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Meta-analysis of the association between autobiographical memory specificity and exposure to trauma and the role of trauma timing.

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Abstract

Cognitive models of emotional disorders suggest that reduced autobiographical memory specificity, resulting from exposure to traumatic events, may play an important role in the aetiology and maintenance of these disorders. However, there has yet to be a comprehensive meta-analysis of the association between trauma exposure and memory specificity, and the role of posttraumatic stress symptoms on this association.

PsycINFO and MEDLINE databases were searched and data were extracted from studies regarding the mean number, or proportion of, specific memories that were recalled between participants with and without trauma exposure in the Autobiographical Memory Test. Data on differences between groups in posttraumatic stress and depressive symptoms were also extracted, along with data on trauma timing and participants' ages at the time of assessment.

The effect size of memory specificity between participants with and without exposure to trauma was large (d = .77) and differed significantly from zero (p < .001). In meta-regression, trauma timing was a significant predictor of the heterogeneity in trauma-exposure specificity effect sizes but posttraumatic stress and depressive symptoms were not.

Compromised memory specificity represents an important cognitive consequence of trauma exposure that might have an important influence on risk for, and maintenance of, subsequent emotional pathologies.

Keywords: Trauma; Memory; Overgeneral; Depression; Meta-Analysis

There is a wealth of evidence, accumulated over more than two decades, regarding the association between exposure to traumatic events and the specificity of autobiographical memories. This accumulation has in part been motivated by cognitive models of emotional disorders suggesting that amongst people with, or at risk of, a range of disorders there is a tendency to recall autobiographical memories with little specificity and that this tendency may result from trauma exposure (Williams, 2006; Williams, et al., 2007). Although there are several reviews of this literature (Moore & Zoellner, 2007; Ono, Devilly, & Shum, 2016; Williams, et al., 2007) there has yet to be a comprehensive meta-analytical investigation of the role of trauma exposure in compromised autobiographical memory specificity that includes the breadth of available literature and which considers other potential influencing factors. Such an investigation will provide strong support for our understanding of the pathways through which traumatic events lead to patterns of autobiographical memory that characterise psychological problems.

Individual differences in memory specificity are typically measured using the Autobiographical Memory Test (AMT) (Williams & Broadbent, 1986). AMT participants are instructed to recall an event that they associate with each of several positive and negative cue words. *Specific* memories are those which have been personally experienced at a specific time and place and which lasted for a day or less (e.g., ‘my friend’s birthday party last month’). Individual differences in specificity are then typically quantified as the number of specific memories that are recalled as a proportion of the total number of cues or as a proportion of the number of positive or negative cues separately (Griffith, et al., 2012). Reduced recall of specific memories compared with healthy controls has been associated with the presence of major depressive disorder, other depressive disorders (e.g., postnatal depression), bipolar disorder, posttraumatic stress disorder (PTSD) and acute stress disorder, and eating disorders (Williams, et al., 2007). Longitudinal investigations suggest that reduced

specificity also precedes diagnoses and predicts the course of symptoms across time (Kleim & Ehlers, 2008; Sumner, Griffith, & Mineka, 2010).

Williams (2006) seminal CaRFAX model suggests three pathways by which reduced specificity occurs: capture and rumination, functional avoidance and impaired executive control. As others have noted (Griffith, et al., 2016) each pathway can be associated with coping following exposure to a trauma. For example, when one is trying to retrieve memories of relatively benign events they may activate the memory of a trauma, perhaps due to semantic associations (e.g., someone who has experienced sexual abuse may find that retrieval of other memories involving men evokes retrieval of memories regarding the abuse). Alternatively, if the trauma had a particularly significant effect on their view of themselves and their world, the person may activate more conceptual memories that are illustrative of these negative beliefs (e.g., attempts to retrieve memories of a social gathering may evoke retrieval of thoughts related to being unlovable). Capture and rumination on these thoughts can abort the retrieval of the other, non-trauma-related, memory and so only limited detail of this memory is retrieved. Also, a person exposed to trauma might avoid specific details of a trauma memory to limit the emotional experience that follows recall of these details – so called functional avoidance. This tendency might also generalise to other autobiographical memories given the semantic association between memories that are related or unrelated to trauma (Dalgleish, Rolfe, Golden, Dunn, & Barnard, 2008; Hermans, et al., 2008). As such a person might come to avoid the detail of all memories for fear that such detail may itself be negatively valenced or that retrieval of these details may evoke retrieval of further trauma memories. Williams et al (2007) suggest that these processes can be further exacerbated by dysfunctional executive control. For example, a person may have poor memory specificity because, when they are retrieving memories of events from their past, they are unable to inhibit distraction by other semantically-related, and perhaps more negative, thoughts. Also,

for those memories that they do retrieve they may be less able than people with strong executive control to hold all of the details of this memory in mind at the same time and so they only report a limited amount of detail instead. It is in this way that one expects that the specificity of memories retrieved within the AMT is expected to be compromised for people who have experienced trauma, compared with those who have not. This tendency to recall memories in this way is posited to be involved in both the emergence and maintenance of psychopathology by affecting a person's ability to problem solve and plan for the future on the basis of their past experiences, whilst also affecting how people regulate their emotions in the presence of significant life events (Williams et al., 2007).

In a previous evaluative review of literature where trauma exposure and specificity were explored, Moore and Zoellner (2007) concluded that exposure to trauma is not sufficient to compromise memory specificity. In their review of 24 studies they suggest that any association between trauma and memory specificity is likely to be explained by an association between posttraumatic stress symptoms and specificity. Put otherwise, they suggest that not everyone who is exposed to trauma will exhibit compromised memory specificity. Instead, reductions in specificity are suggested to be greatest amongst people who experience significant post-traumatic stress symptoms or those who are impacted most by the trauma. Moore and Zoellner (2007) also concluded that the presence of depression has a much greater impact on memory specificity than trauma exposure and that the timing of a trauma – in childhood versus adulthood – has no bearing on compromised specificity. This latter finding is of particular interest as one might expect that traumas which occur during sensitive periods of development when the brain is still highly plastic would have a greater impact on cognition than traumas which occur in adulthood (Nelson, 2000). Also, early findings from AMT studies with people exposed to childhood and adult traumas suggest that specificity is lowest for people with childhood trauma exposure (Stokes, Dritschel, &

Bekerian, 2004; Willebrand, et al., 2002). However, this review merely discussed the available literature and did not statistically analyse the size of any of the hypothesised effects.

More recently, Ono, et al. (2016) conducted a meta-analysis wherein they made several effect size comparisons for specificity scores between trauma exposed participants and controls, trauma exposed participants without PTSD and controls, trauma exposed participants with PTSD and controls, and participants with diagnoses of depression and controls. In contrast to the suggestions of Moore and Zoellner (2007), when pooling the data across nine studies exposure to trauma was associated with reduced specificity relative to controls without exposure (*Hedges' g* = .78) and this was particularly so when recalling memories cued by negative words (*Hedges' g* = .79) (Ono, et al., 2016). The strict inclusion criteria and analytical strategy used by Ono, et al. (2016), and in particular their plan to compare participants with and without diagnoses of PTSD or Depression, meant that in their 2013 search they did not sample the entire breadth of available literature regarding the association between trauma exposure and memory specificity. Instead they included fewer trauma-related studies (k = 9) than the original review by Moore and Zoellner (2007) a decade earlier (k = 24). In their analysis, they also excluded studies where participants had been exposed to trauma and who had depression, rather than including depressive symptoms within their analysis. They also only included studies with adult participants, again rather than statistically controlling for participant age within their analysis, and did not explore the possible effects of trauma timing. They nonetheless provide a robust categorical analysis of PTSD and depressive disorders and their association with memory specificity.

