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Research paper

Lower memory specificity in individuals with dysphoria is not specific to autobiographical memory☆

Matthew J. King [a], Kesia Courtenay [a], Bruce K. Christensen [c], Aaron S. Benjamin [b], Todd A. Girard [a,*]

[a] Department of Psychology, Toronto Metropolitan University, Toronto, ON, Canada
[b] Department of Psychology, University of Illinois at Urbana-Champaign, Champaign, IL, USA
[c] Research School of Psychology, Australian National University, Canberra, ACT, Australia

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ABSTRACT

Background: A core cognitive attribute of depression is lower specificity in the expression of autobiographical memories. Despite interventions targeting memory specificity in depression, its underlying mechanisms are not yet fully understood. Depression also relates to poorer memory for episodic details; here we examine whether reduced specificity might simply reflect broader episodic memory deficits and weakened memory traces with the passage of time.

Methods: Undergraduate students with and without symptoms of depression completed the Autobiographical Interview and prose-reading episodic memory tasks to assess both same-day and delayed memory.

Results: Dysphoria and nondysphoria groups performed similarly on the tasks of immediate episodic and autobiographical memory; notably, the dysphoria group did not display evidence of lower specificity at this time point. After a delay, however, both groups demonstrated less specific memory responses on both memory tasks, and these declines were more pronounced in the group with dysphoria. That is, after a delay, individuals high in dysphoria showed a greater decrease in the quantity of specific event details reported on both the episodic and the autobiographical memory task. Additional analyses incorporating other clinical and cognitive measures indicated that these relations are largely unique to symptoms of depression.

Limitations: The sample comprised mostly female students; the study should be replicated with more diverse samples.

Conclusions: These findings support the claim that lower memory specificity is not peculiar to autobiographical memory, but rather, reflects impoverished memory more generally. This is an important consideration for theories and remedial strategies targeting memory specificity.

1. Introduction

Lower memory specificity (or overgeneral memory) is one of the most commonly reported memory impairments in depression. Reduced memory specificity is characterized by the decreased production of specific events or details when requested, a tendency to provide vague descriptions of events, or a tendency to report past events that occurred repeatedly or over a long period of time (King et al., 2010). Importantly, lower memory specificity predicts the severity of depression in clinical and subthreshold samples (i.e., dysphoria; Hallford et al., 2021; Matsumoto and Mochizuki, 2019) and intervention approaches target this memory impairment for improving depressive symptoms (see Barry et al., 2019; Forooshani et al., 2020). Thus, continued efforts to understand the mechanisms of reduced memory specificity accompanying depression are of considerably urgency.

Research documenting memory specificity in depression conventionally uses the Autobiographical Memory Test (AMT, Williams and Broadbent, 1986). Autobiographical memory (AM) encompasses the recollection of one's past lived events and involves both episodic and semantic details (Levine et al., 2002). In the AMT, individuals are asked to list specific autobiographical events (i.e., tied to a specific day and place) in response to positively or negatively valenced cue words (e.g.,

☆ The study was conducted while BKC was at McMaster University, Hamilton, ON, Canada.

* Corresponding author at: Department of Psychology, Toronto Metropolitan University, 350 Victoria St, Toronto, ON M5B 2K3, Canada.
E-mail address: todd.girard@torontomu.ca (T.A. Girard).

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happy: "birth of my first child"). Studies using this task have shown that individuals with clinical depression consistently report fewer specific events, more generalised events (e.g., "walking my dog"), and/or have greater response latencies than healthy individuals (Liu et al., 2013; Williams et al., 2007). Although the AMT has produced more mixed findings with nonclinical samples with dysphoria, variants of the task that allow for uncued free recall of autobiographical events yield more consistent reductions in specificity with subthreshold samples (see Matsumoto and Mochizuki, 2017, 2019; Romero et al., 2014 for discussions).

Addressing a call to extend these findings beyond the AMT in depression research (Williams et al., 2007), Salmon et al. (2021) distinguished between the macrolevel approach of the AMT and paradigms that offer microlevel assessment of the types of details in AM free recall, such as the Autobiographical Interview (AI; Levine et al., 2002). Moreover, they suggested that this microlevel assessment is more sensitive in subclinical samples. In contrast to the AMT, the AI is considered less dependent on domain-general executive functioning and is sensitive to a more nuanced assessment of *internal* (episodic) versus *external* (more semantic) details in the narrative recall among those with depression (Söderlund et al., 2014). Briefly, in the AI, participants are asked to freely recall specified events (e.g., a birthday party) from their lived experiences in as much detail as possible. Internal details reflect specific episodic descriptions of the event itself, including sensory details and thoughts associated with the event (e.g., "I vividly remember driving my blue car to my 17th birthday party with my friend when…"); whereas external details include semantic statements (fact-based details, generalised or repeated events; e.g., "I owned a blue car when I was in school") or details tangential to the particular event being recalled.

