysis method was used to reflect how certain factors may moderate the strength of the relationship between a risk factor and PTSD symptoms. Meta-regression analyses of trauma type (interpersonal vs non-interpersonal), gender (percentage female), mean age, study type (cross-sectional vs prospective), study quality, and time between trauma occurrence and assessment of peritraumatic factors and PTSD, were planned to explore the possible moderating effects of these variables on the strength of the relationship between peritraumatic factors and PTSD. These analyses focussed upon the main group of studies of subjective threat and fear effect sizes, as this group constituted the largest number of studies; insufficient number of studies were found reporting effect sizes for data-driven processing to conduct meta-regressions, and meta-regressions for the few studies related to dissociation were conducted with cautious suggestions regarding conclusions drawn.

## 1.4 Results

### 1.4.1 Study characteristics

Thirty-two studies were included, providing a total of 47 effect sizes for the planned meta-analyses estimating the overall strength of the relationship between peritraumatic psychological processes and PTSD symptoms. Table 1 (appendix A.3) summarises the characteristics of the studies included. Four studies were included which exceeded the typical upper age of 18 years for child and adolescent studies; two studies included participants up to age 19 (Nordanger, et al., 2014; Polusny, et al., 2011), one study included those age 20 (Elklit & Kurdahl, 2013), and one study included up to age 26 (Filkuková, Hafstad, & Jensen, 2016). Our initial database searches, which allowed for the inclusion of studies spanning child and adult populations to identify those which may report on both samples, perhaps allowed for the identification of studies with over-inclusive child and adolescent age bracket. It was decided to include these four studies as all indicated a mean age of their sample aged 18 years or below, and it was deemed that they provided valuable information about the development of PTSD in adolescent populations. Mean age was planned to be assessed with moderation analyses, and so any effect of older age on the relationship between peritraumatic factors and PTSD would also be indicated. One study was included which exceeded the inclusion criteria of peritraumatic factors being assessed within two years post-trauma: Cénat and Derivois (2015) assessed participants 2.5 years after they experienced a trauma, but again it was deemed that this study provided valuable information about the relationship between peritraumatic factors and PTSD development. Furthermore, risk of bias due to possible forgetting of peritraumatic experiences, was reflected in the study's quality score. No other studies were excluded based on peritraumatic factors being assessed up to 2.5 years since trauma.

All studies included assessed single event traumas, except two which were regarding trauma related to war or ongoing terror, in which case the number of traumas experienced by individuals was not clear, though both focussed on the participants' experience within the past eight to ten months. The studies spanned a range of trauma types: eleven recruited participants who had experienced an acute physical injury requiring a visit to hospital (resulting from road traffic accidents, other accidental injuries or assaults); twelve studies recruited participants exposed to a severe natural disaster (such as an earthquake or hurricane); and nine studies recruited participants who were exposed to or had witnessed acts of severe human conflict (war, terror, or homicide). Nineteen studies were cross-sectional, assessing peritraumatic factors and PTSD symptoms concurrently; thirteen were prospective longitudinal studies, assessing peritraumatic factors soon after trauma (initial assessments ranged from less than one week to five months after trauma) and assessing PTSD up to six years later. See Appendix A.3 for a full summary table of study characteristics.

#### **1.4.2 Measurement of peritraumatic factors and effect sizes**

Table 2 summarises the methods used in each study to assess each peritraumatic factor, how effect sizes were grouped according to the peritraumatic experience(s) assessed, and the effect sizes sourced or calculated from the study data. The large majority of studies assessed peritraumatic fear, perceived life threat or helplessness, or a combination of these experiences (k=28). Twelve of these studies assessed perceived life threat very specifically, and a further small number assessed peritraumatic dissociation or data-driven processing.

Insert Table 2 here

#### **1.4.3 Assessment of study quality and risk of bias**

All thirty-two included studies were scored against the quality assessment framework. Those with scores indicating high quality were deemed to have low risk of bias. Nine studies were rated as high quality, 19 were rated as medium quality, and four studies were rated as low quality (high risk of bias). Inter-rater reliability was calculated with twenty percent of studies ( $n=7$ ), which indicated 83.3% agreement on all items ( $\kappa=.74$ ).

#### **1.4.4 Meta-analyses: peritraumatic subjective threat**

A meta-analysis of all effect sizes related to the experience of the PTSD DSM-5 ‘A2’ criteria, namely peritraumatic fear, horror, helplessness and perceived life threat, included effect sizes from 28 studies with an overall sample size of 27,357. An overall effect size of  $r=.37$  (95% CI=0.31-0.42,  $z=11.82$ ,  $p<0.0001$ ) was estimated by the random effects model.

Estimates of heterogeneity showed that there was significant variance across the studies ( $Q=493.02$ ,  $df=27$ ,  $p<0.001$ ), and the  $I^2$  statistic indicated 94.5% of the variation was due to true variance. Figure 2 illustrates the spread of effect sizes derived from each study. A funnel plot using the ‘trim-and-fill’ method was generated and inspected for estimated missing null studies or asymmetry to indicate publication bias in the study sample; minimal asymmetry was identified and just two null studies were estimated as possibly missing. A regression test for funnel plot asymmetry indicated no publication bias ( $t=-0.21$ ,  $df=26$ ,  $p=0.832$ ), and Kendall’s tau also indicated no significant asymmetry ( $\tau=0.14$ ,  $p=0.298$ ). A calculation of the ‘fail-safe  $n$ ’ for this meta-analysis suggested that 20,834 non-significant studies would be necessary to make the overall estimate found in the meta-analysis non-significant (Oswald & Plonsky, 2010).

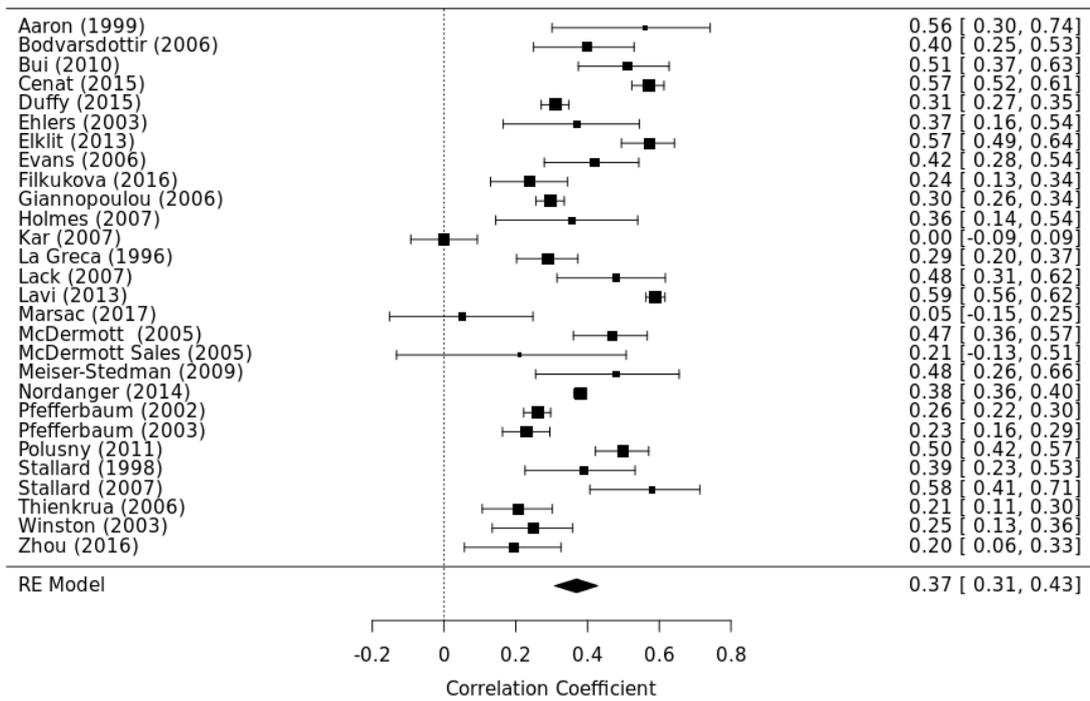


Figure 2. Forest plot for meta-analysis of peritraumatic subjective threat. Illustrating effect sizes ( $r$ ) sourced from each study, and the estimated overall effect size of the relationship between peritraumatic subjective threat and PTSD symptoms in children and adolescents.

**1.4.5 Moderators of the relationship between peritraumatic subjective threat and PTSD.** A number of meta-regression analyses were conducted to assess whether gender, age, trauma type, study type, study quality, or time between trauma and assessment of peritraumatic factors or PTSD, had any moderating effect on the strength of the relationship between peritraumatic factors and PTSD symptoms. The results of these analyses are summarised in Table 3. Age was not found to significantly account for variance in peritraumatic fear or perceived life threat effect size estimates between studies, therefore is unlikely to moderate the relationship between peritraumatic fear and perceived life threat, and likelihood of PTSD. Female gender did appear to play a role in the relationship between peritraumatic subjective threat and fear experiences and the likelihood of PTSD, with greater proportion of females in a study sample leading to larger effect sizes. Cross-sectional studies were also found to be more likely to report

larger effect sizes for peritraumatic fear and perceived threat compared to prospective studies. Importantly, study quality ratings did not have a significant affect upon effect size estimates; suggesting that the population effect size estimate would be the same even if studies with high risk of bias were excluded.

Insert Table 3 here

#### **1.4.6 Perceived life threat**

A number of studies assessed perceived life threat as a specific single item measure (see Table 2); these effect sizes were incorporated in the main meta-analysis reported, but were analysed separately to identify an overall estimate of effect size for perceived life threat alone. Twelve studies were included, giving an overall sample size of 15,432. An overall effect size of  $r=.37$  (95% CI=0.32 – 0.41,  $z=15.25$ ,  $p<0.0001$ ) was estimated by the random effects model (see Figure 3). Estimates of heterogeneity again showed that there was significant variance across the studies ( $Q=51.55$ ,  $df=11$ ,  $p<0.001$ ), although the  $I^2$  statistic indicated a slightly lower percentage of this variation attributed to true variance (78.7%). Inspection of a funnel plot and measures of asymmetry again indicated no significant likely publication bias ( $t=-0.36$ ,  $df=10$ ,  $p=0.725$ ; Kendall's tau = 0.09,  $p=0.737$ ). The 'fail-safe n' for this analysis was estimated as 5573.

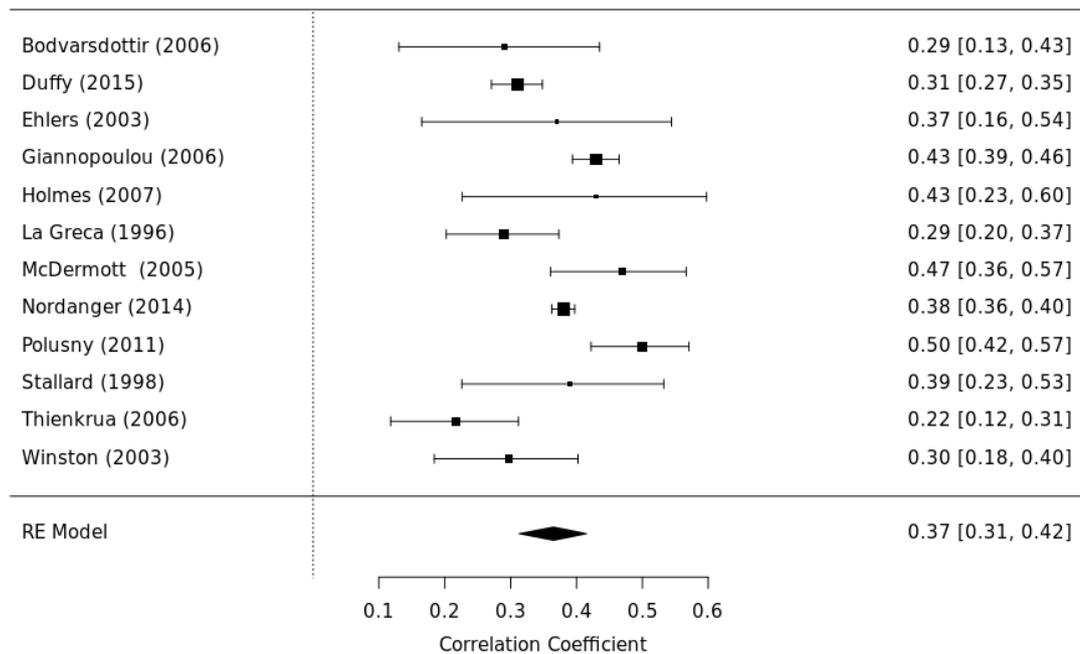


Figure 3. Forest plot for meta-analysis of peritraumatic perceived life threat. Illustrating effect sizes ( $r$ ) from each study and the overall estimate of the effect size for the relationship between peritraumatic perceived life threat and PTSD symptoms in children and adolescents.

#### 1.4.7 Peritraumatic dissociation

Five studies reported assessments of the relationship between peritraumatic dissociation and PTSD symptoms in children and adolescents, with a total sample of 566. All these studies were prospective studies, assessing peritraumatic dissociation between one to eight weeks post-trauma, and PTSD symptoms up to six months post-trauma. The majority of the samples were individuals who had experienced an acute physical injury (RTA, other accident, or assault), one study related to witnessing a terror event, and none of these studies related to natural disasters. An overall effect size of  $r=.17$  (95% CI=0.03 – 0.29,  $z=2.44$ ,  $p<0.05$ ) was estimated by the random effects model (see Figure 4). Estimates of heterogeneity suggested some variance across the studies,

approaching significance ( $Q=9.27$ ,  $df=4$ ,  $p=0.055$ ), and the  $I^2$  statistic indicated a moderate degree of heterogeneity with an estimated 56.8% of variation due to true variance. Inspection of a funnel plot and measures of asymmetry again indicated no significant likely publication bias ( $t=0.099$ ,  $df=3$ ,  $p=0.928$ ; Kendall's tau = 0.2,  $p=0.817$ ). The 'fail-safe n' for this analysis was estimated as just 23 studies required to challenge the significance of this overall estimate.

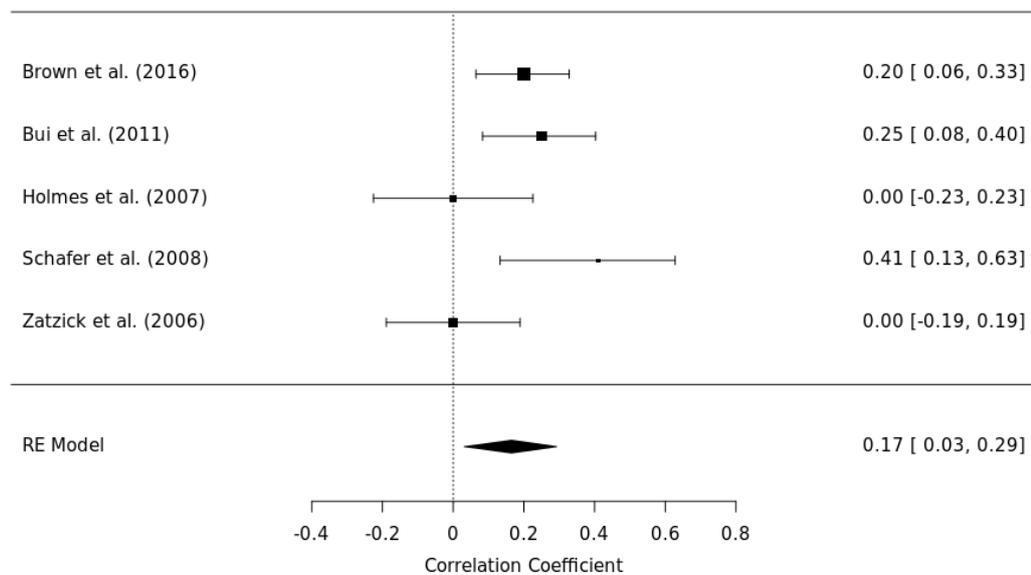


Figure 4. Forest plot for meta-analysis of peritraumatic dissociation. Illustrating effect sizes ( $r$ ) from each study and the overall estimate of the effect size for the relationship between peritraumatic dissociation and PTSD symptoms in children and adolescents.

**1.4.8 Moderators of the relationship between peritraumatic dissociation and PTSD.** Meta-regression analyses were conducted to assess whether age, gender, time between trauma and assessment, and study quality had any moderating effect on the relationship between peritraumatic dissociation and PTSD. These results are tentative and conclusions should be drawn with caution considering the small number of studies included in this analysis; increasing age and increasing time between trauma occurrence and assessment of peritraumatic dissociation appeared to have a negative moderating

effect on the strength of the relationship between peritraumatic dissociation and PTSD symptoms. Table 4 summarises the results.

Insert Table 4 here

### 1.4.9 Data-driven processing

Two studies were identified which reported results indicative of the relationship between data-driven processing (feeling muddled or confused during or immediately after the trauma) and PTSD symptoms in children and adolescents. Result from a random-effects model: ( $k=2$ ) suggested an overall effect size estimate  $r=.29$  (95% CI= 0.138 – 0.429,  $z=3.66$ ,  $p<0.001$ ). Estimates of heterogeneity showed that there was very little variance between the two studies ( $Q=0.02$ ,  $df=1$ ,  $p=0.894$ ,  $I^2= 0\%$ ). A fail-safe  $n$  could not be calculated for such a small sample; however, clearly this is an area requiring more research.

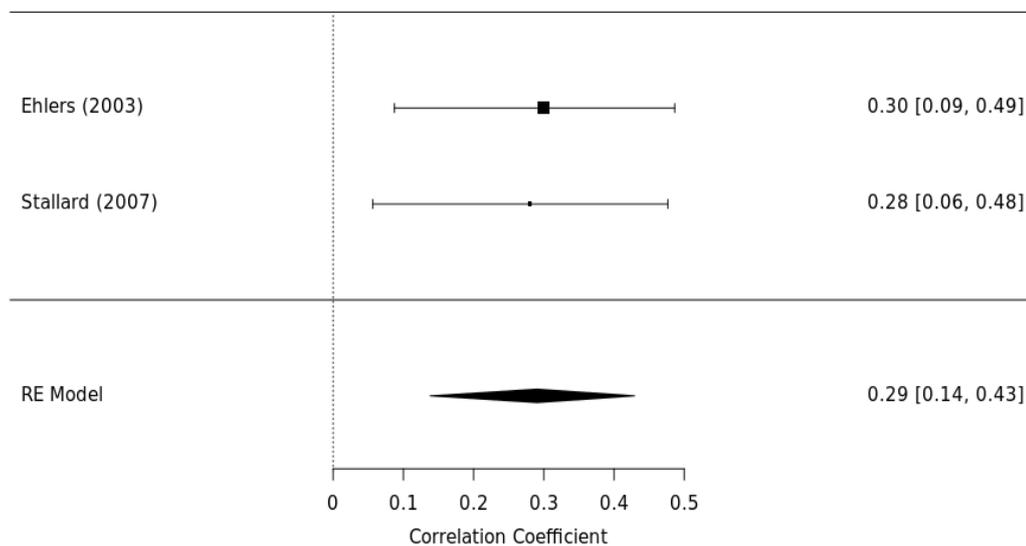


Figure 5. Forest plot for meta-analysis of data-driven processing. Illustrating effect sizes ( $r$ ) and the overall estimate of the effect size for the relationship between data-driven processing and PTSD symptoms in children and adolescents.

## 1.5 Discussion

### 1.5.1 Overall findings

The current review provided a summary and update of the research currently available pertaining to the relationship between a number of peritraumatic psychological factors and PTSD in children and adolescents. Thirty-two studies published since the DSM first defined PTSD in 1980 were identified as having explored the predictive power of peritraumatic factors in the development of PTSD in children and adolescents, providing 47 effect size estimates for the strength of the relationships between different peritraumatic factors and PTSD. The results obtained from this pooled sample of studies were grouped according to the specific peritraumatic process or experience assessed. A number of meta-regression analyses provided insight into possible moderating factors on the size of effect size from each peritraumatic factor. The main group of peritraumatic factors assessed subjective threat and fear, reflecting the DSM-IV 'A2' criteria for PTSD; feeling fear, horror, helplessness, and/or perceived life threat during or immediately after the trauma. A small sample of studies were found to assess peritraumatic dissociation and data-driven processing.

**Subjective threat, fear and perceived threat to life.** The large proportion of studies identified explored factors which reflected subjective threat and fear responses during trauma as predictors of PTSD symptoms. This may be in part due to the previous diagnostic expectation that these peritraumatic experiences were necessary for the diagnosis of PTSD, and therefore have been likely candidates for assessment in research studies. Cognitive models of PTSD have also depicted the integral role of fear responses, perceived helplessness and perceived threat at the time of trauma in the development of PTSD. The meta-analysis demonstrated that subjective threat and fear response yielded a moderate to large overall population estimate of effect size as a

predictor of PTSD. The effect size estimate of .37 ( $k=28$ ) was similar to the estimate of peritraumatic fear reported by Trickey et al. (2012), reporting a population estimate of effect size of .36 ( $k=6$ ) for this predictor of PTSD in children and adolescents.

Exploration of the effect sizes which were related to the specific measurement of perceive threat to life alone also produced a moderate to large population effect size of .37; this result again being very similar to the population estimates reported previously both by Trickey et al. (2012) of .36 and Cox et al. (2008) of .38. These previous meta-analyses were based on much fewer studies and so a smaller overall sample size. This result further confirms the validity of the previous conclusions made by Trickey et al. and Cox et al. as a greater number of studies were identified and meta-analysed in the current review. The possible inclusion of more studies in the current review also increased the breadth and depth of trauma and participant characteristics assessed: the age range of the overall sample included was aged 3-26 years; the trauma types included a range of natural disasters, terror attacks, children suffering physical injury from RTAs, assaults, and other accidents; traumas occurred in the US, Asia, Europe and Africa; and PTSD symptoms were assessed from one month up to six years post-trauma. This range of study and participant characteristics, and the random effects analysis model used, supports the generalisability of conclusions to wide ranging populations and trauma experiences, although does also bear some possible limitations and caveats. Overall, the result supports the assertion that the experience of peritraumatic fear responses and perceived threat are likely to play a role in the likelihood of the development of PTSD in children and adolescents. However, it is also important to reflect that the effect sizes for the role of subjective threat and fear responses reported by studies were fairly consistently moderate; which, if understood in the context of our conceptualisation of traumatic events and PTSD, may not be what is expected. One might reasonably consider that the experience of perceived life threat and feeling fear during an event

occurs in most cases where PTSD develops, and so effect sizes reflecting the relationship between these experiences and PTSD should be consistently large. The results of this current meta-analytic review suggest that there may be a limit to what the assessment of peritraumatic factors can explain in the development of PTSD. With moderate effect sizes indicated, it may be that some individuals experience these peritraumatic factors and do not develop PTSD, and conversely, that some individuals develop PTSD without the experience of these factors. The removal of 'A2' criteria from DSM-IV to DSM-5 also reflects this possibility (Friedman, et al., 2011). Therefore, while these factors are likely to play a role in the development of PTSD, and may be helpful in screening individuals at greater risk for PTSD after trauma, there is a limit to their utility and it is clearly important to consider what other factors are involved.

**Peritraumatic dissociation.** Dissociative responses to trauma have been conceptualised as responses which significantly hamper the adaptive processing of a traumatic event as it occurs, and can increase feelings of helplessness and powerlessness associated with the trauma. It is therefore typically understood to be an experience which can increase one's vulnerability to developing PTSD. Conversely, dissociation can also be conceptualised as an adaptive response to situations of extreme threat, as the body shuts down responsiveness and conserves resources. Despite just five studies being identified within child and adolescent populations, and an overall sample size of 556 individuals, the meta-analysis estimated a small population effect size, which may be deemed lower than expected considering the previously implicated role of dissociation in previous research and theory of PTSD. The heterogeneity of the studies included was moderate, and much less than the heterogeneity found generally in the studies included in this review, suggesting that the measurement of peritraumatic dissociation, the study characteristics and context of its assessment was not large. Two

studies reported a non-significant effect of dissociation but reported no effect size, so 0 was entered as the statistic for these studies. This method was aimed to reduce over-estimation of effect sizes and avoid bias in the absence of reported statistics, however it may be that in this small sample of studies this may have made the population estimate of effect size more conservative. Considering the small number of studies identified, and that this current review is the first meta-analytic review of peritraumatic dissociation in children and adolescents to date, this may be an area for more research. The results of this current review suggest that this factor may not be a significant predictor of PTSD in this population.

**Data-driven processing.** With just two studies identified as measuring peritraumatic muddled or confused processing of the event, conclusions regarding the power of this experience as a risk factor for PTSD are also limited. However, the results of an estimated moderate to large effect size, with two particularly homogenous studies, does indicate that further research into this factor is clearly warranted and necessary to aid better understanding of its role in the development of PTSD.

#### **Moderators of the relationship between peritraumatic factors and PTSD.**

Our moderation analysis suggested studies with younger populations were likely to find just as large effect size estimates for subjective threat and fear response as those with older populations. However, meta-regression analysis of variance in effect size estimates for peritraumatic dissociation suggested that age had a significant negative effect; suggesting that as age increases the size of the effect of peritraumatic dissociation in predicting PTSD decreases. A caveat of conclusions drawn from meta-regression analyses of age is related to the limitation of utilising sample mean age for these analyses; mean age does not comprehensively capture the full range and spread of ages in the samples included.

As may have been expected (as female gender has been shown to be a consistent but small predictor of PTSD), more females in a study sample lead to an increased likelihood of a greater effect size to be reported. This moderating relationship was not, however, found to be significant for peritraumatic dissociation. Cross-sectional studies indicating greater effect sizes may have been a reflection that cross-sectional studies provide insight into correlation and association, whereas prospective studies are better measures of prediction over time, and that the predictive power of a peritraumatic factor reduces over time. However, it is worth noting that peritraumatic factors were found to have a large effect size even when the time between peritraumatic factor assessment and PTSD symptom assessment spanned a number of years. The size of effect of peritraumatic dissociation as a risk factor for PTSD appeared to decrease significantly over longer times periods between the trauma occurring and the assessment of peritraumatic dissociation. It may be that if a child experiences dissociation during a trauma, their memory of this experience is particularly vulnerable to being forgotten, whereas the active experience of fear or perceived life threat is held in the memory of the trauma more clearly. However, any suggestions from the meta-regression analyses of peritraumatic dissociation effect size should be taken with caution; with just five studies available on peritraumatic dissociation, meta-regression analyses are likely to be underpowered and could lead to false positive results (Hedges & Pigott, 2004; Higgins & Thompson, 2004).

Finally, meta-regressions exploring the relationship between effect size and study quality suggested no significant difference between the size of the effect found in high versus medium or low quality studies. This indicated that even if the results of low quality studies (those with high risk of bias) were removed from the analyses, the overall estimates of effect size would largely remain the same.

### **1.5.2 Limitations**

Despite providing a valuable and novel review of the role of peritraumatic risk factors in the development of PTSD in children and adolescents, a number of limitations are important to note with regards to this study and conclusions drawn. Firstly, the identification of studies assessing what would be deemed a ‘peritraumatic’ factor was potentially problematic. Many authors will not label a peritraumatic factor as such; careful reading of the study methodology and wording of assessment measures was often needed to determine if a factor was reflecting an experience which occurred during or immediately after a trauma. For example, perceived life threat can be a post-trauma appraisal rather than a peritraumatic perception. This difficulty meant that our search and screening process was required to be comprehensive and perhaps atypical, and it may be likely that other authors have assessed peritraumatic experiences but not clearly described them as such, or they were missed. Furthermore, the assessment of peritraumatic factors varied greatly; we identified that most studies used single-item measures of a certain experience, such as feeling fear or data-driven processing, and there were few full and validated measures of peritraumatic experiences. This meant that the measurement of peritraumatic factors was not standardised; the most appropriate method of accounting for this was by grouping factors based on the content or vocabulary used in each measure. Despite this, variance in effect sizes could have been partially attributed to variance in the assessment of these experiences. This highlights the need for the development of standardised measurement of peritraumatic factors.

The second limitation relates to the generalisability of the findings; despite random effects analysis methods being applied to increase this, we cannot confidently conclude that these results may apply to the experience of multiple traumas as all studies included related to single-event traumas, except a handful of studies which

related to war. Our study exclusion criteria requiring peritraumatic factors to be assessed within a reasonable time after trauma may have led to the disproportionate exclusion of the types of studies which explore ongoing or multiple trauma experiences in childhood, particularly experiences of abuse, as these children seem to be more likely to be assessed years after the trauma occurrence. It is also likely that our current evidence base related to this population of children is limited; concrete, single event traumas such as RTAs, natural disasters or terror attacks seem to lend themselves more easily to recruitment for prospective studies. It may be valuable for researchers to consider more novel ways of identifying and recruiting children who have experienced multiple traumas such as abuse and consider how peritraumatic factors may reliably be assessed in this group.

A final limitation of this review is the relatively small number of studies included; despite this being a significant increase in the number of studies reported by previous meta-analytic reviews of this population (six studies identified by Trickey et al., 2012, and four by Cox et al., 2008). It has been argued that only two studies are needed for a meta-analysis, which supported our analysis of the effect sizes sourced for data-driven processing, nevertheless there are limitations related to the power of the analysis (Valentine, Pigott, & Rothstein, 2010). There is no concrete recommended number of studies needed for a meta-analysis of correlation coefficients or for meta-regression analyses, however, consideration of the limitations of conclusions from a small number of studies is advised. It is particularly important to consider the heterogeneity of studies. The conclusions drawn from the meta-regression analyses of dissociation effect sizes are particularly limited in this review. Valentine et al. (2010) argue that retrospective power analyses in meta-analysis can be uninformative, and a better indicator of inferences made from 'small n' meta-analyses may be by use of confidence intervals. They also argue that a meta-analysis of even two studies is more

informative and more likely to be valid than other synthesis techniques (such as narrative synthesis of study findings). Large heterogeneity between studies and relatively small numbers of studies is a typical situation for many meta-analyses and meta-regressions (Higgins & Thompson, 2004), therefore we conclude that pragmatic consideration of the limitations of conclusions is appropriate but that the results may still add value to the field.

### **1.5.3 Implications**

The current meta-analytic review supports previous suggestions that certain peritraumatic experiences are likely to be important risk factors for the development of PTSD. The results particularly provide support for the role of these factors in children and adolescents: experiencing feelings of extreme fear, perceived life threat and confused and muddled processing of the event at the time of the trauma may increase a child's likelihood of developing symptoms of PTSD. This supports cognitive models of PTSD which describe how fear responses, perceived threat and poor processing of the event play a role in the development of post-trauma stress symptoms, with post-trauma cognitive processing and behaviours playing a role in the maintenance of PTSD (Brewin, et al., 1996; Ehlers & Clark, 2000). In contrast, the evidence from this review suggests a less pronounced role of peritraumatic dissociation as a risk factor for PTSD. This understanding of the key experiences during trauma which are associated with an increased risk of PTSD may help clinicians and researchers identify which children may be at greater risk of developing PTSD in the acute phase following trauma, by identifying if they had these peritraumatic experiences.