There has yet to be a broad meta-analysis, including all available studies comparing the memory specificity of participants with and without trauma exposure. Although Moore and Zoellner (2007) provided the most inclusive review of the available literature to-date, they only discussed this evidence and did not offer a statistical meta-analysis to explore the

size of the effect of trauma exposure on specificity. Ono, et al. (2016) provided a more recent meta-analysis, although as previously discussed, they did not include the breadth of available literature at the time of their search and the evidence base has also continued to grow since this time. The present meta-analysis sought to fill this gap by providing the most comprehensive analysis to-date of the size of the effect of trauma exposure on memory specificity. We also sought to clarify the role of potential moderators on this effect using meta-regression. In particular, we provide the first meta-analytical investigation of whether the *severity* of posttraumatic stress and depressive symptoms explain the variance between studies in their effect sizes for the relationship between trauma exposure and specificity. We also explore the extent to which trauma timing explains this between study variance. Although Moore and Zoellner (2007) provide preliminary evidence in this regard, there has yet to be a meta-analysis of the possible association between trauma timing and specificity.

In line with the effect sizes evident within Ono and colleagues' (2016) analysis, we hypothesised that in our larger sample of studies there would be moderate to large effects of trauma exposure on the number of specific autobiographical memories recalled. The AMT has been shown to have one factor in many studies (Griffith, et al., 2009; Griffith, et al., 2012; Heron, et al., 2012; Takano, Gutenbrunner, Martens, Salmon, & Raes, 2017). However, to explore whether the relationship found by Ono, et al. (2016) is replicable in a larger and updated meta-analysis, we explored the effects of trauma exposure when considering recall from positive and negative cues separately. However, given recent evidence that the AMT is best characterised as having a single factor rather than separate factors for positive and negative cues, we predicted that effect sizes for the association between trauma exposure and specificity would be similar for positive and negative cues when they are analysed separately. In our moderator analyses, given that psychological coping following trauma exposure is presumed to influence the effects of exposure on

specificity (Williams, et al., 2007) we expected that individual differences in posttraumatic stress and depressive symptoms would explain variability in the effect of trauma exposure on specificity. Finally, in line with the conclusions drawn elsewhere (Moore & Zoellner, 2007), we hypothesised that trauma timing would *not* predict between study variability.

Method

Search strategy and inclusion/exclusion criteria

The search strategy of Moore and Zoellner (2007) was replicated: TJB searched PsycINFO and MEDLINE databases using the term *autobiographical memory* and either *specificity* or *overgeneral*, in addition to the terms *trauma*, *abuse*, *assault*, *rape*, *combat* and *accident* (e.g., autobiographical memory AND specificity AND trauma; autobiographical memory AND specificity AND abuse, etc.; autobiographical memory AND overgeneral AND trauma, etc.)

The authors' libraries and the references of included articles were also searched for additional articles. Studies which assessed performance in a standard AMT in a trauma-exposed group relative to a non-trauma-exposed control group were included. As the majority of studies that were sampled were published prior to the release of DSM-5, trauma exposure was defined in line with DSM-IV-TR (American Psychiatric Association, 2000) criterion A: an event where the individual experiences, witnesses or is confronted by the threat of death, serious injury or a threat to the physical integrity of oneself or others and in which the individual experiences intense fear, helplessness or horror. In addition, in line with Moore & Zoellner (2007) as definitions and reporting of specific details regarding trauma are inconsistent across the literature, we also included studies where the events described are likely to have met the DSM criteria but where there was no assessment of the subjective experience criteria. A standard AMT refers to one in which for each of several positive and negative cue words participants are instructed to retrieve a specific memory (Williams &

Broadbent, 1986). To include the maximum amount of available studies with trauma-exposed participants, studies with clinical and healthy control groups were included. Several studies were excluded because they used an atypical AMT such as instructing participants to recall only trauma memories, or where they were asked to recall multiple memories for single cue words, where cue words were chosen by participants given their self-relevance or where participants were interviewed and further probing of their memories took place. See Figure 1 for a flow diagram of the search and inclusion and exclusion data.

Data extraction, handling and analysis

The following were extracted from each article by T.J.B: 1) Author(s) names; 2) publication year; 3) sample size; 4) participants' mean age and the proportion of females; 5) trauma exposure (yes/no); 6) whether exposure occurred in childhood or adulthood (coded as 0 and 1, respectively, as continuous data in this regard were mostly not recorded/reported); 7) the type of trauma; and mean and standard deviations for 8) posttraumatic stress symptoms; 9) depressive symptoms; 10) (proportion or number of) specific memories recalled in AMT, either as a sum total of all cues or separately for positive and negative cues. Where data for positive and negative cues were given but the total was not given, the total for both cues combined was computed.

STATA 14.2 metan and metareg package were used for analyses. Pooled standard mean differences (*d*) were computed using Cohen's method. The presence of publication bias in the overall specificity effect sizes was examined by visually inspecting funnel plots and then using Egger's test to assess whether the effect size of a given study was related to the size of that study. Vevea and Woods (2005) sensitivity analysis was then performed, which adjusts pooled effect sizes based on the presence of moderate and severe one- and two-tailed selection biases. Similarity in the size of the adjusted and unadjusted effect sizes is then used as evidence of there being little or no publication bias.

Throughout our analyses, a random effects framework was used, making the assumption that the effect of trauma-exposure on specificity varies between studies. The extent to which pooled *d*s differed from zero was calculated using a *Z* test. The DerSimonian and Laird method was used to estimate between study heterogeneity. Following the recommendations of Borenstein and colleagues (2017), tau-squared (τ^2) served as the index of between study heterogeneity. Two random-effects meta-regressions were used to examine whether the heterogeneity between the effects of trauma exposure on specificity could be explained by some other variable. We computed standardised mean difference variables between exposure and control groups in their posttraumatic stress and depressive symptoms separately. This enabled us to compare studies that used different symptom measures. The first meta-regression then used these variables to examine the extent to which heterogeneity in trauma exposure effect sizes were explained by differences between trauma-exposed participants and controls without trauma exposure in posttraumatic stress symptoms and depressive symptoms. The second meta-regression tested whether the heterogeneity was explained by age during trauma exposure whilst statistically controlling for participants age at the time of assessment. The significance of these effects was calculated using a *Z* test. Two, rather than one, meta-regressions were performed due to substantial variability in the number of studies which reported trauma timing and posttraumatic stress and depression symptoms.

