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Depression and Episodic Memory Across the Adult Lifespan: A Meta-Analytic Review

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Episodic memory deficits have increasingly been recognized as a cognitive feature of depression. To quantify these deficits and determine how they are moderated by various tasks (e.g., stimulus valence) and participant (e.g., age, depression diagnosis) variables, we conducted a three-level meta-analysis on 995 effect sizes derived from 205 studies with 236 unique comparisons between depressive and control groups on episodic memory measures. Overall, depression was associated with small to moderate deficits in episodic memory, Hedges' $g = -0.36$, 95% CI [−0.41 to −0.31]. Effects were larger in older age, in diagnosed compared to subthreshold depression, and in those taking medication for depression; effects did not differ between those with current and remitted symptoms. Stimulus valence moderated the effects, such that depression-related deficits were particularly pronounced for positive and neutral stimuli, but not for negative stimuli. Educational attainment served as a sort of protective factor, in that at higher levels of education, depressed group performance was more similar to that of controls. These findings confirm the episodic memory deficits in depression but highlight the important differences in the size of these deficits across a number of task- and participant-related variables.

Public Significance Statement
This meta-analysis demonstrated small to moderate depression-related deficits in episodic memory. Deficits were more pronounced in older age, in clinical depression, and in those receiving pharmacological treatment, while fewer deficits were observed in memory for negative material and with higher educational attainment. By shedding light on depression's nuanced relationship with episodic memory, the present work encourages a greater consideration of cognitive symptoms when diagnosing and treating depression.

Keywords: episodic memory, major depression, subthreshold depression, aging, meta-analysis

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The annotated data set and R code used in this meta-analysis are available on the Open Science Framework: https://osf.io/t8ynd/?view_only=05c342695faa476795218c6a31ecfec5

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Depression is a serious public health issue. In 2019, 19.4 million adults reported experiencing at least one major depressive episode in the past year in the United States alone, and of those, 67.5% were associated with severe impairment in their daily lives (Substance Abuse and Mental Health Services Administration, 2020). Major depressive disorder (MDD) is highly heterogenous (Ostergaard et al., 2011). Impairments resulting from depression can resonate through all areas of individuals' lives, affecting relationships, work and school performance, and even day-to-day leisure activities (see Papakostas et al., 2004; Sheehan et al., 2017, for reviews). Although much of the research is rightfully focused on the telltale signs of changes in mood and well-being, depression is also often accompanied by impairments in cognitive functioning (see Austin et al., 2001; Cambridge et al., 2018, for reviews). In the present meta-analysis, we focus on one of the more prominent of these impairments, namely, the deficit in episodic memory functioning.

Episodic Memory Deficits in Depression

Episodic memory is the richly detailed memory for not only the events we experience but also the associated details such as the

contexts in which the events occur. As is common in the field (e.g., Atkinson & Shiffrin, 1968), we view episodic memory as distinct from working memory or short-term memory, and thus exclude tests for the latter two constructs from our analysis. Likewise, like Tulving (1972, 2002), we consider episodic memory distinct from semantic memory, or memory for general knowledge; thus, we exclude tests for the latter as well. The best measure of episodic memory is retrieval performance after a delay, while episodic encoding is associated with additional neural processes beyond those used in delayed measures, such as processing speed and executive functioning (Casaletto et al., 2017). However, because studies have often used immediate retrieval as a measure of episodic memory encoding, we include these measures in our operationalization of episodic memory in this meta-analysis. Our aim in focusing on episodic memory is not to understate the interconnectedness of episodic memory and other cognitive domains frequently impaired in depression, such as executive functioning (Dotson et al., 2020). An analysis of all of these domains is beyond the scope of a single meta-analysis. However, by better defining the relationship between depression and episodic memory, we hope to encourage further research into common mechanisms that may underly a wider range of cognitive deficits.