This identification of those at heightened risk may help to target intervention strategies. Furthermore, this supports the focus of intervention strategies, such as

trauma-focussed CBT for children and adolescents, incorporating cognitive re-processing of trauma-related appraisals and reducing fear responses associated with trauma-related stimuli (Cohen, Deblinger, Mannarino, & Steer, 2004; Smith, et al., 2013). Finally, the conclusions of the present review may be helpfully considered in line with the recent change in the diagnostic requirements of PTSD from DSM-IV to DSM-5, which no longer stipulates the requirement for experiencing fear, helplessness and/or perceived life threat at the time of trauma. The previous measurement of these factors may have been due to researchers' conceptualisation of these experiences as diagnostic necessities, however, these peritraumatic experiences may be more appropriately considered as risk factors for the development of symptoms. We encourage the further exploration of peritraumatic experiences, better standardised measurement of them, and timely identification of whether children and adolescents' experience of trauma was particularly characterised by fear, perceived life threat, confused or muddled processing to further develop our academic understanding of this debilitating disorder and its appropriate treatment. Researchers and clinicians are also encouraged to consider the importance of peritraumatic factors as relative in comparison to other pre-trauma, trauma-related and post-trauma factors. The effect sizes indicated for peritraumatic factors were small to moderate; hence, they are unlikely to account for a large proportion of variance in PTSD symptoms suggesting other factors with equal or greater predictive power. Whilst there is a limit to the information provided by peritraumatic experiences and the clinical value they hold, they may be most helpfully considered in conjunction with other psychosocial, cognitive and emotional factors involved in the development of PTSD for an holistic consideration of the pathways to PTSD, and the psychological processes and experiences which may make an individual child at higher risk of developing chronic symptoms of PTSD.

## 1.6 Tables

**Table 1** summarising study characteristics can be found in the supplementary materials (Appendix A.3).

**Table 2.** Description of how peritraumatic factors were assessed, with effect sizes extracted from each study

(References for studies included in the meta-analyses which may be listed in this table but are not cited within the main article text are listed in supplemental materials. See Appendix 4 for this reference list.)

<b>Peritraumatic factor</b>	<b>Study</b>	<b>Description of measure</b>	<b>k</b>	<b>Mean r</b>
<i>'A2' criteria; including fear, horror, helplessness or perceived life threat (PLT)</i>				
Fear	Aaron, et al. (1999)	Narrative account from the child about the event, their feelings during and immediately after, and Likert scale questions addressing level of fear and perceived life threat. Index of overall fear was created by summing the scores from their self-reported fear and life threat items.	1	0.56
Fear	Evans & Oehler-Stinnett (2006)	Self-report item rating how scared they felt during the tornado	1	0.42
Fear	Filkuková, et al. (2016)	Semi-structured interview of trauma experience; IPA to ascertain themes, including fear during and immediately after the attack	1	0.24
Fear	Lack & Sullivan (2007)	One item scale: 5 responses from not at all scared to terrified	1	0.48
Fear	McDermott Sales, et al. (2005)	Child rated how scared, upset or frightened or relaxed and happy they felt during the event, by indicating which of two puppets (frightened vs relaxed) they felt like, and then asked to indicate how much they felt like that (1 to 4 response scale from extremely happy and good to extremely frightened and upset).	1	0.21

Fear	Zhou, et al. (2016)	One item from a self-report questionnaire assessing trauma experiences: 'Did you feel scared when the earthquake happened?'	1	0.19
Fear & PLT	Ehlers, et al. (2003)	Child indicated whether they thought they were going to get hurt or die, and the extent to which they felt scared/frightened during the event (scale 1 'not scared' to 3 'a lot'); fear response score was the maximum of these two answers.	1	0.37
Fear & PLT	Kar, et al. (2007)	Unclear: "(child)... had extreme degree of fear with perceived life threat during the cyclone"	1	0
Fear & PLT	Meiser-Stedman, et al. (2009)	3 item measure including perceived life threat, perceived threat of harm and feeling scared	1	0.48
Fear & PLT	Stallard & Smith (2007)	Average score of three questions 'How serious was your accident?', 'Did you think that you were going to get hurt/die during the accident', and 'Did you feel frightened/scared during the accident?'	1	0.58
Fear & PLT	Winston, et al. (2003)	STEPP questionnaire items 'when you got hurt, or right afterwards, did you feel really afraid?' and 'when you got hurt, or right afterwards, did you think you might die?'	2	0.25
PLT	Duffy, et al. (2015)	Unclear- possibly two items from 10-item exposure questionnaire rating whether the person 'thought he/she was going to die' and if they 'saw others who they thought were going to die'.	2	0.31
PLT	La Greca, et al. (1996)	One item from hurricane-related traumatic experiences (HURTE) scale: 'at any point during the hurricane, did you think you might die?'	1	0.29
PLT	McDermott, et al. (2005)	Unclear- possibly two items from a wildfires experiences questionnaire 'thought I might die' and 'thought family member might die'	2	0.47
PLT	Nordanger, et al. (2014)	One item measure: 'To what extent did you perceive the terror events as a threat to your own life or the lives of someone close to you?'	1	0.38
PLT	Polusny, et al. (2011)	Sum of three items from HURTE (Hurricane-Related Traumatic	1	0.5

		Experiences) questionnaire; 'did you get hurt in the storm?', 'were you afraid you would be injured in the storm?' and 'were you afraid you would be killed in the storm?' (with few endorsing sustaining injury, so predominantly a measure of perceived threat of harm or life threat).		
PLT	Stallard, et al. (1998)	Semi-structured interview asking children to describe what happened during and immediately after the event. Reported as 'thought I would die'.	1	0.39
Terror, helplessness, PLT & fear	Bödvarsdóttir, et al. (2006)	Questionnaire about stressors during the earthquake: one item assessing fear of death; one item assessing feelings of terror; and one item assessing helplessness felt during the earthquake.	3	0.4
PLT & distress	Giannopoulou, et al. (2006)	Index of perceived life threat: sum of endorsed items including fear of death, concern for the safety of others. Index of distress: sum of endorsed items including distress at witnessing scenes in the neighbourhood and distress at viewing scenes on TV.	2	0.29
Fear, PLT & helplessness	Holmes, et al. (2007)	Self-report items: 'when you saw the attack did you feel scared?', 'did you feel like your life was in danger?' and 'did you feel like there was nothing you could do?'	3	0.36
Fear, PLT & helplessness	Thienkrua, et al. (2006)	Tsunami modified version of the 'PsyStart Rapid triage system' used to ask questions about trauma experiences: 'felt one's own or a family members life in danger', 'felt unable to escape', and 'felt extreme panic or fear'.	3	0.21
Distress	Bui, et al. (2011)	Peritraumatic Distress Inventory: 13 self-report items assessing the A2 criteria of DSM-IV PTSD, including: criteria sadness/grief, frustrated/angry, afraid for own safety, guilt, ashamed of emotional reaction, worried for the safety of others, afraid of losing control of emotions, difficulty controlling bladder, horror, physical symptoms	2	0.51

		of panic, fear of passing out, and perceived life threat.		
Distress	Cénat & Derivois (2015)	Peri-traumatic distress inventory, as described above.	1	0.57
A2	Elklit & Kurdahl (2013)	Initial response to the event involving fear, helplessness, horror or perceived life threat.	1	0.57
A2	Lavi, et al. (2013)	Sense of fear during the war assessed by five statements in accordance with the A2 criteria for PTSD e.g. 'During the war I felt that my life was in danger'	1	0.59
A2	Marsac, et al. (2017)	Trauma-related appraisals: from ASC-Kids peri-trauma 4 item subscale ('it was shocking/awful horrible'; 'wanted to make it stop but couldn't'; 'felt scared'; 'thought might die')	1	0.05
Peritraumatic response	Pfefferbaum, et al. (2002)	'Peri-traumatic response scale'; included 12 items addressing peritraumatic responses of fear, arousal and dissociation: 'thought I would die; trembling/shaking; heart beat fast; nervous or afraid; made me jump; on automatic pilot; scared someone in my family would be hurt; scared a friend/a teacher would be hurt; frightened by how scared my teachers acted; upset by how I acted; helpless.' Total score indicating greater peritraumatic response.	1	0.26
Peritraumatic response	Pfefferbaum, et al. (2003)	'Peritraumatic reaction scale', described as above; 13 items on how the child felt when the bomb went off.	1	0.23
<b>Peritraumatic dissociation</b>				
	Brown, et al. (2016)	Peritraumatic dissociation items from the DICA-ASD summed to create a continuous dissociation total score	1	0.2
	Bui, et al. (2011)	Peritraumatic dissociative experiences questionnaire; 10 item questionnaire with items describing dissociative experiences	1	0.25
	Holmes, et al. (2007)	One item self-report: 'did it feel like it wasn't real?'	1	0
	Schäfer, et al. (2004)	Peritraumatic dissociation: children rated the presence of each of the	1	0.41

	symptoms in ASD criteria with the following items: ‘Did the world around you seem strange or unreal?’; ‘Did your body feel strange, as if it was not really yours?’; ‘Have you been less aware of what was happening?’; ‘Did you feel numb or did you have no feelings at all?’; ‘Are there any important details which you cannot remember?’.		
Zatzick, et al. (2006)	Unclear- no description of how this was assessed	1	0
<b>Data-driven processing</b>			
Ehlers, et al. (2003)	One item question indicating the extent to which they were muddled/confused during the accident	1	0.3
Stallard & Smith (2007)	One item ‘Did you feel confused or muddled during the accident?’	1	0.28

**Table 3.** Results of meta-regression analyses of moderators on the strength of the relationship between peritraumatic subjective threat ('A2') factors and PTSD symptoms.

<b>Moderator</b>	<b>Estimate</b>	<b>SE</b>	<b>l.CI</b>	<b>u.CI</b>	<b>p</b>
	<b>(r)</b>				
<b><i>Continuous moderators</i></b>					
% Female	0.008	0.004	0.001	0.016	0.036
Mean age	0.017	0.014	-0.010	0.044	0.218
Time between trauma and assessment of peritraumatic factors	0.002	0.001	-0.001	0.004	0.154
Time between peritraumatic factor assessed and PTSD assessed	-0.001	<0.001	-0.002	0.001	0.336
<b><i>Categorical moderators</i></b>					
<b>Study Quality (High vs other)</b>					
High (k=6)	0.351	0.081	0.206	0.482	
Medium or Low (k=22)	0.374	0.039	0.306	0.437	
Meta-regression coefficient	0.026	0.090	-0.149	0.198	0.773
<b>Study Type</b>					
Cross-sectional (k=18)	0.404	0.041	0.336	0.468	
Prospective (k=10)	0.294	0.059	0.185	0.395	
Meta-regression coefficient	-0.125	0.071	-0.259	0.015	0.079
<b>Trauma Type (Interpersonal vs non-interpersonal)</b>					
Non-interpersonal (k=19)	0.361	0.043	0.285	0.433	
Interpersonal (k=8)	0.378	0.062	0.268	0.477	
Meta-regression coefficient	0.018	0.076	-0.129	0.165	0.808

**Table 4.** Results of meta-regression analyses of moderators on the strength of the relationship between peritraumatic dissociation and PTSD symptoms.

<b>Moderator</b>	<b>Estimate (r)</b>	<b>SE</b>	<b>l.CI</b>	<b>u.CL</b>	<b>p</b>
% Female	0.000	0.006	-0.012	0.012	0.99
Mean age	-0.066	0.031	-0.126	-0.005	0.034
Time between trauma and assessment of peritraumatic factors	-0.04	0.018	-0.075	-0.004	0.029
Time between peritraumatic factor assessed and PTSD assessed	-0.003	0.014	-0.029	0.024	0.851
<b>Study Quality (High vs medium)</b>					
High (k=3)	0.174	0.075	0.028	0.313	
Medium (k=2)	0.148	0.093	-0.033	0.319	
Meta-regression coefficient	-0.027	0.119	-0.255	0.205	0.823

## 1.7 Chapter 1 References

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## Chapter 2. Bridging Chapter

The current review outlined in Chapter 1 provided a systematic overview of the available evidence to date regarding the role of psychological peritraumatic risk factors in the development of PTSD in children and adolescents. The evidence presented in this review was a significant addition to the previous reviews including meta-analytic summary of peritraumatic risk factors in children and adolescents (Cox, Kenardy, & Hendrikz, 2008; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012); however, it may still be concluded that the evidence base in children is limited. Our review had a major focus on peritraumatic factors related to subjective perception of threat and fear response, as a majority of studies reporting effect sizes related to peritraumatic factors (28 of 32) had assessed these types of peritraumatic experiences. The data synthesis and meta-analyses demonstrated that experiencing peritraumatic fear and perceived life threat appear to be key risk factors for the development of PTSD symptoms, yielding moderate to large estimates of population effect size. Similarly, data-driven processing was also estimated to have a moderate to large population effect size as a risk factor for the development of PTSD symptoms in children and adolescents, however, just two studies were identified which reported on data-driven processing as a risk factor in this sample and eligible for inclusion in our review. Only five studies had assessed peritraumatic dissociation, and the overall estimate of population effect size was perhaps smaller than expected considering the suggested importance of this experience in the development of PTSD (Breh & Seidler, 2007). Estimates of between study variation indicated high heterogeneity between studies, particularly in the main group of studies assessing subjective threat and fear, with lesser heterogeneity between studies assessing peritraumatic dissociation and data-driven processing. This variance may have been related to difference in study samples, trauma types, or methodology. Our

exploration of how peritraumatic factors were assessed was a key outcome of the review. A common methodology used by researchers was the use of single item measures of specific experiences during trauma. However, there was arguably little standardisation of measurement of these factors or well-validated developed full measures of peritraumatic factors. Overall, the findings of this meta-analytic review demonstrated a warranted need for further research regarding peritraumatic psychological experiences in children and adolescents and the development of PTSD symptoms.

The aims of the current empirical study, outlined in Chapter 3, served to address some of the areas of need highlighted in the conclusions of the review, and further build upon this direction of research pertaining to predictors or risk factors of PTSD in children and adolescents. We were presented with the valuable opportunity to source data from a pre-existing study which recruited a large sample of children and adolescents following their experience of a single-event trauma and presentation at an Emergency Department (The 'ASPECTS' study; Meiser-Stedman, et al., 2017). This study sample represents one of the largest recruitment and prospective longitudinal assessment of children and adolescents following an acute physical injury resulting from a trauma to date (see chapter one for a summary of similar pre-existing studies). The participants had completed two assessments; at approximately two weeks and nine weeks post-trauma, involving a battery of self-report and structured interview measures completed with both the child and their parents. Initial screening of the measures used in this study demonstrated an opportunity to source data and plan analyses to address some areas of research need identified from our literature review.

The literature review completed for the meta-analysis outlined in chapter one, in addition to a brief review of literature completed to inform the development of the

empirical study, demonstrated that studies have assessed a multitude of pre-trauma, trauma-related, and post-trauma variables to develop our understanding of PTSD, however, often studies of risk factors for PTSD assess a list of variables as risk factors with little coherence between studies regarding why each factor is relevant, and the factors are related. It has been acknowledged that the variation and conflict in evidence supporting and challenging the role of different risk factors for PTSD, suggests that there are likely to be multiple causal pathways to PTSD (King, Vogt, & King, 2004). Investigating how risk factors work together, considering multicollinearity or independence, mediating or moderating effects has also been argued to aid appropriate identification of at-risk individuals and design suitable interventions (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). The use of theoretically driven models of predictors of PTSD can also inform how factors are analysed and understood in relation to other factors. For example, the assessment of data-driven processing may be helpfully considered in relation to other cognitive factors informed by cognitive models of the development and maintenance of PTSD symptoms (Ehlers & Clark, 2000). It is unlikely that a certain risk factor acts in isolation; therefore, it was identified that it is important to understand the role of peritraumatic and other risk factors within models of predictors of PTSD.

Secondly, it was identified that there is a need for further consideration of how these factors may also predict other poor mental health outcomes of trauma. PTSD is not the only academically or clinically documented unfortunate outcome of experiencing a trauma in childhood, however it is often the focus of research, with other disorders typically studied as comorbid presentations. Depression, other anxiety disorders, and behavioural difficulties can arise following the experience of trauma in childhood (Pine & Cohen, 2002), and there is recent increased attention related to the

development of ‘Complex PTSD’, with the impending publication of the new ICD-11 diagnostic manual incorporating this as a new diagnosis applicable to both children and adults (Maercker et al., 2013). Complex PTSD, in particular, has received very little research to date in child and adolescent populations. The expected publication of the new ICD-11 in 2018 will bring a new diagnosis which clinicians will be required to consider as an appropriate diagnosis for children and adolescents presenting with mental health difficulties following trauma. Understanding of the psychological processes and how trauma-related experiences are involved in the development of this disorder, will aid clinicians in their application of this new diagnosis in clinical practice and consider appropriate intervention strategies. However, to date, just two studies have been published which have assessed CPTSD in children and adolescents; offering some valuable validation of this diagnostic category, some suggestion of possible risk factors for complex PTSD presentations as opposed to typical PTSD presentations, and some evidence to support the use of psychological therapy to treat this disorder in this population (Perkonigg et al., 2016; Sachser, Keller, & Goldbeck, 2017). Understandably, more research related to CPTSD in children and adolescents is of vital need.

Thirdly, the literature review process highlighted the value of assessment of peritraumatic and other trauma-related factors soon after trauma, and the benefit of longitudinal follow-up to assess mental health disorder symptoms after a reasonable amount of time post-trauma. Studies which have recruited and assessed participants soon after trauma, are more likely to gather accurate and valid data pertaining to the individual’s experience of the trauma, than studies which assess trauma-related experiences after an extended period. In such studies, there is less risk that the individual’s report of what happened, and their thoughts and feelings about the trauma,

will have been affected by forgetting (Candel & Merckelbach, 2004), post-traumatic processing of the trauma, any intervention or effects of support, or experience of further traumas which have occurred in the interim time between initial trauma and assessment. Prospective longitudinal studies assessing risk factors soon after trauma, and the development of symptoms at a later time, also have a benefit over cross-sectional studies, as they allow for the separation of the assessment of risk factors and outcomes over time. This subsequently enables analyses which may provide results indicative of predictive relationships between factors and outcomes, rather than simple correlations. It has been emphasised in psychiatric research of risk factors for disorder that there is a requirement in the definition of a risk factor that it precedes the outcome of interest; therefore, it is advisable to measure a risk factor in participants and then follow up to assess outcomes in prospective longitudinal studies (Kraemer et al., 1997). This information regarding factors which are associated with increased risk of symptoms weeks or months post-trauma is valuable in understanding the development of a disorder, and what key indicators may be present for increased risk soon after trauma occurrence. In most individuals, it is most likely that initial symptoms of distress post-trauma will subside in the few weeks following the event (Le Brocque, Hendrikz and Kenardy, 2009); it is therefore important to allow time to pass before assessing which individuals are likely to have developed chronic symptoms of disorder.

The data available from this pre-existing study sample provided the opportunity to explore the role of different models of risk factors, including peritraumatic factors, in the development of PTSD, Depression and Generalised Anxiety Disorder (GAD), and Complex PTSD, in a prospective longitudinal assessment of children and adolescents who had recently experienced a single event trauma. We aimed to build upon the existing understanding of peritraumatic risk factors of fear, perceived life threat,

dissociation and data-driven processing, and models of psychosocial risk factors and event-related risk factors to understand the comparative role of these factors in the development of different mental health outcomes of trauma in children and adolescents.

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**Chapter 3. Predictive models of PTSD, Complex PTSD, depression and anxiety in children and adolescents following a single-event trauma**

(Written up to be published in Journal of Consulting and Clinical Psychology. See Appendix B.1 for a summary of author guidelines for manuscript preparation)

Abbreviated title:

Predictive models of PTSD & CPTSD in children and adolescents

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### 3.1 Abstract

**Objective:** This study examined the power of research and theory-derived predictive models of the development of PTSD, Complex PTSD, depression and anxiety in children and adolescents who had experienced a single-event trauma.

**Methods:** Children (n=260, aged 8 – 17 years) recruited from local Emergency Departments were assessed at two and nine weeks post-trauma. Data obtained from self-report questionnaires completed by the child, telephone interviews with parents and hospital data were used to develop four predictive models of risk factors for PTSD, CPTSD, depression and Generalised Anxiety Disorder. ICD-11 proposed diagnostic criteria were used to generate measures for CPTSD and PTSD, to assess for risk factors and identify the sample prevalence of these disorders.

**Results:** At nine weeks post-trauma, 64% did not meet criteria for any disorder, 23.5% met criteria for PTSD, 5.2% met criteria for CPTSD, 23.9% and 10.7% had developed clinically significant symptoms of depression and GAD, respectively. A cognitive model was implicated to be the most powerful predictive model; a psychosocial model was weak, and subjective markers of event severity were more powerful than objective measures.

**Conclusions:** The development of symptoms of CPTSD may occur in children and adolescents who have experienced a single-event trauma; validating this new ICD-11 diagnostic category and encouraging the conceptualisation of its development after single trauma. The cognitive model of PTSD shows utility in identifying predictors of PTSD, CPTSD, depression and GAD, particularly the role of trauma-related negative appraisals. This supports the application of cognitive interventions which focus upon re-appraising trauma-related beliefs in children and adolescents.

**Keywords:** PTSD, children, adolescents, predictors, trauma

### **3.2 Public Health Significance Statement**

This study demonstrates the relevance of considering the development of complex PTSD symptoms in children and adolescents who have experienced a single-event trauma, whilst also considering other mental health outcomes of trauma including ‘typical’ PTSD, depression or anxiety. Children who have experienced interpersonal traumas, fear, panic or dissociation at the time of the trauma, and have particularly negative appraisals of the event, and/or engage in rumination about the event, may be at increased risk of developing PTSD, CPTSD and other mental health difficulties.

### **3.3 Introduction**

#### **3.3.1 Post-traumatic Stress Disorder and related disorders in children and adolescents**

A broad range of adverse psychopathological outcomes have been studied following trauma in children, including PTSD or acute stress disorder (ASD), depression, conduct and behavioural difficulties, separation anxiety, phobias, and generalised anxiety disorder (GAD); with depression and anxiety disorders as most commonly developing following trauma over other types of disorders (Pine & Cohen, 2002). The focus of research on psychopathology following trauma has largely been on PTSD and the related presentation of ASD, and other disorders resulting from trauma have typically been studied as comorbidities of post-traumatic stress, or as secondary outcomes of PTSD (Goenjian et al., 1995). However, evidence from adult studies indicates that individuals may develop disorders other than PTSD, such as phobias or depression, and not develop symptoms of PTSD at all (Ehring, Ehlers, & Glucksman,

2008; O'Donnell, Creamer, & Pattison, 2004). In addition to depression, anxiety, 'typical' PTSD and ASD presentations and other disorders, there has also recently been greater acknowledgement of the development of 'complex PTSD' (CPTSD) as a diagnosis relevant to a proportion of individuals following trauma.

The 11<sup>th</sup> edition of the International Classification of Diseases (ICD-11) is due for completion in 2018 and has proposed the inclusion of CPTSD as a new diagnosis that is related to but separate from PTSD. The concept of CPSTD has long been prevalent in literature and clinical practice, initially being described as a reaction to chronic stress or 'prolonged victimisation', and manifesting as difficulties with emotion regulation, self-organisation, self-perception, and interpersonal functioning (Herman, 1995). Childhood interpersonal trauma has been identified as a risk factor for developing CPTSD compared to PTSD, with a dose-response type relationship of exposure to multiple forms of interpersonal trauma and increased risk to CPTSD (Hyland et al., 2017a). However, it has also been argued that CPTSD is not exclusively associated with multiple and long-term trauma, and can develop following other types of trauma. The diagnosis of CPTSD in the proposed ICD-11 is not determined by the nature of the traumatic stressor; the experience of a trauma acts as a 'gate' for PTSD or CPTSD to be considered according to the resulting symptom profile (Cloitre, Garvert, Brewin, Bryant, & Maercker, 2013). The ICD-11 taskforce stated that CPTSD can arise after exposure to a single traumatic stressor (Maercker et al., 2013); a history of prolonged trauma may therefore be best conceptualised as a risk factor, rather than a determining requirement, for CPTSD (Hyland et al., 2017b; Sachser, Keller, & Goldbeck, 2017). To meet criteria for a diagnosis of CPTSD, in addition to meeting the core criteria for PTSD (at least one symptom per symptom cluster of re-experiencing, avoidance, and perceived threat), at least one symptom is required in three CPTSD

specific symptom clusters: affect dysregulation, negative self-concept, and interpersonal difficulties. This diagnostic category is intended to be applicable for all ages.

As the diagnostic category of CPTSD is still proposed rather than confirmed, there have been a small number of studies exploring this specific diagnosis, and most research to date has focussed on determining the construct validity of CPTSD. Evidence has been gathered for CPTSD being a valid diagnosis and distinct from PTSD in adults (Cloitre et al., 2013; Cloitre, Garvert, Weiss, Carlson, & Bryant, 2014; Elklit, Hyland, & Shevlin, 2014; Knefel, Garvert, Cloitre, & Lueger-Schuster, 2015; Knefel & Lueger-Schuster, 2013; Perkonig et al., 2016). Two studies validating CPTSD as a diagnostic category in children and adolescents have been published to date, and have also demonstrated the effectiveness of Trauma-Focussed Cognitive Behavioural Therapy (TF-CBT) as a treatment for CPTSD in this population (Perkonig et al., 2016; Sachser et al., 2017).

Identification of the course of any psychopathology developed following a trauma can be detected in the weeks and months following a trauma; providing an opportunity to identify which young people do not develop any symptoms, those who develop brief symptoms but recover, and those who develop chronic symptoms (deRoon-Cassini, Mancini, Rusch, & Bonanno, 2010; Meiser-Stedman et al., 2017). The exploration of factors determining these symptom trajectories is a key question in designing and targeting appropriate therapeutic interventions. Ehring et al. (2008) highlighted the need to investigate predictors which may differentiate between the development of different psychopathological presentations following trauma, noting that few studies had tested what factors predict different psychological outcomes of trauma. They argued that cognitive theories of emotional disorders have utility in differentiating between emotional disorders by their content of cognitive themes and

cognitive biases. Their study of post-traumatic psychological problems in adults following motor-vehicle accidents demonstrated the power of cognitive models in differentially predicting PTSD, travel phobia, and depression. To date, no study has utilised a similar methodology to analyse the differential power of predictive models in understanding the risk factors of children and adolescents developing PTSD and other disorders following trauma.

### **3.3.2 Models and predictors of PTSD and CPTSD**

The exploration of predictors of PTSD has focussed on three key areas; psychosocial or ‘pre-trauma’ vulnerability factors, event-related factors, and theory derived cognitive factors. All areas have highlighted relevant predictor variables in the development and maintenance of PTSD, however there has been little comparative assessment of them.

**Psychosocial stressors and risk factors.** The evidence available for psychosocial predictors of PTSD in children is variable, with meta-analyses indicating the need for further assessment. Social support, prior life events, low intelligence, socioeconomic status, low self-esteem, and female gender were deemed to be shown as fairly consistent predictors of PTSD but only small to medium effect sizes. Younger age was found not to be a predictor of PTSD in children and adolescents (Cox, Kenardy, & Hendrikz, 2008; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Overall, meta-analyses have concluded that pre-trauma factors yielded small to medium effect sizes as predictors of PTSD.

A number of psychosocial risk factors are commonly referred to in the conceptualisation of CPTSD in adults, including multiple prior traumas, interpersonal traumas or interpersonal stressful life events (Herman, 1995; Hyland et al., 2017a), however, research assessing these as predictors in relation to the new diagnosis is

limited. To date, only one study has explored the predictors of CPTSD in children and adolescents; female gender and type of trauma (interpersonal) were found to predict CPTSD versus PTSD (Sachser et al., 2017). Exposure to child abuse, and multiple types of abuse, has been shown to increase an adult's likelihood of meeting criteria for CPTSD rather than PTSD (Cloitre et al., 2013; Powers et al., 2017).

**Cognitive models.** Ehlers and Clark (2000), Foa, Steketee and Rothbaum (1989) and Brewin, Dalgleish and Joseph (1996) have been major proponents of key cognitive models formulating the development and maintenance of PTSD. These models implicate the nature of trauma memories and the cognitive processes by which these memories are formed as being key in the development of PTSD. Brewin et al. (1996) formulated a dual representation model based on cognitive neuroscience perspectives, suggesting how memories of a trauma are processed and stored differently to non-trauma memories. Within this model, the development and continuation of PTSD symptoms arises due to an inability to fully process and integrate highly sensory and verbal memories representing the individual's appraisals, emotional and physiological reactions to the trauma. Poor social support, any prior or ongoing trauma, aversive secondary emotions, the severity of the trauma, and prior psychopathology are all deemed as risk factors to the inhibition of adaptive processing of the trauma memories, and can lead to ongoing PTSD.

Similarly, Ehlers and Clark (2000) postulate a conceptualisation of PTSD which depicts two pertinent cognitive factors in the development of PTSD: the nature of the trauma memory and the appraisals developed related to the trauma experienced. In cases where PTSD is developed, peritraumatic cognitive processes, including data-driven processing (the focus on sensory experience reducing understanding of what was going on in the situation, experienced as feeling muddled or confused), result in the

development of trauma memories lacking contextual detail, but with strong conditioned associations. These features lead to the memory being easily and involuntarily brought into consciousness, with a poor sense of it being an event which occurred in the past, and little contextual information. If overly negative appraisals of the event are also developed, trauma memories are accompanied by negative intrusions and distressing emotional responses, for example guilt, self-blame, or anger. A range of cognitive and behavioural responses to this distressing re-experiencing, e.g. rumination, then maintains the PTSD. The authors of both these models also stipulate that some pre-trauma experiences and psychosocial factors also play a role as predictors of the development of PTSD. They argue that the prevention of adaptive processing of trauma memories leads to increased risk of individuals suffering from more chronic PTSD. Both these cognitive models of PTSD were developed in relation to PTSD in adults, however, a review of research and understanding of PTSD in children and adolescents found that the concepts described by each model map onto the presentation of PTSD in children and adolescents, supporting the applicability of these models across the life span (Meiser-Stedman, 2002).

Assessment of the predictive power of this cognitive model has been demonstrated in samples of children experiencing road traffic accidents (RTAs); 53-65% of the variance in PTSD symptoms has been shown to be predicted by models which included assessment of cognitive factors outlined in Ehlers and Clark's (2000) model, including data-driven processing, negative appraisals of the trauma, rumination and thought suppression (Ehlers, Mayou, & Bryant, 2003; Meiser-Stedman, Dalgleish, Glucksman, Yule, & Smith, 2009; Stallard & Smith, 2007). Cognitive models have also shown to be disorder specific in predicting the psychopathology outcomes of experiencing a trauma, and differentiating between whether an individual may develop

one disorder presentation over another; despite the overlaps in symptoms across PTSD, depression, and other anxiety disorders, and many shared named risk factors (Ehring et al., 2008).