Results

Study characteristics

33 effect sizes were computed for differences in memory specificity between trauma-exposed participants and controls, overall across positive and negative cues. There were 18 effect sizes for comparisons of specificity following positive and negative cues each. Only four studies compared posttraumatic stress symptoms between trauma-exposed people and

non-exposed controls. Two used the Impact of Events Scale (IES) and one used the revised edition (IES-R) and one used the Child Posttraumatic Stress Scale (CPSS). 17 studies compared depressive symptoms between trauma-exposed and non-exposed people. Five used the Beck Depression Inventory version I (BDI) and five used version II (BDI-II). The depression subscales of the Profile of Mood States (POMS), the Karolinska Affective and Borderline Symptoms Scale (KABOSS), the Depression Anxiety Stress Scale (DASS), Hospital Anxiety Depression Scale (HADS), were each used once; as were the Children's Depression Inventory (CDI), the Hamilton Depression Rating Scale (HDRS) and the Birleson Depression Scale (BDS). There was no difference in the average age of the samples of trauma-exposed participants (n = 1011; *Mean*: 33.75 years; *SD*: 16.65) compared with those with non-exposed participants (n = 643; *Mean*: 31.47 years; *SD*: 15.72), $t(50) = -.49$, $p = .626$. There was also no difference in the average percentage of females in exposed (*Mean*: 69.0% *SD*: 31.3) and non-exposed samples (*Mean*: 67.8%; *SD*: 30.8), $t(45) = -.13$, $p = .894$. Of the overall 33 effect sizes, nine of these came from studies involving sexual abuse, one involved emotional abuse, five involved exposure to physical illness (e.g., cancer), one involved exposure to an accident, six involved exposure to war and 11 involved mixed samples. Also, 22 of these involved childhood traumas and eight involved traumas experienced in adulthood (three did not report trauma age).

Meta-analysis

The pooled effect size across all studies, when considering recall from all cues together, (k = 33) differed significantly from zero, $d = .769$, 95% CI [.423, 1.115], $Z = 4.35$, $p < .001$ (cf. Figure 2 for forest plot of effect sizes for each study). As hypothesised, participants exposed to trauma recalled fewer specific memories than those who were not exposed to trauma. There was a substantial amount of heterogeneity between studies, $\tau^2 = 1.009$, $\chi^2(32) = 1837.95$, $p < .001$. One study had an effect size that was greater than three

standard deviations from the average effect size for all studies, however, even when this study was removed from the analysis the pooled effect size still differed significantly from zero, d = .607, 95% CI [.415, .800], Z = 6.19, p < .001. In both of these tests, the overall 95% prediction interval surrounding the pooled effect size overlapped partially with zero, suggesting that the heterogeneity between study effect sizes was such that there is a small likelihood that subsequent research in this area may return null findings regarding the association between trauma exposure and memory specificity.

Pooled effect sizes between trauma-exposed and non-trauma-exposed individuals for specificity following positive cue words, k = 18, d = .396, 95% CI [.192, .600], Z = 3.81, p < .001, and negative cue words, k = 18, d = .314, 95% CI [.091, .536], Z = 2.76, p = .006, were both significantly different from zero (cf. Figures 3 and 4). Again, there was substantial heterogeneity between studies within these analyses (Positive: τ^2 = .174, $\chi^2(17)$ = 184.26, p < .001; Negative: τ^2 = .211, $\chi^2(17)$ = 220.37, p < .001). In line with our hypotheses regarding the psychometric properties of the AMT and its single factor structure, the effect sizes for positive and negative cue words did not differ from one another given the overlap of their confidence intervals and they were similar to the effect size in the overall meta-analysis. Participants who were exposed to trauma recalled fewer specific memories than participants who had not been exposed to trauma when recalling autobiographical memories irrespective of the valence of the cue word.

Meta-regression

In these subsequent analyses, we consider specificity effect sizes for total AMT scores rather than per-valence, given that our analyses suggest that there is no difference in recall between these cues.

The first meta-regression explored the effects of differences in posttraumatic stress and depressive symptoms between people with and without trauma exposure. Contrary to our

hypotheses, the extent to which trauma exposed and non-exposed individuals, within a given study, differed in their posttraumatic stress symptoms and depressive symptoms only explained trend-level amounts of heterogeneity in trauma-exposure specificity effect sizes (Posttraumatic stress: B = .152, SE = .086, Z = 1.77, p = .076; Depression: B = -.205, SE = .110, Z = -1.86, p = .062). A separate meta-regression explored the effects of the age at which the trauma occurred on the heterogeneity of trauma-exposure specificity effect sizes whilst participants' mean age was also input into the regression. Mean sample age was not a significant predictor of the heterogeneity of effect sizes (B = -.004, SE = .012, Z = -.36, p = .718) whereas trauma timing predicted a significant amount of heterogeneity (B = 1.128, SE = .449, Z = 2.51, p = .012).

Contrary to our expectations, samples involving participants who were exposed to trauma in adulthood were more likely than samples of participants exposed to trauma in childhood to show larger differences in the specificity of their recall in the AMT compared with participants without exposure. Put otherwise, recall of fewer specific memories was most evident for people whose trauma occurred during adulthood than if it occurred during childhood. This effect was independent of participants' mean age at the time of taking the AMT.

Publication bias

Funnel plots of effect sizes for specificity between trauma-exposed and non-exposed samples of participants against standard errors for these effects suggested that there may be evidence of publication bias (cf. Figure 5). However, follow-up analyses using Egger's test suggested that this was not the case, all p's > .05. This was further confirmed by inspection of Vevea and Woods (2005) adjusted effect sizes accounting for severe two-tailed bias. These adjusted values were similar to the unadjusted effect sizes (All cues: .652; Positive: .302; Negative: .238). These adjusted effect sizes would not alter our interpretation of the

main meta-analysis results, suggesting that the unadjusted effect sizes are unlikely to be severely influenced by unpublished studies.

Discussion

The present meta-analysis sought to clarify the suggestions made by Williams and colleagues (Williams, 2006; Williams, et al., 2007) regarding the association between exposure to trauma and the presence of reduced specificity. Effect sizes for differences in specificity between individuals with and without exposure to trauma were computed and pooled across studies. Our findings are in contrast to the conclusions made by Moore and Zoellner (2007), that exposure to trauma is not sufficient to influence individual differences in specificity. Instead, by offering a broader analysis of existing literature, as well as meta-analytic methodology, our findings offer further support to the findings of Ono, et al. (2016) that trauma-exposed individuals show compromised memory specificity compared with individuals without exposure. This also further supports the conclusions of Williams and colleagues (Williams, 2006; Williams, et al., 2007) who suggested that reduced specificity might be associated with exposure to trauma.

However, it must be noted that our analysis does not support the suggestion that low specificity is particularly evident for trauma exposed participants when recalling memories following a negative cue word (Ono, et al., 2016). In our study, the confidence intervals for the specificity effect sizes for positive and negative cue words overlapped substantially, although the effect size for negative cue words was somewhat smaller than for positive cues. This finding is in line with those of others that the AMT has a single factor (Griffith, et al., 2009; Griffith, et al., 2012; Heron, et al., 2012; Takano, et al., 2017). The earlier finding by Ono, et al. (2016) may have been due to the smaller sample size used in this previous meta-analysis.

It must also be noted that the presence of significant heterogeneity in effect sizes is in accordance with the conclusions of Williams, et al. (2007) that there may be other factors beyond mere exposure to trauma that contribute towards reduced memory specificity. As Ono, et al. (2016) rightly remark, some of this heterogeneity is attributable to methodological differences between studies (e.g., differences in the number of cue words used and differences in length of time given to recall memories).