Converging evidence suggests that episodic memory may be disrupted in depression (Ahern & Semkovska, 2017; Bora et al., 2013; Burt et al., 1995; Kindermann & Brown, 1997; Lee et al., 2012; McDermott & Ebmeier, 2009; Rock et al., 2014). Previous meta-analyses have consistently found that relative to healthy controls, individuals with depression tend to show reduced memory performance. Researchers have in turn proposed a range of theoretical explanations for impaired memory in depression. For example, recall is generally found to be more affected than is recognition (Bora et al., 2013; Burt et al., 1995), in line with the resource allocation theory. This hypothesis posits that depression, or more specifically, the maladaptive ruminative thinking patterns that are a feature of depression (Nolen-Hoeksema et al., 2008), occupy a portion of the depressed individual's limited cognitive resources, thereby reducing the resources available to perform other cognitive tasks (Ellis & Ashbrook, 1988; see Gotlib & Joormann, 2010, for review). The claim of the resource allocation view is that, because depression reduces one's cognitive capacity, the greatest deficits should be observed in more effortful processes, such as generating one's own retrieval cues as required in recall tasks, resulting in particularly deep deficits in these types of tasks. This is not, however, universally found. Notably, in their meta-analysis, Kindermann and Brown (1997) obtained the opposite pattern: Recognition deficits were larger than those in free, cued, and incidental recall, though this meta-analysis was restricted to studies investigating depression in older adults (sample $M_{age} \geq 55$).

Another possibility is that depression and memory deficits could both arise from a common factor, such as chronic stress. The social signal transduction theory of depression argues that the upregulation of proinflammatory cytokines in response to stressful life events can drive the onset of depressive symptoms (Slavich & Irwin, 2014). At the same time, increased inflammation has been linked to neurodegeneration and associated memory impairments seen in depression and increasing age (Hurley & Tizabi, 2013). The combined emotional, cognitive, and somatic effects of inflammation would consequently manifest concomitantly. If this were the case, then treatment with anti-inflammatory agents might prove effective at simultaneously lessening the severity of depression and memory deficits.

Depression may also develop in response to cognitive deficits like impaired memory. A recent population-based longitudinal study identified mild cognitive impairment (MCI) as a potential risk factor for the onset of depression, suggesting that declining cognitive function could itself give rise to the negative feelings characteristic of depression (Mirza et al., 2017). Although Mirza and colleagues looked specifically at MCI in individuals 55 and older, it is also possible that younger individuals experience a period of cognitive decline prior to the onset of clinical depression.

The above findings suggest that age is potentially an important factor to consider when exploring memory deficits in depression. In typical aging, greater deficits are often observed for recall than recognition, and this deficit is often ascribed to a decrease in cognitive resources (Danckert & Craik, 2013; Rhodes et al., 2019). Additionally, older adults frequently demonstrate a decreased ability to spontaneously self-initiate effective encoding and retrieval operations. This deficit is often seen as a sign of resource allocation difficulty, brought on by a decrease in overall cognitive resources (see Craik & Rose, 2012, for review)—paralleling the deficit that is also frequently reported in depression (Channon & Green, 1999; Elderkin-Thompson et al., 2007; Hertel, 2000; Joormann et al., 2009; Taconnat et al., 2010). A few meta-analyses have investigated whether age moderates the relationship between depression and memory, with conflicting conclusions. Burt et al. (1995) found that recall and recognition were more impaired earlier in life. Similarly, Kindermann and Brown (1997) found larger depression-related memory impairments in studies that contained individuals younger than age 45. Others (Ahern & Semkovska, 2017; Bora et al., 2013) have not found a modulating effect of age, though Bora et al. (2013) did find that older age of depression onset was associated with more severe verbal memory deficits. One benefit of using a large sample size and a wide age range, as we did in the current meta-analysis, is that we were able to explore the effects of participant age at the time of study and age of depression onset as separate moderating variables. Although the use of cross-sectional data necessarily limits our ability to arbitrate between different causal theories of depression and memory deficits, moderator analyses can help provide preliminary support for one theory over others.

Though several meta-analyses have assessed memory deficits in depression, the characteristics of the samples differ greatly, making it difficult to discern the patterns of these deficits. For example, Ahern and Semkovska (2017) restricted their meta-analysis to studies with first-episode MDD, while Burt et al. (1995) included studies with unipolar and bipolar participants, and Bora et al. (2013) analyzed only studies with euthymic MDD participants. In the current meta-analysis, we excluded bipolar depression, as episodic memory has more often been assessed in unipolar depression in the literature; additionally, this reduced diagnostic variance across studies, allowing us to more clearly delineate the effects of depression proper.