**Event severity.** There has been some argument and evidence for the nature and severity of a traumatic event to be a predictor of PTSD; however, the focus on these factors has included both objective and subjective appraisals of event severity, with poor differentiation between, and little consistency in what constitutes markers of severity (Trickey et al., 2012). Recent research and reviews of both child and adult research of predictors of PTSD demonstrate that valid markers of event severity may include: interpersonal (vs non-interpersonal) trauma; the event resulting in a death; injury severity; levels of pain; and peritraumatic dissociation, perceived threat, fear and panic responses (Cox et al., 2008; Ozer, Best, Lipsey, & Weiss, 2003; Saxe et al., 2005; Trickey et al., 2012; Vogt, King, & King, 2007). However, a recent meta-analysis demonstrated the great heterogeneity existing between studies assessing trauma severity as a predictor of child PTSD. The authors also highlighted the importance of considering ‘trauma severity’ in a trauma-specific manner; one index of severity may simply be a marker of exposure in certain types of trauma, for example injury severity in natural disasters may vary greatly in accordance with exposure, whereas in RTAs, injury severity may be a clearer marker of severity of the incident (Trickey et al., 2012). The differentiation between objective and subjective measures of trauma severity has also been argued to be important when considering this as a risk factors for PTSD in children; for example, injury severity as measured by hospital data and admission versus a child’s perception of threat and their fear response (Ehlers et al., 2003). Foa et al.’s (1989) model of the maladaptive formation of a ‘fear network’ in response to a traumatic experience can inform how these subjective fear responses may lead to

increased likelihood of PTSD. A 'fear network' stores mental representations about sources of threat. These are easily activated by internal or external cues related to the trauma, and have a strong associated fear response due to the feelings of panic at the time of the trauma. Foa et al., (1989) suggested that perceived threat is therefore a key predictor of PTSD. This model has also been applied to children and adolescents to conceptualise child PTSD (Salmon & Bryant, 2002). These subjective responses to an event as a marker for event severity are likely to have different mechanisms of action as risk factors for PTSD than objective markers of event severity, though these two concepts may also be associated. It is therefore important to assess their role in the development of PTSD independently.

### **3.3.2 Aims of the current study**

There have been few studies, other than that of Ehring et al. (2008), which have compared predictive models and their goodness of fit across different psychopathological outcomes of trauma. Furthermore, samples of children and adolescents who have experienced a recent traumatic event are rarely easily recruited and engaged in research studies. This in addition to the recent development of CPTSD as a diagnostic category, means that our current understanding of valid predictors of psychopathological outcomes of children experiencing a trauma is in need of further exploration.

This current study utilised data collected from a large prospective longitudinal study of PTSD in children and adolescents following a recent trauma. Firstly, this study provided the opportunity to explore the possible presentation of CPTSD following a single trauma, which may validate or challenge the ICD-11 stipulation that CPTSD may arise following a single trauma. Secondly, if CPTSD presentations are found within this child and adolescent population, the study aimed to identify other trauma-related,

psychosocial or cognitive factors which are risk factors for developing CPTSD in comparison to PTSD, depression and anxiety. Finally, this study aimed to assess the goodness of fit of predictive models of PTSD and CPTSD in children and adolescents, in comparison to depression and anxiety, and to highlight any differences in predictors of the specific disorders. Predictive models of these four outcome disorders will be developed based on the theory and empirical research outlined above, including models based on: psychosocial factors, cognitive factors, subjective event severity and objective event severity factors. Understanding differential predictors of PTSD, CPTSD, depression or anxiety as outcomes following childhood trauma, will aid the development and implementation of appropriate interventions.

### **3.3.3 Hypotheses**

Firstly, it was hypothesised that peri- and post-trauma factors will be greater predictors of PTSD and CPTSD than pre-trauma psychosocial factors. Secondly, it was hypothesised that the cognitive model of predictors will have the best model fit in predicting PTSD and CPTSD. Finally, it was hypothesised that the cognitive model will have better power in differentiating between PTSD, CPTSD, depression and anxiety as outcomes of trauma in children and adolescents.

## **3.4 Methods**

### **3.4.1 Participants**

This study consecutively recruited 260 8-17 year olds from four Emergency Departments (ED) in the East of England between September 2010 and April 2013, who were identified by research nurses in the ED as presenting due to having experienced a single event trauma. 'Trauma' was defined in accordance with DSM-5 criteria of an

event involving threat of death or serious injury (American Psychiatric Association, 2013). The DSM-5 criteria were proposed at the time of assessment, prior to DMS-5 final publication, but also overlap with DSM-IV definition of trauma. Eligible participants were given study information sheets, and provided informed consent at point of entry into the study. This consent was to take part in a longitudinal research study, exploring PTSD development in children and adolescents. (See Appendix B.2 for details of author and study team involvement in data collection and analysis).

Staff identified 774 eligible children; 168 (21.7%) could not be contacted; of the 605 families contacted, 315 (52%) did not wish to participate, 30 (5%) did not meet eligibility criteria and the final 260 (43%) agreed to participate. Initial assessments at two weeks post-trauma (T1) were completed by 226 participants (completing either a semi-structured interview and/or self-report questionnaires), and the remainder of the recruited participants (34) completed the nine-week assessment only. In total, 234 completed the assessment (interview and/or questionnaires) at nine weeks post-trauma (T2). This current study utilised self-report questionnaire data only; these were completed by 217 participants at T1 and 234 participants at T2, with 204 completing questionnaires at both time points (13 individuals completed the interview part of the assessment only, and so were not included in the current analysis). There were no significant differences between responders and non-responders in terms of age, gender, ethnicity, or a number of measures of injury severity and hospital treatment. However, responders were found to be more likely than non-responders to experience more pain, admission to hospital and to have experienced an assault (vs other) trauma (Meiser-Stedman et al., 2017). See Meiser-Stedman et al. (2017) for full study details.

Participants' mean age was 14.1 years (SD=2.9) and 96 (42.5%) were female. Inclusion criteria were: exposure to a road traffic accident, a one-off assault, or another

discrete traumatic stressor. Exclusion criteria were utilised to consider ability to engage in the assessment process including: intellectual disability; non-fluency in English; being unconscious for more than 15 minutes following the event; a history of brain damage or moderate to severe traumatic brain injury as a result of the trauma. A few exclusion criteria were also adhered to in order to consider patient risk factors, including: assaults involving a caregiver or close relative as the assailant; ongoing exposure to threat; any significant risk of self-harm or A&E attendance resulting from deliberate self-harm; being under the care of social services or a child protection issue related to the presentation; and any current symptoms of PTSD following a previous trauma. However, from those identified by nurses at point of recruitment, it seemed that none were excluded based on presentation being due to ongoing trauma.

### **3.4.2 Measures**

This study utilised a number of measures; semi-structured interviews with parents and self-report questionnaires completed by the child (the study also conducted semi-structured interviews with the children but the current analysis utilised only questionnaire data from the children).

**Assessment of predictors.** For the assessment of predictive factors, data were sourced from self-report questionnaires and structured interviews completed by parents and children two weeks post-trauma, and from hospital data gathered by nurses in the Emergency Department at point of admission. See Table 5 for a summary of measures used to assess factors for each predictive model.

**Psychosocial factors.** Participant demographic data were collected at point of recruitment and was collated with information gathered by the hospital during their

admission, including trauma type (assault, road traffic accident or assault) and injury characteristics. Parents' education level was ascertained during a structured telephone interview; asking parents what was the highest level of qualification or training they achieved. This was categorised as those achieving up to GCSE or equivalent, and those achieving some sort of higher education or training. Parents were also asked about any prior traumas the child had experienced over the course of their lifetime, and any life stressors they had experienced over the past year, by answering positively to a list of possible events. For the purpose of the current analysis, the total number of traumas and total number of life stressors reported were used as predictor variables. Prior poor well-being was identified by parents answering positively to a single question "Before the trauma, have you had concerns for your child's emotional well-being (e.g., anxiety, depression or emotional problems)?" Children's perception of their social support and quality of their friendships and family relationships was assessed at two weeks post-trauma using a self-report questionnaire; the Multidimensional Scale of Perceived Social Support (MSPSS: Zimet, Dahlem, Zimet, & Farley, 1988; Cronbach's  $\alpha = .93$  in this sample).

**Cognitive factors.** The Ehlers and Clark (2000) cognitive model of PTSD informed the selection of six variables to form a cognitive model of the development and maintenance of psychopathological outcomes of trauma in this study population. These factors included cognitive processing during the trauma (Children's Data-Driven Processing Questionnaire (CDDPQ): McKinnon, Nixon, & Brewer, 2008; Cronbach's  $\alpha = .89$  in this sample); unhelpful trauma-related appraisals (Child Post-traumatic Cognitions Inventory (CPTCI): Meiser-Stedman, Dalgleish, et al., 2009; Cronbach's  $\alpha = .95$  in this sample); trauma memory characteristics (Trauma Memory Qualities Questionnaire (TMQQ): Meiser-Stedman, Smith, Yule, & Dalgleish, 2007; Cronbach's

$\alpha = .83$  in this sample); post-traumatic dissociation (Child PTSD Symptoms Scale (CPSS) post-traumatic dissociation subscale: Foa, Johnson, Feeny, & Treadwell, 2001; Cronbach's  $\alpha = .78$  in this sample); and trauma-related rumination and self-blame (Child Rumination and Self-blame Questionnaire (CRSQ); three and two item scales, with Cronbach's  $\alpha = .77$  and  $.91$  in this sample, respectively: Meiser-Stedman et al., 2017).

**Subjective event severity.** Variables which may capture a conditioned fear response related to subjective event severity and the mechanisms of a fear network as outlined by Foa et al., (1989) formed this third predictive model of psychopathology following trauma. This focussed on peritraumatic processes, including: panic responses (Child Peritraumatic Panic scale (CPP): Meiser-Stedman et al., 2017; Cronbach's  $\alpha = .72$  in this sample); peritraumatic dissociation (CPSS peritraumatic dissociation subscale: Foa et al., 2001; Cronbach's  $\alpha = .67$  in this sample); and three items were entered individually, which assessed peritraumatic perceived threat and fear (four scale Likert responses from 'disagree a lot' to 'agree a lot' to the statements 'I really thought I was going to die', 'I thought I was going to be very badly hurt' and 'I was really scared').

**Objective event severity.** The final predictive model focussed on injury and event severity utilising information gathered from the child's presentation at the ED, including the number of injuries they had sustained, whether they had sustained a head injury, whether they were given opiate pain-relief in ED, whether they were admitted to hospital. The child's rating of pain during the event was also included in this model ('How much pain were you in at the time of the accident?' with four response ratings from 'not at all' to 'a lot').

Insert Table 5 here

**Assessment of outcomes.** For the identification of participants scoring positively on symptoms of PTSD and CPTSD, the CPSS (Foa et al., 2001), the CPTCI (Meiser-Stedman, Smith, et al., 2009), and the CRSQ (Meiser-Stedman et al., 2017) were utilised. Items from these questionnaires were selected to capture each symptom criteria outlined by the World Health Organisation proposed diagnostic criteria in ICD-11 for PTSD and CPTSD (Maercker, et al., 2013). The rationale of selecting self-report items to correspond with each symptom criteria, in the absence of a validated measure of ICD-11 CPTSD, was informed by the methodology used in a recent study validating CPTSD in children and adolescents (Sachser et al., 2017). All items were coded 0-3 in relation to the four Likert scale responses given. A total score from these items generated a continuous measure of PTSD and CPTSD symptom severity; and a dichotomous diagnosis variable was also generated to identify those who scored positively on the necessary items in each symptom category and met diagnostic criteria outlined by ICD-11. For the computation of individuals meeting diagnostic criteria, a symptom was deemed to be positive if the corresponding item was scored one or higher (i.e. once per week or more), in line with methodology used by Sachser et al. (2017). The CPSS was primarily utilised for the development of a continuous measure of PTSD, as this is a well-validated scale for this purpose (Nixon et al., 2013). To generate a ‘pure’ PTSD measure, only items which corresponded directly to the ICD-11 PTSD criteria were used (excluding items which correspond to CPTSD criteria). This generated a nine-item continuous measure of core PTSD symptom severity, with a possible score range of 0-27. Items from the CPSS, CPTCI and CRSQ were selected to represent symptom criteria for CPTSD according to ICD-11, and summed to generate an eight-item continuous measure of CPTSD symptom ‘cluster’ severity, with a possible score range of 0-24. ICD-11 proposed criteria stipulates that in order to meet diagnostic criteria for CPTSD, core symptoms of PTSD must also be met, in addition to

the cluster of complex symptoms. Including the core PTSD items in the CPTSD scale would have generated inherent multicollinearity between these two outcomes, therefore, a continuous scale of complex PTSD symptoms alone was used for the analysis of predictors. Full diagnostic criteria (including the core PTSD symptoms required for CPTSD diagnosis) were used to identify frequencies of participants with likely CPTSD and PTSD at week nine. Table 6 outlines a summary of the items used for this current analysis. The internal consistency for each of these scales were Cronbach's  $\alpha = .90$  and  $.78$  for the PTSD and CPTSD scales, respectively. The pure PTSD scale score showed good correlation (Pearson's correlation coefficient = 0.61) with a diagnostic measure of DSM-IV PTSD as assessed by the Children's PTSD Inventory (Saigh et al., 2000) semi-structured interview in this sample. The correlation between the PTSD scale and the CPTSD scale was  $.63$ .

Insert Table 6 here

The Short Mood and Feelings Questionnaire (SMFQ, Angold, Costello, Messer, & Pickles, 1995; Cronbach's  $\alpha = .92$  in this sample) was used to assess symptoms of depression, with a score of 8 or above indicating depression. The Spence Children's Anxiety Scale (SCAS; Spence, 1998) Generalised Anxiety Disorder (GAD) subscale (Cronbach's  $\alpha = .87$  in this sample) was used to assess symptoms of GAD, with computed 't' scores of above 60 indicating elevated levels of anxiety. These are well-validated measures of depression and anxiety in children and adolescents (Sharp, Goodyer, & Croudace, 2006; Spence, Barrett, & Turner, 2003).

### **3.4.3 Study procedure**

The study was approved by the UK National Research Ethics Service, Cambridgeshire 1 Research Ethics Committee (10/H0304/11). Participants were recruited after presenting at a local ED; parents or caregivers of children who met

eligibility criteria were contacted by letter, enclosing information sheets about the study, within a few days, and then contacted by telephone at one-week post ED attendance. Informed consent from the parent, and assent from the child, was gained to be recruited into the study sample, if eligibility criteria were met. At approximately two weeks following their trauma (T1), participants and their parents were interviewed via telephone and were asked to complete self-report questionnaires. Participants were assessed a second time at approximately nine weeks post-trauma (T2), and the same interview and self-report measures were completed.

#### **3.4.4 Analyses**

All data processing and analysis was completed in Stata/IC Version 13.1 (StataCorp, 2013). Missing data codes were assigned to ensure correct treatment of missing data by Stata. Stata uses complete case analysis by default; if any observation has missing data in any of the variables in the analysis, that observation is excluded. Complete case analysis is deemed to be a valid method of treating missing data, if the missing data is deemed 'missing at random' (MAR) and particularly if the missingness of the data is independent of the outcome of interest. Due to different numbers of participants at each timepoint, many observations were 'missing' in the predictor or outcome variables; 260 parents were interviewed but not all questions were answered, 217 children completed the questionnaires at T1, and 234 children completed the questionnaires at T2. Data available from their admission to EDs were also variable. As the outcome variables were generated from the T2 questionnaires, the observations with complete T2 data but missing T1 data were assessed to identify if this was 'MAR'. T-tests were run to assess for significant differences in outcome measures between participants with missing data and complete data at each time point; no significant

differences were found suggesting that the missingness of the data was independent of the outcomes assessed in this study. T-tests also reported no significant differences in outcome scores in participants who were ‘complete cases’ across both time points and those who were not complete cases; suggesting that complete caseness was also independent of the outcomes assessed. Therefore, it was deemed valid to use complete case analysis. Regression analyses were also constrained to only include participants who had completed the week two questionnaires; this reduced the variation in the number of observations Stata included in each model. Some difference in the model goodness of fit statistics across the models may have partially reflected the different number of observations included in the analysis.

Pre-analysis screening of the normality, skew and homoscedasticity of the data was completed to determine the appropriate methods of analysis. Boxplots and histograms indicated the distribution of the data, and scatterplots were generated to observe the relationship between all predictor and outcome variables, to ascertain linearity. Normality of residuals were tested with the Kolmogorov-Smirnov test, and homoscedasticity was assessed by plotting the standardised residuals against their predicted values, with a funnel shaped plot indicating heteroscedasticity. Many variables were skewed, residuals were not normally distributed, and the variance of residuals was heteroscedastic, violating the assumptions of parametric tests, therefore non-parametric or other appropriate considerations were made in the analyses used.

Spearman’s rank correlation coefficients ( $\rho$ ) were used to ascertain the strength of associations between all continuous variables, and point biserial correlation coefficients were computed for dichotomous variables, to identify correlations between predictor variables and outcome variables, and to identify any multicollinearity between predictor variables. For the analysis of the four models to predict psychopathology

(PTSD, CPTSD, depression and GAD), non-parametric adjustments were made to multiple linear regression models by using bootstrapping. This method allowed for the estimation of coefficients and standard errors without relying upon assumptions of distribution or homoscedasticity in the data (Chernick, 2008). Bootstrapping approximates what estimates might be generated if the whole population was sampled by repeatedly resampling the study sample; the number of resamples was set to 1000. Linear regression modelling with bootstrapping in Stata produces unstandardised regression coefficients (Acock, 2008); to also generate standardised coefficients, all variables were transformed into standardised formation, and the regression was re-run to generate an equivalent to a beta coefficient. Both unstandardised and standardised coefficients are reported.

The predictive power and goodness of fit of the models of predictor variables were compared by computing Akaike and Bayesian Information Criteria (AIC and BIC), adjusted R-squared values and the overall model chi-square value and *p* statistic. Low AIC and BIC values indicate better model fit, and higher R-squared values indicate a greater proportion of variance in outcomes being accounted for by the predictor variables (Akaike, 1998; Raftery, 1995).

**Statistical power.** A few equations have been suggested to calculate the required sample size for a reasonably powered multiple regression analysis, including  $N \geq 50 + 8m$  (where *m* is the number of predictors); using this equation with 7 predictor variables, 106 participants would be required (Green, 1991). Consideration of effect size within the calculation of sample size has also been recommended by Green (1991); to detect a large effect size with seven predictor variables and a power of 0.8 ( $\alpha = 0.05$ ) using a parametric multiple correlation analysis, is 44 participants; and to detect a medium effect size with similar power, 103 participants are required. A

minimum of 189 participants were included in the analyses completed in this study, and no more than seven predictor variables were used in each model.

## **3.5 Results**

### **3.5.1 Sample characteristics**

Of the 260 children who participated in either assessment, 118 (45%) had experienced a RTA, 43 (17%) had experienced an assault, 82 (32%) had an accidental injury, 15 (6%) experienced a dog attack, and 2 (1%) had an acute medical emergency. Sample characteristics including age (mean 13.9), gender (43.5% female), psychosocial features, trauma characteristics, and the overall sample presentation in each of the predictor and outcome variables are summarised in Table 7. This table also demonstrates the associations between all variables (Spearman's rho).

The cognitive variables and peritraumatic factors showed the highest correlations with the four psychopathology outcome variables. Trauma memory qualities and trauma-related appraisals were the highest correlated of the predictor factors with PTSD; and appraisals and rumination were the highest correlated factors with CPTSD. Depression and GAD scores were also most highly correlated with trauma-related appraisals and rumination. Sustaining more injuries and self-blame related to the trauma were most highly correlated with CPTSD of the four outcome variables.

Insert Table 7 here

At nine weeks post-trauma, 55 (23.5%) participants met criteria for PTSD, 20 (8.5%) met criteria for complex PTSD cluster symptoms, and 12 (5.2%) met criteria for full CPTSD, according to our 'pure' and complex PTSD measures generated in

accordance with proposed ICD-11 criteria. Seventeen of the 55 participants meeting criteria for PTSD experienced a moderate to severe level of symptoms (endorsing symptom items as occurring twice or more times per week). Five of the 20 participants presenting with complex PTSD symptoms experienced these symptoms twice or more times per week, and one individual met full diagnostic criteria for moderate to severe CPTSD. Table 8 indicates the number of participants meeting each symptom criteria and diagnostic criteria at week two and week nine post trauma. Fifty-six individuals (23.9%) scored highly on the SMFQ to indicate likely depression, and 25 (10.7%) scored highly on the SCAS subscale to indicate likely GAD. Figure Six demonstrates the spread of participants meeting the criteria for each disorder; the totals in each area indicate the absolute frequency i.e. each individual only falls into one category. However, the requirement for core PTSD symptoms in addition to complex symptoms to meet CPTSD diagnostic criteria meant that, by definition, no participants would fall into the 'CPTSD only' category. Eighty-four participants (36%) met diagnostic criteria for one of the four diagnoses studied (they may have met criteria for other disorders, but this was not assessed within this study); 149 (64%) of participants did not meet criteria for PTSD, CPTSD, depression or GAD at nine weeks post trauma.

Insert Table 8 & Figure 6 here

### **3.5.2 Predictors of PTSD**

The psychosocial model accounted for 5% of the variance in PTSD symptom severity (adjusted  $R^2 = 0.055$ ,  $\chi^2(8)=15.2$ ,  $p=0.056$ ), with female gender ( $\beta = .14$ ,  $p<0.05$ ) and experiencing an interpersonal index trauma ( $\beta = .27$ ,  $p<0.01$ ) being the only significant predictors within the model. The objective event severity models accounted for the lowest variance (adjusted  $R^2 = 0.03$ ,  $\chi^2(5)=12.4$ ,  $p<0.05$ ) in PTSD symptom severity of all the four models, with pain being the only significant predictor

( $\beta = .22, p < 0.01$ ). The subjective event severity model accounted for 33% of variance (adjusted  $R^2 = 0.33, \chi^2(5) = 104.12, p = 0.001$ ), and indicated that panic ( $\beta = .37, p < 0.001$ ), feeling scared ( $\beta = .15, p < 0.05$ ) and dissociation ( $\beta = .23, p = 0.001$ ) at the time of the trauma were significant predictors of PTSD. The model which accounted for the greatest variance in PTSD symptom severity was the cognitive model (adjusted  $R^2 = 0.55, \chi^2(6) = 194.02, p < 0.001$ ); indicating that greater post-traumatic dissociation ( $\beta = .17, p < 0.05$ ), poorer trauma memory quality ( $\beta = .2, p < 0.01$ ) and maladaptive appraisals of the trauma ( $\beta = .33, p < 0.01$ ) lead to increased PTSD symptoms. Interestingly, higher event-related self-blame appeared to have a slight protective ( $\beta = -.09, p = 0.054$ ) effect against PTSD symptoms. Higher data-driven processing ( $\beta = .11, p = 0.059$ ) and rumination were also near to significant ( $\beta = .26, p = 0.06$ ) predictors of PTSD.

### **3.5.3 Predictors of CPTSD**

The psychosocial model accounted for greater (13%) variance in CPTSD symptom severity (adjusted  $R^2 = 0.13, \chi^2(8) = 25.6, p < 0.01$ ) than PTSD, and the predictors showing significance within the model were slightly different; greater number of prior traumas lead to increased complex symptoms ( $\beta = .15, p < 0.055$ ), perceived social support had a protective effect on later complex symptoms ( $\beta = -.17, p < 0.05$ ), and similarly to PTSD, experiencing interpersonal index trauma also lead to increased complex symptoms ( $\beta = .29, p < 0.01$ ). Within the event severity models, panic ( $\beta = .39$ ), dissociation ( $\beta = .2$ ) and pain ( $\beta = .2$ ) at the time of the trauma were significant predictors of later complex symptoms; the subjective severity model again accounting for much greater variance (26%; adjusted  $R^2 = 0.26, \chi^2(5) = 52.8, p < 0.001$ ) than the objective (4%) model (adjusted  $R^2 = 0.04, \chi^2(5) = 14.4, p < 0.05$ ). Fifty-five percent of the variance was again accounted for by the cognitive model (adjusted  $R^2 =$

0.55,  $\chi^2(6)=117.02$ ,  $p<0.001$ ), with post-traumatic dissociation ( $\beta = .15$ ,  $p=0.057$ ), maladaptive appraisals ( $\beta = .59$ ,  $p<0.001$ ) and increased self-blame ( $\beta = .17$ ,  $p<0.01$ ) being the only significant predictors within the model (all other cognitive factors were not near to significant).

### **3.5.4 Predictors of depression**

Female gender ( $\beta = .2$ ,  $p<0.01$ ), prior traumas ( $\beta = .16$ ,  $p<0.05$ ), interpersonal index trauma ( $\beta = .28$ ,  $p<0.01$ ) and poorer perceived social support ( $\beta = -.2$ ,  $p<0.05$ ) were significant predictors of later depression in the psychosocial model (accounting for 13% of variance in depression scores; adjusted  $R^2 = 0.13$ ,  $\chi^2(8)=27.2$ ,  $p<0.001$ ). Within the cognitive model, only increased maladaptive trauma appraisals was a significant predictor of later depression symptoms, but this factor demonstrated a large coefficient ( $\beta = .68$ ,  $p<0.001$ ) and despite all other cognitive factors not being anywhere near to significant, the model still accounted for 56% of the variance in depression at week nine (adjusted  $R^2 = 0.56$ ,  $\chi^2(6)=193.1$ ,  $p<0.001$ ). Panic ( $\beta = .3$ ,  $p<0.001$ ) and dissociation at the time of the event ( $\beta = .23$ ,  $p<0.01$ ) were significant predictors within the subjective event severity model (accounting for 24% of variance in depression; adjusted  $R^2 = 0.24$ ,  $\chi^2(5)=54.8$ ,  $p<0.001$ ); and pain ( $\beta = .27$ ,  $p<0.001$ ) and sustaining a head injury ( $\beta = .17$ ,  $p<0.01$ ) were significant predictors within the objective event severity model (accounting for just 8% of variance; adjusted  $R^2 = 0.08$ ,  $\chi^2(5)=25.3$ ,  $p<0.001$ ).

### **3.5.5 Predictors of GAD**

Female gender ( $\beta = .25$ ,  $p<0.01$ ) and experiencing an interpersonal index trauma ( $\beta = .27$ ,  $p<0.05$ ) significantly predicted later GAD symptoms, with all other

psychosocial variables showing non-significant effects within the model and overall it accounted for 13% of variance in symptoms (adjusted  $R^2 = 0.13$ ,  $\chi^2(8)=22.6$ ,  $p<0.01$ ). The cognitive model again showed strong predictive power with 54% variance in GAD symptoms accounted for (adjusted  $R^2 = 0.54$ ,  $\chi^2(6)=120.2$ ,  $p<0.001$ ), and trauma appraisals ( $\beta = .53$ ,  $p<0.001$ ) significantly predicting later GAD symptoms, and dissociation ( $\beta = .18$ ,  $p=0.053$ ) and rumination ( $\beta = .12$ ,  $p=0.065$ ) showing near to significant predictive effect within the model. The subjective event severity model again accounted for the second greatest amount of variance in symptoms (28%; adjusted  $R^2 = 0.28$ ,  $\chi^2(5)=52.04$ ,  $p<0.001$ ), with panic ( $\beta = .35$ ,  $p<0.001$ ) and peritraumatic dissociation ( $\beta = .24$ ,  $p<0.001$ ) again being significant predictors of later symptoms. Within the objective model, increased pain leading to increased GAD symptom severity ( $\beta = .31$ ,  $p<0.001$ ). Interestingly, a greater number of injuries sustained appeared to have a protective effect on later GAD symptoms ( $\beta = -.13$ ,  $p<0.05$ ). This final model accounted for 9% of variance in GAD symptoms (adjusted  $R^2 = 0.09$ ,  $\chi^2(5)=23.8$ ,  $p<0.001$ ). Full details of model results for all regression analyses can be found in Tables ten to thirteen in the supplemental materials (Appendix B.2).

### **3.5.6 Overall model comparisons**

Table nine summarises the goodness of fit statistics for each model predicting each disorder. The cognitive model consistently accounted for the greatest variance in symptoms, achieved the best (lowest) AIC and BIC statistics, and the  $\chi^2$  and  $p$  statistics indicated that this model was significantly better than a model with no predictors for each disorder. The order of best fitting models after the cognitive model was also similar across disorders; the subjective event severity model was second best, the psychosocial model was third best, and the objective event severity model was

consistently poorest. However, comparison across disorders indicates that the psychosocial model was comparably strongest in predicting CPTSD, depression and GAD but was much weaker in predicting pure PTSD. The subjective event severity model was strongest in predicting pure PTSD and weakest in predicting depression, and the objective model was comparable in predicting depression and GAD but weakest in predicting pure PTSD.

## **3.6 Discussion**

### **3.6.1 Overall findings**

Two of our three hypotheses were supported. Firstly, models which contained largely peri- and post-traumatic factors were found to be more powerful predictors of all mental health outcomes than the psychosocial model which was largely pre-trauma factors, supporting hypothesis one. Peritraumatic cognitive factors were also more powerful predictors than event-related objective measures, such as injuries sustained. Hypothesis two, that the cognitive model would provide the best model fit for PTSD and CPTSD, was well supported. However, the cognitive model also derived the best model fit over other models for depression and GAD also. This generalised power of the cognitive model indicated in our results did not support hypothesis three, which expected that the cognitive model would differentiate between the disorders. Overall, poor disorder specificity was indicated, with a similar pattern of goodness of fit indices and some overlap in which individual factors were significant predictors of each disorder outcome. However, some differences between results for each disorder was indicated.

### **3.6.2 Increasing our understanding of CPTSD in children**

With ICD-11 yet to be published there are very few studies of CPTSD in children and adolescents, and none to date exploring predictors of CPTSD. This study provides seminal evidence for the existence of complex PTSD symptom presentations in our young populations. The results provide evidence for ICD-11 diagnostic categories of PTSD and CPTSD as related but distinct presentations, with different predictors and correlates, in a sample of children and adolescents. Furthermore, the presentation of CPTSD being demonstrated in a population of children experiencing single event traumas, such as RTAs, accidental injury and single assaults, is also highly informative in our conceptualisation of this disorder; with it typically being characterised as a presentation likely to occur following multiple and ongoing trauma. The results suggesting a predictive role of experiencing prior traumas in later CPTSD but not ‘pure’ PTSD, supports the historic conceptualisation of complexity in PTSD symptoms arising following multiple traumas. Further exploration of the experience of prior traumas in individuals meeting diagnostic criteria for CPTSD highlighted support for Hyland and colleagues’ (2017) assertion that prior trauma is a risk factor not a diagnostic necessity for CPTSD, as 50% (n=6) had not experienced any prior traumas (two individuals had experienced one prior trauma; two experienced two, and two had experienced three prior traumas). The relevance of interpersonal factors in the development of CPTSD was also highlighted by the predictive role of experiencing an interpersonal index trauma and perceiving themselves to have poorer social support. Theories of CPTSD have referred to the role of disruption of attachments which leads to the negative perceptions of the self, emotion regulation difficulties and the ongoing interpersonal problems characterising CPTSD (Cloitre et al., 2009). Female gender and

prior poor well-being were not found to be predictive of complex symptoms in this sample, which is not in line with previous research.