In the present analysis, we found only trend-level evidence that greater difference in posttraumatic stress symptom severity or depression severity between trauma-exposed and non-exposed groups explained heterogeneity between these groups in their specificity effect sizes. This finding contrasts somewhat with previous suggestions that the extent to which individuals are affected by a trauma is likely to play an important role in memory specificity (Moore & Zoellner, 2007). This finding also to some extent contrasts with previous studies showing a categorical association between depressive disorders and compromised specificity (Ono, et al., 2016). We had expected that participants with more severe depressive symptoms would show lower specificity than participants with less severe symptoms, however, this was not the case. It may be that individual differences in posttraumatic stress and depressive symptoms are poorly associated with differences in specificity and that compromised specificity is only evident when comparing people with diagnoses of PTSD or depressive disorders with control participants or when analysing this association within at-risk samples of participants such as those who show other cognitive vulnerabilities (Gutenbrunner, Salmon, & Jose, 2017; Raes, Hermans, Williams, & Eelen, 2007). It may that in the studies sampled, there was insufficient variability between participants in their posttraumatic stress and depressive symptoms and as such it was not possible to adequately explore the association between these symptoms and differences in specificity. Also, the analysis of posttraumatic stress symptoms was underpowered with few studies reporting comparisons of

posttraumatic stress symptoms between trauma-exposed participants and controls. This might be due to the assumption that an individual who has not been exposed to trauma would not show symptoms of posttraumatic stress symptoms. However, it is worth noting that in many of the sampled studies, the control participants had merely not been exposed to a negative life event that met the DSM. That is not to say that these control participants would not experience any posttraumatic stress symptoms, perhaps due to a less significant trauma. The studies that did measure posttraumatic symptoms in controls illustrate that this is possible. Such symptoms might have important effects on specificity and other aspects of cognition so it is important that future studies assess these symptoms even in control participants.

We also found that the timing of trauma was associated with specificity effect sizes such that participants with trauma exposure were more likely to recall even fewer specific memories than controls if their trauma occurred during adulthood than if it occurred during childhood. This finding stands in contrast to another conclusion of Williams, et al. (2007) who after synthesising the findings of Willebrand, et al. (2002) and Stokes, et al. (2004) concluded that traumas experienced in childhood may be most likely to compromise memory specificity. One possibility was that our finding was because participants with childhood trauma had experienced more time since their trauma at the time of the AMT assessment, relative to adult trauma survivors, and that the effects of the trauma on specificity had perhaps reduced over time. However, even when participants' mean age was input into the regression model, trauma timing was still a predictor of specificity effect sizes. We speculate that this pattern of results is due to the relative contemporaneity of trauma and the AMT for those who experience trauma in adulthood versus childhood. There are other possibilities that might be explored in further research, however. It may be that for children, the full meaning and consequences of a trauma might be appreciated less so, relative to adults. Thus, it would be of interest to further study not only when trauma occurred, but also how it was processed

and interpreted *at that time*. It is also possible that available coping resources and social supports differ, on average, for adults versus children. Our analysis would have been more robust if it were more common in the literature to record the approximate time that trauma took place so that we might have explored the effects of this in a continuous manner. Instead our analysis was confined to an analysis of traumas experienced in childhood versus adulthood, which precludes us from making more robust conclusions regarding the exact timing of trauma and also how distant this was from the time of AMT assessment. It is of note that our analysis did not control for AMT parameters which may have differed between studies, such as the number of cue words used or the time given to participants for them to retrieve their memory for each cue word (van Vreeswijk & de Wilde, 2004). Differences between studies in these parameters may have explained additional heterogeneity in effect sizes and may have limited the amount of heterogeneity explained by trauma timing. Finally, it is also of note that the specific types of trauma are likely to differ for adults versus children and this may also explain some of the heterogeneity between studies and particularly that which was explained by trauma timing. However, given inconsistencies across studies in the reporting of traumas and our limited sample size for some traumas, we were unable to analyse trauma-specific effects. To this end, future research should report such details in a consistent manner whilst considering the possible effects of deficits in retrospective recall in recalling the type and severity of a trauma. To avoid such issues, there is a need for longitudinal designs that monitor, for equal periods of time, people who have experienced traumas in childhood and adulthood where we might then investigate whether such exposure is associated with reductions in memory specificity and if this is moderated by trauma timing. One might expect on the basis of the present findings that reductions in specificity would be most evident among adult trauma survivors. The form and severity of these traumas might

also be quantified at the time of exposure and during a period of adjustment afterwards so that trauma-specific effects might also be explored.

Given the cross-sectional nature of the data that were sampled, it remains possible that reduced specificity may have been present at the time of the trauma rather than as a result of the trauma. Future longitudinal research examining changes in specificity prior to and following trauma is warranted. This is also not to say that everyone who is exposed to trauma will experience a reduction in their memory specificity or, amongst those who experience such a reduction that this will inevitably lead to the emergence of psychopathology or serve to maintain existing psychopathologies. Many of the studies that were included in the analysis identified their participants through clinics. Such a sample may not represent all people exposed to trauma and might only represent those whose trauma is so severe that they seek help. Nevertheless, given our findings regarding trauma exposure and specificity, and evidence from elsewhere that reduced specificity can precede the emergence of emotional disorders (Kleim & Ehlers, 2008; Sumner, et al., 2010) this might suggest that individual differences in memory specificity measured soon after a trauma might be used to identify those who are most at risk of acquiring a disorder. We might then target these people with an intervention to improve memory specificity (Moradi, et al., 2014; Hamid Taher Neshat Doost, et al., 2013; Raes, Williams, & Hermans, 2009) with the intention of building resilience.

It is of note that we excluded several studies because they included atypical AMTs. These studies examined other aspects of memory specificity (e.g., specificity of memories cued by trauma-related or self-relevant words) or used additional probing of memories within an interview format. These studies were excluded due to the additional heterogeneity that such differences in methodology would have brought to the meta-analysis. That is not to say that such studies do not provide valuable data on the role of trauma exposure in memory

specificity. However, the present study provides an analysis of the effect of trauma exposure on the specificity of autobiographical memories that are retrieved following cue words that are seemingly unrelated to possible traumas and which are retrieved without additional probing by an experimenter. That trauma exposure could influence specificity even for memories that are unlikely to be related to a trauma, supports the suggestion that the cognitive effects of trauma exposure, perhaps related to aspects of the CaRFAX model, impact memory processes more broadly and not just memories for traumas.

The findings presented here add to a growing literature on the effects of trauma exposure on cognitive factors that are associated with the emergence and maintenance of psychopathology. Previous work in this area suggests that exposure to trauma can lead to ongoing problems in the appraisal of potential threats and heightened accessibility and retrieval of negative, trauma-related memories and that this can have important influences on one's self view and narrative (Berntsen & Rubin, 2007; Brewin, Gregory, Lipton, & Burgess, 2010; Ehlers & Clark, 2000). The data presented here suggest that the experience of trauma might also influence the retrieval of autobiographical memories even when they are cued by words that are not trauma-related and may even be positively valenced. Longitudinal research is now needed, where there is an assessment of AMT performance and posttraumatic stress and depressive symptoms at the time of trauma exposure and six months or more afterward, during which traumatic coping is quantified as well as other aspects of the CaRFAX model. It might then be possible to draw stronger conclusions regarding the mediating role of traumatic coping on changes in AMT performance and subsequent psychopathology. It is clear from our analysis though that mere exposure to trauma is sufficient to compromise autobiographical memory specificity.

