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The Predictive Power of Autobiographical Memory in Shaping the Mental Health of Young People: An Individual Participant Data Meta-Analysis

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Author note: Due to ethical and governance restrictions, we do not have permission from contributing data owners to share the data publicly. However, analysis scripts are available through the Open Science Framework (https://osf.io/tfxbw/?view_only=ce4d2e1243c5462f82969a07e86ee7d9).

Abstract

Reduced autobiographical memory (AM) specificity, characterized by difficulty recalling specific past events, is a feature of multiple psychiatric disorders. While meta-analyses indicate that reduced AM specificity can predict future symptom severity, its role as a premorbid risk factor for mental illness onset in young people remains unclear. Our pre-registered individual participant data meta-analysis (PROSPERO; CRD42022287786) synthesized longitudinal data from 14 community-based studies of children and adolescents (N = 9165). Most studies reported symptom severity (92.9%), with one-third also reporting diagnostic status (35.7%). Assessment timing ranged from 2 months to 8.3 years following the Autobiographical Memory Task. Multivariate mixed-effect models found no support for AM specificity predicting future symptom severity in depression or post-traumatic stress disorder. Contrary to expectations, higher memory specificity significantly predicted higher anxiety symptoms, but the quality of evidence was low. Intriguingly, reduced AM specificity significantly predicted an increased risk of receiving a psychiatric diagnosis, and an earlier onset of disorder (hazard ratio = 0.55, $p = .018$). This risk was most robust for depressive disorders (hazard ratio = 0.21, $p < .001$). Findings suggest reduced AM specificity could be a risk factor for the onset of functionally impairing psychiatric disorders, but it does not consistently predict elevated symptoms in community-based samples. Further theoretical development is needed.

Keywords: overgeneral memory; memory specificity; risk factor; depression; anxiety; eating disorders; individual participant data meta-analysis; ALSPAC

Public Significance Statement

Modifiable risk factors that contribute to multiple mental disorders are crucial targets for early intervention. Analyzing a multi-year dataset of over 9,000 youth, we found that impaired autobiographical memory specificity—the ability to retrieve specific past events—predicted a higher risk of psychiatric diagnoses, particularly depression, and an earlier onset of these disorders. These findings have significant implications for public health, as they suggest that bolstering memory skills in children and adolescents could help prevent mental illness. Investing in appropriate interventions could potentially reduce the prevalence of depression. Future research should investigate the mechanisms linking memory and mental health and develop effective strategies to improve autobiographical memory across different populations.

The Predictive Power of Autobiographical Memory in Shaping the Mental Health of Young People: An Individual Participant Data Meta-Analysis

Mental illness is a leading cause of disease burden globally (Becker & Kleinman, 2013). Early detection and intervention can lessen disorder severity or prevent its onset altogether (van Doorn et al., 2021). A recent review highlighted the pressing need for research to better understand the processes that predispose individuals to mental illness (Arango et al., 2018). Childhood and adolescence are key periods for psychopathology development and would benefit markedly from early intervention (Arango et al., 2018). Understanding transdiagnostic risk factors—those that predict multiple disorders—will best inform preventive approaches. Disruptions in cognitive processing are a defining feature of many mental health disorders. Cognitive distortions predict symptom persistence (Everaert et al., 2022) and are a key target of gold-standard treatments (e.g., Cognitive Behavioral Therapy; Otte et al., 2016). Yet, it remains unclear whether distorted cognitive processes (e.g., emotional bias in explicit memory) predate disorder onset and represent a malleable target for preventive intervention (Dalgleish & Hitchcock, 2024; Everaert et al., 2022). Evaluating malleable cognitive markers within large-scale, longitudinal data from children and adolescents is critical to advancing preventive efforts, particularly given the limited effectiveness of current universal preventive interventions (Foulkes & Stringaris, 2023; Kuyken et al., 2022).

Disrupted retrieval of autobiographical memories is one cognitive process that might represent a transdiagnostic risk factor for mental illness (Dalgleish & Hitchcock, 2023). Autobiographical memory is a long-term memory system that contains personal life experiences. Autobiographical memory contains highly detailed, specific, single-incident events, and also more generalized summaries of extended periods of time, themes within

one's life (e.g., relationships), or categories of repeated events (Conway et al., 2019). Memory disruptions such as preferencing retrieval of negative autobiographical information over positive information, or retrieving generalized summaries more readily than event memories, are consistently associated with poor mental health (Barry et al., 2021; Dalgleish & Hitchcock, 2023). Here, we focus on the reduced ability to retrieve specific, single-incident events (that last for <24 hours) which characterizes multiple disorders. Specific memory retrieval is commonly measured using the Autobiographical Memory Task (AMT, Williams & Broadbent, 1986), which requires individuals to recall a specific event in response to cue words of positive, negative, or neutral emotional valence. Memory specificity refers to the number (or proportion) of specific memories that the individual retrieves. Reduced memory specificity indicates that the individual has a reduced ability to retrieve specific memories.

The dominant theory of memory specificity and psychopathology is the CaRFAX model (Williams et al., 2007). The CaRFAX model suggests three mechanisms which produce reduced specificity: capture of attention and rumination on negative information yielded during the memory search, functional avoidance of negative autobiographical information, and failures in the executive functions that govern memory retrieval. Empirical support for the model has been mixed (Chiu et al., 2018; Gutenbrunner et al., 2019; Stewart et al., 2017). Other recent reviews (e.g., Dalgleish & Hitchcock, 2023) have proposed alternate mechanisms underpinning reduced memory specificity. In this review we evaluate evidence for reduced memory specificity as a risk factor that predicts the first onset of mental illness.

Existing Evidence for Memory Specificity as a Prospective Predictor of Mental Illness

Cross-sectional meta-analysis indicates that people with any psychiatric diagnosis retrieve significantly fewer specific memories than people without any diagnosis (Barry et al., 2021). For individual disorders, memory specificity was significantly lower, relative to

controls, for people with mood and stress-related disorders, but not anxiety disorders (Barry et al., 2021). Another meta-analysis demonstrated that memory specificity remains reduced in individuals remitted from depression, compared to never-depressed individuals (Hallford et al., 2022). Persistence after symptoms have remitted suggests that reduced memory specificity is distinct from the effects of current depressive mood on memory recall (Blaney, 1986). However, cross-sectional studies, particularly with adults (i.e., after the median age of onset of psychiatric disorders; Solmi et al., 2022) cannot elucidate directionality between memory specificity and psychiatric disorders. Determining whether reduced specificity predates disorder is critical to understanding causality. Although meta-analysis suggests that reduced memory specificity is a transdiagnostic cognitive marker (Barry et al., 2021), the synthesized studies largely recruit disorder-specific samples, and report associations between reduced specificity and symptoms of depression, anxiety, posttraumatic stress, psychosis, and disordered eating. We therefore provide an overview of the existing research regarding prospective relationships in a disorder-specific manner.

There is existing evidence that reduced memory specificity prospectively predicts more severe depressive symptoms. Meta-analysis of adult data indicates a small, significant effect of memory specificity on future depressive symptoms - specificity predicted symptoms up to a year later, when controlling for baseline symptoms (Hallford et al., 2021). In children and adolescents, individual longitudinal studies report that reduced memory specificity predicts later depressive symptoms (e.g., Hipwell et al, 2011), along with the onset of depressive disorders (Rawal & Rice, 2012). However, a predictive effect is more consistently observed in individuals with a history of adverse childhood events (Puetz et al., 2021; Stange et al., 2013).

There is mixed evidence that memory specificity predicts future posttraumatic stress (Ono et al., 2016). Prospective, longitudinal studies have found that reduced memory

specificity significantly predicts the onset of posttraumatic stress disorder (PTSD) in adults recently exposed to trauma (Kleim & Ehlers, 2008). In terms of symptom severity, reduced specificity also predicts higher PTSD symptoms in adults who have experienced multiple traumatic events (Bryant et al., 2007). Only a small number of studies have been completed with trauma-exposed young people. Reduced memory specificity has been found to predict higher PTSD symptoms 6-months after trauma exposure (Hitchcock et al., 2014), but the inverse relationship also has been observed 1-month post-trauma (Nixon et al., 2013). Although both studies suggest that memory specificity is associated with future posttraumatic stress in children and adolescents, evidence is inconsistent regarding the direction and strength of this relationship, over time.

There are few studies investigating the relationship between memory specificity and anxiety (Morgan, 2010). A systematic review of cross-sectional data demonstrated little evidence of an association between memory specificity and anxiety (Williams et al., 2007). In an individual study with a community sample of adolescents, there was no significant prospective relationship between baseline memory specificity and anxiety symptoms over three-year follow-ups (Gutenbrunner et al., 2018). Interestingly, a significant relationship was observed in exploratory analysis, whereby reduced memory specificity predicted increased anxiety symptoms in adolescents with high levels of rumination (Gutenbrunner et al., 2018). For adolescents with a family history of mental illness, there is also evidence that reduced memory specificity at baseline significantly increases the likelihood of having a new anxiety disorder diagnosis at one-year follow-up (Rawal & Rice, 2012). However, high co-morbidity between anxiety and depressive disorders in the sample (Rawal & Rice, 2012) meant it was difficult to determine whether this risk was unique to anxiety onset. Taken together, the current evidence for the role of memory specificity as a prospective predictor of anxiety is

largely equivocal. At-risk status - having a family history of psychiatric disorders, or high rumination - might influence the relationship between anxiety and memory specificity.

For eating disorders, there is cross-sectional evidence of reduced specificity in individuals with an eating disorder relative to community-based controls (Keeler et al., 2022). A greater reduction in specificity has been associated with a longer duration of illness (Nandrino et al., 2006). In samples without a formal diagnosis, reduced memory specificity has been cross-sectionally associated with restrained and disordered eating (Ball et al., 2010; Ridout et al., 2015). Prospective evaluation of the relationship between memory specificity and eating disorders is lacking.