### **3.6.3 The predictive power of the cognitive model**

The cognitive model of predictors based on Ehlers and Clark's (2000) model of PTSD demonstrated the best model fit indices and greatest proportion of variance accounted for in PTSD, CPTSD, depression, and GAD. Maladaptive appraisals of a traumatic event showed to be a strong cognitive predictor of all disorders, but variation in the significance and strength of other cognitive predictors highlighted some differentiation in the predictive power and applicability of all features of this model to different disorders. For example, all cognitive factors had significant or near to significant roles in predicting a greater likelihood of 'pure' PTSD symptoms; whereas, data-driven processing, trauma memory quality and rumination clearly had no effect in predicting complex PTSD symptoms, and self-blame appeared to have significant but opposite effects in predicting pure and complex symptoms. This finding supports the validity of 'pure' and 'complex' PTSD as distinct presentations. Similarly, only greater trauma-related misappraisals were significantly predictive of depression and GAD. Interestingly, rumination appeared to have the most relevance as a predictive factor for GAD and pure PTSD, and showed little predictive value for depression in this sample. Rumination is a cognitive process which has been implicated as a core maintaining feature of both GAD and depression; in our results it showed a significant correlation with depressive symptoms but appeared not to have a role in predicting severity of symptoms nine weeks post-trauma when also set against other cognitive factors.

Challenge to the cognitive model may be drawn from the apparent comparable success in predicting severity of all disorders; it resulted in similar estimates of variance

accounted for (adjusted  $R^2$ ) and model fit indices for all disorders, suggesting poor specificity of it as a cognitive model of PTSD. However, each of the disorders studied have theoretical models implicating cognitive factors in their development and maintenance, with some overlap across disorder specific models. The model specificity and goodness of fit results within our study may have reflected greater differences between the disorders had we developed different models defining specific cognitive features of each disorder a priori. This method was demonstrated by Ehrling et al. (2008), for example, in their depression model they defined ‘depressive rumination’ and in contrast, their PTSD model incorporated ‘rumination about the trauma’. Clinically, this may point towards the importance of firstly highlighting if children are presenting with particular maladaptive cognitive processes, such as misappraisals. Secondly, it may be helpful to explore the content and nature these cognitions to elucidate to which specific disorder-related symptoms they may be most vulnerable.

#### **3.6.4 Psychosocial and event-related predictors of psychopathology following trauma**

Experiencing an interpersonal index trauma rather than a RTA, or some other accidental injury, appeared to lead to increased risk of developing any of the disorder symptoms. If event severity is conceptualised as related to the likelihood of post-traumatic psychopathology, the index trauma being interpersonal rather than non-interpersonal may be a relevant marker of severity. Younger age was not found to be a significant predictor of any disorder within our sample, consistent with findings from a meta-analysis of predictors of PTSD in children (Trickey et al., 2012). Both CPTSD and depression were predicted by poor perceived social support, highlighting the relevance of good interpersonal networks in protecting against both these disorders. Experiencing

traumas prior to the index trauma also predicted CPTSD and depression but not GAD or PTSD; multiple childhood traumas have been implicated in developmental research exploring later psychopathology, particularly depression and the types of complex interpersonal difficulties and negative self-concept captured by CPTSD (Suliman, Mkabile, Fincham, Ahmed, Stein, & Seedat, 2009; Cloitre et al., 2009). Conversely, GAD and PTSD may be conceptualised as ‘less severe’ psychopathological presentations and so their development may be less related to a disruption in development caused by early traumas. It would be pertinent to explore further the suggestion of a ‘dose-response’ type relationship between childhood traumas and complexity of symptoms in children and adolescents.

There was a clear distinction between the relative predictive power of objective versus subjective event severity markers; with subjective experiences of greater fear, panic, and perceived threat during the trauma showing greater relevance in predicting later psychopathology than markers of injury severity or requirement for hospital admission. Perceived life threat was not found to be predictive of PTSD, or other disorders, which is a contraindication of previous research (Trickey et al., 2012). Feeling scared, panicked or even dissociating at the time of the event appeared more important in this sample, suggesting that the emotional experience and fear response may be more indicative of later psychopathology than clear appraisals of threat. Peritraumatic pain was a significant predictor of all disorders, however this was measured post-trauma (hospital data on pain rating was only available for a small proportion of participants), and so could have been a proxy of the child’s post-traumatic appraisal of the event.

### **3.7 Conclusions**

These findings present a key addition to the field of understanding the predictors of PTSD and related disorders in children and adolescents. The results show support for the cognitive model of PTSD, but may also highlight weaknesses in a lack of disorder specificity of the model. Consideration of disorder specific cognitions, or the prediction of an overall ‘distress’ factor post-trauma, may be pertinent in further exploring this finding. Overall, the significance of subjective peritraumatic factors and post-traumatic cognitive processes, over and above the consideration of objective demographic or trauma-related factors, consistently demonstrated the importance of the assessment of how a child experienced an event in understanding their potential susceptibility to developing a range of psychopathological and distressing symptoms.

### **3.7.1 Limitations and future directions**

The analysis strategy in this study focussed on endpoint analysis, exploring factors assessed at time one predicting outcomes at time two, and was limited to complete cases. Multiple imputation or maximum likelihood estimation to account for missing data with alternative longitudinal data analysis incorporating both time one and time two symptoms may have increased the potential information gained from this study. Time one symptoms were not included in the main analysis to avoid reducing the power of our regression models due to increased predictor variables and high multicollinearity between symptom scores. However, studies using endpoint analysis still add valid information and value to the field.

At the point of methodological planning for this study, CPTSD was not yet a proposed diagnostic category, and was not an original focus of this study. The lack of understanding and assessment of CPTSD at the time meant that a specific and validated measure of CPTSD was not included in the study. To date, there is no developed measure of CPTSD in children and adolescents; this is an area for future research focus,

particularly considering the imminent publication of the ICD-11 diagnostic manual. Clinicians and researchers will benefit from a validated tool to assess for these symptoms to aid appropriate intervention. To date, one study has presented findings demonstrating the clinical effectiveness of psychological treatment strategies (TF-CBT; Sachser et al., 2017) for children and adolescents presenting with CPTSD. Our study also supports the validation of this diagnostic category within this population, and the importance of assessing and treating maladaptive cognitive processes to potentially reduce the distressing symptoms of CPTSD, PTSD, depression, or anxiety. This field is clearly in its infancy and requires significant further exploration.

### 3.8 Tables and Figures

**Table 5.** Summary of measures or items used for each predictor variable within each model

<b>Model &amp; Factors</b>	<b>Measure</b>
<b>Psychosocial</b>	
Age	Sociodemographic questionnaire
Gender (female)	
Mother's education	Semi-structured interview with parent
Interpersonal index trauma	Information gathered at admission in ED
Prior trauma (lifetime frequency)	Semi-structured interview with parent
Prior life stressors (past year frequency)	Semi-structured interview with parent
Prior well-being concerns	Semi-structured interview with parent
Perceived social support	MSPSS total score
<b>Cognitive Model</b>	
Post-traumatic dissociation	CPSS post-trauma dissociation items total score
Data-driven processing	CDDPQ total score
Trauma memory quality	TMQQ total score
Trauma-related appraisals	CPTCI total score
Rumination	CRSQ items 1-3 total score
Self-blame	CRSQ items 4-5 total score
<b>Conditioned Fear / Subjective event severity</b>	
Peritraumatic panic	CPP total score
Peritraumatic perceived life threat	CPT: item 1 'thought I will die'
Peritraumatic perceived harm	CPT: item 2 'thought I would be badly hurt'
Peritraumatic fear	CPT: item 3 'very scared'
Peritraumatic dissociation	CPSS peritraumatic dissociation items total score
<b>Objective event severity</b>	
Pain	Child Pain Scale (peritraumatic)
Number of injuries sustained	Information recorded by nurses during admission to ED
Head injury sustained	
Admitted to hospital	
Opiates administered in ED	

*Abbreviations:* MSPSS: Multidimensional Scale of Perceived Social Support; CPSS:

Child PTSD Symptoms Scale; CDDPQ: Children's Data-Driven Processing

Questionnaire; TMQQ: Trauma Memory Quality Questionnaire; CPTCI: Child Post-

Traumatic Cognitions Inventory; CRSQ: Child Rumination and Self-blame

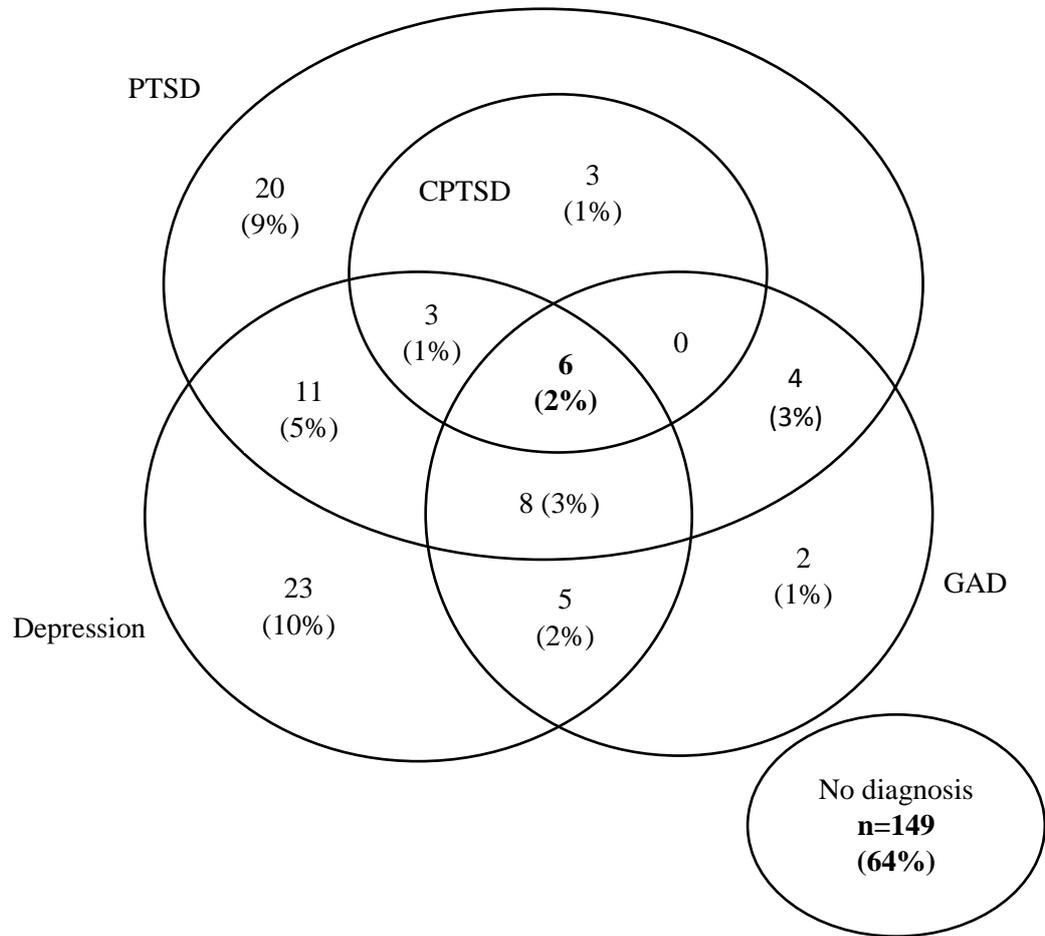
Questionnaire; CPP: Child Peritraumatic Panic scale; CPT: Child Peritraumatic Threat

scale.

**Table 6.** Items used to generate a measure of ICD-11 PTSD and CPTSD

ICD-11 symptom	Item selected
<b>PTSD</b>	
<i>Re-experiencing</i>	
Flashbacks	CPSS item 3: Acting or feeling as if the event was happening again (hearing something or seeing a picture about it and feeling as if I am there again)
Intrusive memories	CPSS item 1: Having upsetting thoughts or images about the event that came into your head when you didn't want them to
Nightmares	CPSS item 2: Having bad dreams or nightmares
Fear, horror, physical sensations or same emotions as during event	CPSS item 4: Feeling upset when you think or hear about the event (for example, feeling scared, angry, sad, guilty etc). CPSS item 5: Having feelings in your body when you think about or hear about the event (for example, breaking out in a sweat, heart beating fast).
<i>Avoidance</i>	
Of thoughts or memories	CPSS item 6: Trying not to think about, talk about, or have feelings about the event.
Of activities, situations or people	CPSS item 7: Trying to avoid activities, people, or places that remind you of the traumatic event.
<i>Current threat perception</i>	
Hypervigilance	CPSS item 16: Being overly careful (for example, checking to see who is around you and what is around you).
Enhanced startle response	CPSS item 17: Being jumpy or easily startled (for example, when someone walks up behind you).
<b>Complex PTSD</b>	
<i>Affect regulation problems</i>	
Anger	CPSS item 14: Feeling irritable or having fits of anger.
Violent or reckless behaviour	CPSS item 21: Taking more risks and being reckless or dangerous.
Emotional reactivity, or a lack of emotion	CPSS item 11: Not being able to have strong feelings (for example, being unable to cry or unable to feel very happy).
<i>Negative beliefs about self</i>	
Diminished, or defeated	CPSS item 12: Feeling as if your future plans or hopes will not come true (for example, you will not have a job or get married or have kids).
Worthless	CPTCI item 7: I am no good
Feelings of shame, guilt, or failure (related to the event)	CRSQ 5: It was my fault the event happened
<i>Interpersonal difficulties</i>	
Difficulties sustaining relationships	CPTCI item 5: I don't trust other people
Difficulties feeling close to others	CPSS item 10: Not feeling close to people around you.

**Figure 6.** Venn diagram summarising number of participants meeting criteria for likely diagnoses of PTSD, CPTSD, depression and GAD at nine weeks post-trauma



**Table 7.** Sample characteristics and correlations between week two predictor variables and outcomes at week nine post trauma. ( $^{\vee} p < 0.1$ ,  $*p < 0.05$ ,  $**p < 0.01$ ,  $***p < 0.001$ )

	<i>Mean (SD)/ frequency (%)</i>		<i>Range</i>	<b>PTSD</b>	<b>CPTSD</b>	<b>Depression</b>	<b>GAD</b>
<i>Week nine outcomes</i>							
<b>PTSD</b>	4.6	(5.9)	0-24	1			
<b>CPTSD</b>	2.9	(3.9)	0-21	0.54***	1		
<b>Depression</b>	4.5	(5.4)	0-23	0.56***	0.69***	1	
<b>GAD</b>	45.9	(9.1)	40-100	0.54***	0.58***	0.67***	1
<i>Week two predictors</i>							
<i>Psychosocial factors</i>							
<b>Age</b>	13.9	(2.9)	8.0-17.9	-0.09	0.08	0.02	0.13*
<b>Gender</b>	108	(42.5%)		0.08	0.02	0.11 <sup>v</sup>	0.14*
<b>Mother's education</b>	147	(58.3%)		0.02	0.05	0.07	0.10
<b>Frequency of prior traumas</b>	0.9	(1.0)	0-5	0.06	0.14*	0.15*	0.15*
<b>Frequency of prior life stressors</b>	0.9	(1.2)	0-6	0.14*	0.10	0.09	0.20**
<b>Prior wellbeing concerns</b>	62	(24.2%)		0.08	0.19**	0.12*	0.11 <sup>v</sup>
<b>Interpersonal Index Trauma</b>	43	(16.5%)		0.24***	0.27***	0.22***	0.26***
<b>Perceived Social Support</b>	69.7	(12.8)	25 -84	-0.09	-0.18*	-0.20**	-0.19*
<i>Cognitive factors</i>							
<b>Post- traumatic dissociation</b>	1.4	(2.3)	0-12	0.46***	0.37***	0.40***	0.41***
<b>Data-driven processing</b>	15.7	(6.1)	7 -28	0.48***	0.34***	0.40***	0.43***
<b>Trauma Memory Quality</b>	21.9	(6.8)	11-44	0.58***	0.43***	0.49***	0.52***

<b>Trauma-related appraisals</b>	37.6	(14.3)	25-90	0.58***	0.54***	0.66***	0.65***
<b>Rumination</b>	7.5	(2.8)	3-12	0.57***	0.46***	0.55***	0.58***
<b>Self-blame</b>	3.6	(2.1)	2-8	-0.002	0.30***	0.15*	0.11
<i>Subjective event severity and fear response factors (all peritraumatic)</i>							
<b>Panic</b>	3.6	(2.4)	0-10	0.47***	0.45***	0.45***	0.48***
<b>Perceived life threat</b>	1.9	(1.1)	1-4	0.28***	0.30***	0.35***	0.31***
<b>Perceived harm</b>	2.9	(1.0)	1-4	0.26***	0.24***	0.23**	0.17*
<b>Fear</b>	3	(1.1)	1-4	0.43***	0.20**	0.30***	0.35***
<b>Dissociation</b>	3.9	(3.1)	0-12	0.34***	0.30***	0.38***	0.35***
<i>Objective event severity factors</i>							
<b>Pain</b>	3.1	(1.1)	1-4	0.19**	0.19**	0.22**	0.23**
<b>Admission to hospital</b>	73	(28.1%)		-0.13*	-0.09	-0.11 <sup>y</sup>	-0.11 <sup>y</sup>
<b>Head injury</b>	97	(38.1%)		0.02	0.08	0.10	0.08
<b>Number of injuries</b>	1.7	(0.9)	0-5	0.001	0.15*	0.06	0.03
<b>Opiates given in ED</b>	44	(17.9%)		-0.04	-0.08	-0.08	-0.06

**Table 8.** Frequency of participants meeting symptom and diagnostic criteria at week two and week nine post-trauma

Symptom/diagnosis	Week 2 n=217		Week 9 n=234	
<b>ICD-11 PTSD</b>				
<i>Re-experiencing</i>				
Flashbacks (CPSS 3)	76	35%	62	26.5%
Intrusive memories (CPSS 1)	117	53.9%	87	37.2%
Nightmares (CPSS 2)	75	34.6%	62	26.5%
Fear, horror, physical sensations (CPSS 4) or same emotions as during event (CPSS 5)	118	54.4%	83	35.5%
<i>Re-experiencing criteria met (1 or 2 or 3, and 4 or 5)</i>	108	49.8%	76	32.5%
<i>Avoidance</i>				
Of thoughts or memories (CPSS 6)	108	49.8%	79	33.7%
Of activities, situations or people (CPSS 7)	70	32.3%	59	25.2%
<i>Avoidance criteria met (6 or 7)</i>	119	54.8%	94	40.2%
<i>Current threat perception</i>				
Hypervigilance (CPSS 16)	119	54.8%	94	40.2%
Enhanced startle response (CPSS 17)	82	37.8%	68	29.1%
<i>Threat criteria met (16 or 17)</i>	131	61.5%	110	47%
<b>ICD-11 PTSD criteria met</b>				
Score of 1 or higher on items (1 or 2 or 3) + (4 or 5) + (6 or 7) + (16 or 17)	<b>73</b>	<b>33.6%</b>	<b>55</b>	<b>23.5%</b>
<b>ICD-11 Complex PTSD</b>				
<i>Affect regulation problems</i>				
Anger (CPSS 14)	80	36.9%	57	24.4%
Violent or reckless behaviour (CPSS 21)	26	11.9%	26	11.1%
Emotional reactivity or lack of emotion (CPSS 11)	46	21.2%	34	14.5%
<i>Affect regulation criteria met (14 or 21 or 11)</i>	101	46.5%	80	34.2%
<i>Negative beliefs about self</i>				
Diminished or defeated (CPSS 12)	30	13.8%	23	9.8%
Worthlessness (CPTCI 7)	41	18.9%	52	22.2%
Guilt, shame or failure (CRSQ 5)	90	41.5%	82	35%
<i>Negative beliefs criteria met (12 or 7, and 5)</i>	37	17.1%	34	14.5%
<i>Interpersonal difficulties</i>				
Difficulties sustaining relationships (CPTCI 5)	80	36.9%	92	39.3%
Difficulties feeling close to others (CPSS 10)	45	20.7%	37	15.8%
<i>Interpersonal difficulties criteria met</i>	91	41.9%	99	42.3%
<b>ICD-11 CPTSD symptom cluster criteria met</b>	<b>25</b>	<b>11.5%</b>	<b>20</b>	<b>8.5%</b>
<b>Full ICD-11 CPTSD diagnostic criteria met (core PTSD symptoms plus CPTSD cluster)</b>	<b>18</b>	<b>8.3%</b>	<b>12</b>	<b>5.1%</b>
<b>Depression</b>				
<i>(SMFQ total score cut-off 8/&lt;)</i>	<b>54</b>	<b>24.9%</b>	<b>56</b>	<b>24%</b>
<b>Generalised Anxiety Disorder</b>				
<i>(SCAS GAD t-score cut-off)</i>	<b>29</b>	<b>13.4%</b>	<b>25</b>	<b>10.7%</b>

**Table 9.** Overall goodness of fit and model statistics for multiple linear regression analyses of predictors of each disorder

<b>Disorder</b>	<b>Model</b>	<b>Adj R<sup>2</sup></b>	<b>AIC</b>	<b>BIC</b>	<b>(df) <math>\chi^2</math></b>	<b><i>p</i></b>
<b>Core</b>	Psychosocial	0.055	1249.867	1279.324	(8) 15.18	0.0557
<b>PTSD</b>	<b>Cognitive</b>	<b>0.551</b>	<b>1139.585</b>	<b>1162.743</b>	(6) 194.02	0.000
	SES	0.326	1225.927	1245.806	(5) 104.12	0.000
	OES	0.029	1221.135	1240.617	(5) 12.42	0.029
<b>CPTSD cluster</b>	Psychosocial	0.127	1062.265	1091.722	(8) 25.57	0.0012
	<b>Cognitive</b>	<b>0.550</b>	<b>958.419</b>	<b>981.577</b>	(6) 117.02	0.000
	SES	0.255	1064.068	1083.947	(5) 52.75	0.000
	OES	0.043	1034.344	1053.826	(5) 14.39	0.013
<b>Depression</b>	Psychosocial	0.128	1196.391	1225.802	(8) 27.17	0.0007
	<b>Cognitive</b>	<b>0.559</b>	<b>1095.815</b>	<b>1118.938</b>	(6) 193.13	0.000
	SES	0.235	1211.155	1231.005	(5) 54.83	0.000
	OES	0.084	1169.831	1189.281	(5) 25.31	0.0001
<b>GAD</b>	Psychosocial	0.125	1412.535	1441.945	(8) 22.61	0.0039
	<b>Cognitive</b>	<b>0.544</b>	<b>1325.549</b>	<b>1348.672</b>	(6) 120.19	0.000
	SES	0.280	1422.851	1442.7	(5) 52.04	0.000
	OES	0.091	1380.137	1399.587	(5) 23.82	0.0002

*SES = Subjective event severity; OES = Objective event severity. Model with fit indices suggesting the best goodness of fit and highest variance in outcome accounted for highlighted in bold. N observations included in each model analysis varied as such: psychosocial n=194; cognitive n=201; SES n=202; OES n=189.*

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## Chapter 4. Additional Methodology

This section comprises of further details of methodology utilised for the review paper outlined in Chapter 1.

### 4.1 Data extraction form and quality assessment framework

The following form was used to extract data from the studies, assess study quality and generate a quality score as a measure of study risk of bias. Section one was completed for all studies, and incorporated gathering of general study information, information about the measures used for the assessment of peritraumatic factors and PTSD, and ratings for the study quality assessment framework. Within section one, section 1.2.4 was completed for longitudinal prospective studies only. Section two was completed for cross-sectional studies only, and section three was completed for longitudinal prospective studies only; section two and three gathered data required for the data synthesis and study review.

Item no.	Item	Data
<b>1</b>	<b>Section 1: complete for all studies</b>	
<b>1.1</b>	<b>Study information</b>	
<b>1.1.1</b>	Coder Initials	
<b>1.1.2</b>	Date form completed	
<b>1.1.3</b>	Double coded? Y/N	
<b>1.1.4</b>	Study ID number	
<b>1.1.5</b>	First Author	
<b>1.1.6</b>	Journal name	
<b>1.1.7</b>	Year of publication	
<hr/>		
<b>1.2</b>	<b>Quality Assessment and Risk of Bias Tool</b>	
<b>1.2.1</b>	<b>Was the study population clearly defined? (<i>consider clear description of age, gender, location, ethnicity, demographics</i>)</b>	
	Yes- descriptive statistics reported on participant demographics (including age range and mean, gender split) and trauma characteristics (type of trauma, injuries or impact, if natural disaster indicates some level of exposure)	2

Some descriptive statistics reported but some missing information.	1
No clear description of sample and trauma characteristics	0

**1.2.2 Was some form of random selection used to select the sample or a method of sampling appropriate to the study? (consider random, cluster, or systematic sampling, consecutive recruitment if appropriate, or approached all eligible participants if possible, for example approached all students involved in a specific trauma occurring at one school)**

Clear report given on random selection method or appropriate recruitment strategy	2
Some sampling method used, but not totally random	1
Unclear whether appropriate sampling method was used, or inappropriate or non-random sampling method used	0

**1.2.3 Was non-response bias minimal or accounted for? (consider if the response rate was >40%. If response rate was an analysis was <40%, consider if authors assessed and reported no significant difference between responders and non-responders in key indicators e.g. age, gender, trauma type)**

Yes; more than 40% of eligible and approached participants took part and, if reported, there were no significant differences between those who took part and those who did not.	2
No but accounted for; less than 40% of those approached took part, but there were no significant differences between those who participated and those who did not.	1
No; less than 40% of those approached took part, and differences between those who took part and those who did not were not reported or highlighted significant differences. Or, response rate was not reported.	0

**1.2.4 For longitudinal/prospective studies: was loss to follow-up 20% or less?**

Yes; participant drop-out or non-response was less than 20%.	2
No, but accounted for; loss to follow up was more than 20% (but less than 40%) but differences between those who completed the full study and those who did not were assessed and reported as showing no significant differences in key indicators (e.g. in age, gender, trauma characteristics or symptoms)	1
No; loss to follow up was more than 20% and difference between complete cases and incomplete	0

cases were not assessed or reported, or showed significant differences.	
Not applicable; this was a cross-sectional study	N/A

<b>1.2.5</b>	<b>Was the measure of PTSD valid and reliable? (consider if they reference the use of the measure in other research; if they report internal consistency; Cronbach's alpha, as at least 0.7; if this was interview based or self-report; and if they reference the measure as being informed by diagnostic manual criteria for PTSD)</b>	
	Yes; a well-validated interview or self-report measure based on diagnostic manual criteria was used.	2
	A validated interview or self-report measure was used but was not based on diagnostic manual criteria of PTSD	1
	No; a poorly validated or unknown measure of PTSD was used.	0

<b>1.2.6.i</b>	<b>Was the measure of peri-traumatic factors reliable? (Consider how well the authors described this measurement; if a validated full-scale, or multiple items from another scale, or just a single item was used to assess each peri-traumatic factor; and consider if this was assessed by interview or self-report measure)</b> <b>*If multiple peri-traumatic factors are assessed in one study, please complete this question for each factor, labelling each factor assessed here: Peri-traumatic factor (e.g. fear, perceived life threat...):</b>	
	A specific and validated full-scale measure (self-report or interview), or multiple items from a semi-structured interview was used to assess peri-traumatic factors.	2
	A total or mean score from multiple self-report items, either designed specifically for the study or taken from within an existing measure (with good internal consistency for these items, if reported) or A score from a single item from an existing and validated measure	1
	Response on a single item or another single way of assessing a peri-traumatic factors was used, or poor description was given of how this factor was assessed.	0

<b>1.2.6.ii</b>	<b>Was the measure of peri-traumatic factors reliable? (Consider how well the authors described this measurement; if a validated full-scale, or multiple items from another scale, or just a single item was used to assess each peri-traumatic factor; and consider if this was assessed by interview or self-report measure)</b> <b>*If multiple peri-traumatic factors are assessed in one study, please complete this question for each factor, labelling each factor assessed here: Peri-traumatic factor (e.g. fear, perceived life threat...):</b> __
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A specific and validated full-scale measure (self-report or interview), or multiple items from a semi-structured interview was used to assess peri-traumatic factors.	2
A total or mean score from multiple self-report items, either designed specifically for the study or taken from within an existing measure (with good internal consistency for these items, if reported) or A score from a single item from an existing and validated measure	1
Response on a single item or another single way of assessing a peri-traumatic factors was used, or poor description was given of how this factor was assessed.	0

**1.2.6.iii Was the measure of peri-traumatic factors reliable? (Consider how well the authors described this measurement; if a validated full-scale, or multiple items from another scale, or just a single item was used to assess each peri-traumatic factor; and consider if this was assessed by interview or self-report measure)**

*\*If multiple peri-traumatic factors are assessed in one study, please complete this question for each factor, labelling each factor assessed here:*

*Peri-traumatic factor (e.g. fear, perceived life threat...):* \_\_\_\_\_

A specific and validated full-scale measure (self-report or interview), or multiple items from a semi-structured interview was used to assess peri-traumatic factors.	2
A total or mean score from multiple self-report items, either designed specifically for the study or taken from within an existing measure (with good internal consistency for these items, if reported) or A score from a single item from an existing and validated measure	1
Response on a single item or another single way of assessing a peri-traumatic factors was used, or poor description was given of how this factor was assessed.	0

**1.2.7 Was the measure of peri-traumatic factors taken within a reasonable time period after the trauma?**

Yes; peri-traumatic factors were assessed within 2 weeks since the trauma	2
Peri-traumatic factors were assessed >2 weeks but <4 weeks since the trauma	1
Peri-traumatic factors were assessed > 1 month since the trauma	0

**1.3**

**Total Quality Assessment score**

*(\*note if different total score according to different peri-traumatic factor)*

<b>For longitudinal studies:</b> ____ / 14 = ____ %	<b>For cross-sectional studies:</b> ____ / 12 = ____ %
>70% = high quality study	
50-70% = medium quality study	
<50% = low quality study	

<b>1.4 Study Characteristics</b>	
<b>1.4.1</b>	Country of origin
<b>1.4.2</b>	Type of publication (e.g. peer reviewed article)
<b>1.4.3</b>	Sample description
<b>1.4.4</b>	Study design ( <i>cross-sectional or prospective longitudinal</i> )
<b>1.4.5</b>	Recruitment source
<b>1.4.6</b>	Trauma type ( <i>what was the nature of the traumatic event?</i> )
<b>1.4.7</b>	Intentional or unintentional trauma

<b>1.5 PTSD measurement</b>	
<b>1.5.1</b>	PTSD measure 1 name, first author and publication date
<b>1.5.2</b>	PTSD measure 1 type ( <i>interview or self-report</i> )
<b>1.5.3</b>	PTSD measure 1 continuous (symptom severity) or categorical (diagnostic)
<b>1.5.4</b>	PTSD measure 2 name, first author and publication date
<b>1.5.5</b>	PTSD measure 2 type ( <i>interview or self-report</i> )
<b>1.5.6</b>	PTSD measure 2 continuous (symptom severity) or categorical (diagnostic)

<b>1.6 Peritraumatic factors and measurement</b>	
<b>1.6.1</b>	Peritraumatic factor 1 ( <i>what peritraumatic factor was assessed?</i> )
<b>1.6.2</b>	Peritraumatic factor 1 measure ( <i>how was this factor assessed</i> )
<b>1.6.3</b>	Description of peritraumatic factor 1 measure ( <i>e.g. wording of self-report item</i> )
<b>1.6.4</b>	Peritraumatic factor 1 measure type ( <i>interview or self-report</i> )
<b>1.6.5</b>	Peritraumatic factor 1 assessed by single item, multiple items, or full measure?
<b>1.6.6</b>	Peritraumatic factor 2 ( <i>what peritraumatic factor was assessed?</i> )
<b>1.6.7</b>	Peritraumatic factor 2 measure ( <i>how was this factor assessed</i> )
<b>1.6.8</b>	Description of peritraumatic factor 2 measure ( <i>e.g. wording of self-report item</i> )

<b>1.6.9</b>	Peritraumatic factor 2 measure type ( <i>interview or self-report</i> )	
<b>1.6.10</b>	Peritraumatic factor 2 assessed by single item, multiple items, or full measure?	
	Enter additional fields for any further peritraumatic factors assessed	

<b>2</b>	<b>Complete for cross-sectional studies only</b>	
<b>2.1</b>	Section 2.1: cross-sectional study details	
<b>2.1.2</b>	Sample size	
<b>2.1.3</b>	Percentage of responders vs non-responders (those invited who were successfully recruited and participated)	
<b>2.1.4</b>	Age range of sample	
<b>2.1.5</b>	Mean age of sample	
<b>2.1.6</b>	Percentage female	
<b>2.1.7</b>	Time peritraumatic factor assessed ( <i>number of weeks/months since trauma</i> )	
<b>2.1.8</b>	Time PTSD assessed ( <i>number of weeks/months since trauma</i> )	
<b>2.2</b>	<b>Cross-sectional study peritraumatic factor effect size data</b>	
<b>2.2.1</b>	Correlation (r) between peritraumatic factor 1 and PTSD score	
<b>2.2.1b</b>	If r is not provided, enter alternative result statistics and effect size for conversion	
<b>2.2.2</b>	Correlation (r) between peritraumatic factor 2 and PTSD score	
<b>2.2.2b</b>	If r is not provided, enter alternative result statistics and effect size for conversion	
	Add additional fields for any further peritraumatic factor or time point assessed	

<b>3</b>	<b>Complete for longitudinal/prospective studies only</b>	
<b>3.1</b>	Section 3.1: prospective study details	
<b>3.1.1</b>	How many follow-up assessments were completed? <i>Detail number of follow-ups and time since trauma for each assessment</i>	Initial assessment: x days/months since trauma  First follow-up:  Second follow-up:
<b>3.1.2</b>	Sample size at each assessment	Initial assessment n=  First follow-up n=
<b>3.1.3</b>	Mean age (and standard deviation) of sample at initial assessment	
<b>3.1.4</b>	Age range of sample	

<b>3.1.5</b>	Percentage female	
<b>3.1.6</b>	Time peritraumatic factors assessed ( <i>number of weeks or months since trauma</i> )	
<b>3.1.7</b>	Time PTSD assessed ( <i>number of weeks or months since trauma</i> )	
<b>3.2 Longitudinal study effect size data</b>		
<b>3.2.1</b>	Correlation (r) between initial assessment (T1) peritraumatic factor 1 and first assessment of PTSD	
<b>3.2.1b</b>	If r is not provided, enter alternative result statistics and effect size for conversion	
<b>3.2.2</b>	Correlation (r) between initial assessment (T1) peritraumatic factor 1 and first assessment of PTSD	
<b>3.2.2b</b>	If r is not provided, enter alternative result statistics and effect size for conversion	
<b>3.2.3</b>	Correlation (r) between initial assessment (T1) peritraumatic factor 1 and second/follow-up assessment of PTSD	
<b>3.2.3b</b>	If r is not provided, enter alternative result statistics and effect size for conversion	
	Add additional fields for any further peritraumatic factor or time point assessed	

#### **4.2 Converting effect sizes if r is not reported.**

As described in Chapter 1, the meta-analysis was conducted using correlation coefficient  $r$ , as this was most commonly reported by the studies as a measure of risk factor association with PTSD outcome. This effect size statistic is also commonly and simply converted from other analysis statistics when  $r$  has not been computed (Rosnow, Rosenthal, & Rubin, 2000). The following equations were adhered to in order to convert statistics from  $t$  tests (Cohen's  $d$  as the measure of effect size), ANOVA, odds ratio, and beta ( $\beta$ ) statistics.