As reflected in the memory tasks outlined above, research examining reduced memory specificity focuses almost exclusively on its occurrence within AM. Indeed, this body of literature has operated within the implicit assumption that reduced memory specificity is a phenomenon specific to AM in depression (e.g., Burnside et al., 2004; Kuyken and Howell, 2000). However, the definition of reduced memory specificity, which delineates an impairment in the ability to recall specific events or details unique to an event, begs the question of whether reduced memory specificity in depression might reflect poor *event memory* more broadly. For instance, reduced memory specificity might be more parsimoniously explained by a weakened memory trace with the passage of time (i.e., forgetting), irrespective of whether the event to be recalled is personally relevant (i.e., autobiographical) or simply episodic. Thus, here we assess the hypothesis that if forgetting contributes to reduced memory specificity, it should be observable in both non self-relevant episodic memory (EM) and AM.

The hypothesis that reduced memory specificity reflects a more general memory impairment, such that it should be seen in EM and AM, is supported by several lines of evidence. For one, cognitive impairments in depression are consistent with a generalised pattern rather than isolated domain-specific or selective deficits (Porter et al., 2015). That is, impairments are seen across a range of cognitive domains, including EM (Ahern and Semkovska, 2017; Pauls et al., 2015; Rock et al., 2014). Therefore, it is plausible that more general processes might underlie lower memory specificity, which may be detectable in both EM and AM reports.

Further indirect support is found in a leading theory of reduced memory specificity, the Capture and Rumination, Functional Avoidance, and Executive control model (CaR-FA-X; Williams et al., 2007), which, in part, proposes that cognitive deficits extraneous to AM contribute to this phenomenon. The CaR-FA-X model proposes that impaired attention and executive function processes in the face of limited cognitive resources contribute to poorer search and retrieval of AM. It seems probable that if such deficits contribute to impaired AM, they would also impair episodic recall more broadly; indeed, some work has demonstrated that executive function deficits are related to EM (Pauls et al., 2015) and reduced memory specificity in depression (Raes et al., 2006).

More direct support for the proposition that reduced memory specificity may reflect general memory processes is found in a study conducted by Raes et al. (2006), who explored whether reduced memory specificity in AM was related to other aspects of memory in depression. They reported that poor source memory on an EM task correlated with reduced memory specificity on the AMT, though reduced memory specificity did not correlate strongly with other EM tasks (e.g., verbal memory) or semantic memory tasks. Source memory refers to memory for the contextual details of an event at the time of encoding (e.g., spatial, temporal, social contexts) and thus can be taken to reflect the ability to recall episodic details. In another study, Ramponi et al. (2004) also observed that impaired recall of contextual details for an EM task was related to deficits in memory specificity among individuals with dysphoria. The authors from both studies concluded that reduced memory specificity in AM may relate to a broader memory deficit in recollecting events. Nonetheless, this issue has received relatively little attention, despite its theoretical and practical importance in relation to remediation strategies that remain focused on AM.

In the current study, we test the hypothesis that reduced memory specificity among those with depressive symptoms is reflective of more generalised forgetting processes and can, therefore, be measured in both EM and AM tasks. To this end, we administered two memory tasks that tested same-day and delayed EM and AM in individuals high and low in dysphoria. Undergraduate students experiencing dysphoria were included rather than a clinically depressed sample to address a related goal of replicating the phenomenon of reduced memory specificity in subthreshold depression, which has been less studied. We hypothesized that both groups would report fewer internal details in the delayed conditions for both EM and AM events (i.e., reduced memory specificity), but disproportionately so among those with dysphoria. We also apply conditional process analysis (Hayes, 2022) to assess the possibility that the dysphoria-related differences in AM internal details recalled at delay relate indirectly through shared variance with EM specificity at the level of the individual subject. Finally, we assessed whether reduced memory specificity accompanying dysphoria would be present for recall of recent information (same day internal details) or only after a delay, reflecting delay-dependent mnemonic processes. Each of these features of our design and analytic strategy provides an opportunity to evaluate the generality of the phenomenon of reduced memory specificity in dysphoric individuals across a broader base of tasks, individuals, and levels of memory strength than has been used in previous work.

2. Method

2.1. Participants

Participants included 59 undergraduate students enrolled in an introductory psychology course who were assigned to one of two groups based on levels of depressive symptoms reported in the past week: dysphoria (N = 25) and nondysphoria (N = 34). Based on a sensitivity analysis in G*Power (Faul et al., 2007) our sample was powered (α = 0.05, power = 0.80) to detect at least a medium-large effect size of d = 0.75 for group differences in recall with t-tests and a conservative estimate of d = 0.81 for nonparametric contrasts (applying a lower-bound asymptotic relative efficiency for Mann-Whitney tests). Meta-analytic reviews indicate large effect sizes for lower memory specificity on the AMT in samples with depression (g = 1.05, Liu et al., 2013; d = 1.12, Williams et al., 2007), including a student sample with dysphoria (d = 1.12, Goddard et al., 1997). Similarly large effects have also been observed with the AI in depressed (d = 1.09 at 2 weeks, Söderlund et al., 2014) and older adult samples (d = 1.46, Levine et al., 2002). Using Superpower (Lakens and Caldwell, 2021) for an ordinal between-within interaction with a group difference of d = 0.75 only at delayed recall with a conservative n = 25/group, our study offered an estimated power of at least 50–74 % power for the interaction based on at least small (r = 0.10) or large (r = 0.50) correlations between immediate and delayed

memory conditions, respectively.