Similarly, there exists cross-sectional, but not longitudinal, evidence of a relationship between specificity and psychosis. A meta-analysis of 20 studies reported a large reduction in memory specificity for people with schizophrenia spectrum disorders, compared to control populations (Berna et al., 2016). However, associations between memory specificity and psychosis symptoms lack clarity. One article suggests that reduced specificity is associated with positive but not negative symptoms of psychosis (D'Argembeau et al., 2008). In contrast, another article reports that reduced specificity is only associated with negative symptoms (Harrison & Fowler, 2004). Divergent findings suggest the relationship between memory specificity and psychosis is complex, and reducing heterogeneity (e.g., age, symptom severity, comorbidity) within the sample may help to clarify the relationship. We could not identify any prior evaluations of memory specificity as a longitudinal, prospective risk factor for onset of psychosis in young people.

In sum, prior meta-analyses suggest that reduced memory specificity is a transdiagnostic marker of concurrent psychiatric disorder (Barry et al., 2021) and a single-disorder predictor of later depressive symptoms (Hallford et al., 2021). Yet, a critical question remains: Is reduced memory specificity a risk factor that can predict the first onset

of disorder? If so, which disorders? Reduced specificity is malleable to intervention (for review see Barry et al., 2019a; Dalgleish & Hitchcock, 2023; Hitchcock et al., 2017) and evidence that specificity can predict the onset of multiple mental health disorders would support repurposing existing memory-based treatment programs for preventive intervention.

Potential Moderators of Effect

Participant Characteristics. There are several individual-level factors that appear to moderate the relationship between memory specificity and symptoms. One potential moderator is age. Older children are more specific in their memory retrieval than younger children (O'Carroll et al., 2006). However, it is unclear whether age changes the association between memory specificity and mental illness in young people and better understanding of this relationship will indicate the ideal age group for preventive intervention. Similarly, prior research has suggested that memory specificity is more strongly associated with future depressive symptoms in females than males following stressful life events (Hamlat et al., 2015). Yet, other studies with adolescents have reported no sex differences when examining the effect of baseline memory specificity on depressive symptoms at follow-ups (Puetz et al., 2021; Stange et al., 2013; Warne et al., 2020).

Most prolifically, the relationship between memory specificity and symptom severity appears to be strongest for those already at risk of mental illness. Family history of depression (Woody et al., 2022), own prior experience of depressive symptomology (Hipwell et al., 2011), prior trauma (McCrory et al., 2017), and stressful life events (Askelund et al., 2019; Rawal & Rice, 2012) have all been identified as possible moderators of the predictive effect on depression. That is, memory specificity may be a stronger predictor of symptoms in participants with other risk factors for mental illness. However, synthesis of evidence is needed before strong conclusions can be drawn. Aggregate-level meta-analyses (e.g., Barry et al., 2021; Hallford et al., 2021) are susceptible to aggregation bias, whereby interactions that

occur at the person-level and study-level are conflated (Riley et al., 2021). There is therefore a need to synthesize data at the individual-level to gain a clear understanding of moderation effects.

Measurement Characteristics. The AMT uses a mix of cue words of positive, negative, and sometimes neutral emotional valence. Meta-analysis has reported that for adults, the valence of AMT cues does not significantly impact the relationship between memory specificity and mental health (Barry et al., 2021). However, children and adolescents retrieve more specific memories for positive cues relative to negative cues, suggesting that the emotional valence of cue words may be more important at younger ages (Preko et al., 2023). Reduced access to positive specific memories predicts development of the negative self-cognitions which drive disorder (Askelund et al., 2019). Thus, it is important to consider whether a reduced ability to retrieve positive memories may be more important in the onset of disorder, relative to negative memories (or vice versa).

The Present Meta-Analysis

To determine whether reduced specificity is a risk factor for disorder, we analysed individual participant data from longitudinal studies with community-based samples of children and adolescents. Our rationale for focusing on children and adolescents is three-fold. First, we aimed to measure memory specificity prior to the first onset of disorder. Thus, measurement prior to the median age of first onset of disorder (18 years; Solmi et al., 2022) increased our chances of obtaining premorbid data. Second, there are established developmental differences between adults and young people on both memory specificity (Preko et al., 2023) and the executive functions that underpin specific memory retrieval (notably, the mechanisms of the CaRFAX model; Stewart et al., 2017). Finally, if a modifiable risk factor can be identified and targeted by preventive intervention (Arango et al.,

2018) in this developmental period it can change the lifetime trajectory of mental health. A focus on children and adolescents thus maximizes the practical application of our results.

Individual participant data meta-analysis (IPD-MA) synthesizes data at the individual-level, rather than study-level as completed in traditional aggregate meta-analysis. IPD-MA therefore overcomes aggregation bias (where information across studies distorts the interaction estimate) and provides stronger statistical power to rigorously assess individual-level moderators (Tierney et al., 2021). Multivariate IPD-MA also allows estimation of the correlations between mental health outcomes over time for each participant, which cannot be derived from aggregate data alone (Riley et al., 2015). Use of IPD-MA therefore offers multiple advantages for understanding how memory specificity can predict the trajectory of psychopathology.

Our primary interest was to determine whether memory specificity at baseline significantly predicted the onset of any psychiatric disorder during childhood and adolescence. To increase the interpretability of results, we also individually evaluated primary outcomes of a) depressive, b) anxiety, and c) posttraumatic stress disorders, and secondary outcomes of d) psychotic disorders and e) eating disorders. We hypothesized that reduced memory specificity would predict increased risk of any psychiatric diagnosis and greater symptom severity. To capture both fluctuation in symptoms and vulnerability to developing psychiatric disorders, we included two mental health outcomes: continuous symptom scores and dichotomous presence of psychiatric diagnosis at each follow-up time-point.

Our exploratory moderator analysis aimed to investigate whether cue valence, age, sex assigned at birth, family history of psychiatric disorder, prior trauma exposure, or elevated baseline symptoms moderated the effect of memory specificity on future symptoms.

As prior research is mixed, we did not have predictions regarding the direction of moderation effects.

Method

Transparency and Openness

Our research questions, search strategy, study selection criteria, and analysis plan were pre-registered on PROSPERO before starting the literature search (CRD42022287786). We note deviations from the protocol elsewhere in the Method. Ethics approval was obtained from the University of Melbourne (2022-23243-34518-1). We followed PRISMA-IPD guidelines in reporting this review (see Table S1 in supplementary materials for the checklist). Data owners did not consent to the public sharing of data, due to ethical restrictions. Nonetheless, analysis scripts are available through the Open Science Framework (https://osf.io/tfxbw/?view_only=ce4d2e1243c5462f82969a07e86ee7d9.).

Selection Criteria and Systematic Search Strategy

As reported in our pre-registration, we obtained known datasets from an international collaborative network (see Figure 1), including unpublished and non-peer-reviewed studies. We contacted key authors by email to request any unpublished datasets, along with calling for data in social media posts. These data supplemented formal literature searches (see Figure 1) in APA PsycINFO, Web of Science (Core Collection), EMBASE, and MEDLINE performed on 11 January 2022 using the terms ‘autobiographical memory AND adolescen* OR child*’. The search terms were shaped in accordance with the PICO framework and the full search terms are included in the Supplementary Material. Only articles published after Williams and Broadbent’s (1986) seminal article on autobiographical memory specificity were included. The inclusion criteria were (a) a measure of autobiographical memory

specificity, (b) participants aged 5-20 years[1] at baseline assessment, (c) a follow-up assessment completed at least 1-month after baseline that included at least one of our primary or secondary outcomes, (d) community-based sample (i.e., not recruited based on having a psychiatric diagnosis at baseline). We excluded studies that were non-empirical or implemented a memory-based intervention. All full texts were screened for inclusion by two independent raters (DH and LM). Raters initially agreed on reasons for exclusion for 93.8% of studies, and following discussion, the five conflicts were resolved via discussion with CH. The primary reason for disagreement was due to studies reporting on datasets that we had already identified and included. An updated literature search on 31 January 2024, and another updated search adding the terms 'OR paediatric OR pediatric' on 11 June 2024, yielded no additional articles that met our inclusion criteria.

Data Extraction and Harmonization

The corresponding author was emailed to request access to data. When needed, data transfer agreements (n = 4) and additional approval from the original ethics committee (n = 2) were arranged. For studies that reported on data extracted from a larger dataset (e.g., Warne et al. reported ALSPAC data from Boyd et al., 2013; Fraser et al., 2013), we contacted owners of the original, larger dataset. We requested un-imputed datasets (please see pre-registration for full list of requested variables). During data extraction, points for clarification were discussed with study authors. Final datasets were received September 21, 2022.

Primary Predictors and Outcomes

Memory Specificity. All identified studies used the AMT (Williams & Broadbent, 1986), where participants received either written or verbal instructions to recall a specific memory (single incident event lasting less than 24 hours) in response to a cue word of

[1] Note that we allowed for study participants to be up to 20 years old at baseline, based on an initial screen of the literature, which suggested that some studies with adolescent samples had 20 years as the upper age limit. However, ultimately all included participants were aged 18 years or younger at baseline.

negative, positive, or neutral valence. The AMT has demonstrated reliability and validity, and is widely regarded as the gold-standard measure of memory specificity (Griffith et al., 2009). The total number of cue words (n = 10-12) varied between studies. All included negative and positive cue words, with a subset also including neutral cues. Memory responses were coded by the original researchers as one of five different memory types: omission (blank or erroneous response), semantic associate (response contained content with similar meaning to the cue word, but was not a memory), categoric (memory described a category of repeated events), extended (memory of an event that lasted more than 24 hours), and specific (memory of an event that lasted less than 24 hours). Our primary predictor of memory specificity was indexed as the proportion of specific memories, calculated by dividing the number of specific memories by the total number of cues. We also separately calculated the proportion of specific memories for positive and negative cues.

Symptom Severity. As per our pre-registration, self-report and caregiver-report symptom scores on depression, anxiety and posttraumatic stress were included as three co-primary outcomes. We examined predictive effects for different symptom clusters separately. We created one variable for symptom severity per study, using self-report when data were available, and caregiver-report if there were no self-reported data. Each study used a range of different symptom scales (see Table 1). Therefore, all symptom scores were converted into z-scores, per study, to produce a standardized effect that could be used to compare effect sizes between symptom scales, studies, and statistical models. Therefore, we interpret regression coefficients (b) and 95% CI in standard deviation units.