References

Abdollahi, M., Moradi, A., Hasani, J., & Jobson, L. (2012). Investigating the relationships between autobiographical remembering, the self and posttraumatic stress disorder in individuals with HIV. *Memory, 20*(8), 872-881.

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of mental disorders (4th ed. Text Revision)*. Washington, DC: Author.

Berntsen, D., & Rubin, D.C. (2007). When a trauma becomes a key to identity: enhanced integration of trauma memories predicts posttraumatic stress disorder symptoms. *Applied Cognitive Psychology, 21*(4), 417-431.

Brennen, T., Hasanovic, M., Zotovic, M., Blix, I., Solheim Skar, A.-M., Kravic Prelic, N., et al. (2010). Trauma Exposure in Childhood Impairs the Ability to Recall Specific Autobiographical Memories in Late Adolescence. *Journal of Traumatic Stress, 23*, 240-247.

Brewin, C.R., Gregory, J.D., Lipton, M., & Burgess, N. (2010). Intrusive images in psychological disorders: characteristics, neural mechanisms, and treatment implications. *Psychol Rev, 117*(1), 210-232.

Dalgleish, T., Rolfe, J., Golden, A.-M., Dunn, B.D., & Barnard, P.J. (2008). Reduced autobiographical memory specificity and posttraumatic stress: Exploring the contributions of impaired executive control and affect regulation. *117*, 236-241.

Ehlers, A., & Clark, D.M. (2000). A cognitive model of posttraumatic stress disorder. *Behav Res Ther, 38*(4), 319-345.

Griffith, J.W., Claes, S., Hompes, T., Vrieze, E., Vermote, S., Debeer, E., et al. (2016). Effects of Childhood Abuse on Overgeneral Autobiographical Memory in Current Major Depressive Disorder. *Cognitive Therapy and Research, 40*(6), 774-782.

Griffith, J.W., Sumner, J.A., Debeer, E., Raes, F., Hermans, D., Mineka, S., et al. (2009). An item response theory/confirmatory factor analysis of the Autobiographical Memory Test. *Memory, 17*(6), 609-623.

Griffith, J.W., Sumner, J.A., Raes, F., Barnhofer, T., Debeer, E., & Hermans, D. (2012). Current psychometric and methodological issues in the measurement of overgeneral autobiographical memory. *J Behav Ther Exp Psychiatry, 43 Suppl 1*, S21-31.

Gutenbrunner, C., Salmon, K., & Jose, P.E. (2017). Do Overgeneral Autobiographical Memories Predict Increased Psychopathological Symptoms in Community Youth? A 3-Year Longitudinal Investigation. *J Abnorm Child Psychol*.

Hauer, B.J., Wessel, I., Geraerts, E., Merckelbach, H., & Dalgleish, T. (2008). Autobiographical memory specificity after manipulating retrieval cues in adults reporting childhood sexual abuse. *J Abnorm Psychol, 117*(2), 444-453.

Henderson, D., Hargreaves, I., Gregory, S., & Williams, J.M.G. (2002). Autobiographical memory and emotion in a non-clinical sample of women with and without a reported history of childhood sexual abuse. *British Journal of Clinical Psychology, 41*, 129-141.

Hermans, D., de Decker, A., de Peuter, S., Raes, F., Eelen, P., & Williams, J.M. (2008). Autobiographical memory specificity and affect regulation: coping with a negative life event. *Depress Anxiety, 25*(9), 787-792.

Heron, J., Crane, C., Gunnell, D., Lewis, G., Evans, J., & Williams, J.M. (2012). 40,000 memories in young teenagers: psychometric properties of the Autobiographical Memory Test in a UK cohort study. *Memory, 20*(3), 300-320.

Huntjens, R.J., Wessel, I., Hermans, D., & van Minnen, A. (2014). Autobiographical memory specificity in dissociative identity disorder. *J Abnorm Psychol, 123*(2), 419-428.

Kleim, B., & Ehlers, A. (2008). Reduced autobiographical memory specificity predicts depression and posttraumatic stress disorder after recent trauma. *J Consult Clin Psychol, 76*(2), 231-242.

Kuyken, W., Howell, R., & Dalgleish, T. (2006). Over-general Autobiographical Memory in Depressed Adolescents with versus without a Reported History of Trauma. *Journal of Abnormal Psychology, 115*, 387-296.

Maurex, L., Lekander, M., Nilsonne, A., Andersson, E.E., Asberg, M., & Ohman, A. (2010). Social problem solving, autobiographical memory, trauma, and depression in women with borderline personality disorder and a history of suicide attempts. *Br J Clin Psychol, 49*(Pt 3), 327-342.

McNally, R.J., Clancy, S.A., Barrett, H.M., Parker, H.A., Ristuccia, C.S., & Perlman, C.A. (2006). Autobiographical memory specificity in adults reporting repressed, recovered, or continuous memories of childhood sexual abuse. *Cogn Emot, 20*(3-4), 527-535.

Moore, S.A., & Zoellner, L.A. (2007). Overgeneral autobiographical memory and traumatic events: an evaluative review. *Psychol Bull, 133*(3), 419-437.

Moradi, A.R., Abdi, A., Fathi-Ashtiani, A., Dalgleish, T., & Jobson, L. (2012). Overgeneral autobiographical memory recollection in Iranian combat veterans with posttraumatic stress disorder. *Behav Res Ther, 50*(6), 435-441.

Moradi, A.R., Miraghaei, M.A., Parhon, H., Jabbari, H., & Jobson, L. (2013). Posttraumatic stress disorder, depression, executive functioning, and autobiographical remembering in individuals with HIV and in carers of those with HIV in Iran. *AIDS Care, 25*(3), 281-288.

Moradi, A.R., Moshirpanahi, S., Parhon, H., Mirzaei, J., Dalgleish, T., & Jobson, L. (2014). A pilot randomized controlled trial investigating the efficacy of MEmory Specificity

Training in improving symptoms of posttraumatic stress disorder. *Behav Res Ther, 56*, 68-74.

Mowlds, W., Shannon, C., McCusker, C.G., Meenagh, C., Robinson, D., Wilson, A., et al. (2010). Autobiographical memory specificity, depression, and trauma in bipolar disorder. *Br J Clin Psychol, 49*(Pt 2), 217-233.

Nelson, C.A. (2000). Neural plasticity and human development: the role of early experience in sculpting memory systems. *Developmental Science, 3*(2), 115-130.

Neshat Doost, H.T., Dalgleish, T., Yule, W., Kalantari, M., Ahmadi, S.J., Dyregrov, A., et al. (2013). Enhancing Autobiographical Memory Specificity Through Cognitive Training. *Clinical Psychological Science, 1*(1), 84-92.

Neshat Doost, H.T., Yule, W., Kalantari, M., Rezvani, S.R., Dyregrov, A., & Jobson, L. (2014). Reduced autobiographical memory specificity in bereaved Afghan adolescents. *Memory, 22*(6), 700-709.

Nilsson-Ihrfelt, E., Fjallskog, M.L., Liss, A., Jakobsson, O., Blomqvist, C., & Andersson, G. (2004). Autobiographical memories in patients treated for breast cancer. *J Psychosom Res, 57*(4), 363-366.