One of the most noteworthy omissions in previous meta-analyses of memory performance in depression is that none have included studies in which cases of subthreshold depression (i.e., depressive symptoms that do not meet clinical criteria for MDD diagnosis) were represented in the samples. In a meta-analysis assessing cognitive control deficits in depression, Dotson et al. (2020) found similar deficits between those with clinical and subthreshold depression in

their overall sample; in a subsample where the mean age was 39 or older, those with a clinical diagnosis performed significantly worse than those with subthreshold symptoms. While the effects of clinical versus subthreshold depression on episodic memory have not been previously investigated in a meta-analysis, it is feasible that memory performance may differ as a function of clinical diagnosis due to the reliance of episodic memory on underlying cognitive control processes (also frequently referred to as executive functions), such as attention allocation and information monitoring (see Duarte & Dulas, 2020; Gliebus, 2018, for reviews). Subthreshold depression is quite prevalent in the population, with estimates ranging from 1.4% to 17.2%, and is associated with increased risk of developing major depression (Cuijpers & Smit, 2004; Karsten et al., 2011; Tuithof et al., 2018). Furthermore, while rates of clinical depression decrease with age, rates of subthreshold depression increase (see Polyakova et al., 2014; Szymkowicz et al., 2019, for reviews). For these reasons, among others, it is crucial to understand the extent to which memory deficits observed in MDD are also present in subclinically depressed samples.

That said, it is also worth acknowledging the limitations of a binary distinction between clinical and subthreshold depression, as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). Although the DSM has proven a useful means of diagnosing depression over the past several decades, the symptom cutoffs defined therein are necessarily artificial. At the same time, a single measure of DSM-defined symptomatology can belie the heterogeneity of depression across individuals; two individuals may receive the same diagnosis while having very different manifestations of the disorder (Ostergaard et al., 2011). Recent work has suggested that a dimensional classification scheme could better characterize the nature of depression (Hankin, 2019). Such a model might include a broad indicator of psychopathology (e.g., the *p* factor proposed by Caspi et al., 2014) to account for the overlap between disorders with high comorbidity—such as depression and anxiety disorders—as well as a set of internalizing factors and specific symptoms that separately account for depression (Hankin, 2019).

There are several outstanding questions regarding variables that may impact the relationship between depression and memory. In the following sections, we describe our four main questions and detail how we address each in the current meta-analysis.

Is the Pattern of Episodic Memory Deficits in Depression Consistent Across the Adult Lifespan?

There are a number of reasons to believe that depression-related memory deficits may be magnified in older age. For one, episodic memory is dependent on underlying cognitive control functions, and these functions are impaired in both normal aging and depression. For memories to be encoded accurately, one often needs to selectively attend to relevant information and inhibit irrelevant details, such as conversations of nearby customers when having a conversation with one's dining partner (James et al., 2016; Kuhl & Chun, 2014). For memory retrieval, one may need to monitor the retrieved details with respect to the current context and decision criteria (e.g., "Did I hear this story from my son or daughter?"; Barredo et al., 2015; Benjamin, 2007). Both aging (Campbell et al., 2012; Jacoby et al., 2005; Kray & Ferdinand, 2014; Rey-Mermet & Gade, 2018) and depression (Snyder, 2013; Wagner et al., 2012; Yang et al., 2017; Zaninotto et al., 2016; Zetsche et al., 2018) have been associated with deficits in these kinds of cognitive control functions, and there is evidence for a synergistic effect such that older adults with MDD show the greatest deficits (Dotson et al., 2020).

Another reason to believe that age and depression may have interactive effects on memory is that similar patterns of neural degeneration have been observed in depression and healthy, as well as pathological, aging. In their comprehensive review of structural changes in healthy and pathological aging, Lockhart and DeCarli (2014) describe a linear decline in prefrontal cortex (PFC) volume with advancing age. Hippocampal volume, on the other hand, remains relatively stable until the sixth decade of life, at which point it decreases rapidly. Volumetric studies of depression have delivered similar results, indicating atrophy in both the hippocampus (Butters et al., 2008; Byers & Yaffe, 2011; Juan & Adlard, 2019; Leonard & Myint, 2009; Videbech & Ravnkilde, 2004) and PFC (Hurley & Tizabi, 2013; Leonard & Myint, 2009). Given the importance of the PFC in cognitive control (see Banich, 2009, for review; Braver, 2012; Gratton et al., 2018) and the key role of the hippocampus in episodic memory (de Flores et al., 2015; Eichenbaum et al., 2007), such declines could contribute to the cognitive deficits observed in depression and aging.