Psychiatric Disorder Onset. Diagnostic status was determined by clinical interviews completed with participants and/or their caregiver, by trained research staff, at each time-point. Participants were coded as 1 if they met either International Classification of Diseases (ICD; World Health Organisation, 2022) or Diagnostic and Statistical Manual of Mental

Disorders (DSM; American Psychiatric Association; 2013) criteria (any edition) for diagnosis, and 0 if they did not. Psychiatric disorder onset was determined as the first time-point, either at baseline or follow-up, that participant met criteria for any diagnosis. Included diagnoses were depressive disorders (major depressive disorder, minor depression, dysthymia, depression, and combined minor and major depression), anxiety disorders (generalized anxiety disorder, panic disorder, and social phobia), bipolar disorders (type 1 and type 2 bipolar disorder), stress disorders (acute stress disorder and posttraumatic stress disorder; PTSD), eating disorders (anorexia nervosa, bulimia nervosa, binge eating disorder, and eating disorder not otherwise specified), and obsessive compulsive disorder. On obtaining the individual datasets, we were fortunate to receive previously unpublished data, which allowed us to complete additional analyses for disorders that we had not anticipated.

Meta-Analytic Moderators and Measurement Characteristics

Age and Sex Assigned at Birth. We converted all participants' age into years. All studies included two categories for sex assigned at birth, male (coded as 0) and female (coded as 1).

Pre-existing Depressive Symptoms. For moderation analyses only, we used a binary measure of prior depression, as use of z-scores across the different symptom scales prohibited the use of mean centering (see Analysis Approach section). Because all studies included baseline depressive symptom score, we classified each participant as being above or below the clinical cut-off on the relevant self-report or other-reported measure, determined separately for each measure.

Trauma and Adverse Childhood Experiences. For trauma history, we recorded those individuals identified by either self-report or caregiver-report as having experienced a potentially traumatic event. Across the studies, this was primarily defined as meeting Criterion A of DSM criteria for PTSD and decided by study researchers on the basis of

caregiver interviews or trauma history questionnaires (see Table 1). We coded prior exposure to potentially traumatic events as 1 and no prior exposure as 0. For studies where trauma experience was an inclusion criterion, we coded 1 for trauma experience prior to the indexed event, and 0 for absence of trauma experience prior to the indexed event.

Family History. As reported in our pre-registration, we sought to include family history of any psychiatric disorder. However, in the included studies, only family history of depression was reported. Where one or more parents were reported by either the young person or caregiver as having a history of depression, the participant was coded 1 as having a family history of depression. Participants were coded 0 if neither parent was reported as experiencing depression. Included datasets did not indicate which depressive disorder the parent had experienced, or when.

Risk of Bias Assessment. As we had pre-registered, two raters (DH and LM) independently rated the Risk of Bias of all included studies using the Cochrane Risk of Bias 2 tool (Sterne et al., 2019). Three domains were assessed: bias due to missing outcome data, bias in outcome measurement, and bias in selection of reported results. Domain of bias from randomization and deviation from intended intervention were not assessed as they were not applicable to the studies in this review. Since pre-registration, the Risk Of Bias In Non-randomized Studies - of Exposures (ROBINS-E) tool has been published (ROBINS-E Development Group, 2023), which provides a more appropriate fit to our data. Therefore, we also report risk ratings according to the ROBINS-E (see Table S2 in the Supplementary Material for comprehensive ratings). Funnel plots of the main outcomes were used to visually examine publication bias.

Evidence Certainty Assessment. Confidence in the quality of synthesized evidence was assessed using the GRADE framework (Balshem et al., 2011; Guyatt et al., 2011) for each outcome. Due to residual confounding factors in observational studies, all evidence in

this review starts at low quality, which means that the true effect might be markedly different from the estimated effect. We report any changes to this rating when describing results.

Analysis Approach

Analyses followed recommendations for individual participant data meta-analysis (Riley et al., 2021). We had pre-registered a one-stage analysis approach and used one-stage analysis for disorder onset and assessment of moderators. Small deviations from our pre-registration were required for analysis of other outcomes, as follows; Timing of the follow-up assessments varied from 2-48 months (see Table 1) for symptom severity on our co-primary outcomes. A univariate one-stage approach would necessitate selecting one time-point for analysis and ignore the correlation between the different time-points (Jones et al., 2009; Riley et al., 2007). Thus, a multivariate analysis was deemed more statistically rigorous (Riley et al., 2007; Riley et al., 2015), and multivariate analysis is most effectively facilitated via a two-stage approach (Jones et al., 2009; Riley et al., 2007; Riley et al., 2015). We therefore report multivariate models for symptom severity, and a sensitivity analysis using the pre-registered one-stage approach.

Disorder Onset. To index the transdiagnostic effect of memory specificity, we completed a univariate mixed-effect time-to-event Cox survival model with stratified intercept by study, using maximum likelihood estimation. The outcome was time until first diagnosis of any psychiatric disorder, across all assessment points. The model allowed for the inclusion of all time-points and allowed estimation of the variable hazard ratio of being diagnosed with a psychiatric disorder from baseline to 8.3 years post-baseline. As a follow-up analysis, we then completed separate Cox survival models for depressive, anxiety, eating, and trauma-related disorders. As sensitivity analysis, we additionally completed a random-effect Cox survival model, which allows baseline hazard rate to vary across studies while not directly estimating them, thus reducing the number of parameters estimated. For Cox models,

heterogeneity is quantified using τ^2, which is the variance of the random-effects modelled, such that τ^2 represents the between-study heterogeneity of the predictive effects.

Symptom Severity. We first completed separate linear fixed-effects models for each study. Memory specificity predicted symptoms at different time-points covarying for baseline symptoms. Separate models were conducted for depression, anxiety, and posttraumatic stress. Follow-up assessments were grouped in six-month blocks (see Table 1 for assessment time-points for each study). Next, these estimates and associated standard errors were combined in a multivariate random effects meta-analysis, with random effect adjustment for each study and restricted maximum likelihood estimation. Age at baseline, sex assigned at birth, and IQ were not recorded in all identified studies. We therefore report models without these covariates to allow for inclusion of all obtained data in primary analysis. We did compare results to sensitivity analysis including these variables. We also conducted a sensitivity analysis using only self-report symptoms scores as opposed to combining self-report and observer-report. The self-report only model yielded similar results, and for completeness we report analyses across all raters. Finally, we repeated analyses with unstandardized symptom scores (rather than z scores), which again yielded similar results. Heterogeneity was assessed using the multivariate Q_E-test for residual heterogeneity. This tests whether the variability among the observed outcomes is larger than expected given random sampling variances and covariances among sampling errors. A low *p*-value indicates true outcomes are heterogeneous between studies (Deeks et al, 2023). In addition, we also report I^2, which is the percentage of variability in the observed outcomes that is not due to heterogeneity and chance alone. I^2 of 0% indicates no heterogeneity, less than 40% indicates minimal heterogeneity, and higher than 75% indicates considerable heterogeneity (Deeks et al., 2019).

Moderation Analyses. We completed separate one-stage univariate models for each moderator, with restricted maximum likelihood estimation. Symptom severity was predicted

by memory specificity, the centered within- and across-study interaction terms between memory specificity and the moderator, and baseline symptom severity as covariates. Most studies that reported on our pre-registered moderators also indexed depressive symptoms. We therefore considered modifiers of the effect of memory specificity on future depressive symptoms only. As aggregate data meta-analysis indicates that the predictive effect of memory specificity on depression weakens over time (Hallford et al., 2021), we selected the closest follow-up assessment for each study. Within- and across-study interactions were separated by mean centering the moderator within each study and across all studies, to account for aggregation bias in one-stage, univariate models. Within-study interaction terms were computed from baseline memory specificity and the individual deviation from the study mean (e.g., individual age minus mean age for the study). Across-study interaction terms were computed from baseline memory specificity and the study mean. Across-study interactions were included in analyses to adjust for aggregation bias, but in the results, we report the within-study interactions between baseline memory specificity and each moderator to illustrate the effect at the participant-level. The three main predictors (baseline memory specificity, individual deviation from study mean, and study mean), baseline depressive symptoms, and time of follow-up were included as fixed effects.

Missing Data. All models used restricted maximum likelihood estimation. The complexities of the statistical models prohibited the use of multiple imputation for missing data, which is not uncommon for IPD-MA with large datasets (see Dora et al., 2019). To consider the potential bias arising from missing data, point-biserial correlations investigated whether age, sex, baseline memory specificity, and baseline symptoms were associated with missing symptom severity outcomes or missing diagnostic outcomes.

Results

Overview of Included Studies

Figure 1 shows the PRISMA flowchart for studies at identification, screening, and inclusion stage. Study characteristics for included datasets are presented in Table 1. One eligible dataset (Stewart et al., 2018) was unavailable due to limits to consent under which the data were obtained. Only one study reported on psychosis symptoms, prohibiting meta-analysis. For disordered eating, no studies included self-report symptom measures, but two (14.3%) studies did measure diagnostic status. Obsessive compulsive disorder contributed to the any psychiatric diagnosis analysis, but no other analyses.

The final dataset included 14 studies with 9165 young people aged 6 - 18 years (M = 13.1, SD = 1.3). Most participants (N = 5171, 56.4%) were assigned female at birth. Ten studies (71.4%) used verbal format for the AMT, and four (28.6%) used written format. For our primary outcomes, thirteen studies (92.9%) included symptom severity outcomes, and five studies included diagnostic outcomes (35.7%). For moderators and measurement characteristics, all studies included age and sex assigned at birth, 13 studies (92.9%) included cue valence, six (42.9%) studies had information on family history of depression, and five (35.7%) studies included prior trauma history.