**4.2.1 T-tests and Cohen's  $d$ .** A few studies reported  $t$ -test analyses of PTSD symptom score mean difference between groups of participants who did and did not experience a peritraumatic factor. Where an effect size was not reported, but mean and

standard deviations were reported, Cohen's d for independent groups was calculated using the following equation:

$$d = \frac{|\bar{x}_1 - \bar{x}_2|}{\sqrt{(\sigma_1^2 + \sigma_2^2)/2}}$$

Where  $x_1$  and  $x_2$  are the means of group 1 and group 2, and  $\sigma_1^2$  and  $\sigma_2^2$  are the variances of group 1 and group 2.

Cohen's d was then converted to r using the following equation (Borenstein, Hedges, Higgins, & Rothstein, 2009):

$$r = d \frac{d}{\sqrt{d^2 + \alpha}} \text{ where } \alpha = \frac{(n_1 + n_2)^2}{n_1 n_2}$$

If a 't' statistic was reported, r could be calculated directly from this statistic, using Cohen's (1965) formula:

$$r = \sqrt{\frac{t^2}{t^2 + df_{within}}}$$

where  $df_{within}$  is the degrees of freedom for the t statistic (calculated by  $n - 2$ ;  $n$  = total number of participants in both groups).

**4.2.2 Estimating r from ANOVA F statistic.** If ANOVA statistics were reported to indicate difference in PTSD symptom scores between those who did or did not experience a peritraumatic factor, the following equation was used to convert the F statistics into Cohen's d using a strategy outlined by Rosnow and Rosenthal (1996):

$$d = 2 \sqrt{\frac{df_n \times F}{df_d}}$$

where  $df_n$  denotes the degrees of freedom of the numerator, and  $df_d$  denotes the demoninator degrees of freedom. Cohen's d was then converted into r using the formula outlined in section 4.2.1.

**4.2.3 Estimating r from odds ratio statistics.** Studies which reported odds ratio statistics were used to convert into Cohen's d, using the formula below (Borenstein et al., 2009), and then converted into r using the formula outlined in section 4.2.1.

$$d = \text{LogOddsRatio} \times \frac{\sqrt{3}}{\pi}, \text{ where } \pi \text{ is approximately } 3.14159.$$

**4.2.4 Estimating r from  $\beta$ .** A number of studies reported standardised regression coefficients ( $\beta$ ) to represent the relationship between peritraumatic factors and PTSD symptom severity. It is argued that  $\beta$  is equivalent to r in univariate regression analyses, i.e. where just one peritraumatic risk factor or predictor variable is entered into the regression model (Peterson & Brown, 2005). However, in multiple regression analyses, an adjustment to  $\beta$  is required to convert it to r, as outlined by Peterson and Brown (2005) using the following formula:

$$r = \beta + .05\lambda$$

where  $\lambda=1$  when  $\beta$  is nonnegative, and 0 when  $\beta$  is negative.

**4.2.5 Fisher's r-to-z transformation and combining r values.** If multiple effect sizes for peritraumatic measures needed to be grouped, r values were transformed into Fisher's z values using the following equation (Borenstein et al., 2009):

$$z = 0.5 \times 1n\left(\frac{1+r}{1-r}\right)$$

A mean value of the subsequent z values was generated, which was then transformed back to an r value, using the following equation (Borenstein et al., 2009):

$$r = \frac{e^{2z} - 1}{e^{2z} + 1}$$

### 4.3 Chapter 4 References

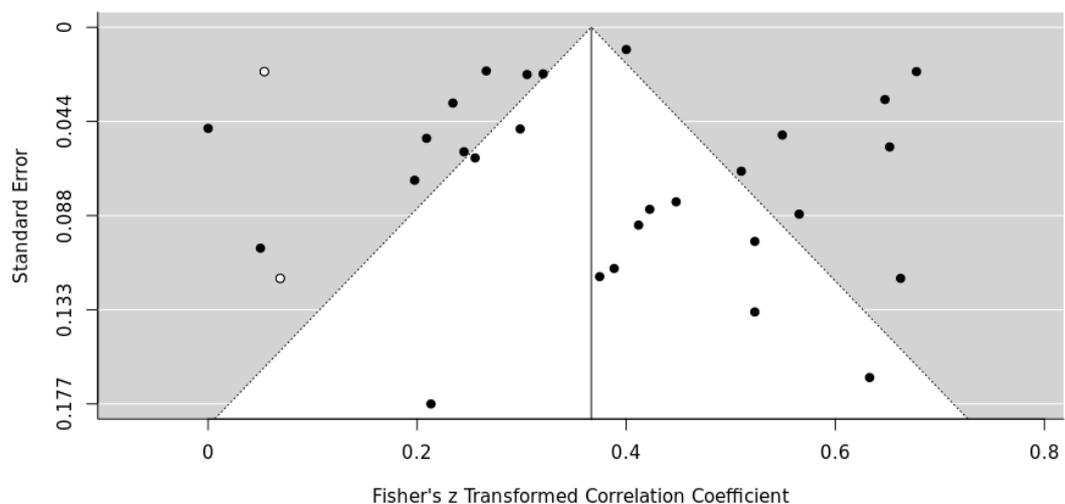
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## Chapter 5. Additional Results

### 5.1 Further analyses and results from the meta-analytic review outlined in Chapter 1

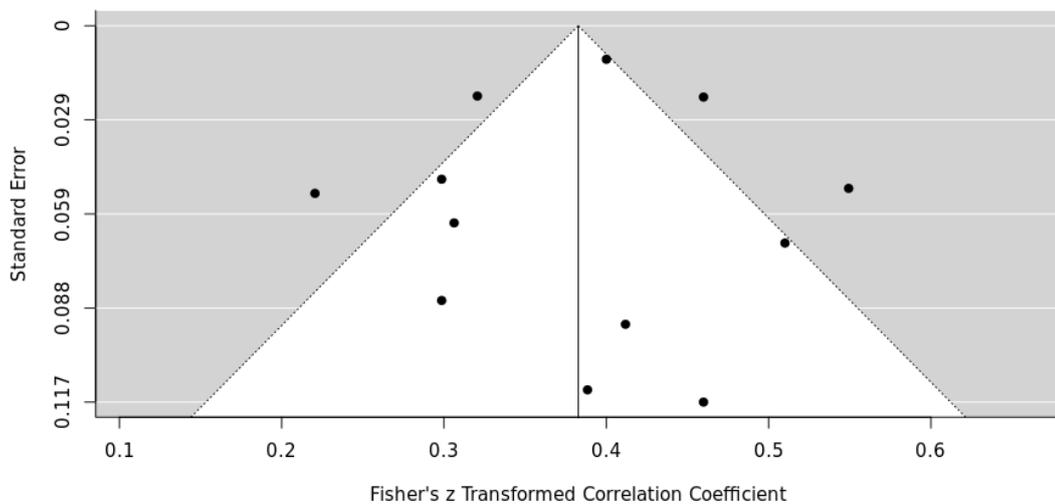
**5.1.1 Funnel plots to assess publication bias.** As outlined in the methods section of Chapter 1, an estimation of possible publication bias within the studies included in each meta-analysis was assessed by generating funnel plots, along with Kendall's tau test of asymmetry. The 'trim-and-fill' method was used to generate the funnel plots, as described by Duval & Tweedie (2000). This method estimates if there may have been any null or weaker studies missing from the meta-analysis; if many are estimated as missing this may be an indicator of publication bias or bias in the included studies. The method augments the observed data by adding estimated missing studies to the funnel plot to make it more symmetric, as asymmetry suggested publication bias; any estimated missing studies are indicated by open circles.

In the main meta-analysis, funnel plots suggested just two estimated null studies missing from the study sample; indicating very low level of possible publication bias (see Figure 7).



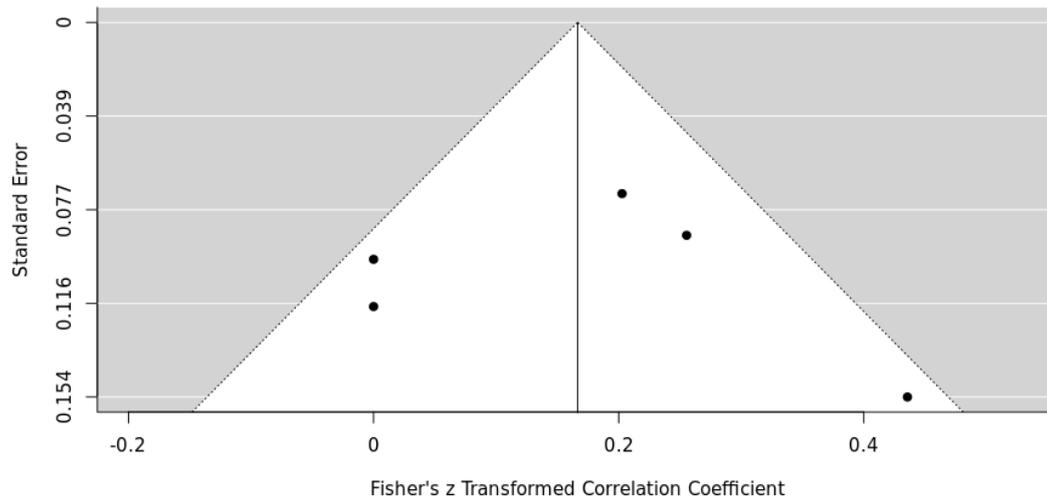
**Figure 7.** Funnel plot generated from Random effects model for the meta-analysis of all peritraumatic subjective threat effect sizes

The second meta-analysis outlined in chapter one analysed effect sizes from studies specifically assessing peritraumatic perceived life threat. The funnel plot below (Figure 8) demonstrates that no null or weaker studies were estimated as missing from this study sample, indicating little to no publication bias.



**Figure 8.** Funnel plot generated from Random effects model for the meta-analysis of all effect sizes related to peritraumatic perceived life threat.

Similarly, the funnel plot generated for the third meta-analysis of effect sizes related to peritraumatic dissociation also indicated no missing null studies, therefore no indicated publication bias in the study sample (see Figure 9). The final meta-analysis of data-driven processing effect sizes included just two studies. A random effects model funnel plot was not able to be generated for this analysis due to the small sample of studies available for inclusion.



**Figure 9.** Funnel plot generated from Random effects model for the meta-analysis of all effect sizes related to peritraumatic dissociation.

## 5.2 Further analyses and results from the empirical study outlined in Chapter 3

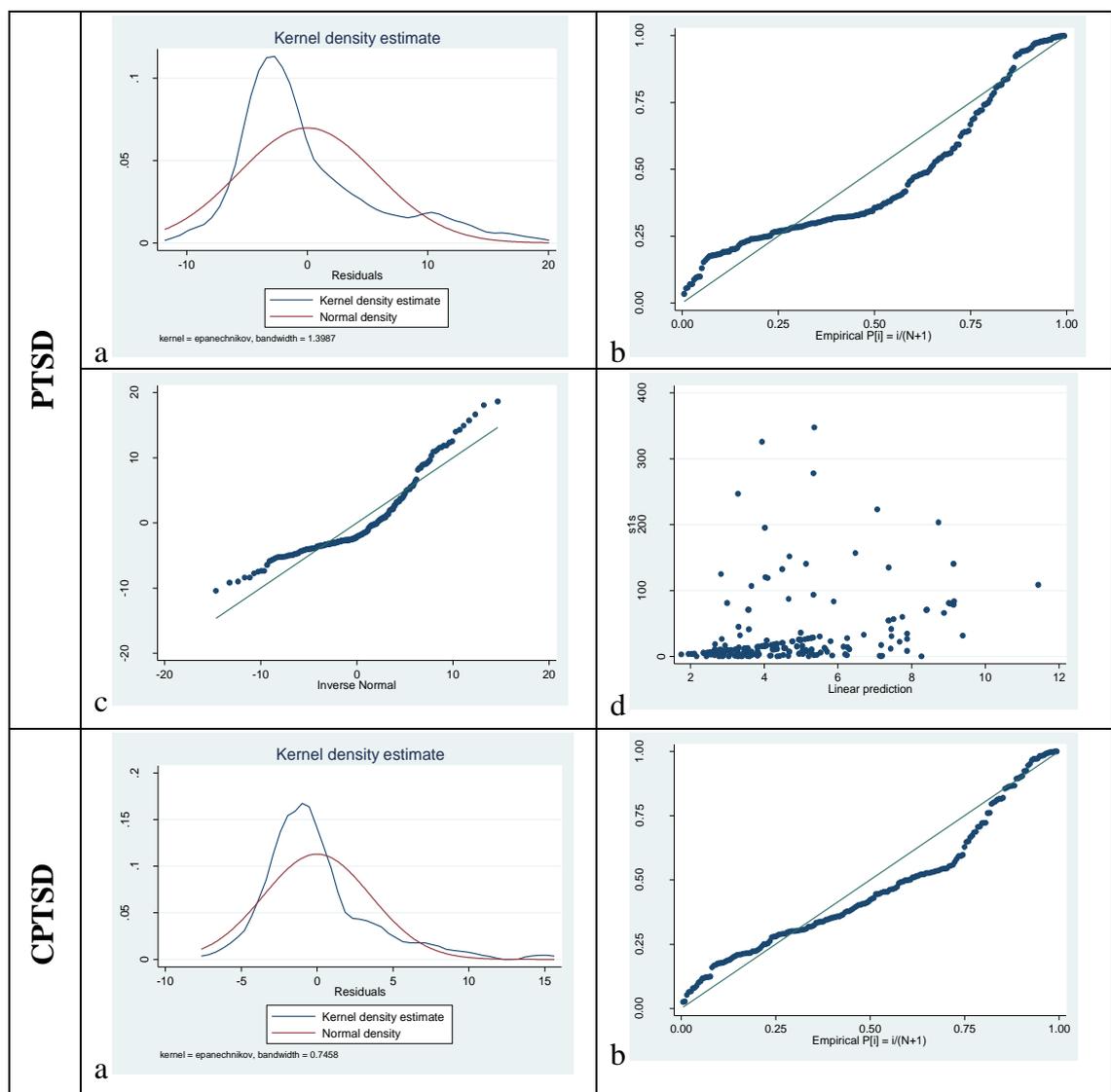
**5.2.1 Pre-analysis data screening.** A series of pre-analysis data screening methods were used to assess whether the data met the assumptions of parametric multiple linear regression. The assumptions of multiple linear regression include: linearity of relationships between predictors and outcome variables; normality of error terms; homoscedasticity indicating constant variance of error terms; and no multicollinearity of predictor variables (UCLA Statistical Consulting Group). Scatterplots with lines of best fit were generated to observe whether there were approximately linear, or non-linear, relationships between all continuous predictor variables and the four outcome variables. Histograms were also generated for all continuous predictor variables to observe the distribution of the data. All scatterplots indicated linear relationships between predictor and outcome variables, however, most continuous predictor variables appeared to have a non-normal and skewed distribution. However, multiple regression does not require normality of data, but does require

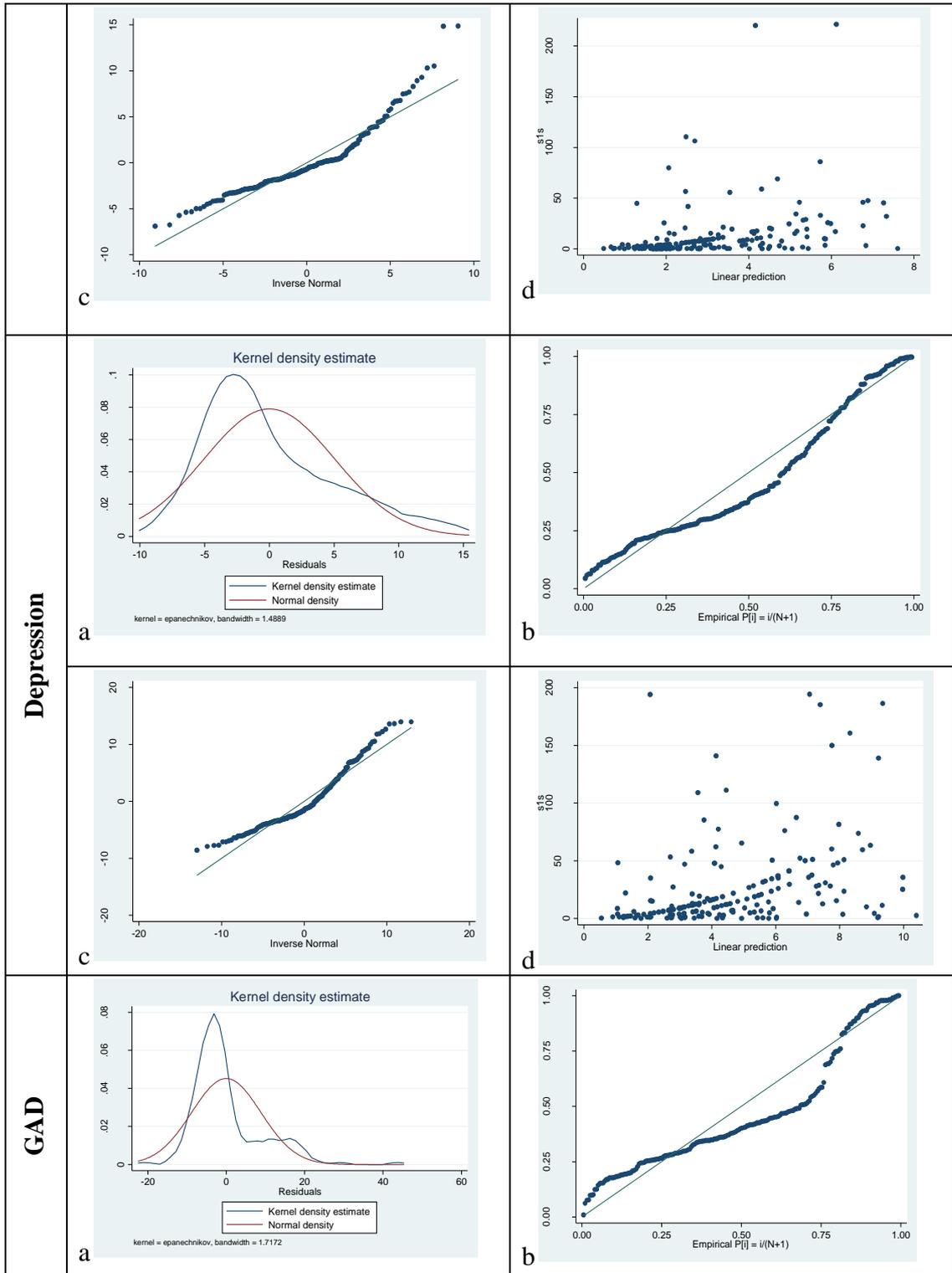
normality of residuals. Multicollinearity between predictor variables was assessed by generating pairwise correlation coefficients between all predictor variables included within each model, and any correlation ( $r$ )  $>.7$  was deemed to indicate a high correlation, suggesting that both predictor variables should not be included in the model. This occurred between just two variables planned to be included in the cognitive model; the two subscales of the trauma appraisals questionnaire were highly correlated ( $r=.82$ ), therefore the subscale scores were not entered as separate predictors, and instead the full scale total score was used. No other predictor variables within a model were highly correlated.

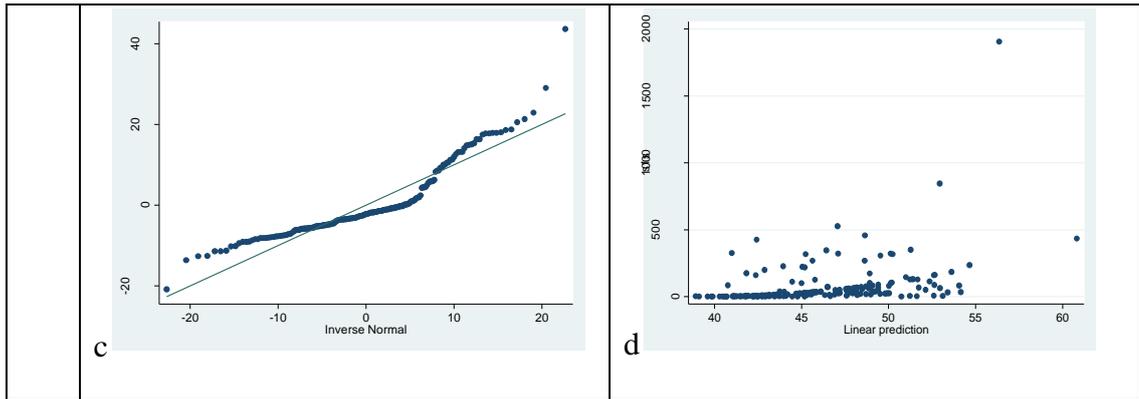
Normality of residuals (error terms) and homogeneity of error variance was assessed by running each multiple regression and using the Stata ‘predict’ command to generate residuals, and the ‘kdensity’, ‘qnorm’ and ‘pnorm’ commands to check the normality of the residuals. The ‘kdensity’ command with the option ‘normal’ generates a kernel density estimate plot with a normal density overlaid to allow for visual comparison of whether the residuals show a normal distribution. The ‘pnorm’ and ‘qnorm’ commands generate a graph of the standardised normal probability and a graph of the quantiles of the variable against the quantiles of a normal distribution, respectively. If the plotted residuals deviate in the centre section in the ‘pnorm’ plot, this suggests non-normality of residuals; likewise, if the plotted residuals deviate at the tail ends of the ‘qnorm’ plot this also suggests non-normality of residuals. Graphical means were also used to observe if the variance of residuals was homogenous. If variance of residuals is constant (homoscedastic), no pattern should be seen when the residuals are plotted against fitted values; conversely, if a funnel shaped pattern is observed this indicates that variance changes as the linear prediction changes suggesting the variance of residuals is heteroscedastic (UCLA: Statistical Consulting Group).

Figures 10.1-10.4 illustrate the plots of residual normality and residual variance generated from the four predictive regression models (psychosocial, cognitive, subjective event severity and objective event severity) for the four disorder outcomes (PTSD, CPTSD, depression and GAD).

**Figure 10.1.** Psychosocial model data screening plots: Kernel density plots (a), probability (pnorm) (b) and quantile normal (qnorm) (c) plots, to assess the normality of residuals, and scatter plots (d) of residual variance to assess homoscedasticity in the psychosocial multiple linear regression model of the four disorder outcomes.

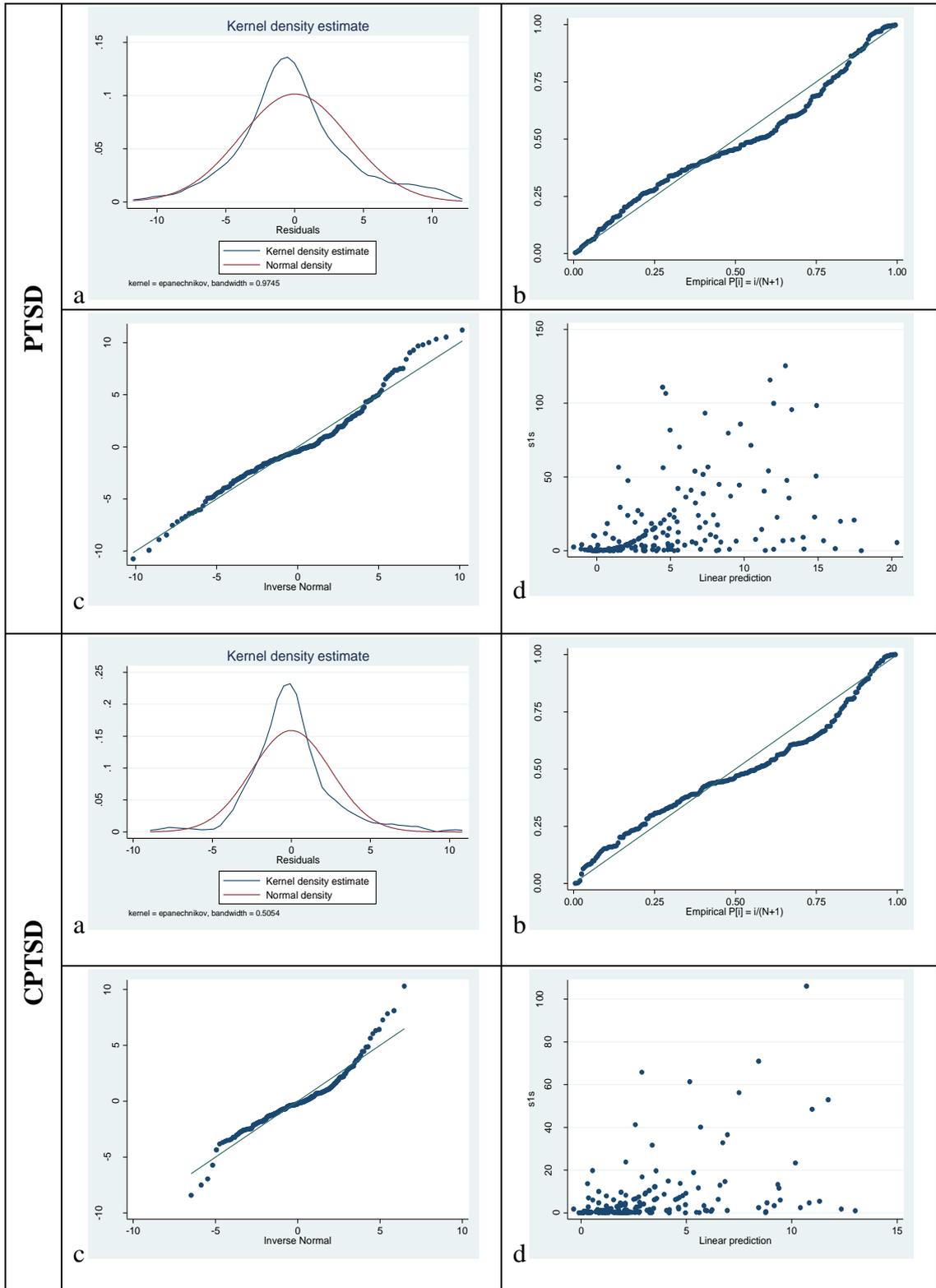


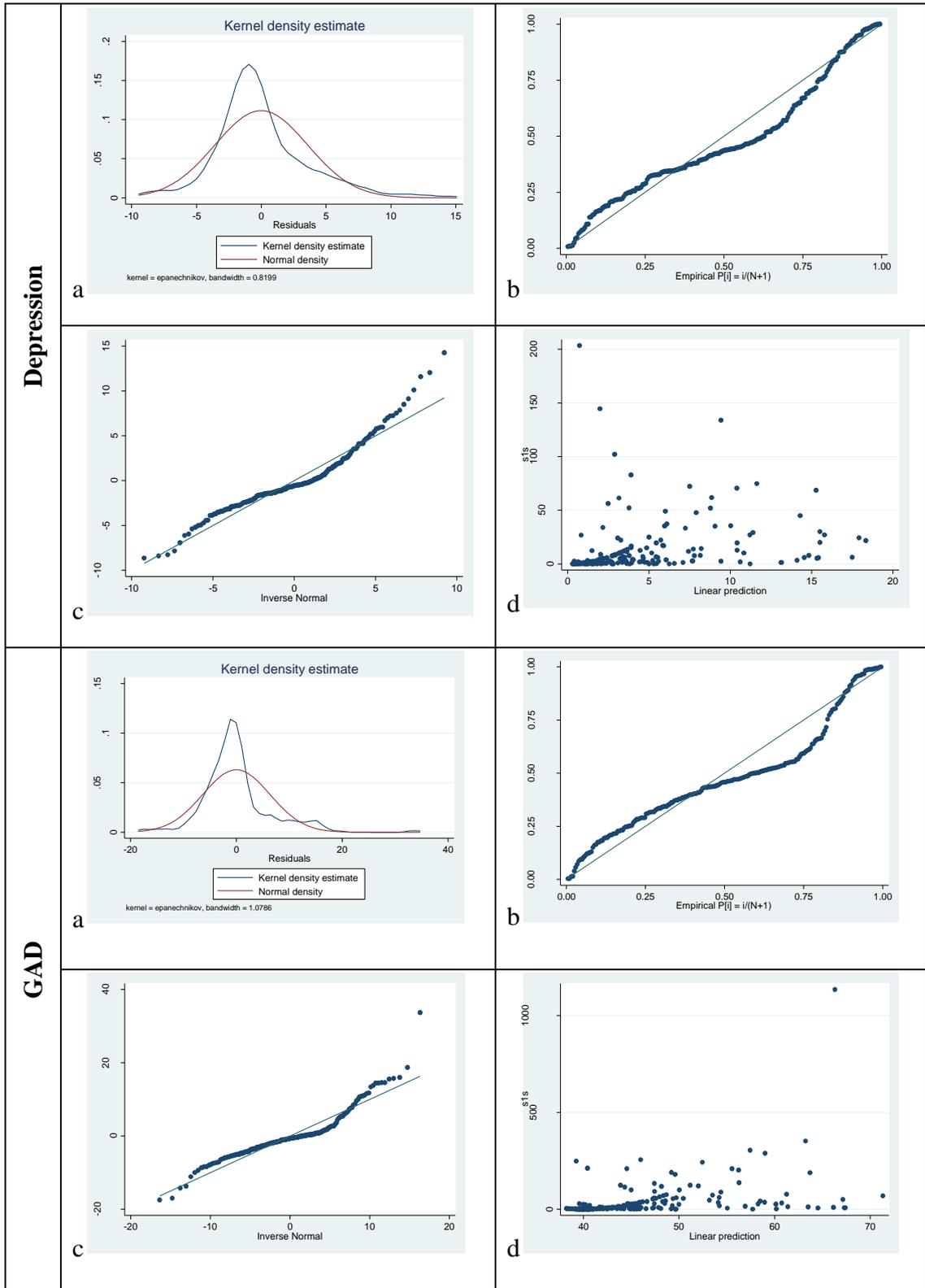




All plots indicated non-normality of residuals as there was clear deviation from the normal distribution in the kernel density plot, and residuals deviated from the straight line in the p and q normal plots. Funnel shaped patterns in all scatter plots suggested homoscedasticity of residual variance. Furthermore, a Shapiro-Wilk W test for normal data generated a very low p value ( $p < 0.0001$ ) for all four analyses, in which case a null hypothesis that the data was normally distributed was rejected (Shapiro & Wilk, 1965). Therefore, the assumptions of multiple linear regression were suggested to be violated and so non-parametric adjustments were required and all regression models using the psychosocial predictors were re-run with bootstrapping, as described in Chapter 3.