Nixon, R.D.V., Ball, S.-A., Sterk, J., Best, T., & Beatty, L. (2013). Autobiographical Memory in Children and Adolescents With Acute Stress and Chronic Posttraumatic Stress Disorder. *Behaviour Change, 30*(03), 180-198.

Ono, M., & Devilly, G.J. (2013). The role of childhood and adulthood trauma and appraisal of self-discrepancy in overgeneral memory retrieval. *Cognition and Emotion, 27*, 979-994.

Ono, M., Devilly, G.J., & Shum, D.H. (2016). A meta-analytic review of overgeneral memory: The role of trauma history, mood, and the presence of posttraumatic stress disorder. *Psychol Trauma, 8*(2), 157-164.

Raes, F., Hermans, D., Williams, J.M., & Eelen, P. (2007). A sentence completion procedure as an alternative to the Autobiographical Memory Test for assessing overgeneral memory in non-clinical populations. *Memory, 15*(5), 495-507.

Raes, F., Hermans, D., Williams, J.M.G., & Eelen, P. (2005). Autobiographical memory specificity and emotional abuse. *British Journal of Clinical Psychology, 44*, 133-138.

Raes, F., Williams, J.M., & Hermans, D. (2009). Reducing cognitive vulnerability to depression: a preliminary investigation of MEmory Specificity Training (MEST) in inpatients with depressive symptomatology. *J Behav Ther Exp Psychiatry, 40*(1), 24-38.

Raymaekers, L., Smeets, T., Peters, M.J., & Merckelbach, H. (2010). Autobiographical memory specificity among people with recovered memories of childhood sexual abuse. *J Behav Ther Exp Psychiatry, 41*(4), 338-344.

Stokes, D.J., Dritschel, B.H., & Bekerian, D.A. (2004). The effect of burn injury on adolescents autobiographical memory. *Behav Res Ther, 42*(11), 1357-1365.

Sumner, J.A., Griffith, J.W., & Mineka, S. (2010). Overgeneral autobiographical memory as a predictor of the course of depression: a meta-analysis. *Behav Res Ther, 48*(7), 614-625.

Takano, K., Gutenbrunner, C., Martens, K., Salmon, K., & Raes, F. (2017). Computerized Scoring Algorithms for the Autobiographical Memory Test. *Psychol Assess*.

van Vreeswijk, M.F., & de Wilde, E.J. (2004). Autobiographical memory specificity, psychopathology, depressed mood and the use of the Autobiographical Memory Test: a meta-analysis. *Behaviour Research and Therapy, 42*(6), 731-743.

Willebrand, M., Norlund, F., Kildal, M., Gerdin, B., Ekselius, L., & Andersson, G. (2002). Cognitive distortions in recovered burn patients: the emotional Stroop task and autobiographical memory test. *Burns, 28*, 465-471.

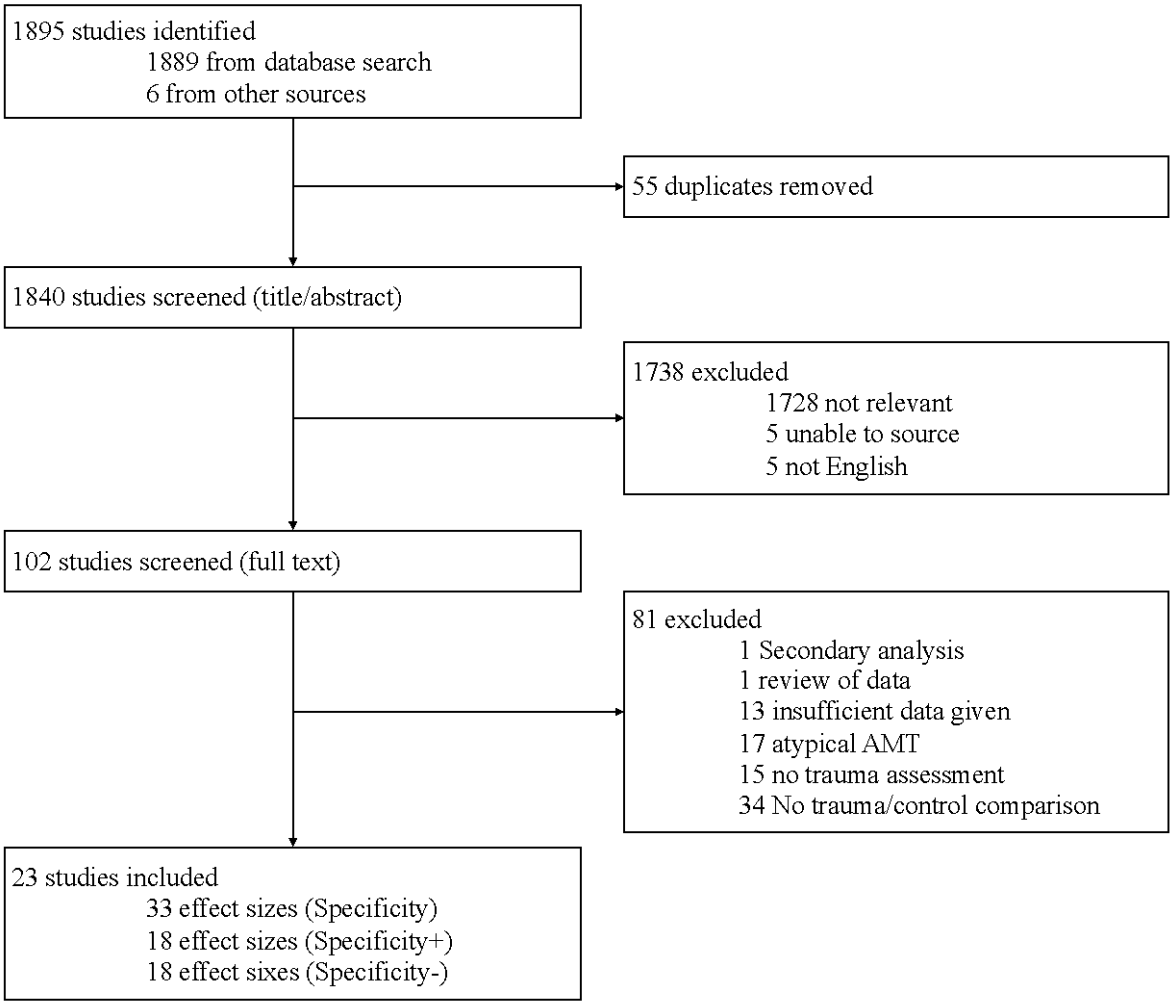
Williams, J.M. (2006). Capture and rumination, functional avoidance, and executive control (CaRFAX): Three processes that underlie overgeneral memory. *Cogn Emot, 20*(3-4), 548-568.

Williams, J.M., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., et al. (2007). Autobiographical memory specificity and emotional disorder. *Psychol Bull, 133*(1), 122-148.

Williams, J.M., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology*, *95*(2), 144-149.