Figure 1

Study Screening and Inclusion

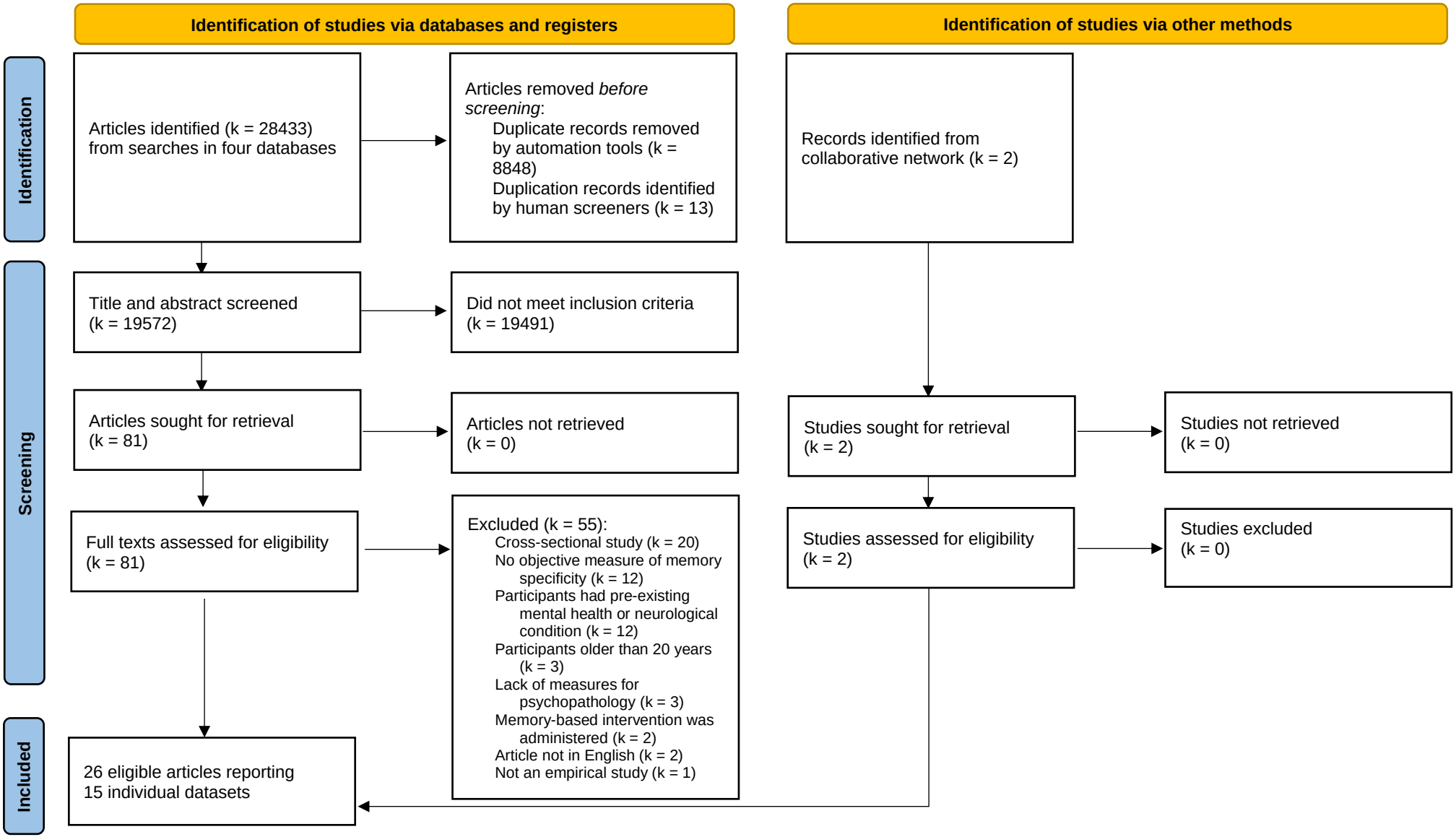


Table 1

Characteristics of Included Studies

Study	Site	Age Range	n	Female (%)	Ethnicity (n)	AMT Characteristics	Follow-up (Month)	Depression Measure	Anxiety Measure	PTSD Measure	Clinical Interview	Trauma History Measure (%)	Family History (%)	RoB/ ROBINS-E
Askelund et al. (2019)	UK	12-16	930	45.16%	-	Written format 60s to respond 6 positive cues, 6 negative cues	6, 12	MFQ	-	-	KSADS	-	43.87%	Some/ Low
Gutenbrunner et al. (2018)	NZ	10-15	269	46.10%	-	Written format 60s to respond 5 positive cues, 5 negative cues	12, 23	CDI-Short form	Revised Children's Manifest Anxiety Scale	-	-	-	-	Some/ Low
Hamlat et al. (2015)	US	11-16	397	52.14%	191 Caucasian 192 African American 14 Other	Verbal format 60s to respond 3 positive cues, 3 neutral cues, 3 negative cues	6, 12, 18, 24, 30, 36, 42, 48, 54, 60, 66, 72, 78, 84, 90, 102	CDI	Multi-dimensional Anxiety Scale for Children	-	KSADS	-	11.08%	Low/ Low
Hiller et al. Unpublished	UK	10-16	28	64.29%	NI	Verbal format 60s to respond 5 positive cues 5 negative cues	6	Revised Child Anxiety and Depression Scale	-	-	-	CATS 75.00%	-	
Hipwell et al. (2011)	US	10-12	235	100%	65 European American 156 African American	Verbal format 30s to respond 5 positive cues, 5 negative cues	12	K-SADS	-	-	KSADS	-	-	Low/ Some

Study	Site	Age Range	n	Female (%)	Ethnicity (n)	AMT Characteristics	Follow-up (Month)	Depression Measure	Anxiety Measure	PTSD Measure	Clinical Interview	Trauma History Measure (%)	Family History (%)	RoB/ ROBINS-E
Hitchcock et al. (2014)	Aus	7-17	75	34.67%	Not assessed	Verbal format 60s to respond 5 positive cues, 5 negative cues	2, 5	CDI-Short Form	-	CPSS	-	CLDM (66.22%)	-	Some/ Low
Hitchcock 2 Unpublished	UK	9-17	195	53.33%	Not assessed	Written format 60s to respond 5 positive cues, 5 negative cues	8	CDI	Spence Children's Anxiety Scale	CPSS	-	-	-	
Nixon et al. (2013) Study 1	Aus	7-17	67	37.31%	55 White 2 Indigenous Australian 2 Other	Verbal format 60s to respond 5 positive cues, 5 negative cues	2	CDI	-	CPSS, CPTRI	-	Parent interview (53.85%)	26.15%	Some/ Low
Nixon et al. (2013) Study 2	Aus	6-16	67	41.79%	64 White 1 Arab 1 Indigenous Australian	Verbal format 60s to respond 5 positive cues, 5 negative cues	2	CDI	-	CPSS	-	Parent interview (67.16%)	40.30%	Some/ Low
Puetz et al. (2021)	UK	9-14	41	51.22%	25 Caucasian 16 Non-Caucasian	Verbal format 60s to respond 10 positive cues, 10 negative cues	27	CASI-Subscale	CASI-Subscale	-	-	CTQ (46.34%)	-	Low/ Some
Rawal & Rice (2012)	UK	10-18	259	60.23%	257 British 1 Other European 1 Other	Verbal format 30s to respond 6 positive cues, 6 negative cues	12	MFQ	-	-	CAPA	-	-	Low/ Some
Sumner et al. (2014)	US	16-18	333	69.37%	164 Caucasian 11 Asian 41 African American	Verbal format 30s to respond 6-8 positive cues,	4, 16, 28, 40, 52, 64, 76, 88, 100	-	-	-	SCID	-	-	Low/ Low

Study	Site	Age Range	n	Female (%)	Ethnicity (n)	AMT Characteristics	Follow-up (Month)	Depression Measure	Anxiety Measure	PTSD Measure	Clinical Interview	Trauma History Measure (%)	Family History (%)	RoB/ ROBINS-E
					117 Other	6-8 negative cues								
Warne et al. (2020)	UK	12-14	5792	56.84%	5559 Caucasian 43 Asian 37 Black 85 Other	Written format No time limit 5 positive cues, 5 negative cues	48, 72, 108	MFQ-short form	-	-	-	-	17.90%	Some/ Low
Woody et al. (2021)	US	8-15	256	50.39%	203 White 3 Asian 12 Black 1 Indigenous American 29 Other	Verbal format 60s to respond 5 positive cues, 5 negative cues	6, 12, 18, 24	CDI	Multi-dimensional Anxiety Scale for Children	-	-	-	50.20%	Low/ Low

Note. For datasets reported in multiple articles, this table includes one article from which we extracted methodological details and completed risk of bias rating.

AMT = Autobiographical Memory Test; RoB = Cochrane Risk of Bias; ROBINS-E = Risk of Bias in Non-Experimental Studies – of Exposure. CDI = Children's Depression Inventory; MFQ = Moods and Feelings Questionnaire; CASI = Child and Adolescent Symptom Inventory Subscale; KSADS = Kiddie Schedule for Affective Disorders and Schizophrenia; SCID = Structured Clinical Interview for DSM-IV; CAPA= Child and Adolescent Psychiatric Assessment' CPSS = The Child Post-Traumatic Stress Scale; CPTRI = Child Post-Traumatic Stress Reaction Index; CATS = Child and Adolescent Trauma Screen; CLDM = Cambridge Life Development Measure; CTQ = Childhood Trauma Questionnaire. Family history is for depressive disorders.

Onset of Psychiatric Disorder

Missing Data

Missing diagnostic outcome data did not significantly (*ps* > .05) correlate with participant's age (r = -.03), sex (r = -.03), or baseline memory specificity (r = .06).

Any Psychiatric Diagnosis

There were 423 participants who received a diagnosis of any psychiatric disorder, out of 2138 participants who were interviewed up to 102 months post-baseline, across five studies. Memory specificity significantly predicted the first onset of any psychiatric diagnosis in participants (n = 1983) who did not have a diagnosis at baseline (hazard ratio = 0.55, 95% CI [0.33, 0.90], p = .018; Figure 2). To improve the interpretability of the results, we describe the reversed hazard ratio, to explain the effect of *reduced* memory specificity on risk of receiving a diagnosis. Relative to someone who provided a specific memory for every cue on the AMT, every 10% decrease in memory specificity was associated with an 6.2% increase in the hazard of receiving a psychiatric diagnosis.[2] Further, that 10% decrease was associated with receiving the diagnosis 5.8% faster. Heterogeneity across studies was low, $\tau^2 < 0.001$.

Including participants with a diagnosis at baseline can increase accuracy of the estimated hazard ratio (e.g., Abd Elhafeez et al., 2021). The pattern and significance of effects did not change when including participants with a diagnosis at baseline (n = 155 of 2138 participants), hazard ratio = 0.34, 95% CI [0.24, 0.48], p <.001. Every 10% decrease in memory specificity was associated with an 11.5% increase in the hazard of receiving a psychiatric diagnosis and receiving diagnosis 10.3% faster.