**Figure 10.2** Cognitive model data screening plots: Kernel density plots (a), probability (pnorm) (b) and quantile normal (qnorm) plots (c), to assess the normality of residuals, and scatter plots (d) of residual variance to assess homoscedasticity in the cognitive multiple linear regression model of the four disorder outcomes.

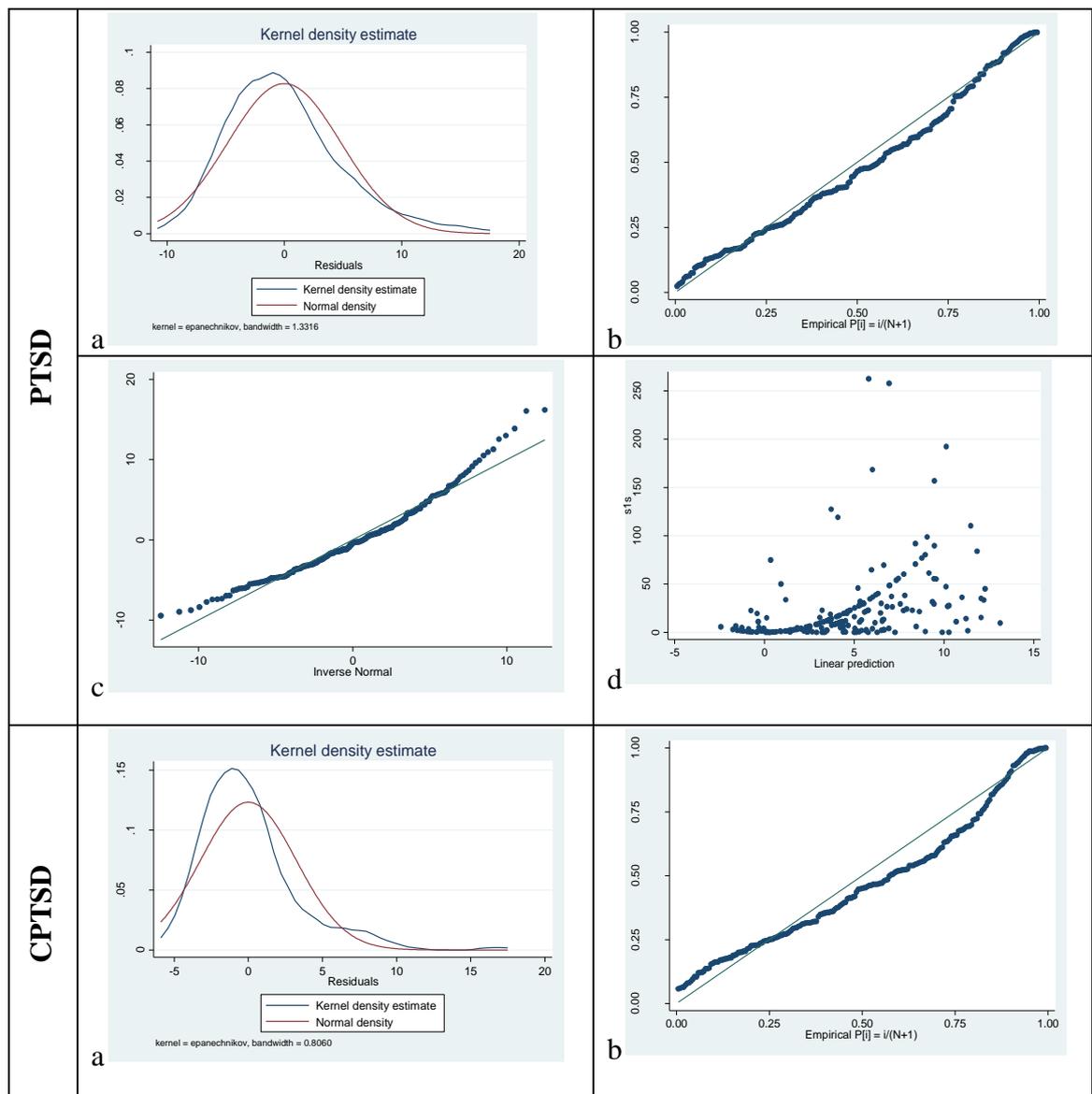


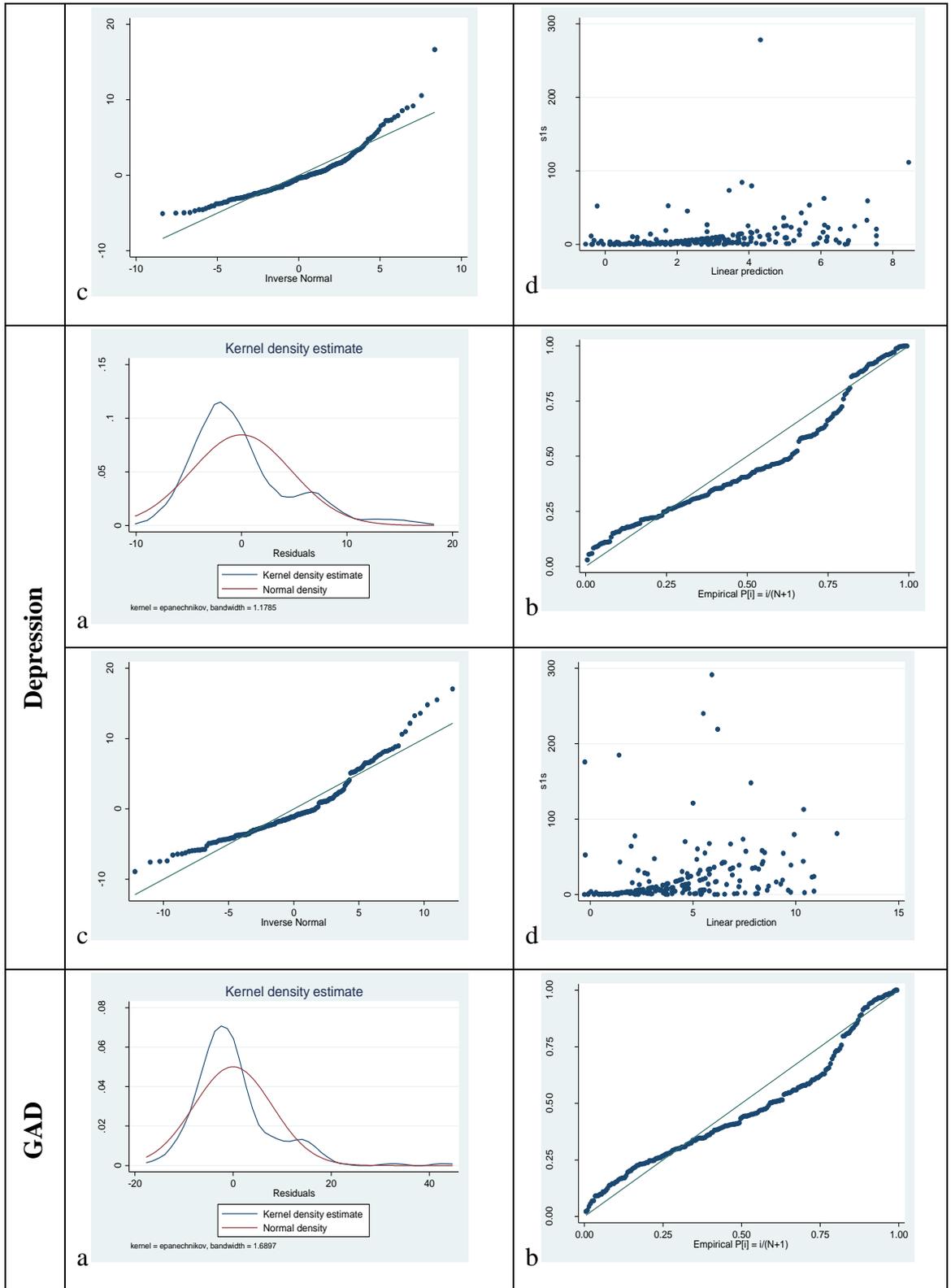


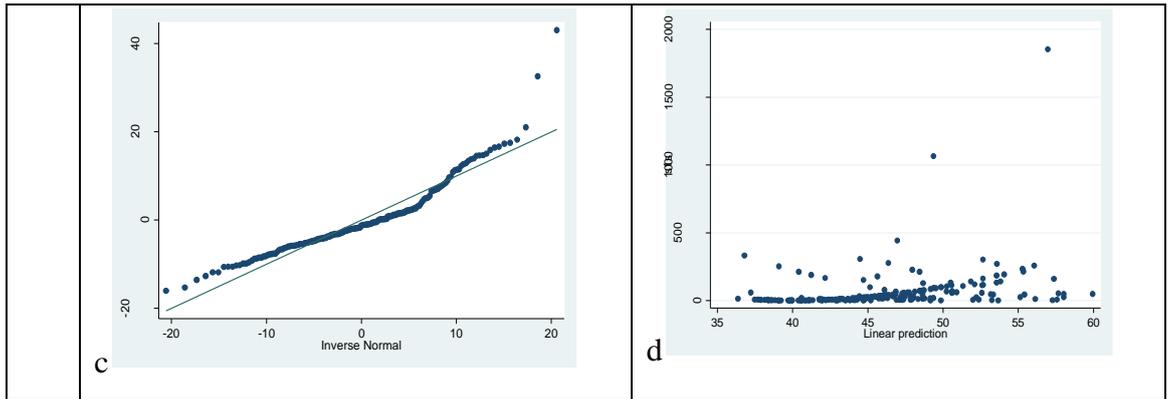
All plots again indicated non-normality of residuals and homoscedasticity of residual variance. The Shapiro-Wilk W tests for normal data again generated a very low p value ( $p < 0.0001$ ) for all four analyses, and so residuals were deemed not normal

(Shapiro & Wilk, 1965). Therefore, the assumptions of multiple linear regression were suggested to be violated and so non-parametric adjustments were required for all cognitive predictive regression models, and these analyses were re-run with bootstrapping, as described in Chapter 3.

**Figure 10.3** Subjective event severity model data screening plots: Kernel density plots (a), probability (pnorm) (b) and quantile normal (qnorm) plots (c), to assess the normality of residuals, and scatter plots (d) of residual variance to assess homoscedasticity in the subjective event severity multiple linear regression model of the four disorder outcomes.

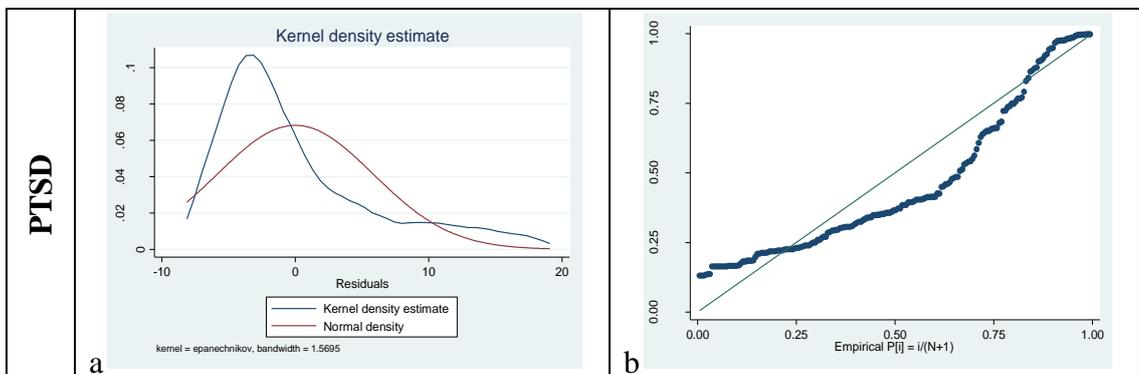


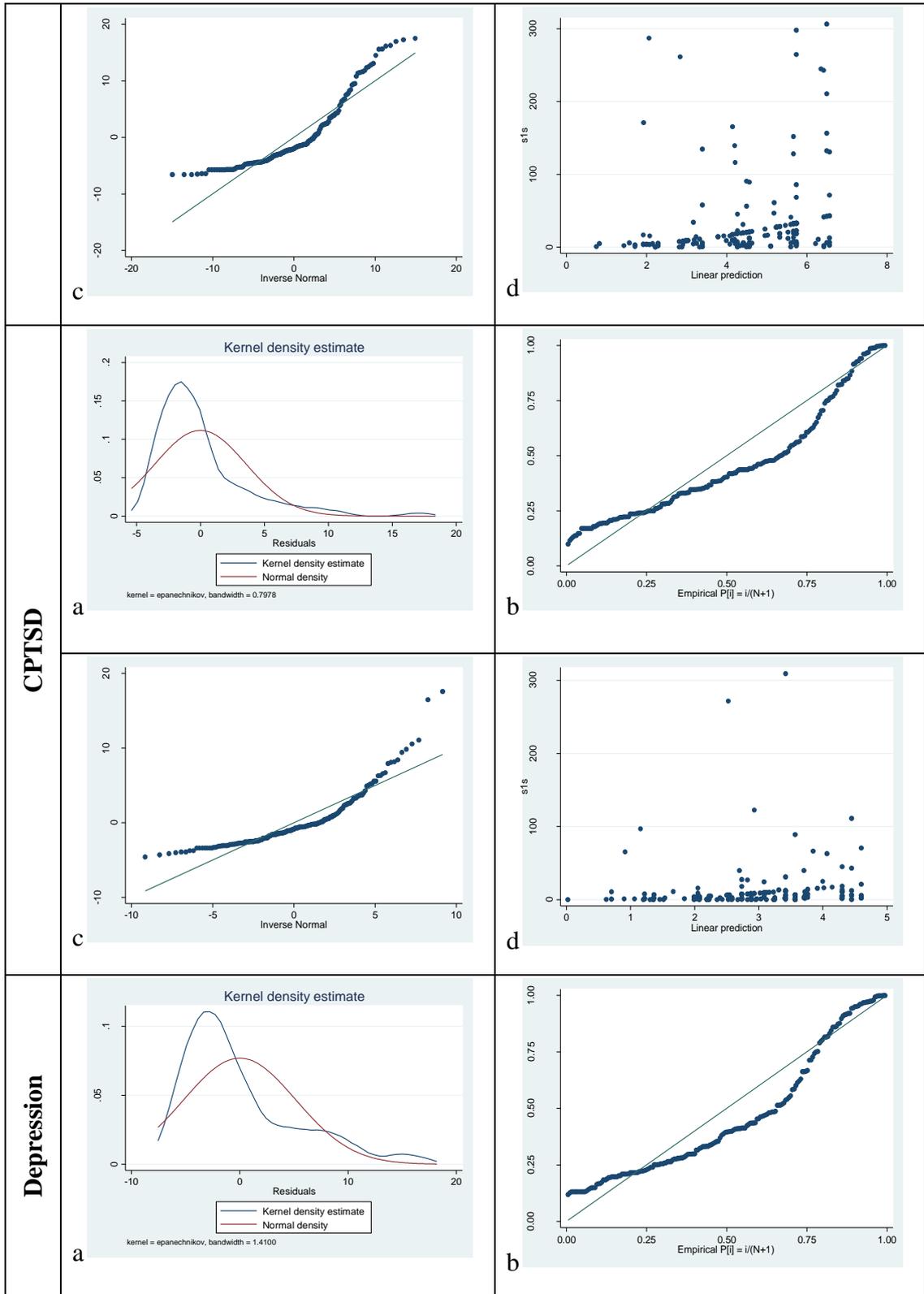


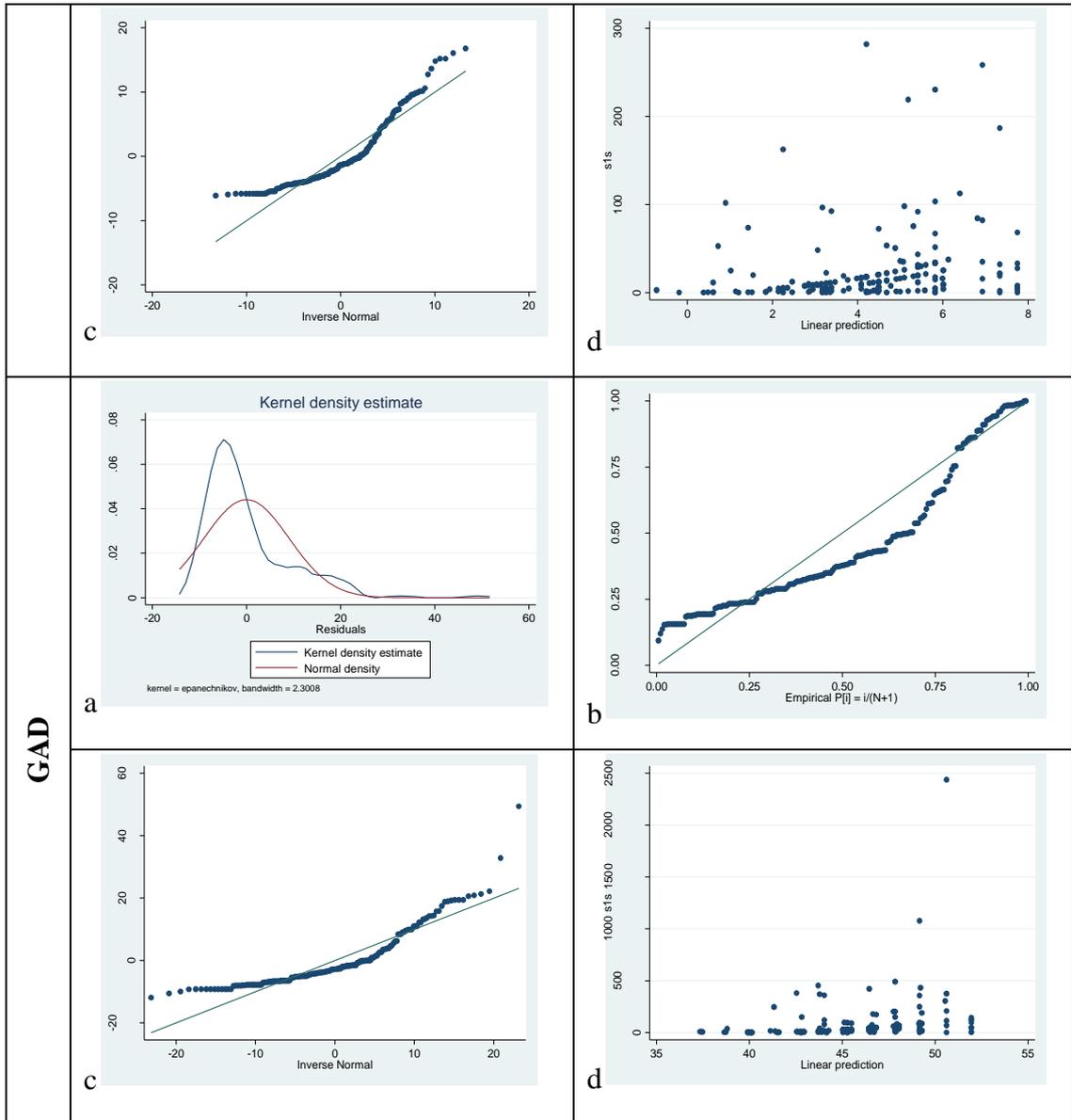


All plots again indicated non-normality of residuals and homoscedasticity of residual variance. The Shapiro-Wilk W tests for normal data again generated a very low p value ( $p < 0.0001$ ) for all four analyses, and so residuals were deemed not normal (Shapiro & Wilk, 1965). Therefore, the assumptions of multiple linear regression were suggested to be violated and so non-parametric adjustments were required for all regression models incorporating the subjective event severity factors, and these analyses were re-run with bootstrapping, as described in Chapter 3.

**Figure 10.4** Objective event severity model data screening plots: Kernel density plots (a), probability (pnorm) (b) and quantile normal (qnorm) plots (c), to assess the normality of residuals, and scatter plots (d) of residual variance to assess homoscedasticity in the objective event severity multiple linear regression model of the four disorder outcomes.







Assessment of data quality in the final predictive model also indicated non-normality of residuals and homoscedasticity from inspection of the plots shown in Figure 10.4. The Shapiro-Wilk W tests for normal data again generated a very low p value ( $p < 0.0001$ ) for all four analyses, and so residuals were deemed not normal (Shapiro & Wilk, 1965). Therefore, the assumptions of multiple linear regression were suggested to be violated and so non-parametric adjustments were required for this final model assessing all four outcomes, and these analyses were re-run with bootstrapping, as described in Chapter 3.

### **5.2.2 Multiple regression analyses incorporating Time 1 symptoms**

The multiple regression analyses described in Chapter 3 incorporated risk factors assessed at two weeks post-trauma and outcomes (symptom severity scores in four disorder domains) at nine weeks post-trauma. The results described in Chapter 3 summarised which risk factors within each predictive model significantly predicted symptoms at nine weeks post-trauma. There is evidence to suggest that acute symptom levels after trauma are a strong predictor of later symptom severity or the development of chronic disorder, therefore, it can be helpful to identify if other acute risk factors add predictive value over and above acute symptom scores (Ehring, Ehlers, & Glucksman, 2008). To assess whether our results would retain significance when symptom severity assessed at two weeks post-trauma was also entered into each model as a predictive factor, the regression analyses were re-run, using exactly the same methods as previously described but with the addition of the two-week symptom scores corresponding to each outcome being measured. For example, to assess if any psychosocial factors would predict PTSD symptoms at nine weeks post-trauma over and above acute PTSD symptom scores, the same continuous measure of ICD-11 PTSD symptom severity was generated using data gathered at the initial assessment two weeks post-trauma, and entered into the psychosocial regression model for PTSD. This method was conducted for all disorders and models. Figure 11 summarises the results.

**Figure 11.** A summary of the factors within each model which retained significance when acute symptom scores were included in the model. Each box represents the relevant predictive model (psychosocial, cognitive, subjective event severity (SES) and objective event severity (OES) models) for each disorder outcome. Any blank boxes indicate that no factors within that model were found to be significant when acute symptom scores were also entered into the model. Any factors which retained a significant effect are noted, with summary statistics indicating their effect.

	Psychosocial	Cognitive	SES	OES
<b>PTSD</b>		Trauma-related appraisals ( $\beta=0.26$ , $p=0.013$ )	Peritraumatic panic ( $\beta=0.17$ , $p=0.013$ )  Peritraumatic perceived harm ( $\beta= -0.1$ , $p=0.06$ )  Peritraumatic dissociation ( $\beta=0.1$ , $p=0.08$ )	
<b>CPTSD</b>		Trauma-related appraisals ( $\beta=0.43$ , $p<0.001$ )	Peritraumatic panic ( $\beta=0.18$ , $p=0.007$ )	Head injury ( $\beta=-0.12$ , $p=0.058$ )
<b>Depression</b>		Trauma-related appraisals ( $\beta=0.41$ , $p<0.001$ )		Opiates ( $\beta=-0.09$ , $p=0.08$ )
<b>GAD</b>		Trauma-related appraisals ( $\beta=0.41$ , $p<0.001$ )	Peritraumatic panic ( $\beta=0.15$ , $p=0.025$ )  Peritraumatic dissociation ( $\beta=0.1$ , $p=0.09$ )	Peri-traumatic pain ( $\beta=0.07$ , $p=0.09$ )

These results demonstrate that no psychosocial factor added value in predicting any of the disorders at nine weeks post-trauma over and above initial disorder symptom scores. However, trauma-related appraisals within the cognitive model consistently retained predictive value for all disorder outcomes with moderate regression coefficients. Interestingly, the PTSD regression coefficient was the smallest, perhaps suggesting the initial symptoms of PTSD are comparatively more pertinent as a risk factor for later PTSD than initial symptoms of CPTSD, depression or anxiety in the long-term development of those disorders. A number of subjective event severity or fear response factors retained predictive value for all disorders apart from depression. Peritraumatic panic appeared to be a significant predictor of PTSD even when acute symptoms are included in the model, and perceived harm and dissociation were near to significant. Just peritraumatic panic remained a significant predictor of CPTSD, and GAD was still predicted by panic, and dissociation had a near to significant effect. All coefficients were small, however, suggesting a significant but small effect of these experiences increasing risk for later disorder when initial symptoms are also considered. No objective measures of event severity retained significance at  $p < 0.05$  level. Interestingly, experiencing a head injury appeared to still have a slight negative impact on the risk of developing CPTSD. No objective measures of event severity retained significance in predicting PTSD over and above acute symptom scores.

### 5.3 Chapter 5 References

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## Chapter 6. Discussion and Critical Evaluation

### 6.1 Overall findings

Our meta-analytic review and empirical study have produced some interesting and valuable results, and generated some important points for consideration. The processes of completing each of these projects were complementary, with each aiding the development and interpretation of the other, although their individual methodological restraints determined a certain amount of limitation on the extent of the conclusions that can be drawn. The systematic literature review and meta-analysis of peritraumatic risk factors for PTSD demonstrated a significant association between peritraumatic psychological processes, in particular, perceived life threat, fear and data-driven processing, and increased likelihood of PTSD symptoms in children and adolescents who had experienced a wide range of traumatic experiences. The evidence available related to peritraumatic dissociation and its association with increased risk of PTSD symptoms suggested an estimated overall effect size which was perhaps weaker than expected, however, with just five studies and two reporting non-significant effects but no effect size data, this result may be overturned with further research evidencing stronger effect sizes for peritraumatic dissociation as a risk factor. Very small ‘fail-safe  $n$ ’s were estimated indicating the number of studies published with non-significant findings required to significantly challenge the overall estimates of effect size calculated for peritraumatic dissociation and data-driven processing. This implicates that there is limited certainty with which conclusions can be drawn from the meta-analytic results relating to these two risk factors. Estimates of between-study heterogeneity were not large for studies of peritraumatic dissociation or data-driven processing, but indicated significant variation between the larger group of studies assessing subjective threat and fear. This difference in heterogeneity between these groups of studies assessing

different factors may be in part due to there being fewer studies assessing data-driven processing and dissociation, the measurement of these factors being more similar (either by use of standardised measures, for example the peritraumatic dissociative experiences questionnaire, or single items worded very similarly), or the study types being similar (trauma type, cross-sectional and prospective studies). We also identified a greater variation in how subjective threat and fear experiences were assessed; with some studies overlapping and assessing multiple aspects of peritraumatic experiences together as a proxy of overall peritraumatic 'response', and other studies using more specific assessment of individual factors, such as perceived life threat or feeling helpless. In order to account for this difference in methodologies of assessing peritraumatic experiences, we used the DSM-IV grouping of subjective threat and fear response (which includes perceived life threat, feelings of fear, helplessness or horror) as an overarching peritraumatic experience, which was also a way of grouping factors that a number of the included studies used. Ideally, future research may serve to develop our understanding of these different experiences if researchers use methods to clearly assess each factor individually, and report effect sizes separately for perceived life threat, feeling fear, helplessness or horror. We may then be better able to identify if between study heterogeneity is a methodological issue of measurement and grouping of factors, and if these individual experiences have different predictive powers as risk factors for PTSD. Within the methodology for the empirical study analysis of predictors of PTSD and other disorders, we were mindful of this methodological reflection, and so entered peritraumatic experiences of perceived life threat, perceived risk of physical harm, and feeling scared, individually into the predictive model. This allowed observation of differential effects of each item within the model and across different disorders.