We assessed dysphoria levels using the Depression Anxiety and Stress Scale (DASS-21; Lovibond and Lovibond, 1995). Individuals in the dysphoria group where those who scored 10 or higher on the depression subscale, which marks the lower boundary of mild symptoms (Lovibond and Lovibond, 1995). This aligns with previous work defining dysphoria as mild symptoms of depression (e.g., see Williams et al., 2007). As shown in Table 1, dysphoria participants had significantly higher depression scores on the DASS-21 than the nondysphoria group and scored in the moderate symptom range on average. To more fully characterize the samples, we administered additional cognitive and clinical measures. As shown in Table 1, the groups were matched on age, sex, and global cognition, but the dysphoria group scored higher on all clinical measures with the exceptions of problematic use of alcohol and drugs. While matched on age overall, $z = -0.16$, $p = .876$, the dysphoria group included some older individuals (range 18–55) than the nondysphoria group (18–27). The range difference is attributable to two older individuals in the former group (aged 32 and 55). To assess their potential influence on the results, we reran the analyses without them. Their exclusion had no impact on the outcomes of the statistical tests reported here; as a consequence, their data were retained.

2.2. EM task

We assessed EM using a prose-reading task that presented participants with two 2500-word fictitious short stories to be remembered in as much detail as possible. This study comprised two testing sessions. Both short stories were read at the first session, but only one was recalled at session 1 (immediate memory) and the other was recalled one week later (delayed). The assignment of story to delay was counterbalanced such that half of the participants in each group were tested on story A at immediate recall and story B at delayed recall; the other half of participants were tested on story B at session 1 and story A at session 2. The short stories had several quantitative targets to be remembered (e.g., times) that were presented with appropriate lexical descriptors (e.g., 7:05, after sunrise). Story A involved a police interview following a night out and story B was about an individual looking to buy a house. The stories scored 80.8/100 and 84.3/100 on the Flesch-Kincaid Reading Ease formula, respectively, indicating easy levels of readability. Immediately after reading the first of the two short stories, participants freely recalled as many details from the story as possible within a maximum of 10 min and then provided a subjective confidence rating for each statement made, as well as a qualitative rating of episodic reexperiencing. Participants then read the second story, which they recalled at session 2 one week later.

Table 1
Demographic, cognitive and clinical characteristics of the participants.

	Nondysphoria ($n = 34$)	Dysphoria ($n = 25$)	d
Demographic data			
Female/male (*n*)	29/5	21/4	0.06
Age (*Mdn, IQR)*	19 (18–19.75)	19 (18–19)	0.11
Cognitive performance			
WAIS-III DQ (*M, SD*)	105.8 (10.5)	109.5 (9.1)	0.38
Clinical characteristics (*M, SD)*			
PAS	39.8 (24.1)	57.0 (28.3)	0.65**
PAI ALC	46.8 (7.3)	49.6 (7.4)	0.38
PAI DRG	48.2 (9.9)	48.6 (7.6)	0.05
PAI PIM	41.1 (9.4)	36.2 (8.9)	−0.54*
PAI NIM	49.6 (6.0)	54.7 (10.1)	0.61*
DASS-21 Depression	3.9 (2.3)	15.6 (5.7)	2.69***
DASS-21 Anxiety	5.9 (4.8)	11.1 (8.2)	0.77**
DASS-21 Stress	13.0 (8.1)	18.8 (8.6)	0.69*

Abbreviations: WAIS-III DQ – Deviation quotient based on Information and Matrix Reasoning subtests of the Wechsler Adult Intelligence Scale (Third Edition; Wechsler, 1997); PAI (Personality Assessment Inventory; Morey, 1991); PAS (Personality Assessment Screener) P scores; ALC (Alcohol Use), DRG (Drug Use), PIM (Positive Impression Management), and NIM (Negative Impression Management) T scores; DASS-21 - Depression, Anxiety, and Stress Scales (Lovibond and Lovibond, 1995).

* $p < .05$.
** $p < .01$.
*** $p < .001$.

2.3. Autobiographical interview (AI)

We used the AI (Levine et al., 2002) to assess AM at immediate and delayed recall. In the current study, participants recalled their morning from the present day (immediate AM) or from three days prior (delayed AM). Participants completed these delay conditions in a counterbalanced fashion across sessions, such that half of each group performed the immediate AM condition at session 1 and the delayed AM at session 2, and the other half the delayed and immediate conditions at sessions 1 and 2, respectively. The 3-day delay was selected to avoid having participants recall events from one week prior (as done for the EM task) to ensure that those recalling delayed AM at session 2 would not report events from the morning that they completed session 1, as well as to avoid recall of weekends (testing was on Thursdays and Fridays), both of which may have influenced schematic support in recall. Participants were first asked to engage in free recall with no interruptions until they arrived at a natural end point. They were then probed to elicit additional detail of the events (e.g., "Is that all you remember?"). These general probes elicited less than one additional detail on average ($M_{diff} = 0.9$) and had a minimal effect on the results; given their redundancy, these data are not reported.