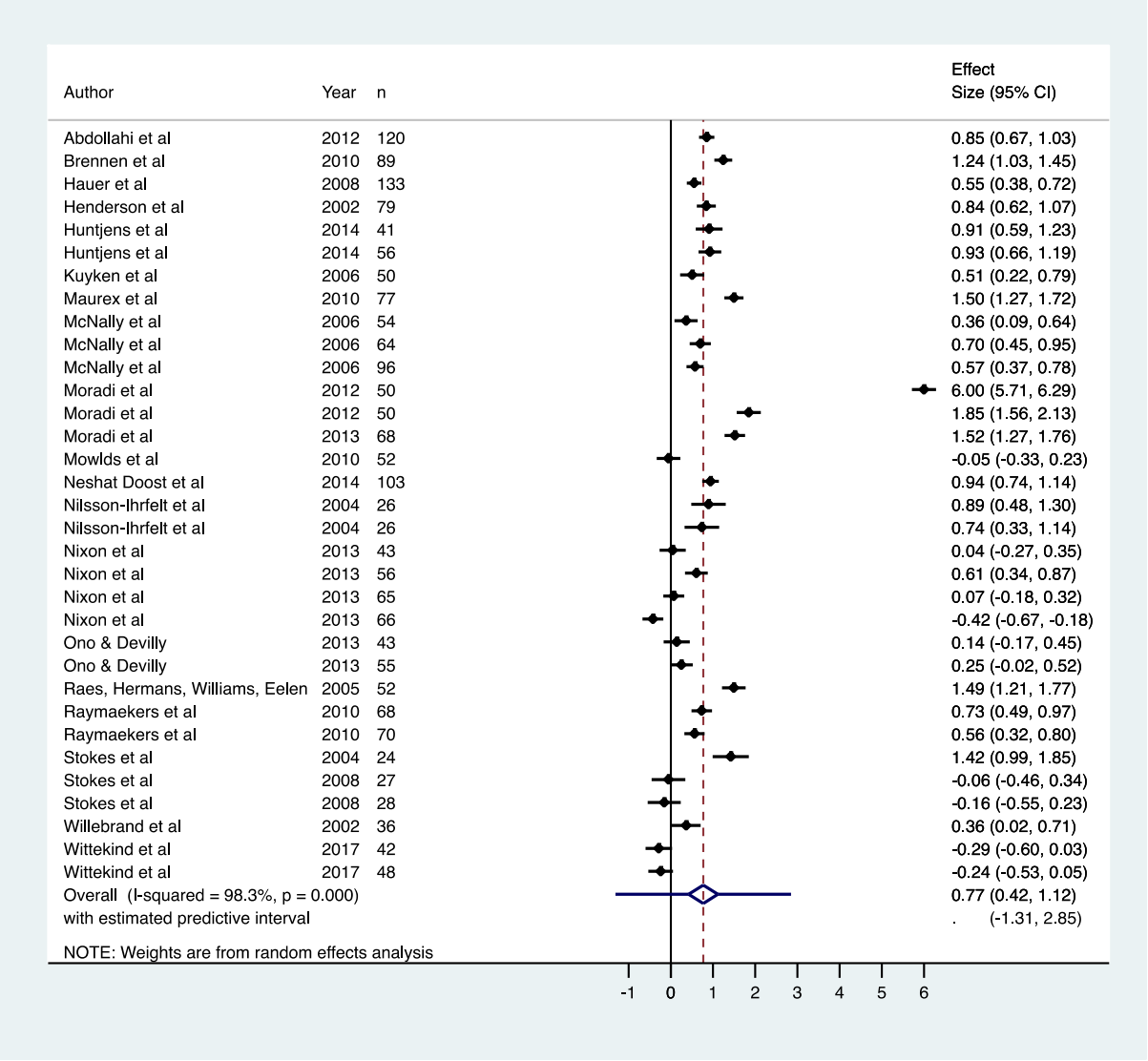
Wittekind, C.E., Jelinek, L., Kleim, B., Muhtz, C., Moritz, S., & Berna, F. (2017). Age effect on autobiographical memory specificity: A study on autobiographical memory specificity in elderly survivors of childhood trauma. *J Behav Ther Exp Psychiatry, 54*, 247-253.

Figure 1.



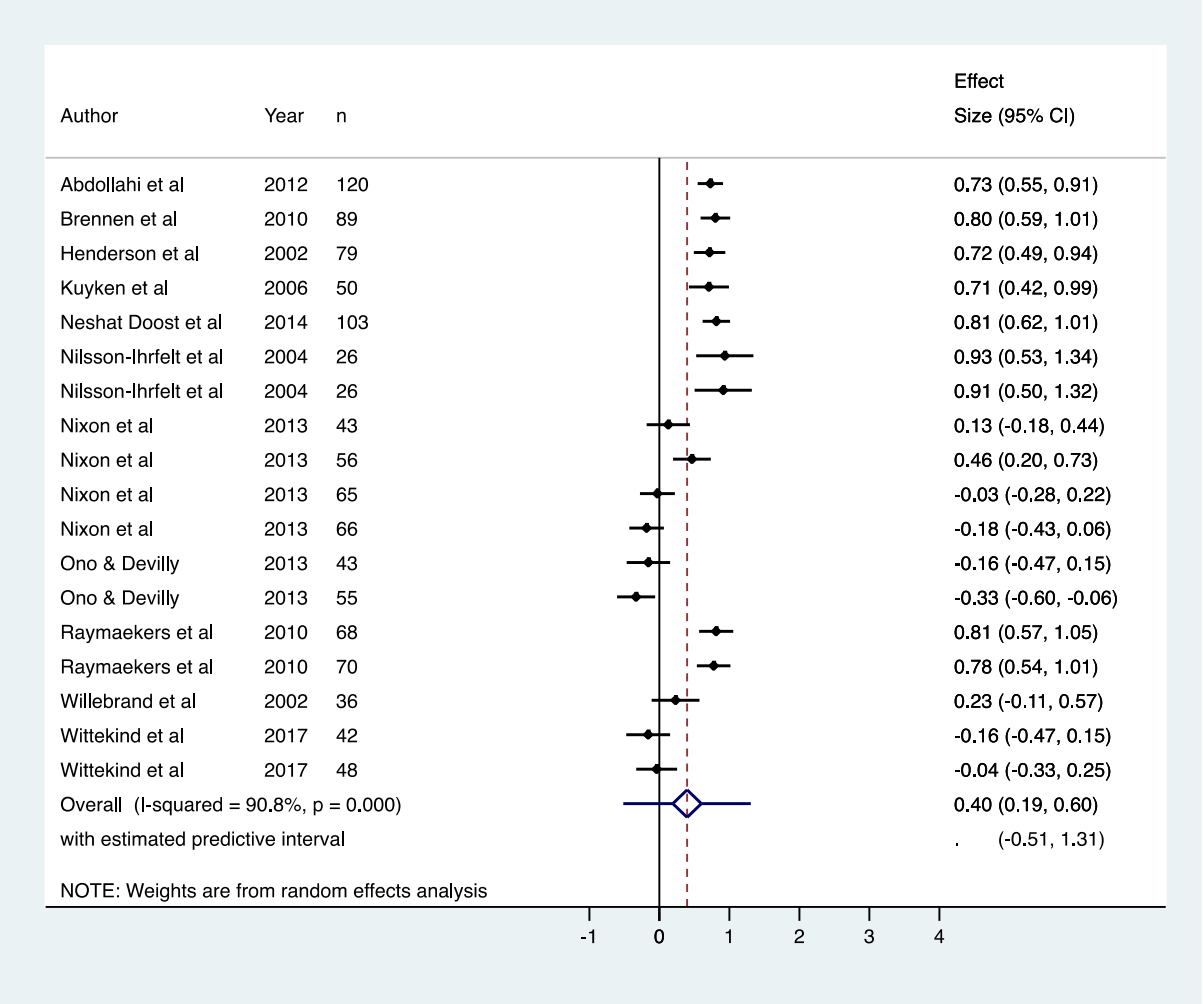
Note. Flow chart of article inclusion and exclusion. Specificity refers to the overall analysis of effect sizes comparing memory specificity across both types of cue valence between trauma exposed and non-exposed groups. Specificity+ and Specific- refer to the effect sizes for comparisons of positive and negative cues separately.