Within our study of children and adolescents following a single-event trauma, we were able to compare and contrast different models of risk factors for PTSD and other disorders, including peritraumatic experiences. The peritraumatic subjective threat and fear model predicted 33% of variance in PTSD symptoms, and also predicted 26% of variance in CPTSD, 24% of variance in depression symptoms and 28% of variance in GAD symptoms nine weeks post-trauma in this population. The correlation coefficients (Pearson's  $r$ ) estimated for peritraumatic risk factors assessed at two weeks and symptoms at nine weeks in the study population were not largely dissimilar to the overall population estimates of effect size estimated in the meta-analysis. Perceived life threat, perceived threat of harm, and fear demonstrated correlations with PTSD symptoms of .28, .26, and .43 respectively in our sample, which when averaged (using Fisher's  $z$  transformation) generates an overall subjective threat and fear response correlation with PTSD symptoms of .33; compared to the population estimate of effect size (Pearson's  $r$ ) of .37 estimated in the meta-analysis. However, data-driven processing and PTSD symptoms showed a greater correlation in the study sample (.48 compared to .29 derived from the meta-analysis), as did peritraumatic dissociation (.34 in the study sample compared to .17 population estimate from the meta-analysis). Importantly, our empirical study analysis of predictive models of PTSD demonstrated the importance of considering risk factors together within analyses, as risk factors may not be totally independent in their mechanism of action on the increased risk of development of symptoms. When assessed in isolation, the meta-analysis and our empirical study results indicated that many individual risk factors appear to have a strong correlation with later symptom scores. However, when entered into a model of peritraumatic predictors of PTSD including perceived life threat, panic, feeling scared, perceived threat of harm, and dissociation, perceived life threat did not have a significant effect whereas dissociation, fear and panic during the trauma were

significant predictors of later symptoms. Similarly, when entered into a model alongside other cognitive factors outlined in cognitive models of PTSD, data-driven processing was not a significant predictor of PTSD.

A major finding of our empirical study was the relative power of cognitive factors and the overall cognitive model in predicting not only PTSD but also CPTSD, depression, and GAD as outcomes of trauma in children and adolescents. This supported our hypothesis that the cognitive model would be the strongest model in predicting PTSD and CPTSD, however did not fully support our hypothesis that this model would demonstrate disorder specificity and power in differentiating between disorders. Individual correlation coefficients for cognitive factors and PTSD symptoms were large for all the cognitive factors outlined by the Ehlers and Clark (2000) model of PTSD; rumination, negative trauma-related appraisals, trauma memory quality, ongoing dissociation and self-blame. However, within the predictive model, trauma memory quality, appraisals, and ongoing dissociation were significant positive predictors of increased likelihood of PTSD symptoms, whereas self-blame was a significant negative predictor of PTSD. It may be interesting for future research to measure both self-blame and blame of others in relation to a trauma as meta-analytic assessment of previous studies demonstrates that blame of others has been found to be a significant positive predictor for PTSD (Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). This finding within our study may be demonstrative of self-blame being an opposing proxy to blame of others. Interestingly, a key difference was identified between risk factors for PTSD versus CPTSD in relation to self-blame as this was a significant positive predictor for CPTSD, and was also had no significant effect in predicting depression or GAD. The objective and subjective event severity models demonstrated similar significant within-model predictors across all disorders; peritraumatic panic,

dissociation and pain were implicated as risk factors which predicted PTSD, CPTSD, GAD, and depression. The objective event severity model was a weak model for all disorders, but appeared to account for more variance in depression and GAD symptoms compared to PTSD and CPTSD. There were few other significant differentiators between the models, the effect of the factors within them or their goodness of fit between the four disorders. Overall, the analyses conducted suggested that subjective and cognitive factors hold more importance than objective measures of trauma severity or psychosocial factors in predicting in the acute post-trauma phase which children or adolescents may later develop symptoms of PTSD, CPTSD, depression or anxiety.

## **6.2 Strengths and limitations**

**6.2.1 A comprehensive literature review.** A highly comprehensive search strategy screening a vast number of studies enabled a significant addition to the field of understanding relating to peritraumatic risk factors for PTSD in children and adolescents. A greater number of studies than expected were identified which had explored this area, considering that a meta-analysis published five years previous to the current searches yielded just six studies of peritraumatic factors, and the current review identified 32 relevant studies. Despite some challenges related to the identification of studies assessing peritraumatic factors, a systematic screening method was used which allowed us to most carefully consider any studies identified in the searches which may have been relevant. Reference to previous meta-analyses and literature regarding meta-analytic methods helped to inform the appropriate selection of analysis strategies. Furthermore, by developing and utilising a considered quality assessment framework we were able to consider risk of bias within the meta-analysis results, which currently is rare practice for meta-analyses not related to randomised controlled or treatment trials.

Meta-regression and subgroup analyses demonstrated that the results were not likely to change even if studies with high risk of bias were excluded, or only high-quality studies were assessed. Funnel plots and assessment of publication bias also gave confidence that risk of bias related to under-representation of weaker research data, due to the increased likelihood of significant results being published, was low in our analysis. This review also provided an insight into the range of methodologies typically used by researchers to assess peritraumatic experiences, which may be helpful for future researchers to consider developing or increasing the use of standardised measures, and to encourage the use of the term ‘peritraumatic’ when reporting experiences or processes occurring during or immediately after a trauma. The conceptualisation of certain peritraumatic experiences as risk factors, rather than diagnostic criteria, is also in line with the changes from DSM-IV to DSM-V, which no longer requires these experiences of fear, perceived life threat, horror or helplessness to have occurred for an experience to be deemed traumatic. This change reflects that not all individuals may have, or may remember (for example, due to loss of consciousness) these peritraumatic experiences; as our results also indicate, these peritraumatic experiences are not necessary for the development of trauma. The strength of the predictive effects of these experiences in this current analysis were not so large to suggest they are overly important, however, a child who experiences these factors at the time of trauma may be more likely to develop PTSD.

**6.2.2 Issues of sample size, power and variance.** Our review, despite being a significant addition to the field, was limited by a reasonably small number of studies being eligible for inclusion. This was a particular weakness of the meta-analysis of study data related to data-driven processing and dissociation, and for the meta-regression analyses. Issues related to power and number of studies needed for meta-

analysis and meta-regression were discussed, and warning was given regarding the limitations to conclusions that can be drawn from these analyses. In particular, meta-regression analyses with a small number of studies has been argued to be prone to generating false-positive results (Hedges & Pigott, 2004; Higgins & Thompson, 2004). However, this is a generally accepted common situation for meta-analyses and recommendations to account for this do not suggest that these analyses are invaluable or should be avoided, but do suggest care should be taken with regards to over-stating conclusions, or for the application of a statistical test to further explore the ‘true’ significance of a meta-regression finding (Higgins & Thompson, 2004). This ‘permutation test’ to assess the true significant of the meta-regression p-values was not available in the software used to run the current meta-analysis. An improvement to this study may have been to utilise a statistical software which would allow for this extra test.

A second limitation of the meta-analysis related to the high heterogeneity between the studies included in the main analysis; possible reasons for this heterogeneity were discussed, with meta-regression analyses providing some insight into possible reasons for the variation. High estimates of heterogeneity between studies indicates a possible lack of consistency and genuine differences underlying the results of the studies, rather than variation in findings being due to chance alone. Within the main group of studies, the estimate of heterogeneity was 94%, suggesting a large degree of inconsistency in results. It has been estimated that 25% of meta-analyses indicate  $I^2$  values of over 50%, therefore this is not an uncommon issue and should be expected (Higgins, Thompson, Deeks, & Altman, 2003). Higgins et al. (2003) suggested that investigating the reasons for heterogeneity and considering any clinical implications of the degree and nature of variation is the advisable strategy for dealing with high

heterogeneity. Importantly, within this meta-analysis, effect sizes sourced from the studies did not reflect results suggesting different directions of effect for the peritraumatic risk factors assessed, which supports a clinically meaningful conclusion that peritraumatic experiences of fear, perceived life threat, dissociation and data-driven processing generally increase the likelihood of developing PTSD symptoms. A further limitation regarding the variation in studies sourced for the review was that all were peer-reviewed journal articles, no unpublished material was included. This may have increased the risk of publication bias being represented in the overall results, however, efforts were made to assess likelihood of publication bias and these analyses indicated low risk of bias. The inclusion of data from a few unpublished studies or projects reporting weaker results may have improved the risk of bias in our review.

**6.2.3 Variation in measurement techniques.** Issues related to measurement of factors arose in relation to both the review study and the empirical study. Research authors typically take great care in designing methodology and selecting measures to use, however, this does not fully reduce between-studies or within-study variation in measurement techniques of risk factors. Within the meta-analysis, single-item self-report questionnaire measures, averages or totals of multiple self-report items, full questionnaires, and structured interview questions (including one study utilising puppets to adapt questions for young children) were used to assess peritraumatic factors. Careful consideration of the content of each measure was made to attempt to group them based on the underlying factor which they were deemed to assess. Consideration was also made at the point of data sourcing with regards to the quality of measures used, for example, only using self-report from the child (no parent report). However, less variation in these measurement techniques may have improved consistency and validity of the overall results. Similarly, within the empirical study of risk factors for PTSD and

other diagnoses, data derived for the factors within the predictive models had been gathered in different ways, including: hospital data, parent report (self-report questionnaires and structured telephone interviews), and child self-report (mainly questionnaires in this current study). The cognitive model of predictors included all child self-report questionnaire data, whereas the objective event severity model included data sourced from hospital records and child self-report data, and the psychosocial model incorporated more data sourced from parents. We considered whether a limitation of our conclusions from the analysis may have been the predictive power of the cognitive model being a proxy of the meaningfulness of self-report data from the child, as other models included data related to the child but from other sources. However, the subjective event severity model also incorporated all self-report data and was not as powerful as a predictive model as the cognitive model, reflecting that the content of the cognitive model was important.

**6.2.4 Strengths of the empirical study.** The opportunity to complete an empirical study utilising a pre-existing dataset (from the ‘ASPECTS’ study; Meiser-Stedman et al., 2017) provided huge strengths; this was a highly valuable and difficult to recruit study population, who had engaged in a longitudinal research project incorporating a comprehensive battery of measures conducted by a team of research and clinical professionals. Our meta-analysis identified just one longitudinal study of children and adolescents following an acute physical injury and single-event trauma which recruited a fractionally larger sample ( $n=269$  compared to  $n=260$  in our study sample), conducted by Winston, Kassam-Adams, García-España, Ittenbach, and Cnaan (2003) in the US, indicating that this current sample may be the largest of its kind in the UK. Access to this large study sample enabled us to consider well-powered and comprehensive analyses to explore the research questions we had. The prospective

longitudinal design also enabled the appropriate consideration of risk factors, as suggested by Kraemer et al. (1997), as these factors were assessed first, shortly after trauma, and outcomes were measured after a time period at follow-up. These strengths were attributable to the initial study design, implementation and efforts by the study team, but were of huge value to what was then possible for this thesis project and within the feasibility of a DCLinPsy thesis.

A strength in the design and methodology of this thesis project was the use of theoretically and research driven models of predictors of different mental health outcomes of trauma. Typically, research has focussed on PTSD as the main outcome, have utilised single models of predictors, or explored a list of predictors independently of each other. We identified a strength in developing a number of predictive models incorporating appropriately grouped risk factors, and were able to implement this analysis by carefully sourcing data for each risk factor and applying regression models to assess the four outcomes. Despite it being a comparatively large sample to other studies, we considered statistical power, the nature of the study data and identified where there were violations of assumptions required for parametric analyses. We employed bootstrapping techniques to account for these data violations, leading to more reliable and standardised result statistics. The generation of model fit statistics to assess the goodness of fit of each of the predictive models also allowed for comparison between the models and account for the variables not within each model but tested in other models. A final major strength of this thesis project was the information we have gained from studying Complex PTSD in this population. The development and analysis of an outcome reflecting the proposed ICD-11 criteria for CPTSD was informed by two studies validating this diagnostic category in children and adolescents, and the methodology used to develop a measure for CPTSD post-study design and

implementation (Perkonigg et al., 2016; Sachser, Keller, & Goldbeck, 2017). The comprehensive analysis of predictors of CPTSD using a longitudinal sample of children and adolescents who had experienced a single-event trauma was the first of its kind. This analysis and project report was therefore highly novel and timely considering the proposed publication of ICD-11 in 2018; and so may help to inform academic debate regarding the conceptualisation of this disorder, its identification and presentation in children and adolescents. Our sample prevalence statistics suggested that 8.5% of children (n=20) presented with complex symptoms outlined in ICD-11 nine weeks after trauma, and 5% (n=12) met full diagnostic criteria for CPTSD. This is a significant contribution to the conceptualisation of this disorder as it indicates that complex symptoms and disorder can arise in children and adolescents after a single-event trauma.

**6.2.5 Limitations of the empirical study.** A limitation related to the analysis conducted in our empirical study was the difficulty in separating comorbidity of symptom presentation; the sample available did not allow for the assessment of predictive models for outcomes of PTSD alone (no depression, complex symptoms or GAD), and likewise for the other disorders. Our summary of frequencies of individuals meeting each disorder demonstrated that there were many individuals within the sample who had symptoms of more than one of the disorders. Therefore, those scoring highly on each continuous measure of the disorders used as outcome in the regression analyses may have also concurrently be scoring positively on the continuous measure of another disorder. This comorbidity may explain some of the similarity in results found with regards to significant predictors across the disorders, and may suggest that results were reflecting a ‘general distress’ factor. However, some differences in the correlates and predictors of each disorder were identified, and there were variations in the models’ goodness of fit indices. An improvement of this methodology would be the recruitment

of a larger sample size, and conduct analyses which control for the presentation of other disorders. This may improve the theoretical conclusions able to be drawn with regards to disorder specific predictive models, but clinically may be less valid, as patients are most likely to present with symptoms which do overlap a number of disorders.

### **6.2.6 The context and process of the completing the thesis portfolio**

Some further reflections regarding the strengths and limitations of these thesis projects relate to the process and experience of completing the thesis. The empirical study completed was planned and designed following an initial project proposal being rejected by a research ethics committee (REC); this original study aimed to source data from pre-existing interview recordings from the same ASPECTS study sample to assess qualities of trauma narratives as predictors of PTSD. The challenge from the ethics committee related to the consent which participants had given, as recordings were taken initially for quality-control rather than analysis purposes. This process was a useful learning experience with regards to the procedure of making an application to a REC, and considering ethical issues with using pre-existing study data and considering the original remit of a study. Despite some disappointment due to the original proposal being rendered unfeasible as the committee recommended re-contacting and re-consenting participants to access and analyse interview recordings, the experience overall was positive and helpful. In particular, the REC were highly encouraging of research in this area, understood the time limitations of a DCLinPsy thesis project, and encouraged the consideration of an alternative project within the remit of the original study. The current project was planned and proposed to the university, and as it was using only the quantitative dataset which participants had consented to be gathered and analysed and fell in the original project remit, no further ethical applications were

needed beyond the original ASPECTS study ethics application previously granted approval. Completing an empirical study utilising previously gathered data also enabled a greater focus of time to complete the comprehensive meta-analytic literature review. However, not having an active role in the data collection process may have limited the qualitative insight into the experience of participants which may have aided interpretation of results with more contextual depth. The alternative benefit of having an objective position may have also reduced the likelihood of researcher bias in the analysis and interpretation of the results. Overall, the two research projects conducted were experienced as comprehensive, systematic and complementary projects which developed our understanding of an important area of research and clinical practice.

### **6.3 Theoretical and clinical implications, and areas for future developments**

A number of implications resulting from the conclusions of the meta-analysis and empirical study were discussed in chapters one and three. Overall, the results from both studies supported the validity of cognitive models of PTSD, which depict peritraumatic processes and experiences of feeling fear, panic, perceived threat, and dissociation, and post-trauma cognitive processes relating to how trauma memories are appraised, the nature and quality of the memory, and rumination and self-blame related to the trauma, as key factors involved in the development of PTSD in children and adolescents. Evidence gathered was also in support of the role of prior experiences and the characteristics of trauma, as outlined in the Ehlers and Clark cognitive model of the development of PTSD (Ehlers & Clark, 2000). The results from the empirical study challenged cognitive theories suggesting that data-driven processing is a key factor in the development of PTSD, as this was a non-significant predictor in the model assessed. However, the meta-analytic results, despite limited by study numbers, would warrant

this factor to still be considered within a cognitive model, with the need for further research to explore this further. Future research is encouraged to consider how multiple risk factors may relate and interact, or how the presence of one may negate the predictive action of another. Theoretically, our results imply that models which consider mediating, moderating or multiple pathways of action for predictors, rather than independent paths of action, may be most valid. Path analyses of risk factors and the development of outcomes of interest, such as mental health difficulties, may be best conducted with longitudinal studies of large sample sizes incorporating three time points or more. Further research incorporating mediation, moderation or path analyses would aid the clarification of the role of different risk factors, and may explain the variation in current research findings as related to what other factors were assessed in each study and how risk factors were analysed together.

With regards to implications for clinical and research practice with children who have experienced trauma, a number of considerations and recommendations may be made. When individuals come into contact with services or are recruited into research studies soon after a trauma, early identification of individuals who may have experienced multiple prior traumas, those who have experienced an interpersonal trauma, with prior mental health or wellbeing difficulties, and particular consideration of females, may be useful psychosocial indicators of those at higher risk of developing chronic symptoms of mental health difficulties. These psychosocial factors may be relatively simple to assess, but are not highly predictive of later disorder. In contrast, the assessment of emotional and cognitive factors including peritraumatic responses, ongoing dissociation, the nature of trauma memories, negative appraisals and rumination about the event in the early stages after a trauma may be more valuable and

helpful in identifying those at increased risk of developing PTSD, CPTSD, depression, or anxiety.

Our results suggest that consideration of Complex PTSD when a child has experienced trauma may be valid and helpful, even in situations when a child has experienced a single-event trauma. Referral to the new ICD-11 and related research and guidance will be required for the accurate application of this new diagnosis in clinical settings. Clinicians may not come into contact with patients until symptoms of mental distress are significant and deemed as requiring referral to services for intervention, which may not occur until an extended period following trauma, therefore the early identification of risk factors may not be possible or relevant. This reality of clinical practice limits the potential utility of assessing for early risk factors for the development of disorder, particularly for clinicians such as psychologists and psychiatrists who are most likely to come into contact with individuals long after the experience of trauma for the purposes of diagnosis or formulation, treatment and intervention. The clinical, as opposed to research, benefits of awareness and assessment of early risk factors may more suitably be considered by professionals in the emergency services, such as police officers, paramedics, and hospital Emergency Department staff. These professionals are likely to come into contact with and support individuals in the early aftermath of trauma and therefore may be more able to consider and assess for early risk factors. This awareness then may helpfully enable these professionals to signpost individuals to support services if they are deemed at risk of developing mental health difficulties such as PTSD or CPTSD. It is likely that mental health services will consider referrals for treatment once a clinical level of disorder is present, which for PTSD and CPTSD will be at least one month following the event, however this may enable identification of and referral for individuals earlier in the development of disorder. As such, the early

assessment of risk factors for the development of disorder following trauma may beneficially alter an otherwise potentially slower trajectory of recovery from traumatic stress following a trauma.

Awareness of key risk factors may also inform how diagnosis and treatment is conceptualised. In particular, understanding the peritraumatic and post-trauma cognitive maladaptive processes which are key in the development of disorder may help to inform the focus of intervention. For example, trauma-focussed cognitive behavioural therapy (TF-CBT) involves challenging and shifting unhelpful trauma-related appraisals. The results of the current study support this focus of treatment, as maladaptive trauma appraisals were a consistent and powerful predictor of PTSD, and also CPTSD, depression, and GAD, even when acute symptom scores were incorporated into the predictor model (see Chapter 5 section 2.2). It may be of benefit for clinicians to also consider applying intervention strategies which involve a focus on reappraising trauma-related beliefs, re-processing trauma memories (for example by building a coherent narrative of a trauma), and reducing negative emotional responses to trauma-related stimuli through graded exposure with patients presenting with symptoms of CPTSD, depression, or anxiety following trauma. Finally, the application of our findings to clinical practice and further research to capture the outcomes of these types of interventions will also help to develop our conceptualisation of these cognitive factors as ‘risk factors’ or as ‘causal risk factors’ for these presentations in children and adolescents. As outlined by Kraemer et al., (1997), if altering a risk factor changes the outcome, i.e. the severity of symptoms, we can have greater confidence that it has a causal influence in the development of the disorder.

## 6.4 Overall conclusions

The process of conducting the comprehensive literature reviews, data synthesis, meta-analysis and the empirical study lead to clinically and theoretically helpful additions to the current understanding of PTSD and related disorders in children and adolescents. In particular, the focus of the findings has been upon understanding risk factors for PTSD, but has also informed our understanding of the new diagnostic category of Complex PTSD, and similarities and differences in risk factors for the development of depression and anxiety. The review provided an insight into areas of need for the development and standardisation of measures of peritraumatic experiences, and encouraged the further consideration of these factors in future research. Strong support for cognitive models of PTSD was drawn from both the review and the empirical study, however, it was also highlighted how factors of this model may not be disorder specific and may have relevance to the development of depression and anxiety. However, these conclusions must be taken with caution and clear recommendation for further research required to develop the validity of the results.

## 6.5 Chapter 6 References

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## 7. Appendices

**Appendix A.1.** Author guidelines for manuscript preparation for submission to Clinical Psychology Review Journal. A summary of the relevant manuscript preparation instructions, related to the manuscript style, formatting and referencing requirements is provided below, copied from the journal website. For full author guidelines, see:

<https://www.elsevier.com/journals/clinical-psychology-review/0272-7358/guide-for-authors>

### “Article structure

Manuscripts should be prepared according to the guidelines set forth in the Publication Manual of the American Psychological Association (6th ed., 2009). Of note, section headings should not be numbered.

Manuscripts should ordinarily not exceed 50 pages, *including* references and tabular material. Exceptions may be made with prior approval of the Editor in Chief. Manuscript length can often be managed through the judicious use of appendices. In general the References section should be limited to citations actually discussed in the text. References to articles solely included in meta-analyses should be included in an appendix, which will appear in the on line version of the paper but not in the print copy. Similarly, extensive Tables describing study characteristics, containing material published elsewhere, or presenting formulas and other technical material should also be included in an appendix. Authors can direct readers to the appendices in appropriate places in the text.

It is authors' responsibility to ensure their reviews are comprehensive and as up to date as possible (at least through the prior calendar year) so the data are still current at the time of publication. Authors are referred to the PRISMA Guidelines (<http://www.prisma-statement.org/statement.htm>) for guidance in conducting reviews and preparing manuscripts. Adherence to the Guidelines is not required, but is recommended to enhance quality of submissions and impact of published papers on the field.

### *Appendices*

If there is more than one appendix, they should be identified as A, B, etc. Formulae and equations in appendices should be given separate numbering: Eq. (A.1), Eq. (A.2), etc.; in a subsequent appendix, Eq. (B.1) and so on. Similarly for tables and figures: Table A.1; Fig. A.1, etc.

### **Essential title page information**

*Title.* Concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations and formulae where possible. **Note: The title page should be the first page of the manuscript document indicating the author's names and affiliations and the corresponding author's complete contact information.**

*Author names and affiliations.* Where the family name may be ambiguous (e.g., a double name), please indicate this clearly. Present the authors' affiliation addresses (where the actual work was done) below the names. Indicate all affiliations with a lower-case superscript letter immediately after the author's name and in front of the appropriate address. Provide the full postal address of each affiliation, including the country name, and, if available, the e-mail address of each author within the cover letter.

*Corresponding author.* Clearly indicate who is willing to handle correspondence at all stages of refereeing and publication, also post-publication. **Ensure that telephone and fax numbers (with country and area code) are provided in addition to the e-mail address and the complete postal address.**

### ***Abstract***

A concise and factual abstract is required (not exceeding 200 words). This should be typed on a separate page following the title page. The abstract should state briefly the purpose of the research, the principal results and major conclusions. An abstract is often presented separate from the article, so it must be able to stand alone. References should therefore be avoided, but if essential, they must be cited in full, without reference to the reference list.”

### ***Highlights***

Highlights are mandatory for this journal. They consist of a short collection of bullet points that convey the core findings of the article and should be submitted in a separate editable file in the online submission system. Please use 'Highlights' in the file name and include 3 to 5 bullet points (maximum 85 characters, including spaces, per bullet point). You can view [example Highlights](#) on our information site.

### **Keywords**

Immediately after the abstract, provide a maximum of 6 keywords, using American spelling and avoiding general and plural terms and multiple concepts (avoid, for example, 'and', 'of'). Be sparing with abbreviations: only abbreviations firmly established in the field may be eligible. These keywords will be used for indexing purposes.

### **Tables**

Please submit tables as editable text and not as images. Tables can be placed either next to the relevant text in the article, or on separate page(s) at the end. Number tables consecutively in accordance with their appearance in the text and place any table notes below the table body. Be sparing in the use of tables and ensure that the data presented in them do not duplicate results described elsewhere in the article. Please avoid using vertical rules and shading in table cells.

### ***References***

Citations in the text should follow the referencing style used by the American Psychological Association. You are referred to the Publication Manual of the American Psychological Association, Sixth Edition, ISBN 1-4338-0559-6, copies of which may be ordered from <http://books.apa.org/books.cfm?id=4200067> or APA Order Dept., P.O.B. 2710, Hyattsville, MD 20784, USA or APA, 3 Henrietta Street, London, WC3E 8LU, UK. Details concerning this referencing style can also be found at <http://humanities.byu.edu/linguistics/Henrichsen/APA/APA01.html>

### ***Citation in text***

Please ensure that every reference cited in the text is also present in the reference list (and vice versa). Any references cited in the abstract must be given in full. Unpublished results and personal communications are not recommended in the reference list, but may be mentioned in the text. If these references are included in the reference list they should follow the standard reference style of the journal and should include a substitution of the publication date with either 'Unpublished results' or 'Personal communication'. Citation of a reference as 'in press' implies that the item has been accepted for publication.

### ***Web references***

As a minimum, the full URL should be given and the date when the reference was last accessed. Any further information, if known (DOI, author names, dates, reference to a source publication, etc.), should also be given. Web references can be listed separately (e.g., after the reference list) under a different heading if desired, or can be included in the reference list.

### ***Reference management software***

Most Elsevier journals have their reference template available in many of the most popular reference management software products. These include all products that support [Citation Style Language styles](#), such as [Mendeley](#) and [Zotero](#), as well as [EndNote](#). Using the word processor plug-ins from these products, authors only need to select the appropriate journal template when preparing their article, after which citations and bibliographies will be automatically formatted in the journal's style. If no template is yet available for this journal, please follow the format of the sample references and citations as shown in this Guide.

Users of Mendeley Desktop can easily install the reference style for this journal by clicking the following link:

<http://open.mendeley.com/use-citation-style/clinical-psychology-review>

When preparing your manuscript, you will then be able to select this style using the Mendeley plug-ins for Microsoft Word or LibreOffice.

### **Reference style**

References should be arranged first alphabetically and then further sorted chronologically if necessary. More than one reference from the same author(s) in the same year must be identified by the letters "a", "b", "c", etc., placed after the year of publication. **References should be formatted with a hanging indent (i.e., the first line of each reference is flush left while the subsequent lines are indented).**

*Examples:* Reference to a journal publication: Van der Geer, J., Hanraads, J. A. J., & Lupton R. A. (2000). The art of writing a scientific article. *Journal of Scientific Communications*, 163, 51-59.

Reference to a book: Strunk, W., Jr., & White, E. B. (1979). *The elements of style*. (3rd ed.). New York: Macmillan, (Chapter 4).

Reference to a chapter in an edited book: Mettam, G. R., & Adams, L. B. (1994). How to prepare an electronic version of your article. In B.S. Jones, & R. Z. Smith (Eds.), *Introduction to the electronic age* (pp. 281-304). New York: E-Publishing Inc.

[dataset] Oguro, M., Imahiro, S., Saito, S., Nakashizuka, T. (2015). *Mortality data for Japanese oak wilt disease and surrounding forest compositions*. Mendeley Data, v1. <http://dx.doi.org/10.17632/xwj98nb39r.1>

### **Supplementary material**

Supplementary material such as applications, images and sound clips, can be published with your article to enhance it. Submitted supplementary items are published exactly as they are received (Excel or PowerPoint files will appear as such online). Please submit your material together with the article and supply a concise, descriptive caption for each supplementary file. If you wish to make changes to supplementary material during any stage of the process, please make sure to provide an updated file. Do not annotate any corrections on a previous version. Please switch off the 'Track Changes' option in Microsoft Office files as these will appear in the published version.”

(All author guidelines directly copied from the Clinical Psychology Review journal website: <https://www.elsevier.com/journals/clinical-psychology-review/0272-7358/guide-for-authors> )

**Appendix A.2.** Supplemental material for submission to Clinical Psychology Review

**Highlights** (3-5 bullet points to convey the core findings; max 85 characters including spacing per bullet point; submit in a separate editable file in online submission)

- Peritraumatic fear and perceived threat to life increase risk for PTSD in children.
- Data-driven processing is implicated as a risk factor, but requires more research.
- Peritraumatic dissociation may not be as powerful as a risk factor for PTSD.
- Female gender may moderate effect sizes for peritraumatic risk factors.
- Effect sizes also vary by time between trauma and assessment of risk factors.

### Appendix A.3

Table 1. Characteristics of studies included in the meta-analyses.