Following AM recall, participants provided subjective ratings on their experience recalling memories using a scale from 1 to 10, including the ease with which they experienced recalling the events, and the degrees to which visual/auditory perceptual or thoughts and emotional aspects of the memory were reexperienced. Reduced memory specificity is detected on the AI by a dearth of episodic information specific to the event, time, or place. Namely, the AI indexes reduced specificity by a pattern of recall characterized by few internal details.

2.4. Data analysis

Both the EM and AM tasks were scored by the method recommended by Levine et al. (2002). Briefly, after we transcribed the audio recordings of the two recall tasks at each delay, we coded each report according to the number of internal and external details expressed. The first author (MK) has extensive training administering and scoring the AI, with established reliability on a standard AI training set following Levine et al. (2002). Given the novel application to the EM task and for purposes of the current study, we established reliability with a second rater on a random set of 7 AM and 20 EM reports from our paradigm. For both tasks interrater reliability was high for both internal and external details, ICCs (2,1) ≥ 0.98.

We report the free-recall results at an omnibus level using mixed-factors ANOVAs on the between-subjects factor group (dysphoria, nondysphoria) and within-subjects factors of delay (same-day/immediate, delayed) and detail type (internal, external) on each of the AM and EM tasks using SPSS (v25.0, IBM Corp., Armonk, NY). However, several of the variables for the free-recall tasks, and some difference scores between repeated measures, were positively skewed and leptokurtic. Given these distributional concerns, we corroborated the omnibus ANOVA results using nonparametric contrasts (e.g., the Delay × Group interaction was modeled as a Mann-Whitney contrast between groups on the difference scores between delay conditions); these results closely matched the parametric effects, with one exception for the AM recall as reported below. In addition, we conducted all simple-effects analyses using Mann-Whitney contrasts between groups for each combination of delay and detail type for both tasks, and Wilcoxon matched-pairs signed-

ranks tests to assess delay effects within groups and detail type. We applied the Benjamini-Hochberg method to control the false-discovery rate ($Q = 0.05$) across these eight between-group contrasts and independent set of eight repeated contrasts. All reported significant contrasts survived correction.

We assessed the expected indirect relation between depression severity and internal details for AM at delay through delayed EM using PROCESS v4 in SPSS (Hayes, 2022); we used DASS-depression scores to maximize use of the symptom data. AM and EM internal details at day 1 were entered as covariates, such that the delayed measures in the model reflect the respective decreases (change) observed at delay. The parameter estimates are based on 5000 bootstrap samples and the significance of the indirect effect is interpreted relative to a 95 % confidence interval. All regression assumptions were satisfied.

Given that the subjective ratings of recall differed by task, the assessment of incorrect details (intrusions) was unique to the EM task, and because these variables were of secondary interest, the results for these measures are shared in the supplementary material.

3. Results

3.1. AM: free recall

A 2 Group × 2 Delay × 2 Detail type mixed-factors ANOVA on the number of details recalled during the AI revealed large main effects of delay, $F(1, 57) = 35.22$, $p < .001$, $\eta_p^2 = 0.38$ and detail type, $F(1, 57) = 289.32$, $p < .001$, $\eta_p^2 = 0.84$, reflecting that individuals recalled fewer details after a delay and tended to recall more internal details than external ones (see Figs. 1 and S1). The main effect of group was small and nonsignificant, $F(1, 57) = 2.53$ $p = .117$, $\eta_p^2 = 0.04$, but we observed significant interactions of group with delay, $F(1, 57) = 10.86$, $p = .002$, $\eta_p^2 = 0.16$, and detail type, $F(1, 57) = 4.14$, $p = .047$, $\eta_p^2 = 0.07$. These were further qualified by a three-way interaction of Group × Delay × Detail type, $F(1, 57) = 8.27$, $p = .006$, $\eta_p^2 = 0.13$. Nonparametric contrasts corroborated the above findings with the exception that the Group × Delay interaction failed to reach significance, Mann-Whitney U, $z = -1.93$, $p = .053$.

A follow-up ANOVA on only internal details recalled maintained a large and significant effect of delay, $F(1, 57) = 39.63$, $p < .001$, $\eta_p^2 = 0.41$, and a Group × Delay interaction, $F(1, 57) = 9.82$, $p = .003$, $\eta_p^2 = 0.15$. Wilcoxon matched-pairs signed-ranks tests indicated robust effects of delay for both the nondysphoria, $z = -2.95$, $p = .003$, and dysphoria group $z = -4.13$, $p < .001$. Almost all of the dysphoria group (92 %) and three quarters of the nondysphoria group (77 %) recalled fewer internal details following the delay. Notably, group differences were small and nonsignificant for same-day recall, Mann-Whitney U, $z = 0.06$, $p = .951$; whereas, after a delay, the dysphoria group recalled significantly fewer internal details than the nondysphoria group with a large effect size, $z = -4.31$, $p < .001$ (see Fig. 1).

Similar analysis of external details failed to reveal any significant effects or interactions, $ps > .10$, $\eta_p^2 < 0.04$. Nonparametric tests corroborated the lack of group differences in recall of external details at either the same day, Mann-Whitney U, $z = 0.39$, $p = .694$, or delayed AM recall, $z = -0.52$, $p = .606$; likewise, the effects of delay were not significant for the nondysphoria, Wilcoxon matched-pairs signed-ranks test, $z = 1.66$, $p = .097$, and dysphoria group $z = -0.70$, $p = .485$ (see Fig. S1).