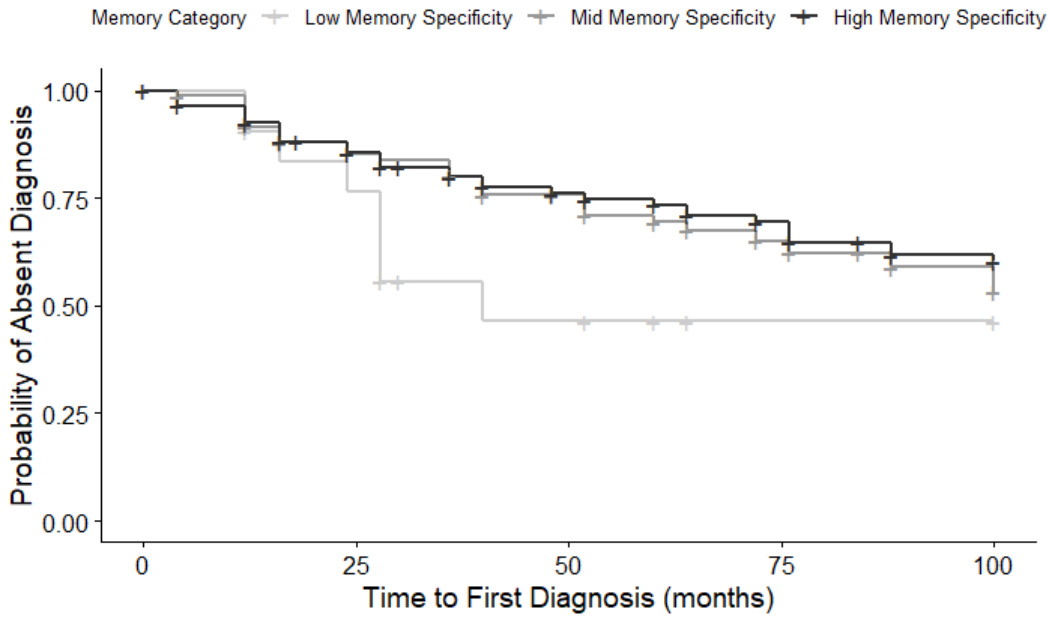
[2] As a reminder, there was variability in the number of cues between datasets, thus we are interpreting the change in the proportion correct, rather than a change for each additional specific memory the individual fails to retrieve. Although a 10% increase in specificity would be one specific memory on an AMT with ten cue words, this would not be true for a twelve cue word AMT.

Using the GRADE criteria, evidence for this outcome was upgraded from low to moderate quality due to low heterogeneity, and as missing data were unlikely to strongly bias this result.

We also explored this relationship separately for specificity to positive and negative cues. Both specificity to positive cues (hazard ratio = 0.38, 95% CI [0.26, 0.53], p <.001) and specificity to negative cues (hazard ratio = 0.51, 95% CI [0.32, 0.81], p = .005) significantly predicted first onset of any psychiatric diagnosis.

Figure 2

Time-To-Event of First Onset of Any Psychiatric Disorder for Adolescents With Low, Mid, And High Memory Specificity



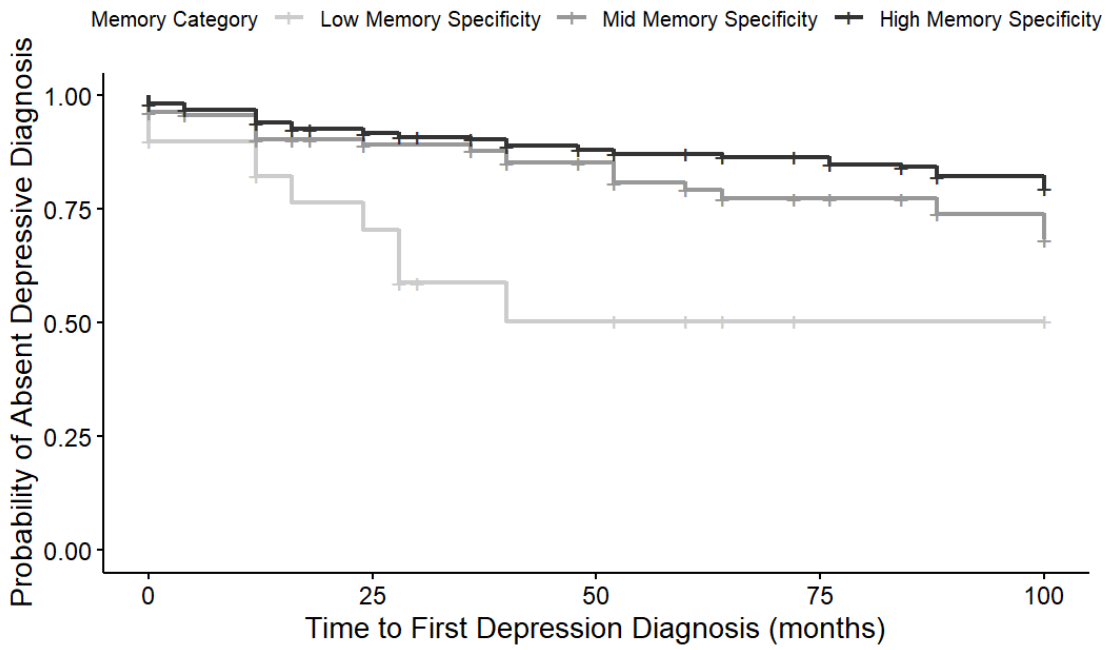
Note. Low, mid, and high memory specificity groups were categorized based on proportion of correct responses on the Autobiographical Memory Test as follows: low memory specificity group ranged between 0.0 - 0.33, mid memory specificity group ranged between 0.34 - 0.66, and high memory specificity ranged between 0.67 - 1.0.

Depressive Disorders. There were 234 participants who received a diagnosis of any depressive disorder, out of 2138 participants who were interviewed up to 102 months post-baseline, across five studies. Baseline memory specificity significantly predicted the first

onset of any depressive disorder (hazard ratio = 0.21, 95% CI [0.13, 0.33], $p < .001$; Figure 3). For every 10% decrease in memory specificity, the hazard of being diagnosed with a depressive disorder increases by 16.9%, while the time to onset reduces by 14.4%. Heterogeneity across studies was low, indicated by the small variation in the random effect of memory specificity by study ($\tau^2 < 0.001$). The evidence for this outcome was again upgraded from low to moderate quality due to the large hazard ratio observed, as per the GRADE criteria.

Figure 3

Time-To-Event for First Depressive Disorder Onset For Adolescents With Low, Mid, And High Memory Specificity



Note. Low, mid, and high memory specificity groups were categorized based on proportion of correct responses on the Autobiographical Memory Test as follows: low memory specificity group ranged between 0.0 - 0.33, mid memory specificity group ranged between 0.34 - 0.66, and high memory specificity ranged between 0.67 - 1.0.

Eating Disorders. Eighteen participants received a diagnosis of any eating disorder, out of 1258 participants who were interviewed up to 100 months post-baseline, across two studies. The mixed-effect Cox survival model converged early, possibly due to the small number of positive cases and the complexity of the mixed-effect model. Therefore, we used a

proportional hazard Cox survival model with baseline hazard rate stratified by study. Baseline memory specificity significantly predicted the first onset of any eating disorder (hazard ratio = 0.12, 95% CI [0.03, 0.58], p = .008). For every 10% decrease in memory specificity, the hazard of being diagnosed with any eating disorder increased by 23.6% while the time to first eating disorder onset decreased by 19.1%.

Other Psychiatric Diagnoses. Memory specificity did not predict the first onset of anxiety disorders (n = 1650, $n_{diagnosed}$ = 218, hazard ratio = 0.59, 95% CI [0.34, 1.01], p = .055), posttraumatic stress disorders (n = 1650, $n_{diagnosed}$ = 12, hazard ratio = 0.62, 95% CI [0.04, 8.55], p = .720), or bipolar disorders (n = 720, $n_{diagnosed}$ = 10, hazard ratio = 29.25, 95% CI [0.17, 5132.22], p = .200).

Sensitivity analysis. To test the robustness of results against different model assumptions and number of parameters estimated, we repeated each analysis using random-effect Cox models without separate stratified hazard per study. Where data was available, age, sex, and IQ were also entered as covariates. The pattern and significance of all disorder onset models did not change in any sensitivity analyses.

Symptom severity

Missing Data

There was no strong association (ps > .05) between missing symptom severity outcomes and participants' age (r = - .04), sex (r = .10), baseline memory specificity (r = .11), or baseline depressive symptoms (r = .04).

Depressive Symptoms

Outcomes were grouped as 2-6 months, 7-12 months, 13-18 months, 19-24 months, and 25-48 months (k = 13 across all time-points). Memory specificity did not significantly predict depressive symptoms at any time-point (see Table 3). Multivariate Cochran Q_E-test for heterogeneity revealed a non-significant degree of heterogeneity between studies, $Q(60)$ =

39.87, $p = .979$, $I^2 = 1.00\%$. See Figure 4 for forest plots of each time-point. According to the GRADE criteria, the strength of the evidence is low.

Table 3

Unstandardized Estimates and 95% Confidence Interval for Effect of Memory Specificity on Each Symptom Cluster.