Figure 2.



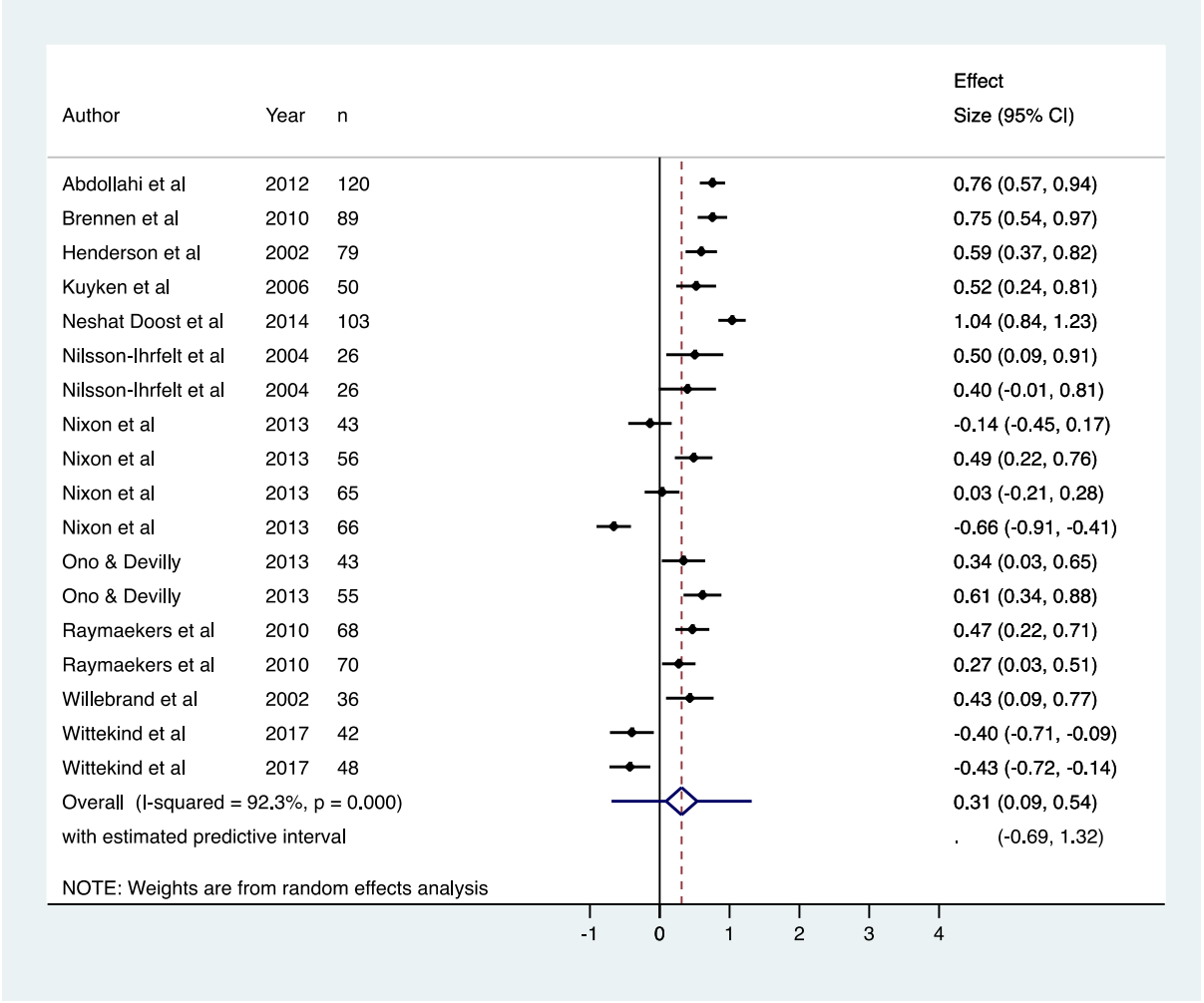
Notes. Forest plot of effect sizes for differences in memory specificity (all cues) between trauma-exposed and non-exposed participants. Diamonds are the effect size estimates (standardised), with 95% confidence intervals. The pooled effect size is also given, with 95% prediction intervals. The pooled effect size for the difference in specificity between trauma-exposed and not-exposed people was large (d = .77) and differed significantly from zero (p < .001).

Figure 3.



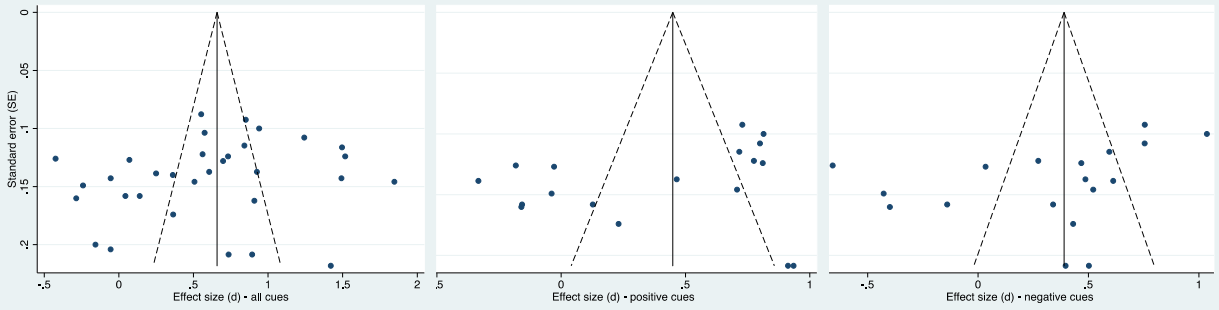
Notes. Forest plot of effect sizes for differences in memory specificity (positive cues) between trauma-exposed and non-exposed participants. Diamonds are the effect size estimates (standardised), with 95% confidence intervals. The pooled effect size is also given, with 95% prediction intervals. The pooled effect size for the difference in specificity, following positive cues, between trauma-exposed and not-exposed people was moderate (d = .40) and differed significantly from zero ($p < .001$).

Figure 4.



Notes. Forest plot of effect sizes for differences in memory specificity (negative cues) between trauma-exposed and non-exposed participants. Diamonds are the effect size estimates (standardised), with 95% confidence intervals. The pooled effect size is also given, with 95% prediction intervals. The pooled effect size for the difference in specificity, following negative cues, between trauma-exposed and not-exposed people was moderate (d = .31) and differed significantly from zero (p = .006).

Figure 5.



Note. Funnel plots of effect sizes for specificity between trauma-exposed and non-exposed samples of participants against standard errors, for each of the meta-analyses (from left to right: effect sizes for specificity for all cues together, for positive cues only and for negative cues only). The plots suggest that there may be evidence of publication bias, however, further analyses using Egger's and Vevea and Woods' procedures suggested that this was not the case.