In sum, all participants had lower recall of internal details after a delay; however, the dysphoria group was more sensitive to this effect. A limitation of null-hypothesis significance testing is that it only speaks to whether the results reject or fail to reject the null, not the extent to which the null hypothesis offers an explanation for interpretation of the nonsignificant group differences at session 1 for internal details, i.e., the conclusion that dysphoria-related deficits in memory specificity are not present following a short delay (same day recall). Thus, we ran Bayesian analyses in JASP (JASP Team, 2021) to estimate the probability of the alternative versus null hypotheses (Bayes Factor, BF_{10}). These Bayesian Mann-Whitney U tests (Cauchy priors: 0, 0.707) provided moderate evidence in favour of equal group performance at session 1, $BF_{10} = 0.27$, $\delta = -0.06$, 95 % high-density interval [−0.53, 0.42], and extreme support for their divergence at delay, $BF_{10} = 100.56$, $\delta = 0.99$ [0.43, 1.54]. Likewise, a Bayesian mixed-factors ANOVA provided strong evidence for the Group × Delay interaction $BF_{INC} = 28.77$.

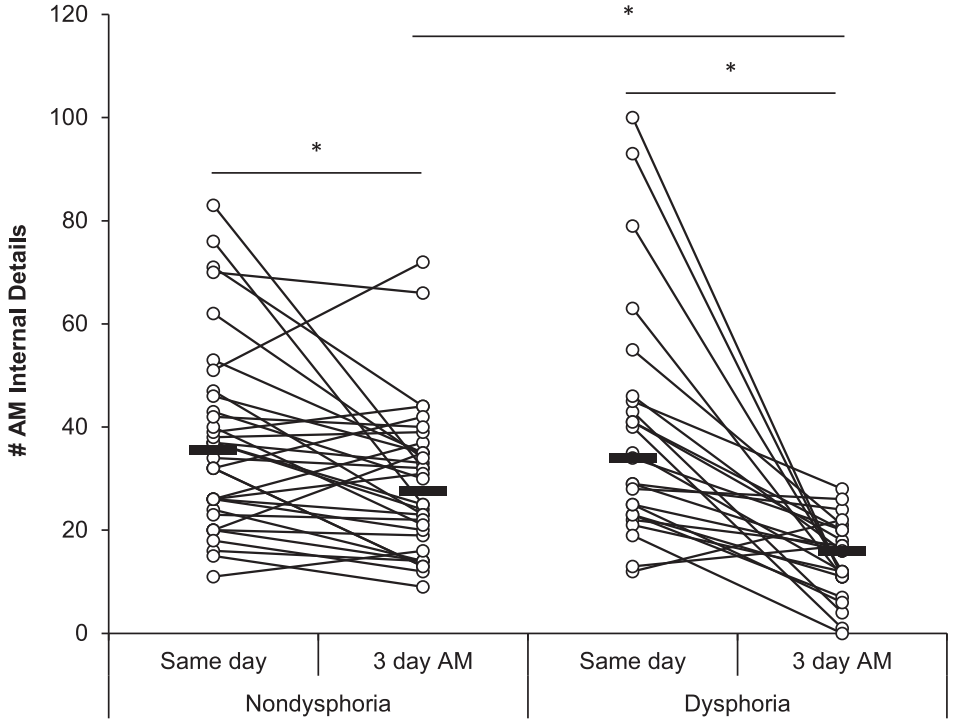

Fig. 1. Internal details recalled during the free recall phase of the autobiographical interview. Most notably, the dysphoria group recalled disproportionately fewer internal details after the delay compared to the nondysphoria group (*$p \leq .003$). Dots represent the number of AM internal details recalled by individual participants from the same morning and that three days prior. Black boxes represent the medians per group at each delay. Dot plot generated following the methods of Weissgerber et al. (2015).

3.2. EM task: correct free recall

A 2 Group × 2 Delay × 2 Detail type mixed-factors ANOVA on the number of details correctly recalled from the transcripts revealed large and significant main effects of delay, F (1, 57) = 118.25, $p < .001$, $\eta_p^2 = 0.68$, detail type, F (1, 57) = 176.94, $p < .001$, $\eta_p^2 = 0.76$, and group, F (1, 57) = 9.98, $p = .003$, $\eta_p^2 = 0.15$, reflecting that individuals recalled more internal details than external ones, fewer details after the delay, and dysphoria related to lower recall overall. The three-way interaction failed to reach significance, F (1, 57) = 2.63, $p = .110$, $\eta_p^2 = 0.04$. The above main effects were qualified by three 2-way interactions: Group × Delay, F (1, 57) = 4.86, $p = .031$, $\eta_p^2 = 0.08$; Group × Detail Type, F (1, 57) = 10.19, $p = .002$, $\eta_p^2 = 0.15$; Delay × Detail Type interaction, F (1, 57) = 33.65, $p < .001$, $\eta_p^2 = 0.37$. Consistent with the analyses for autobiographical recall, we followed up these interactions with separate analyses by detail type.