Symptom type	Assessment time-point (months post-baseline)				
	6-Months	12-Months	18-Months	24-Months	48-Months
Depressive	-0.07 [-0.22,0.08]	0.02 [-0.20,0.24]	-0.22 [-0.47,0.03]	-0.03 [-0.33,0.27]	-0.76 [-2.00,0.48]
Anxiety	0.46 [-0.37,1.28]	0.41 [-0.07,0.88]	0.77 [0.34,1.14]***	0.80 [0.10,1.50]*	-
	2-Months	5-Months	8-Months	-	-
PTSD	-0.36 [-1.03,0.32]	0.33 [-0.37, 1.02]	0.21 [-0.34,0.77]	-	-

Note. Unstandardized regression estimates and their [95% CIs] obtained from multivariate mixed-effect models for each symptom cluster, across all time-points. A positive estimate indicates that specificity predicts higher symptoms, and a negative estimate indicates that specificity predicts lower symptoms.
*$p < .05$, **$p < .01$, ***$p < .001$

Table 4

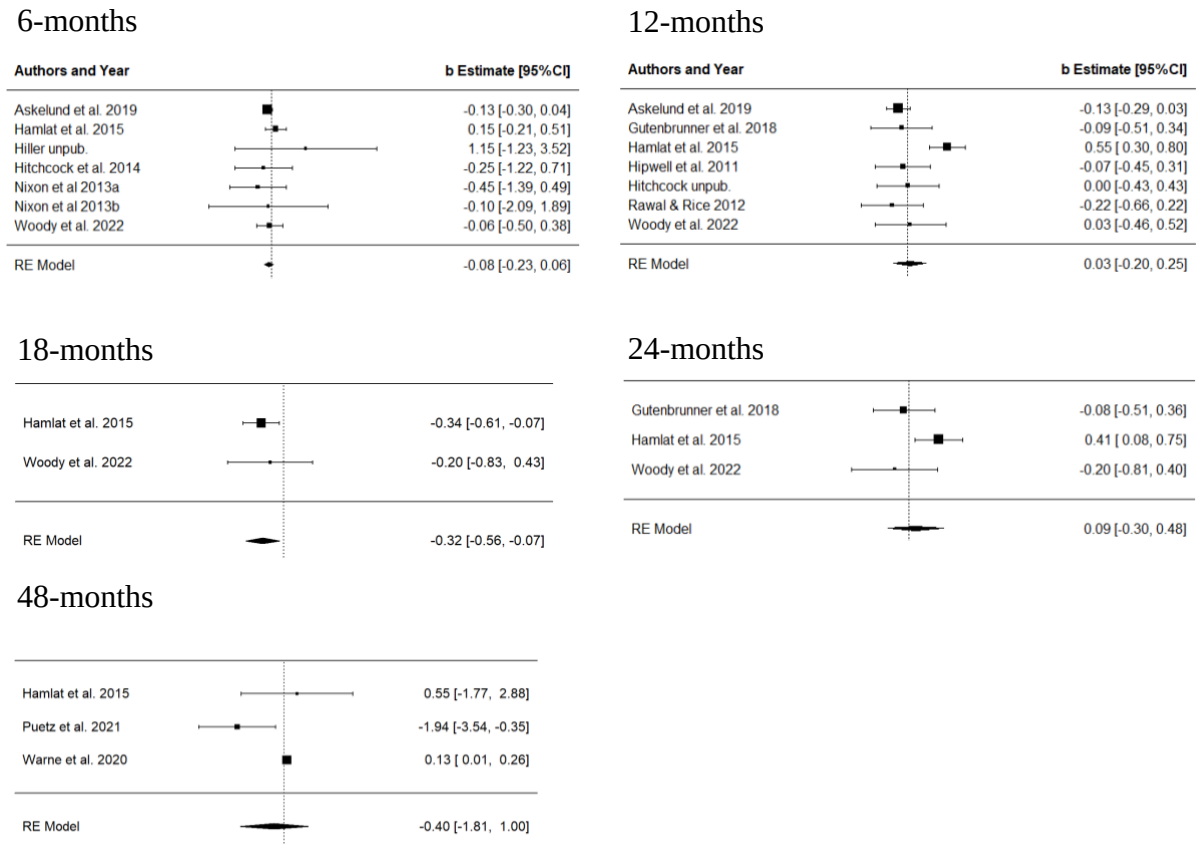
Unstandardized Estimates and 95% Confidence Intervals for Effects of Positive and Negative Memory Specificity on Depressive Symptoms.

Cue Valence	Assessment time-point (months post-baseline)				
	6-Months	12-Months	18-Months	24-Months	48-Months
Positive	-0.05 [-0.18,0.09]	-0.06 [-0.17,0.05]	0.05 [-0.41,0.50]	-0.07 [-0.35,0.22]	0.06 [-0.04,0.17]
Negative	-0.14 [-0.28,-0.0004]*	-0.07 [-0.18,0.05]	-0.42 [-0.91,0.07]	-0.15 [-0.46,0.17]	-0.45 [-1.92,1.10]

Note. Unstandardized regression estimates and their [95% CIs] from multivariate mixed-effect models for each type of cue valence, across all time-points. A positive regression estimate indicates that specificity predicts higher symptoms, and a negative estimate indicates that specificity predicts lower symptoms.
*$p < .05$, **$p < .01$, ***$p < .001$

Figure 4

Memory Specificity Predicting Depressive Symptoms at Each Time-point Post-Baseline, Controlling for Baseline Depressive Symptoms.



Note. The vertical dashed line indicates that the *b* estimate equals 0 (i.e., no effect). The left of the line represents lower depressive symptoms, and the right of the line represents higher depressive symptoms. Error bars represent 95% CIs. As forest plots cannot be created from multivariate models, for illustration purposes, the plots above were plotted from univariate, linear mixed-effect models at each time-point. We display plots for each time-point separately, however, these time-points were all included in one multivariate model such that covariance between multiple time-points within one study was accounted for while estimating coefficients of each outcome.

We next considered the effect of cue valence (see Table 4) by completing two separate multivariate models for memory specificity in response to positive and negative cues (k = 12). Positive memory specificity did not significantly predict depressive symptoms at any time-point. Multivariate Cochran Q_E-test for heterogeneity revealed non-significant heterogeneity between studies, Q_E (55) = 9.80, p = 1.000; I^2 = 1.00%. According to the GRADE criteria, the strength of the evidence is low.

In contrast, greater specificity to negative cues did predict significantly lower depressive symptoms 6-months later, but not at other follow-up time-points. Multivariate Cochran Q_E-test for heterogeneity revealed non-significant heterogeneity, Q_E (55) = 10.99, p = 1.000; I^2 = 1.00%. According to the GRADE criteria, the strength of the evidence is low.

Finally, given the discrepancy in results between the symptom severity and diagnostic analyses, we completed an exploratory analysis to determine whether memory specificity at baseline predicted scores above the clinical cut-off on the depressive symptom scale at follow-up. We used a mixed-effect Cox model, with stratified intercept by study and random effect of memory specificity per study. There were 1675 participants who were above the clinical cut-off, out of 7803 participants who had follow-up data up to 108-months post-baseline, across 11 studies. Baseline memory specificity did not significantly predict the hazard of exceeding the clinical cut-off on a depressive symptom scale (hazard ratio = 0.83, 95% CI [0.58, 1.18], p =.290).

Anxiety Symptoms

In a separate multivariate model (k = 4), we found that memory specificity did not significantly predict anxiety symptoms (see Table 3) at 6-months and 12-months but did predict symptoms 18-months and 24-months later. Contrary to expectations, the effect size indicated that higher memory specificity predicted higher anxiety symptoms. Multivariate Cochran Q_E-test for heterogeneity revealed evidence for moderate heterogeneity between

studies, $Q_E(12) = 22.36$, $p = .034$; $I^2 = 46.30\%$; thus, in accordance with the GRADE criteria, the quality of evidence was downgraded from low to very low.[3] There were insufficient data to explore effects separately for positive and negative cues.

Posttraumatic Stress Symptoms

Memory specificity did not predict posttraumatic stress symptoms ($k = 3$) at any time-point (see Table 3). There was non-significant heterogeneity between studies, Q_E (9) = 0.99, $p = 1.000$; $I^2 = 1.00\%$. According to the GRADE criteria, the strength of the evidence is low. Again, there were insufficient data to explore valence effects.

Sensitivity Analysis

The pattern and significance of the depression, anxiety and posttraumatic stress symptom severity results remained the same when we used univariate models, unstandardized symptom severity scores, and included age, sex, and IQ, with one exception. When covarying for age, sex, and baseline IQ ($k = 3$, $n = 1202$), memory specificity significantly predicted depressive symptoms 12-months later ($b = -0.17$, 95% CI [-0.32, -0.02], $p = .031$). Sex ($b = 0.21$, 95% CI [0.12, 0.30], $p < .001$) and baseline IQ ($b = 0.003$, 95% CI [0.0007, 0.006], $p = .014$) were also significant predictors of depressive symptoms 12-months later.

Finally, to explore a transdiagnostic effect on symptom severity, we completed a multivariate model with different symptom clusters as outcomes ($k = 13$). Where a study had multiple follow-ups for each symptom group, the earliest follow-up assessment was used. Symptoms at baseline were entered as a covariate. Multivariate test of moderators revealed no significant effect of symptom cluster on the relationship between memory specificity and future symptoms, $Q_M(4) = 3.56$, $p = .468$. Memory specificity did not predict future

[3] In exploratory follow-up analyses, we completed a multivariate model with four outcomes: depressive symptoms at 18- and 24-months, and anxiety symptoms at 18- and 24-months, to explore whether this finding held when covariance with depressive symptoms was considered. We observed a significant, positive effect of memory specificity on anxiety symptoms at 18-months ($b = 0.74$, 95% CI [0.22, 1.27], $p = .006$), but not at 24-months ($b = 0.58$, 95% CI [-0.12, 1.27], $p = .104$), nor for depressive symptoms at 18-months ($b = -0.20$, 95% CI [-0.70, 0.29], $p = .416$), and 24-months ($b = 0.01$, 95% CI [-0.39, 0.41], $p = .958$).

depressive symptoms (b = -0.03, 95% CI [-0.15, 0.10], p = .682), anxiety symptoms (b = 0.36, 95% CI [-0.11, 0.82], p = .134), posttraumatic stress symptoms (b = -0.11, 95% CI [-0.60, 0.38], p = .660), or psychosis symptoms (b = -0.01, 95% CI [-0.14, 0.12], p = .874). There was no significant heterogeneity between studies, $Q(49) = 33.72$, p = .941, I^2 = 1.00%.