Article	Peri-traumatic risk factors assessed	Trauma type	N	Age range	Mean age (SD)	% female	Country of trauma	Study type	Time between trauma & initial assessment	Time between assessment of peritraumatic factor and PTSD assessment	PTSD measure	Interview or self-report questionnaire
<b>Aaron, Zaglul, and Emery (1999)</b>	Fear	Acute physical injury (RTA, physical assault or other accidental injury)	40	8-17	13.6 (2.9)	52.5%	US	Cross-sectional	4 weeks	0	The UCLA PTSD Reaction Index (PTSD-RI)	Self-report
<b>Bödvarsdóttir, Elklit, and Gudmundsdóttir (2006)</b>	Fear of dying; Terror; helplessness	Natural disaster: earthquake	140	10-15	12.2 (1.6)	55%	Iceland	Cross-sectional	3 months	0	The Post-traumatic Stress Reaction Index for Children (CPTS-RI)	Self-report
<b>Brown, et al. (2016)</b>	Dissociation	Acute physical injury (burns and other accidental injuries)	204	7-18	13.5 (3.5)	25.7%	US	Prospective longitudinal (follow-up at 3, 6, 12 and 18 months)	<1 week	12 weeks	The Diagnostic Interview for PTSD in Children & Adolescents (DICA-PTSD)	Interview
<b>Bui, et al. (2011)</b>	Distress	RTA	133	8-15	11.7 (2.2)	43.6%	France	Prospective longitudinal	<1 week	5 weeks	CPTS-RI	Self-report

								(follow-up at 5 weeks)				
<b>Cénat and Derivois (2015)</b>	Distress	Natural disaster (earthquake)	872	7-17	14.9 (1.9)	56.3%	Haiti	Cross-sectional	30 months	0	Impact of Events Scale (IES-R)	Self-report
<b>Duffy, et al. (2015)</b>	Perceived life threat	Terror attack	2095	14-18	15.9 (1.2)	52.3%	Ireland	Cross-sectional	15 months	0	Post-traumatic Diagnosis Scale	Self-report
<b>Ehlers, Mayou, and Bryant (2003)</b>	Data-driven processing; perceived life threat; fear	RTA	81	5-16	12.3 (2.9)	45%	UK	Prospective longitudinal (follow-up at 3 & 6 months)	2 weeks	10 weeks	IES-R	Self-report
<b>Elklit and Kurdahl (2013)</b>	PTSD A2 criteria: fear, helplessness, horror, and perceived life threat	Witnessing a homicide	320	16-20	17.9 (1.1)	62.2%	Denmark	Cross-sectional	7 months	0	Harvard Trauma Questionnaire	Self-report
<b>Evans and Oehler-Stinnett (2006)</b>	Fear	Natural disaster (tornado)	152	6-12	9.5	51.3%	US	Cross-sectional	12 months	0	OSU PTSD Scale-CF (developed by the authors, including items from CPTSD-RI, IES and CPSS)	Self-report
<b>Filkuková, et al. (2016)</b>	Fear	Terror attack	296	13-26	18.4	48.6%	Norway	Cross-sectional	4-5 months	0	PTSD-RI	Self-report
<b>Giannopoulou, et al. (2006)</b>	Perceived life threat; distress	Natural disaster (earthquake)	2037	9-17	12.9	48.7%	Greece	Cross-sectional	6-7 months	0	Children's IES-R (CRIES-13)	Self-report

<b>Holmes, et al. (2007)</b>	Fear; perceived life threat; helplessness; derealisation	Witnessing a terror attack (911 on TV)	76	10-11		51.3%	UK	Prospective longitudinal (follow-up at 6 months)	2 months	16 weeks	CPSS	Self-report
<b>Kar, et al. (2007)</b>	Fear of life threat	Natural disaster (cyclone)	447	7-17	12.9 (1.8)	50.7%	India	Cross-sectional	12 months	0	Semi-structured psychiatric interview	Interview and self-report
<b>Lack and Sullivan (2007)</b>	Fear	Natural disaster (tornado)	102	8-12	10.4 (1.2)	52.7%	US	Cross-sectional	13 months	0	PTSD-RI	Self-report
<b>La Greca, Silverman, Vernberg, and Prinstein (1996)</b>	Perceived life threat	Natural disaster (hurricane)	442	(US grade s 3-5)		57.6%	US	Prospective longitudinal (follow-up at 7 & 10 months)	3 months	16 weeks	PTSD-RI	Self-report
<b>Lavi, Green, and Dekel (2013)</b>	Fear	War	2314	12-15	13.5 (0.7)	51.6%	Lebanon	Cross-sectional	8-10 months	0	CPTS-RI	Self-report
<b>Marsac, et al. (2017)</b>	Shock/horror, helplessness, fear, perceived life threat	Acute physical injury (accidental: RTA or other accident)	96	8-13	10.6 (1.7)	35.4%	US	Prospective longitudinal (follow-up at 6 and 12 weeks)	2 weeks	10	CPSS	Self-report
<b>McDermott, Lee, Judd, and Gibbon (2005)</b>	Perceived threat to life	Natural disaster (wildfire)	222	8-18	12.5 (2.5)	54.9%	Canada	Cross-sectional	6 months	0	PTSD-RI	Self-report
<b>McDermott Sales, Fivush, Parker, and Bahrck (2005)</b>	Stress (scared/upset/frightened)	Natural disaster (hurricane)	35	3-4	4.25 (0.6)	40%	US	Prospective longitudinal (follow-up at 6 years)	2-5 months	approx. 300 weeks	Child PTSD-RI	Self-report
<b>Meiser-Stedman, Dalgleish, Glucksman,</b>	Subjective severity of threat	RTA or physical assault	59	10-16	14 (1.9)	45.8%	UK	Prospective longitudinal	2-4 weeks	20-22 weeks	ADIS-C	Interview

<b>Yule, and Smith (2009)</b>	(perceived life threat, threat of harm and scared)							(follow-up at 6 months)				
<b>Nordanger, et al. (2014)</b>	Perceived life threat	Terror attack	9186	17-19	16.9 (0.9)	53%	Norway	Cross-sectional	7 months	0	3 items from the UCLA-PTSD-RI	Self-report
<b>Pfefferbaum, et al. (2003)</b>	Peritraumatic 'reaction'	Terror attack	793	9-17	11.43 (1.5)	57%	Kenya	Cross-sectional	8-14 months	0	PTSS	Self-report
<b>Pfefferbaum, et al. (2002)</b>	Peritraumatic 'reaction'	Terror attack	2381	(US grade 6-8)		56%	US	Cross-sectional	7 weeks	0	PTSS	Self-report
<b>Polusny, et al. (2011)</b>	Perceived life threat	Natural disaster (tornado)	394	12-19	15.3 (1.8)	59%	US	Cross-sectional	6 months	0	IES-R	Self-report
<b>Schäfer, Barkmann, Riedesser, and Schulte-Markwort (2004)</b>	Dissociation	RTA	45	8-18	13 (3.2)	44%	Germany	Prospective longitudinal (follow-up at 3 months)	1 week	11 weeks	IES-R	Self-report
<b>Solomon and Lavi (2005)</b>	Perceived life threat/danger	War/terror	740	11-15		49-54%	Israel	Cross-sectional	unclear (maximum 10 months)	0	CPTSD-RI	Self-report
<b>Stallard, Velleman, and Baldwin (1998)</b>	Perceived life threat	RTA	119	5-18		43%	UK	Cross-sectional	22-79 days (mean 40)	0	CAPS-C	Interview
<b>Stallard and Smith (2007)</b>	Data-driven processing; perceived harm and life threat, how frightened/scared	RTA	75	7-18	14 (3.4)	50.7%	UK	Cross-sectional	8 months	0	CAPS-C	Interview
<b>Thienkrua, et al. (2006)</b>	Perceived life threat; feeling helpless	Natural disaster (tsunami)	371	7-14	10.4	54%	Thailand	Prospective longitudinal	2 months	28 weeks	PTSD-RI	Self-report

	(unable to escape); panic or fear							(follow-up at 9 months)				
<b>Winston, Kassam-Adams, García-España, Ittenbach, and Cnaan (2003)</b>	Perceived life threat; fear	RTA	269	8-17	11.4 (2.6)	23%	US	Prospective longitudinal (follow-up at 3 months)	1 month	8 weeks	CAPS-CA	Interview
<b>Zatzick, et al. (2006)</b>	Dissociation	Acute injury (assault/RTA)	108	12-18	15.9 (1.9)	32%	US	Prospective longitudinal (follow-up at 2, 5 & 12 months)	<3 weeks	5 weeks	PTSD-RI	Self-report
<b>Zhou, Zhang, Wei, Liu, and Hannak (2016)</b>	Fear	Natural disaster (earthquake)	197		13.2 (1.6)	53.3%	China	Prospective longitudinal (follow-up at 2, 6 & 12 months)	2 weeks	6 weeks	PTSD-RI	Self-report

*References for studies included in the meta-analysis and summarised in Table 1, but not mentioned within the article main text, appear in the*

*Supplementary Reference list in Appendix A.4.*

**Appendix A.4.** References for studies included in the meta-analyses but not cited within the article text.

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**Appendix B.1.** Author guidelines for manuscript preparation for submission to Journal of Consulting and Clinical Psychology. Details of relevant manuscript preparation instructions related to the format, style and referencing requirements are copied from the journal website. For full details see: <http://www.apa.org/pubs/journals/ccp/?tab=4>

## Length and Style of Manuscripts

Full-length manuscripts should not exceed 35 pages total (including cover page, abstract, text, references, tables, and figures), with margins of at least 1 inch on all sides and a standard font (e.g., Times New Roman) of 12 points (no smaller). The entire paper (text, references, tables, etc.) must be double spaced.

Instructions on preparing tables, figures, references, metrics, and abstracts appear in the *Publication Manual of the American Psychological Association* (6th edition).

Authors submitting manuscripts that report new data collection, especially randomized clinical trials (RCTs), should comply with the newly developed *APA Journal Article Reporting Standards* (PDF, 98KB) (JARS; see *American Psychologist*, 2008, 63, 839–851 or Appendix in the *APA Publication Manual*).

For papers that exceed 35 pages, authors must justify the extended length in their cover letter (e.g., reporting of multiple studies), and in no case should the paper exceed 45 pages total. Papers that do not conform to these guidelines may be returned without review.

The References section should immediately follow a page break.

## Title of Manuscript

The title of a manuscript should be accurate, fully explanatory, and preferably no longer than 12 words. The title should reflect the content and population studied (e.g., "treatment of generalized anxiety disorders in adults").

If the paper reports a randomized clinical trial (RCT), this should be indicated in the title. Note that JARS criteria must be used for reporting purposes.

## Abstract and Keywords

All manuscripts must include an abstract containing a maximum of 250 words typed on a separate page. After the abstract, please supply up to five keywords or brief phrases.

Manuscripts published in the *Journal of Consulting and Clinical Psychology* will include a structured abstract of up to 250 words.

For studies that report randomized clinical trials or meta-analyses, the abstract also must be consistent with the guidelines set forth by JARS or MARS (Meta-Analysis Reporting Standards) guidelines, respectively. Thus, in preparing a manuscript, please ensure that it is consistent with the guidelines stated below.

Please include an Abstract of up to 250 words, presented in paragraph form. The Abstract should be typed on a separate page (page 2 of the manuscript), and must include each of the following sections:

- **Objective:** A brief statement of the purpose of the study
- **Method:** A detailed summary of the participants (N, age, gender, ethnicity) as well as descriptions of the study design, measures (including names of measures), and procedures
- **Results:** A detailed summary of the primary findings that clearly articulate comparison groups (if relevant), and that indicate significance or confidence intervals for the main findings
- **Conclusions:** A description of the research and clinical implications of the findings

## Public Health Significance Statements

Authors submitting manuscripts to the *Journal of Consulting and Clinical Psychology* are required to provide 2–3 brief sentences regarding the public health significance of the study or meta-analysis described in their paper. This description should be included within the manuscript on the abstract/keywords page. It should be written in language that is easily understood by both professionals and members of the lay public.

When an accepted paper is published, these sentences will be boxed beneath the abstract for easy accessibility. All such descriptions will also be published as part of the Table of Contents, as well as on the journal's web page. This new policy is in keeping with efforts to increase dissemination and usage by larger and diverse audiences.

Examples of these 2–3 sentences include the following:

- "This study strongly suggests that (description of a given psychosocial treatment) is an effective treatment for anxiety, but only if it is of mild to moderate severity. For persons with severe anxiety, additional treatments may be necessary."
- "When treating individuals of (name of a particular ethnic minority group) who are experiencing PTSD, this study demonstrated the importance of taking into account cultural factors, especially those that involve one's spiritual beliefs."
- "This study highlights the importance of directly including one's family in treatment when helping adults diagnosed with cancer overcome their depression."

To be maximally useful, these statements of public health significance should not simply be sentences lifted directly out of the manuscript.

They are meant to be informative and useful to any reader. They should provide a bottom-line, take-home message that is accurate and easily understood. In addition, they should be able to be translated into media-appropriate statements for use in press releases and on social media.

Prior to final acceptance and publication, all public health significance statements will be carefully reviewed to make sure they meet these standards. Authors will be expected to revise statements as necessary.

## Participants: Description and Informed Consent

The Method section of each empirical report must contain a detailed description of the study participants, including (but not limited to) the following: age, gender, ethnicity, SES, clinical diagnoses and comorbidities (as appropriate), and any other relevant demographics.

In the Discussion section of the manuscript, authors should discuss the diversity of their study samples and the generalizability of their findings.

The Method section also must include a statement describing how informed consent was obtained from the participants (or their parents/guardians) and indicate that the study was conducted in compliance with an appropriate Internal Review Board.

## Measures

The Method section of empirical reports must contain a sufficiently detailed description of the measures used so that the reader understands the item content, scoring procedures, and total scores or subscales. Evidence of reliability and validity with similar populations should be provided.

## Statistical Reporting of Clinical Significance

*JCCP* requires the statistical reporting of measures that convey clinical significance. Authors should report means and standard deviations for all continuous study variables and the effect sizes for the primary study findings. (If effect sizes are not available for a particular test, authors should convey this in their cover letter at the time of submission.)

*JCCP* also requires authors to report confidence intervals for any effect sizes involving principal outcomes (see Fidler et al., *Journal of Consulting and Clinical Psychology*, 2005, pp. 136–143 and Odgaard & Fowler, *Journal of Consulting and Clinical Psychology*, 2010, pp.287–297).

In addition, when reporting the results of interventions, authors should include indicators of clinically significant change. Authors may use one of several approaches that have been recommended for capturing clinical significance, including (but not limited to) the reliable change index (i.e., whether the amount of change displayed by a treated individual is large enough to be meaningful; see Jacobson et al., *Journal of Consulting and Clinical Psychology*, 1999), the extent to which dysfunctional individuals show movement into the functional distribution (see Jacobson & Truax, *Journal of Consulting and Clinical Psychology*, 1991), or other normative comparisons (see Kendall et al., *Journal of Consulting and Clinical Psychology*, 1999).

The special section of *JCCP* on "Clinical Significance" (*Journal of Consulting and Clinical Psychology*, 1999, pp. 283–339) contains detailed discussions of clinical significance and its measurement and should be a useful resource (see also Atkins et al., *Journal of Consulting and Clinical Psychology*, 2005, pp. 982–989).

## Discussion of Clinical Implications

Articles must include a discussion of the clinical implications of the study findings or analytic review. The Discussion section should contain a clear statement of the extent of clinical application of the current assessment, prevention, or treatment methods. The extent of application to clinical practice may range from suggestions that the data are too preliminary to support widespread dissemination to descriptions of existing manuals available from the authors or archived materials that would allow full implementation at present.

## Manuscript Preparation

Prepare manuscripts according to the *Publication Manual of the American Psychological Association* (6<sup>th</sup> edition). Manuscripts may be copyedited for bias-free language (see Chapter 3 of the *Publication Manual*).

Review APA's [Checklist for Manuscript Submission](#) before submitting your article.

Double-space all copy. Other formatting instructions, as well as instructions on preparing tables, figures, references, metrics, and abstracts, appear in the *Manual*. Additional guidance on APA Style is available on the [APA Style website](#).

## References

List references in alphabetical order. Each listed reference should be cited in text, and each text citation should be listed in the References section.

Examples of basic reference formats:

▪ **Journal Article:**

Hughes, G., Desantis, A., & Waszak, F. (2013). Mechanisms of intentional binding and sensory attenuation: The role of temporal prediction, temporal control, identity prediction, and motor prediction. *Psychological Bulletin*, 139, 133–151. <http://dx.doi.org/10.1037/a0028566>

▪ **Authored Book:**

Rogers, T. T., & McClelland, J. L. (2004). *Semantic cognition: A parallel distributed processing approach*. Cambridge, MA: MIT Press.

▪ **Chapter in an Edited Book:**

Gill, M. J., & Sypher, B. D. (2009). Workplace incivility and organizational trust. In P. Lutgen-Sandvik & B. D. Sypher (Eds.), *Destructive organizational communication: Processes, consequences, and constructive ways of organizing* (pp. 53–73). New York, NY: Taylor & Francis.

### **Appendix B.2.** Information regarding author (Trainee Psychologist) and ASPECTS study team role in data collection and analysis

The ASPECTS study team consisted of Research Assistants and post-doctoral Researchers, and was led by Dr Richard Meiser-Stedman (thesis supervisor) who was the ASPECTS research coordinator and to whom the research grant for this project was awarded. Dr Meiser-Stedman and the ASPECTS study team developed the original study protocol, recruited participants and collected the raw study data in 2010-2013. I was granted honorary study team membership and access to the anonymised raw dataset for the development of this current research study in 2016. I screened and processed the raw data to source data relevant for the current research questions; this included

identifying appropriate items and measures for each predictor and outcome variable, considering the purpose and nature of measures used and data gathered. For example, this included computing measures of PTSD and CPTSD in accordance with ICD-11 diagnostic criteria by identifying items and measures within the ASPECTS study data corpus, recoding data as needed, and generating continuous and diagnostic (categorical) variables to reflect participants' scores within these new variables. I also then completed all data analysis using this ASPECTS study data as presented in this thesis portfolio.

**Appendix B.3.** Supplemental materials to be submitted with the empirical paper to JCCP

Table 10. Linear regression model statistics for predictors of PTSD

Model	Predictor	Coefficient	$\beta$	Bootstrapped SE	z	p	lower 95%CI	upper 95%CI
PSYCHOSOCIAL FACTORS AND STRESSORS	Age	-0.102	-0.052	0.145	-0.70	0.481	-0.387	0.182
	<b>Female gender</b>	<b>1.173</b>	<b>0.144</b>	<b>0.863</b>	<b>1.98</b>	<b>0.047</b>	<b>0.021</b>	<b>3.405</b>
	Mother's education	0.745	0.062	0.819	0.91	0.363	-0.862	2.352
	Prior traumas	0.287	0.049	0.446	0.64	0.520	-0.587	1.162
	Prior life stressors	0.229	0.045	0.452	0.51	0.613	-0.658	1.115
	<b>Interpersonal index trauma</b>	<b>4.242</b>	<b>0.268</b>	<b>1.561</b>	<b>2.72</b>	<b>0.007</b>	<b>1.182</b>	<b>7.302</b>
	Prior wellbeing concerns	0.687	0.050	1.192	0.58	0.565	-1.649	3.023
	Perceived social support	-0.015	-0.033	0.032	-0.48	0.629	-0.078	0.047
COGNITIVE	<b>Dissociation (ongoing)</b>	<b>0.426</b>	<b>0.167</b>	<b>0.212</b>	<b>2.01</b>	<b>0.044</b>	<b>0.011</b>	<b>0.841</b>
	Data-driven processing	0.106	0.109	0.059	1.77	0.077	-0.011	0.224
	<b>Trauma memory quality</b>	<b>0.175</b>	<b>0.203</b>	<b>0.065</b>	<b>2.7</b>	<b>0.007</b>	<b>0.048</b>	<b>0.302</b>
	<b>Trauma appraisals</b>	<b>0.136</b>	<b>0.329</b>	<b>0.044</b>	<b>3.12</b>	<b>0.002</b>	<b>0.051</b>	<b>0.221</b>
	Rumination	0.257	0.124	0.138	1.86	0.062	-0.013	0.527
	<b>Self-blame</b>	<b>-0.245</b>	<b>-0.085</b>	<b>0.127</b>	<b>-1.93</b>	<b>0.054</b>	<b>-0.493</b>	<b>0.004</b>
SUBJECTIVE EVENT SEVERITY	<b>Peri-traumatic panic</b>	<b>0.916</b>	<b>0.373</b>	<b>0.195</b>	<b>4.7</b>	<b>0.000</b>	<b>0.534</b>	<b>1.298</b>
	Perceived life threat	0.187	0.034	0.453	0.41	0.680	-0.700	1.074
	Perceived harm	-0.338	-0.058	0.378	-0.89	0.372	-1.079	0.404
	<b>Felt scared</b>	<b>0.842</b>	<b>0.150</b>	<b>0.349</b>	<b>2.41</b>	<b>0.016</b>	<b>0.157</b>	<b>1.526</b>
	<b>Peri-traumatic dissociation</b>	<b>0.439</b>	<b>0.231</b>	<b>0.128</b>	<b>3.42</b>	<b>0.001</b>	<b>0.187</b>	<b>0.689</b>
OBJECTIVE EVENT SEVERITY	<b>Peri-traumatic pain</b>	<b>1.174</b>	<b>0.220</b>	<b>0.376</b>	<b>3.12</b>	<b>0.002</b>	<b>0.437</b>	<b>1.909</b>
	Admitted to hospital	-1.329	-0.102	1.011	-1.31	0.189	-3.312	0.653
	Head injury	0.824	0.068	0.985	0.84	0.403	-1.107	2.756
	Number of injuries sustained	-0.068	-0.010	0.510	-0.13	0.894	-1.068	0.932
	Given opiates in ED	-0.141	-0.016	1.209	-0.12	0.907	-2.511	2.229

Table 11. Linear regression model statistics for predictors of Complex PTSD

Model	Predictor	Coefficient	$\beta$	Bootstrapped SE	z	p	lower 95%CI	upper 95%CI
PSYCHOSOCIAL FACTORS AND STRESSORS	Age	0.033	0.026	0.084	0.39	0.695	-0.132	0.198
	Female gender	0.908	0.116	0.537	1.69	0.091	-0.145	1.961
	Mother's education	0.025	0.003	0.518	0.05	0.962	-0.991	1.040
	<b>Prior traumas</b>	<b>0.569</b>	<b>0.147</b>	<b>0.296</b>	<b>1.92</b>	<b>0.055</b>	<b>-0.012</b>	<b>1.149</b>
	Prior life stressors	-0.108	-0.033	0.278	-0.39	0.698	-0.653	0.437
	<b>Interpersonal index trauma</b>	<b>2.951</b>	<b>0.285</b>	<b>1.039</b>	<b>2.84</b>	<b>0.005</b>	<b>0.914</b>	<b>4.988</b>
	Prior wellbeing concerns	0.758	0.084	0.750	1.01	0.312	-0.712	2.228
	<b>Perceived social support</b>	<b>-0.050</b>	<b>-0.167</b>	<b>0.024</b>	<b>-2.12</b>	<b>0.034</b>	<b>-0.097</b>	<b>-0.004</b>
COGNITIVE	<b>Dissociation (ongoing)</b>	<b>0.252</b>	<b>0.151</b>	<b>0.133</b>	<b>1.90</b>	<b>0.057</b>	<b>-0.008</b>	<b>0.512</b>
	Data-driven processing	-0.018	-0.028	0.032	-0.54	0.586	-0.081	0.046
	Trauma memory quality	-0.016	-0.028	0.036	-0.43	0.667	-0.087	0.055
	<b>Trauma appraisals</b>	<b>0.159</b>	<b>0.589</b>	<b>0.025</b>	<b>6.26</b>	<b>0.000</b>	<b>0.109</b>	<b>0.209</b>
	Rumination	0.073	0.054	0.069	1.04	0.297	-0.064	0.209
	<b>Self-blame</b>	<b>0.312</b>	<b>0.166</b>	<b>0.103</b>	<b>3.03</b>	<b>0.002</b>	<b>0.110</b>	<b>0.515</b>
SUBJECTIVE EVENT SEVERITY	<b>Peri-traumatic panic</b>	<b>0.627</b>	<b>0.389</b>	<b>0.139</b>	<b>4.49</b>	<b>0.000</b>	<b>0.353</b>	<b>0.900</b>
	Perceived life threat	0.112	0.031	0.257	0.44	0.663	-0.392	0.615
	Perceived harm	0.152	0.039	0.267	0.57	0.570	-0.372	0.675
	Felt scared	-0.169	-0.046	0.203	-0.83	0.405	-0.568	0.229
	<b>Peri-traumatic dissociation</b>	<b>0.248</b>	<b>0.199</b>	<b>0.098</b>	<b>2.52</b>	<b>0.012</b>	<b>0.055</b>	<b>0.441</b>
OBJECTIVE EVENT SEVERITY	<b>Peri-traumatic pain</b>	<b>0.684</b>	<b>0.196</b>	<b>0.239</b>	<b>2.86</b>	<b>0.004</b>	<b>0.216</b>	<b>1.153</b>
	Admitted to hospital	-0.895	-0.104	0.682	-1.31	0.189	-2.231	0.442
	Head injury	0.881	0.111	0.556	1.59	0.113	-0.208	1.970
	Number of injuries sustained	0.149	0.034	0.321	0.47	0.640	-0.479	0.778
	Given opiates in ED	-0.452	-0.045	0.719	-0.63	0.530	-1.861	0.957

Table 12. Linear regression model statistics for predictors of Depression

Model	Predictor	Coefficient	$\beta$	Bootstrapped SE	z	p	lower 95%CI	upper 95%CI
PSYCHOSOCIAL FACTORS AND STRESSORS	Age	0.016	0.009	0.118	0.13	0.894	-0.216	0.247
	<b>Female gender</b>	<b>2.268</b>	<b>0.206</b>	<b>0.786</b>	<b>2.89</b>	<b>0.004</b>	<b>0.728</b>	<b>3.809</b>
	Mother's education	0.557	0.051	0.776	0.72	0.473	-0.965	2.078
	<b>Prior traumas</b>	<b>0.862</b>	<b>0.159</b>	<b>0.419</b>	<b>2.06</b>	<b>0.040</b>	<b>0.040</b>	<b>1.683</b>
	Prior life stressors	-0.123	-0.026	0.368	-0.33	0.738	-0.844	0.597
	<b>Interpersonal index trauma</b>	<b>4.111</b>	<b>0.281</b>	<b>1.429</b>	<b>2.88</b>	<b>0.004</b>	<b>1.311</b>	<b>6.911</b>
	Prior wellbeing concerns	0.171	0.014	1.013	0.17	0.866	-1.815	2.157
	<b>Perceived social support</b>	<b>-0.086</b>	<b>-0.203</b>	<b>0.035</b>	<b>-2.49</b>	<b>0.013</b>	<b>1.152</b>	<b>-0.018</b>
COGNITIVE	Dissociation (ongoing)	0.088	0.037	0.165	0.53	0.593	-0.235	0.411
	Data-driven processing	0.039	0.043	0.046	0.84	0.402	-0.052	0.129
	Trauma memory quality	-0.032	-0.039	0.052	-0.61	0.540	-0.133	0.069
	<b>Trauma appraisals</b>	<b>0.258</b>	<b>0.677</b>	<b>0.028</b>	<b>9.07</b>	<b>0.000</b>	<b>0.205</b>	<b>0.314</b>
	Rumination	0.135	0.071	0.115	1.17	0.242	-0.091	0.361
	Self-blame	0.139	0.053	0.143	0.98	0.327	-0.139	0.419
SUBJECTIVE EVENT SEVERITY	<b>Peri-traumatic panic</b>	<b>0.686</b>	<b>0.302</b>	<b>0.184</b>	<b>3.73</b>	<b>0.000</b>	<b>0.326</b>	<b>1.047</b>
	Perceived life threat	0.624	0.123	0.369	1.69	0.090	-0.098	1.347
	Perceived harm	-0.159	-0.029	0.343	-0.46	0.643	-0.832	0.513
	Felt scared	0.022	0.004	0.309	0.07	0.945	-0.586	0.628
	<b>Peri-traumatic dissociation</b>	<b>0.402</b>	<b>0.229</b>	<b>0.129</b>	<b>3.10</b>	<b>0.002</b>	<b>0.148</b>	<b>0.656</b>
OBJECTIVE EVENT SEVERITY	<b>Peri-traumatic pain</b>	<b>1.328</b>	<b>0.269</b>	<b>0.354</b>	<b>3.75</b>	<b>0.000</b>	<b>0.633</b>	<b>2.023</b>
	Admitted to hospital	-1.619	-0.134	0.968	-1.67	0.094	-3.518	0.279
	<b>Head injury</b>	<b>1.926</b>	<b>0.172</b>	<b>0.744</b>	<b>2.59</b>	<b>0.010</b>	<b>0.466</b>	<b>3.385</b>
	Number of injuries sustained	-0.410	-0.067	0.486	-0.84	0.398	-1.362	0.541
	Given opiates in ED	-0.940	-0.066	1.045	-0.90	0.368	-2.988	1.107

Table 13. Linear regression model statistics for predictors of GAD

Model	Predictor	Coefficient	$\beta$	Bootstrapped SE	z	p	lower 95%CI	upper 95%CI
PSYCHOSOCIAL FACTORS AND STRESSORS	Age	0.213	0.069	0.208	1.02	0.307	-0.195	0.622
	<b>Female gender</b>	<b>4.662</b>	<b>0.252</b>	<b>1.492</b>	<b>3.13</b>	<b>0.002</b>	<b>1.739</b>	<b>7.586</b>
	Mother's education	2.103	0.114	1.332	1.58	0.114	-0.507	4.714
	Prior traumas	0.995	0.109	0.698	1.42	0.154	0.374	2.364
	Prior life stressors	0.665	0.085	0.798	0.83	0.405	-0.899	2.230
	<b>Interpersonal index trauma</b>	<b>6.576</b>	<b>0.268</b>	<b>2.926</b>	<b>2.25</b>	<b>0.025</b>	<b>0.841</b>	<b>12.312</b>
	Prior wellbeing concerns	0.685	0.032	1.781	0.38	0.701	-2.806	4.176
	Perceived social support	-0.057	-0.080	0.052	-1.11	0.269	-0.158	0.044
COGNITIVE	<b>Dissociation (ongoing)</b>	<b>0.704</b>	<b>0.178</b>	<b>0.363</b>	<b>1.94</b>	<b>0.053</b>	<b>-0.008</b>	<b>1.415</b>
	Data-driven processing	0.084	0.056	0.072	1.15	0.249	-0.058	0.225
	Trauma memory quality	0.031	0.023	0.099	0.31	0.754	-0.163	0.225
	<b>Trauma appraisals</b>	<b>0.338</b>	<b>0.528</b>	<b>0.072</b>	<b>4.68</b>	<b>0.000</b>	<b>0.197</b>	<b>0.480</b>
	<b>Rumination</b>	<b>0.381</b>	<b>0.119</b>	<b>0.207</b>	<b>1.84</b>	<b>0.065</b>	<b>-0.024</b>	<b>0.786</b>
	Self-blame	-0.194	-0.044	0.243	-0.8	0.424	-0.671	0.283
SUBJECTIVE EVENT SEVERITY	<b>Peri-traumatic panic</b>	<b>1.342</b>	<b>0.353</b>	<b>0.306</b>	<b>4.38</b>	<b>0.000</b>	<b>0.741</b>	<b>1.942</b>
	Perceived life threat	1.124	0.132	0.664	1.69	0.090	-0.177	2.425
	Perceived harm	-0.681	-0.075	0.545	-1.25	0.212	-1.749	0.388
	Felt scared	0.432	0.049	0.503	0.86	0.391	-0.554	1.418
	<b>Peri-traumatic dissociation</b>	<b>0.701</b>	<b>0.238</b>	<b>0.217</b>	<b>3.23</b>	<b>0.001</b>	<b>0.275</b>	<b>1.125</b>
OBJECTIVE EVENT SEVERITY	<b>Peri-traumatic pain</b>	<b>2.565</b>	<b>0.311</b>	<b>0.572</b>	<b>4.48</b>	<b>0.000</b>	<b>1.444</b>	<b>3.686</b>
	Admitted to hospital	-2.726	-0.134	1.601	-1.70	0.089	-5.863	0.411
	Head injury	2.785	0.149	1.557	1.79	0.074	-0.267	5.837
	<b>Number of injuries sustained</b>	<b>-1.334</b>	<b>-0.129</b>	<b>0.653</b>	<b>-2.04</b>	<b>0.041</b>	<b>-2.614</b>	<b>-0.054</b>
	Given opiates in ED	-1.407	-0.059	1.782	-0.79	0.430	-4.900	2.085