Analysis of correct internal details recalled maintained a large and significant effect of delay, F(1, 57) = 84.86, $p < .001$, $\eta_p^2 = 0.60$, and a Group × Delay interaction, F(1, 57) = 4.36, $p = .041$, $\eta_p^2 = 0.07$; there was also a main effect of group, F(1, 57) = 12.61, $p = .001$, $\eta_p^2 = 0.18$. Effects of delay were highly significant for both the nondysphoria, Wilcoxon matched-pairs signed ranks test, $z = -4.43$, $p < .001$, and dysphoria group, $z = -4.38$, $p < .001$. All of the dysphoria group (100 %) and most of the nondysphoria group (85 %) recalled fewer internal details following the delay. Again, group differences were small and nonsignificant at immediate recall, Mann-Whitney U test, $z = -1.30$, $p = .192$, but the dysphoria group recalled significantly fewer internal details than the nondysphoria group following the delay, $z = -6.42$, $p < .001$ (see Fig. 2).

Analysis of external details revealed only a significant main effect of lower recall after a delay, F(1, 57) = 69.22, $p < .001$, $\eta_p^2 = 0.55$, but not for group, F(1, 57) = 1.45, $p = .234$, $\eta_p^2 = 0.03$, or their interaction, F(1, 57) = 1.33, $p = .254$, $\eta_p^2 = 0.02$. Mann-Whitney contrasts likewise indicated no difference between groups for immediate recall, $z = -0.63$, $p = .529$, but revealed fewer details recalled by the dysphoria than nondysphoria group at delay, $z = -3.48$, $p = .001$. The effects of delay, however, were highly significant for both the nondysphoria, Wilcoxon matched-pairs signed ranks test, $z = -4.12$, $p < .001$, and dysphoria group, $z = -4.20$, $p < .001$ (see Fig. S2).

In sum, both groups again exhibited lower recall of internal details after a delay, with the dysphoria group revealing this effect more dramatically. These findings parallel those for AM. Again, Bayesian Mann-Whitney U tests provided support in favour of a null group difference at session 1, $BF_{10} = 0.40$, $\delta = 0.21$ [−0.26, 0.71], and extreme evidence for divergence at session 2, $BF_{10} = 4943.09$, $\delta = 1.46$ [0.89, 2.09]. There was also positive support for the Group × Delay interaction $BF_{INC} = 6.92$.

3.3. Intervening effect of EM on the OGM in AM

Conditional process analysis revealed a significant intervening effect of depression severity on AM internal details at delay through EM internal details recalled at delay, controlling for day 1 AM and immediate EM internal details (see Fig. 3). The indirect effect was significant, such that with delayed EM recall in the model, the direct relation between greater depression symptoms and lower delayed recall of AM internal details was no longer significant. Overall, the variables explained 30 % of the variance in delayed recall of AM details, $R^2 = 0.30$, $p < .001$.

4. Discussion

We assessed whether lower memory specificity associated with dysphoria may be more parsimoniously explained by a weakened memory trace (i.e., forgetting) that is observable in both EM and AM, as opposed to a phenomenon specific to AM that may be driven by emotional or more specific cognitive factors. Consistent with this hypothesis, we found that forgetting of episodic details was more pronounced among individuals with dysphoria. Moreover, significant group differences emerged at delayed recall only, suggesting this phenomenon is observable only after a delay in this population. Conditional process analysis further supported that shared variance with EM may explain poorer depression-related AM. In addition, subjective ratings of the