Moderators of Effect

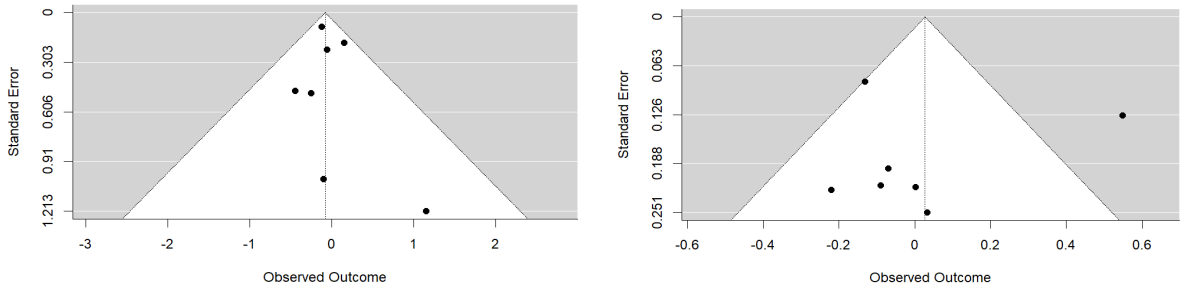
For each hypothesized moderator, we completed a one-stage, univariate random effects model predicting depressive symptoms. Contrary to expectations, the predictive effect of memory specificity on depressive symptoms was not moderated by age of the participant, b = 0.003, SE = 0.05, $t(5901) = 0.06$, p = .950, sex assigned at birth, b = -0.17, SE = 0.10, $t(5896) = -1.68$, p = .094, prior exposure to a potentially traumatic event, b = 0.40, SE = 0.44, $t(185) = 0.92$, p = .357, family history of depression, b = -0.10, SE = 0.16, $t(2933) = -0.63$, p = .532, or own prior experience of depression, b = 0.24, SE = 0.26, $t(5659) = 0.94$, p = .350.

Risk of Bias

Cochrane and ROBINS-E risk of bias ratings for each study are presented in Table 1. All studies demonstrated either low or some risk to bias, with none rated as high risk of bias. Main threats to bias included potentially biased selection of reported outcomes due to the lack of pre-registration, and missing outcome data due to high attrition. To allow comparison to prior aggregate data meta-analyses (Barry et al., 2021; Hallford et al., 2021) we examined funnel plots to assess publication bias in depressive symptoms at six and twelve-months post-baseline. Funnel plot asymmetry demonstrates some evidence for publication bias, although this appears to vary across time-points. This is consistent with prior aggregate data meta-analyses (Barry et al., 2021; Hallford et al., 2021)

Figure 5

Funnel plots



Note. The left and right panel demonstrate the regression coefficient and its standard error for memory specificity predicting depressive symptoms at 6-months and 12-months, respectively.

Discussion

This pre-registered IPD-MA evaluated whether autobiographical memory specificity prospectively predicts the first onset of psychiatric disorders and later symptom severity for depression, anxiety, and posttraumatic stress in over 9,000 young people. Using individual participant data collected from community-based samples of children and adolescents over multiple years also allowed us to explore moderators of prospective relationships, without conflating person- and study-level features.

Disorder Onset

We provided the first meta-analytic evidence that reduced memory specificity on the AMT is a transdiagnostic risk factor for first incidence of psychiatric disorders. Reduced memory specificity significantly increased odds of a future diagnosis of any DSM or ICD-defined psychiatric disorder and significantly reduced the time to first onset of that disorder. Interestingly, this effect was driven by those with the lowest scores for memory specificity. There was very little difference in the risk associated with moderate or high memory specificity, consistent with the theory (Williams et al., 2007) that mental illness only arises if reduced specificity is used in a chronic and entrenched manner. Indeed, it is suggested that sporadic avoidance of specific memories may in fact be functional (c.f., the functional

avoidance hypothesis; Williams et al., 2007). Longitudinal data from community-based samples of young people is a strength of this analysis, as establishing temporal precedence of the risk factor before disorder onset is one (of many) criterion for causal inference. Low heterogeneity supports the consistency across studies and lends confidence to results.

We build upon prior meta-analyses indicating that reduced memory specificity is a transdiagnostic cross-sectional indicator of psychiatric disorder (Barry et al., 2021), by demonstrating that memory specificity is a predictor of disorder onset. Examination of hazard ratios for different disorder types suggested that this effect was strongest for depressive disorders and eating disorders. However, the number of eating disorder cases was very low. Although this may be expected given the low prevalence of eating disorders in the community (Hoek & van Hoeken, 2003), it does limit conclusions. The hazard ratio was non-significant for anxiety and posttraumatic stress disorders. Future research will benefit from large datasets (e.g., embedding memory measures in cohort studies) with a greater number and variety of diagnosed cases. Overall, our findings suggested that reduced memory specificity in childhood and adolescence may represent a transdiagnostic risk factor for first onset of a psychiatric disorder, but most robustly for onset of a depressive disorder.

Symptom Severity

Although predictive effects were observed for disorder diagnosis, reduced memory specificity on the AMT did not predict future symptom severity for depression, or posttraumatic stress. Reduced specificity thus does not appear to be a transdiagnostic predictor of future symptom severity in community-based samples. The difference in results between diagnosis and symptom severity could reflect differences in measurement characteristics. That is, memory specificity may predict clinician ratings of symptoms (as occurs for diagnosis) but not self- or caregiver-report of symptoms (as indexed by symptom severity questionnaires). Indeed, exploratory analyses indicated that memory specificity did

not significantly predict scores above the clinical cut-off on self-report depressive symptom scales. Future consideration of who is reporting symptoms might help to evaluate this possibility. Similarly, it might be interesting to explore the relationship between memory specificity and meta-cognitive awareness, as meta-cognitive impairments are also associated with poor mental health (see Seow et al., 2021).

The same datasets were used in both diagnosis and symptom severity analysis, however, studies with diagnostic data were typically from larger studies with British or American adolescents. Similarly, on average, participants in studies that measured diagnosis were significantly older than participants in studies that examined symptom severity. Indeed, younger children (aged 6-9 years) were not included in the studies with diagnostic data. This is not surprising, as most studies indexed depressive diagnoses which are infrequent in younger children. There was no evidence that age moderates the risk of memory specificity on future depressive symptoms. Yet, it is possible that there are developmental effects, such as differences in cognitive operations in younger populations, that impact the relationship between memory and psychopathology (see Valentino, 2011 for further discussion). Sample characteristics should thus be considered in interpretation of results. Symptom data were available from large sample sizes, we had adequate power to detect a small effect, and results suggested low heterogeneity. The inconsistent findings between diagnosis and symptom severity are thus surprising and reveal interesting possibilities.

The one analysis that did demonstrate high heterogeneity between studies and high unexplained variance was for anxiety symptom severity. While these factors reduce confidence in results, with the GRADE rating being low to very low, higher memory specificity predicted more severe anxiety symptoms 18- and 24-months later. A heightened focus on concrete, body-specific information in memory is maladaptive for anxiety symptoms (von Spreckelsen et al., 2022). Thus, the content of the memory might be important. The

reduced ability to isolate specific episodes in memory, and the level of detail within those episodes, are separable constructs (for discussion see Barry et al., 2023). Yet, it is unclear how episodic detail and specificity interact to predict mental health. Future research is needed to clarify how the content of specific memories influences anxiety in youth.

The interaction between memory specificity and other risk factors for anxiety (e.g., rumination; see Gutenbrunner et al., 2018) also needs further investigation. There are prior cross-sectional studies which demonstrate no significant relationship between specificity and anxiety symptoms (e.g., Wessel et al., 2001), with meta-analysis indicating no significant difference in specificity between anxious and non-anxious control groups (Barry et al., 2021). There may be study-level (e.g., adolescent vs adult samples) and participant-level moderators that influence the relationship between specificity and anxiety. However, with the current data were unable to consider participant-level moderators for anxiety (or posttraumatic stress and eating disorder symptoms). In particular, we were unable to explore whether positive and negative specific memories had different effects on anxiety. Given the high comorbidity between depression and anxiety, we need to better understand how specificity impacts anxiety before using memory specificity interventions for prevention.

It was particularly surprising that specificity did not predict depressive symptoms, as prior meta-analyses have reported a significant predictive effect in depressed adults (Hallford et al., 2021; Sumner et al., 2010). Discrepant findings could be influenced by developmental differences in depressive etiology and symptom presentation between young people and adults. We found little evidence of moderation by participant age, sex assigned at birth, trauma exposure, or own or family history of depression, suggesting that specificity is not simply a risk factor that is only at play for those already at risk of mental illness.

There are several potential explanations for a predictive effect on depressive diagnosis, but not symptom severity. Our results may suggest that reduced memory

specificity can predict the emergence of the more chronic, functionally impairing level of symptoms that are needed for psychiatric diagnosis. But memory specificity does not predict smaller fluctuations in mental health in community-based samples that primarily consist of young people without psychiatric disorders. Reduced specificity was initially observed in those with recurrent suicidality (William & Broadbent, 1986), and appears to represent a marker of more severe, functionally impairing mental health issues.

Our results reiterate prior suggestions that the AMT is not a sensitive measure in community-based samples (e.g., Farina et al., 2019; Raes et al., 2007). Using modified instructions does appear to make the AMT more sensitive for community-based samples. It has been previously reported that reduced specificity on the traditional AMT (which explicitly requests a specific memory in response to the cue word) is not associated with depressive symptoms in community-based samples, but when instructions do not explicitly ask for a specific memory, an association with higher depressive symptoms emerges (Debeer et al., 2009; Raes et al., 2007). In our study, 13 out of 14 analyzed datasets used traditional AMT instructions. Specificity on a minimal instructions version of the AMT may thus yield different results.

Additionally, memory specificity may have an effect on some symptoms (e.g., suicidal ideation, social withdrawal) but not others, such that considering the overall effect across all symptoms dilutes any effect. For example, prior literature has indicated that reduced memory specificity is associated with higher suicidal ideation and also reduces the ability to elicit social support (Barry, Vinograd, et al., 2019). In contrast, associations between reduced memory specificity and more somatic symptoms (i.e., lack of appetite, sleep disturbance, low energy) may be weaker. Future work to untangle which (if any) symptoms are predicted by memory specificity will clarify the clinical implications of our research, as will elaborating the mechanisms through which memory specificity impacts mental health.

Cue Valence

The emotional valence of the cue used to prompt memory retrieval did influence the effects of memory specificity. This was particularly important to consider, as young people more frequently report specific memories to positive cues, relative to negative cues (Preko et al., 2023). Higher specificity to both positive and negative cues was associated with a lower hazard ratio for psychiatric diagnosis. For symptom severity, reduced negative memory specificity predicted higher depressive symptoms at 6-months post-baseline, but not at later time-points. We found little evidence of this effect for positive memories. Reduced memory specificity may therefore be an early indicator of difficulties in processing cognitive-emotional content.