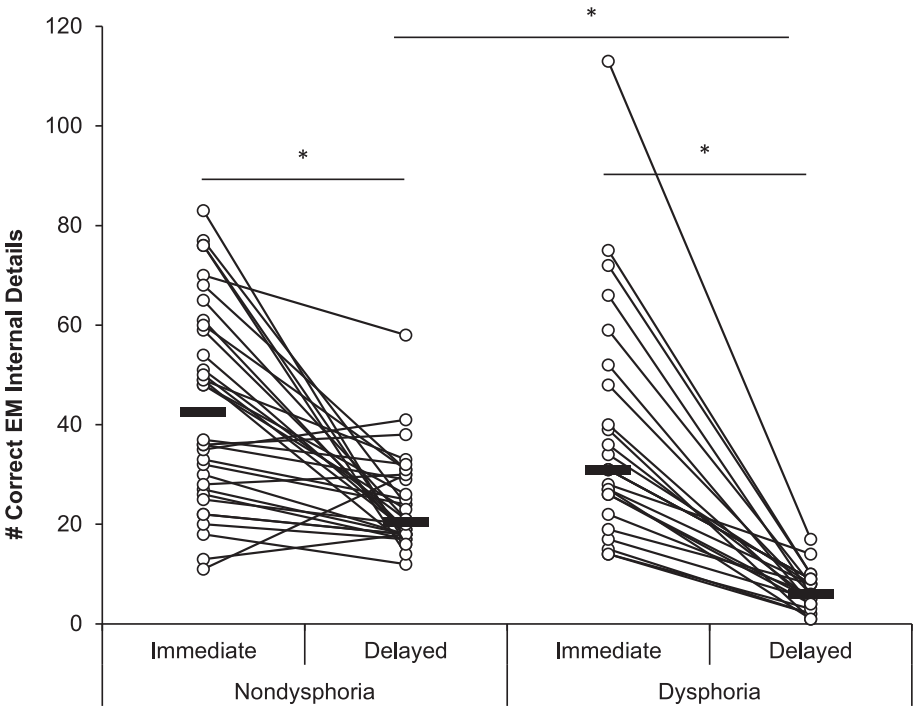

Fig. 2. Correct internal details recalled during the free recall phase of the episodic memory task. Most notably, the dysphoria group recalled disproportionately fewer internal details after the delay compared to the nondysphoria group (*$p < .001$). Dots represent the number of correct EM internal details recalled by individual participants following an immediate and one-week delay. Black boxes represent the medians per group at each delay.

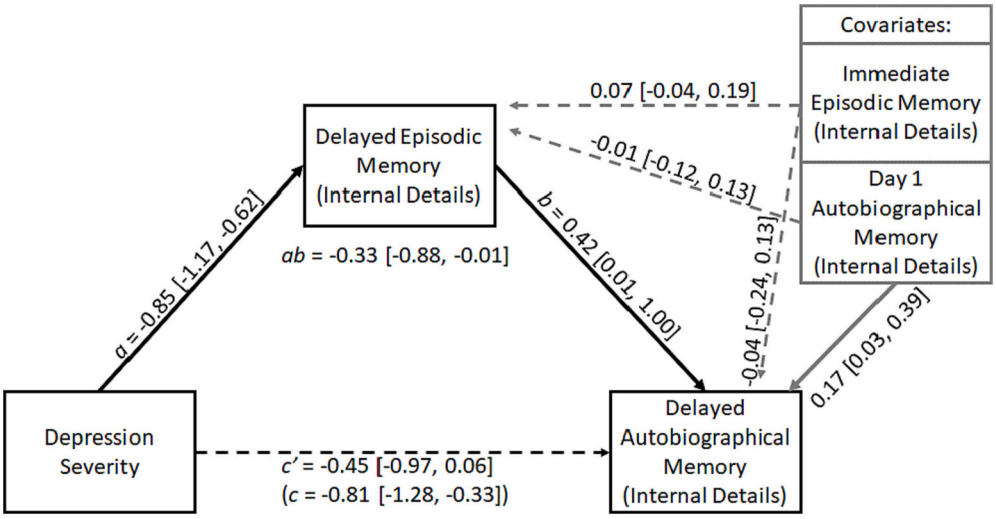

Fig. 3. The intervening effect (ab path) of depression severity on delayed autobiographical memory explained through delayed episodic memory. The memory scores reflect internal details recalled and depression was measured with the DASS-21. The total effect (c) of depression on delayed autobiographical recall of internal details was no longer significant (direct path, c′) after taking into account episodic memory performance (correct internal details). Covariates controlled for variance at delayed recall explained by immediate episodic and day-1 autobiographical recall; only the latter contributed significantly to delayed autobiographical memory. Coefficients represent unstandardized estimates and 95 % confidence intervals based on 5000 bootstrap samples. Solid and dashed lines indicate significant and nonsignificant paths, respectively.

quality of recall on both tasks reflected awareness of worse memory representations after a delay in both groups (see Table S1). These findings support the view that lower memory specificity may emerge with a degrading memory trace over time, as both groups objectively and subjectively experienced greater difficulty recollecting experiences at delayed testing.

The EM task afforded us the opportunity to examine the accuracy of recollected details, which is typically difficult to do on tasks of AM for events occurring outside of the laboratory. The findings from the EM task showed that lower dysphoria-related memory specificity was evidenced by a decline in the number of internal details correctly recalled and mirrored the pattern of findings for internal details reported on the AM task. Fewer external details were reported at session 2 only for the EM task, particularly for the dysphoria group.

Although we replicated the finding of lower memory specificity in dysphoria on delayed recall, a notable aspect of our results is the absence of the effect at immediate testing. The observations at delayed recall support that participants' depressive symptomology was sufficient to detect poorer memory in the dysphoria group. Moreover, this finding lends further support to the notion that forgetting processes may contribute to lower memory specificity in EM and AM. It is possible that more severe levels of depression may yield lower memory specificity at immediate testing, but this remains to be investigated as lower memory specificity is typically assessed by AM for distal past life events. Indeed, to our knowledge, this is the first study to assess memory specificity for same-day events. Examining whether both clinical and subthreshold depression show lower memory specificity only after a delay and not for immediate memory is an important avenue for future research to determine the relative contributions of mnemonic, metacognitive, and other performance-related factors.

Preserved immediate AM in the dysphoria group may also reflect that recent events require less cognitive effort to recollect compared to remote events (e.g., Hartlage et al., 1993). As the task of recollection becomes more challenging for more delayed events, individuals with mood-related symptoms may be less able to recruit the necessary resources to employ effective metamnemonic strategies. More specifically, deficits in delayed memory performance may suggest problems with memory encoding, storage, or more general search and retrieval processes. Indeed, Söderlund et al. (2014) suggested that executive-function demands on the AMT contribute to difficulty accessing and retrieving information in those with depression. Generating effective search strategies is critical for both autobiographical and episodic recall (Benjamin, 2007; Unsworth et al., 2014); those strategies may be compromised or too demanding for dysphoric individuals. Furthermore, the CaR-FA-X model suggests that executive functioning deficits contribute to lower memory specificity. Future research should examine how additional cognitive processes involved in the search and retrieval of memories contribute to memory specificity at different delay times or with different task demands.