Difficulty isolating specific negative events may reflect a tendency to generalize across negative experiences, and negative generalizations are a key driver of depressive symptoms (Beck & Haigh, 2014). We have previously proposed that reduced specificity for past negative events may reflect the activation of generalized, negative self-schema that drive depression, and serve to reinforce the suitability of generalized, negative models about the self (Dalgleish & Hitchcock, 2023). In line with this hypothesis, the short-term nature of the effect (also reported in aggregate meta-analysis with adults; Halford et al., 2021) would be consistent with reduced specificity only driving symptoms when negative self-schemas are active. Assessing the relevance and centrality of retrieved memories to self-identity (Matsumoto et al., 2022) may further elucidate how the emotional valence of memories are associated with mental health status.

Theoretical Implications

Evidence that reduced memory specificity is a risk factor for disorder onset offers important implications for cognitive theories of mental illness. Notably, our findings indicate the importance of further developing models that can explain both a) how reduced memory

specificity first emerges, and b) why it might influence the development of psychiatric disorders.

The current literature is unclear regarding the mechanisms underlying reduced memory specificity. The CaRFAX model (Williams et al., 2007), which suggests that reduced specificity is a result of mechanistic failure of a generative retrieval process, has not been well supported. The experience of trauma also has been proposed to trigger reduced specificity (Williams et al., 2007), with case-controlled studies indicating reduced specificity in trauma-exposed individuals relative to matched controls (Barry, Lenaert, et al., 2018). However, our moderation analysis suggested that the relationship between memory specificity and future symptoms did not vary between those with and without a trauma history. Finally, it has been previously suggested that parental figures impact memory specificity in their children, via prompting and modelling during shared recall (Nelson & Fivush, 2004). For example, parental prompts to elaborate on detail is associated with higher memory specificity in preschool children (Valentino et al., 2014). Other social influences, such as cultural norms regarding shared reminiscence between collectivist and individualist societies, have been shown to influence memory specificity (Humphries & Jobson, 2012). Of course, the ongoing development of higher-order cognitive processes (e.g., executive function) during childhood and adolescence are likely to influence specificity, as might other common factors that predispose depressogenic cognitive styles, such as adverse childhood experiences. Further consideration of the biological, cognitive, social, and environmental factors that contribute to development of reduced specificity will not only inform new theoretical models, but potentially aid the early identification of those at risk of psychiatric disorder.

Our findings also offer implications for broader theoretical understanding of the cognitive factors that drive onset of psychiatric disorders. In particular, our results support

prior assertions that negative and generalized cognitive processing will predispose someone to mental illness (Gotlib & Joormann, 2010). Reduced specificity may be one type of overgeneralization that is typical of poor mental health. Reduced specificity may represent the failure to contextualize experiences (i.e., constrain them to a certain place, time, or situation), in the same way that rumination (another prognostic predictor of mental illness; Grierson et al., 2016; Watkins et al., 2020) involves the abstraction of the causes and meanings of a negative event beyond that one specific experience. In an experimental context, we have previously demonstrated that retrieval of specific event memories serves to place a boundary on negative generalizations about the self (Hitchcock et al., 2017). To further investigate how memory specificity relates to broader overgeneralization processes, additional longitudinal research should explore bidirectional relationships (e.g., using latent change score modelling) between memory specificity and other markers of overgeneralized negative cognition, such as global negative beliefs.

Both cognitive behavioral and psychodynamic (e.g., Parry et al., 2021) theories converge on the idea that the overgeneralized self-beliefs which drive mental illness (e.g., I am worthless; I am a failure) are supported by memories of past experiences. A reduced ability to isolate past, specific experiences in place and time (‘*My mother took my game console away when I showed her my B for Year 8 mathematics’)* might contribute to the development of overgeneralized self-beliefs that are applied across contexts (e.g., ‘*I must be perfect to be loved’)*, as one’s personal past is represented in more generalized, less-contextualized summaries (e.g., *‘I was punished every time I got a grade below A’*). Multi-wave, longitudinal research with children and adolescents with no prior history of mental illness would be needed to explore this possibility. Focusing on the context in which negative events have occurred is a feature of many cognitive behavioral therapy programs (e.g., Watkins et al., 2012). If our reasoning is proven correct, intervention techniques that either

increase contextualization of event specific information (i.e., situate the event within broader life experiences) and/or seek to restrict how widely negative meaning is generalized (i.e., constrain negative meaning to that one event) may also support development of good mental health in children and adolescents.

Clinical Implications

Findings suggest there might be potential for future use of memory specificity interventions as preventive mental health programs for adolescents. There are multiple science-driven interventions that aim to improve access to specific memories and have demonstrated significant treatment effects for a range of psychiatric disorders (for review see Barry et al., 2019a, Dalgleish & Hitchcock, 2023, Hitchcock et al., 2017). There are at least two memory-based interventions that have been shown to improve memory specificity and mental health in adolescents (Mirabolfathi et al., 2023; Pile et al., 2021). However, there are several issues which need to be addressed before repurposing these interventions for prevention.

First, preventive interventions should not be explored until we have a better understanding of whether improved memory specificity may increase later anxiety symptoms. Currently, the use of memory-based intervention to prevent onset of depressive disorders may have unintended potential harms (see Foulkes & Stringaris, 2023). Second, we only evaluated memory specificity on the AMT. In community-based samples (Keats et al., 2023; Keats et al., 2024; Salmon et al., 2021), episodic detail (e.g., such as adjectives and names) and specificity of memories for critical life events (i.e., turning point memories) have been reported to predict higher depressive symptoms. The relationship between symptom severity and memory specificity may thus be impacted by measurement characteristics we have been unable to explore. Further consideration of specificity on tasks other than the AMT will be integral to refining the intervention target.

Third, stronger evidence of causality should be sought, utilizing study designs that control for the confound between exposure-outcome association, such as Mendelian Randomization. Finally, the appropriate delivery mode should be determined. School-based delivery is a sustainable and scalable approach to preventive intervention (Murphy et al., 2017), but may be unsuitable or potentially harmful for high-risk groups (see Stallard et al., 2012). Indeed, universal programs have been largely ineffective in a preventive context (e.g., mindfulness; see Hetrick et al., 2016; Kuyken et al., 2022). Improved accessibility of positive, specific memories may reduce risk of mental health disorders, but also support positive self-image (Hitchcock et al., 2017) and other adaptive cognitive skills that underpin successful academic, social, and emotional functioning (e.g., problem solving, Beaman et al., 2007; facilitation of social support; Barry et al., 2019b). If the listed considerations can be accounted for, exploring a memory-based approach could, in the future, offer a new avenue for prevention. Our results suggest that increasing accessibility of positive, specific memories using existing programs (e.g., Mirabolfathi et al., 2023; Pile et al., 2021) may potentially be a useful approach to prevention.

Future work to explore the utility of a clinical cut-off for memory specificity might also prove useful, if our noted considerations are resolved. Currently, there is no accepted value (e.g., the proportion correct) that constitutes ‘reduced’ memory specificity. Our analysis suggested that more severely impaired specificity was associated with poorer prognosis. That is, there was increasing risk of psychiatric disorder for each additional specific memory that the young person failed to retrieve on the AMT. Although this suggests that anything less than perfect specificity might be associated with increased risk, the plotted Cox analysis indicates the relationship between memory specificity and diagnosis onset may be driven by low (≤33% accuracy) specificity. Utility of a cut-off could be explored by evaluating the health economics of decisions made based on a cut-off, and the sensitivity and

specificity of using memory specificity to identify vulnerable populations. Such research may aid the clinical translation of this research.

Limitations of Current IPD-MA

Most critically, multiple psychiatric disorders were included in analysis, but most studies only reported depressive disorders. There were a very small number of cases (e.g., less than 20 positive cases each for eating and posttraumatic stress disorders) for disorders other than depressive disorders. As we hope to have emphasized, our results may be most representative of the first onset of depressive disorders. Similarly, we had pre-registered an evaluation of predictive effects for psychotic disorders, but data were unavailable and should be obtained in future research. There was considerable variability in the timing of follow-up assessments between disorder types (e.g., latest assessment of 8-months for PTSD versus 48-months for depression), and future studies should seek to measure multiple symptom clusters at all time-points to address this limitation. Inclusion of data from never-diagnosed adults in future analyses would also strengthen conclusions.

Another limitation of the available data is that we were unable to control for demographic factors that are well-established risk factors for mental illness, including socio-economic status and executive function deficits. Reduced executive function is associated with both poor memory performance (Dalgleish et al., 2007) and increased risk of mental illness (Halse et al., 2022). Prior case-controlled studies have shown that the association between reduced memory specificity and mental illness remains significant when samples are matched on executive function with never-unwell controls (Hitchcock et al., 2019; Piltan et al., 2021). Similarly, younger participants may not have understood task instructions, although practice trials on the AMT (prior to the test trials used in analysis) were commonly employed to reduce this possibility. Large sample sizes with varied demographics mitigate these concerns, but accounting for these factors may influence the observed effects. Finally,

all identified data came from Australia, New Zealand, the US, and UK. Memory specificity needs to be evaluated as a risk factor in non-Western, Educated, Industrialized, Rich and Democratic (WEIRD) countries to improve the generalization of our results.

Conclusions

Meta-analysis of over 9000 community-based young people suggested that reduced memory specificity on the AMT is a transdiagnostic risk factor for the onset of psychiatric disorders. Reduced specificity not only increased risk of diagnosis, but increasing impairment was also associated with the earlier onset of disorder. Thus, reduced memory specificity both increases the risk of mental illness and predisposes earlier diagnosis. This relationship was strongest for depressive disorders. Results also indicated that reduced memory specificity is a predictor of mental illness but does not predict more everyday fluctuations in mental health. Key needs for future research are consideration of how memory specificity might have different predictive effects for depression and anxiety symptom severity, and should include measurement on tasks other than the AMT. Finally, further theoretical development is needed to account for how reduced memory specificity emerges, and for the mechanisms through which memory specificity impacts mental health.

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