Another avenue for future research is to examine the relation between forgetting curves and lower memory specificity. Söderlund et al. (2014) found that lower memory specificity in major depressive disorder remained relatively stable, in terms of number of internal details recalled, at delays ranging from 2 weeks to10 years. Additional work should examine when depression-related group differences emerge across the time points observed in the present study (i.e., after 3 days AM or one-week EM) and when their magnitude plateaus in dysphoria.

4.1. *Limitations and future directions*

This study is not without its limitations. Consistent with convenience samples from our undergraduate Psychology participant pool, our sample comprised mostly female students. Aligned with literature on student samples (Ibrahim et al., 2013), the groups were delineated by a minimum cut-off for mild depressive symptoms. As such, it will be important to replicate these results with more diverse nonclinical and clinical samples. We also note that although the groups were matched on demographics and global cognition, the dysphoria group differed on several clinical attributes. Thus, we ran post-hoc analyses to explore potential relations among the 10 clinical, cognitive, and demographic variables in Table 1 (gender, age, WAIS-III DQ, DASS, PAS, and PAI scores) with delayed recall of internal details on the AM and EM tasks. These analyses confirmed their relations with depression severity (AM $r = -0.42$, $p = .001$, EM $r = -0.55$, $p < .001$). The only other significant relation was for problematic alcohol use with the AM task ($r = -0.28$, $p = .029$), but this failed to survive correction for false discovery rate of 5 % (Benjamini-Hochberg $Q = 0.05$ across the set of 10 correlations, applied separately for the AM and EM data) and was not seen for EM ($r = -0.12$, $p = .378$). Thus, the current finding of poorer recall of internal details at delay appears to be a relatively specific relation with depressive symptoms. Nonetheless, lower memory specificity has also been reported among older adults (e.g., Wank et al., 2021) and other clinical conditions, including posttraumatic stress disorder (Ono et al., 2016) and psychosis (Zhang et al., 2019). Thus, it will be informative to further investigate the underlying mechanisms and apparent specificity or transdiagnosticity of lower memory specificity.

It is also important to consider methodological limitations. Our within-subjects design involved different delays for the AM and EM tasks; use of the same delays will be useful for more direct comparisons across these paradigms in future. Additionally, while the similar pattern

of findings across tasks and the conditional process model support that shared variance with EM may contribute to reduced AM specificity, our cross-sectional design cannot support causal inference. It will be useful in future to investigate these relations in a longitudinal design with experimental manipulations. It may also be informative to further interrogate the nature of the memory reports for a better understanding of how lower memory specificity manifests. For instance, individuals with temporal-lobe lesions select events to recall differently than healthy adults, such that they tend to rely more on intact semantic memory processes (Lenton-Brym et al., 2016). It would be interesting to see how such factors may relate to lower memory specificity in samples with depression across different paradigms and by examining changes over time. We assessed free recall for the EM task to map it onto the AM protocol, but future use of recognition paradigms may allow for further understanding of underlying mnemonic and metamnemonic processes.

Given that the vast majority of the literature pertaining to memory specificity in depression is based on the AMT and related cuing tasks (Liu et al., 2013; Salmon et al., 2021; Williams et al., 2007), it will also be important to see whether the current findings can be extended to the AMT. Indeed, more research is required to better understand the extent to which macro- (i.e., AMT) and microlevel approaches (i.e., AI) to studying reduced memory specificity target the same cognitive construct (Salmon et al., 2021). Our current use of the AI was informed by calls to build on the literature with alternative paradigms and suggestions that the AI may be more sensitive for nonclinical samples (Salmon et al., 2021; Williams et al., 2007). The AI was also well-suited for our current aims for a comparison EM recall task, but future work could attempt to develop an AMT-like EM paradigm.

5. Conclusions

These results call into question the notion that lower memory specificity is a phenomenon specific to AM in depression and instead suggest that the phenomenon may arise from more generalised delay-dependent degradation of event memory detectable in both EM and AM, exacerbated by depressive symptoms. This hypothesis is relevant to current theories of memory specificity in depression and presents the need for further research aimed at better understanding its underlying mechanisms. Such work is imperative for advancing treatment approaches in depression that target lower memory specificity.

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CRediT authorship contribution statement

Matthew J. King: Conceptualization and design; data collection, analysis, and visualization; writing, review, and editing
Kesia Courtenay: Data visualization; writing, review, and editing
Bruce K. Christensen: Supervision; conceptualization and design; writing, review, and editing
Aaron S. Benjamin: Conceptualization and design; writing, review, and editing
Todd A. Girard: Supervision; conceptualization and design; data analysis and visualization; writing, review, and editing.

All authors contributed to and have approved the final manuscript.

Conflict of interest

All authors declare that they have no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2023.01.040.

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