Some longitudinal studies have posited a link between depression and accelerated cognitive aging. For example, depressive symptoms are predictive of higher rates of incident cognitive impairment (Köhler et al., 2010; Wilson et al., 2014); steeper declines in Mini-Mental State Examination (MMSE) scores (Rapp et al., 2011); and worse performance over time on tests of verbal memory, prospective memory, verbal fluency, and attention (Gale et al., 2012). Together, these results suggest that depression may move forward the point at which cognitive declines characteristic of aging appear. In the current meta-analysis, we assess the moderating effect of age to investigate whether episodic memory deficits are consistent across the adult lifespan, or if they increase as a function of age.

What Are the Effects of Valence on Episodic Memory in Depression?

One way in which memory impairments in normal aging are unlike those typically seen in depression is in emotional memory. Specifically, while older adults, relative to younger adults, have been shown to exhibit better memory for positive than negative events (Carstensen & Mikels, 2005; Mather, 2012; Thomas & Hasher, 2006), negative events are typically better remembered in depression (e.g., Marchetti et al., 2018). Mood-congruency effects in memory—wherein an individual's affective state biases them toward stimuli of the same valence—are well documented in past research (Bower, 1981; Matt et al., 1992; Van Vleet et al., 2019). Depressed participants frequently exhibit impaired memory performance for positive and neutral stimuli both in episodic (Marchetti et al., 2018) and autobiographical (Young et al., 2012) memory tests. The current meta-analysis includes valence as a moderator variable to assess its impact on memory in depression across different task demands.

Are Depression-Related Memory Deficits Tied to One's Current Depressive State?

If memory impairments in depression are linked to the current depressive state, impairments would be expected to vary as a

function of symptom severity regardless of diagnosis, and for memory performance to improve once symptoms have remitted. If, on the other hand, memory deficits are independent of depressive state, one would expect those deficits to be more pronounced in clinically diagnosed depression than in subthreshold depression, and that they would persist following treatment into symptom remission. Currently, these remain open questions and we address them in this meta-analysis in the following ways.

Are Memory Deficits Similar Across Clinical and Subthreshold Depression?

Studies have shown negative associations between total depressive symptoms and gray matter volume in the hippocampus (O'Shea et al., 2018), as well as other areas known to be affected in depression such the PFC (see Besteher et al., 2020, for review). Additionally, like those with clinical depression (Cao et al., 2012), individuals with subthreshold symptoms show weaker functional connectivity between hippocampal and frontal regions compared to healthy controls (Zhu et al., 2014). Research into whether these abnormalities in subthreshold depression translate into episodic memory deficits, and how those deficits compare to those observed in MDD, has yielded inconsistent results. Some studies have indicated that symptom severity in those with subthreshold depression is negatively correlated with memory performance (Dotson et al., 2008; Elderkin-Thompson et al., 2007) and positively correlated with memory bias (Everaert et al., 2013; Marchetti et al., 2018; Reid et al., 2006), such that those with higher severity show better memory for negative relative to positive material. However, other researchers have not shown such correlations (Jermann et al., 2008; Spalletta et al., 2014), and others have shown that subthreshold depression is not associated with impaired episodic memory relative to controls (Airaksinen et al., 2004). The current meta-analysis will be the first to comprehensively evaluate how episodic memory impairments in subclinical depression compare to those associated with MDD.

Do Memory Deficits Persist Into Remission?

While there are well-attested deficits in a range of cognitive control functions in remitted depression (Hasselbalch et al., 2011; Nakano et al., 2008; Paelecke-Habermann et al., 2005; Preiss et al., 2009; Rock et al., 2014; Smith et al., 2006), findings are less consistent for episodic memory. Some past work has reported memory deficits in participants in the euthymic stage of depression, regardless of age (Airaksinen et al., 2006; Preiss et al., 2009; Sheline et al., 1999), while one meta-analysis found deficits were greater in those with remitted late-onset depression (defined as onset after age 50–65, depending on the study) than in those with early-onset (Bora et al., 2013). By contrast, other researchers have reported minimal memory deficits in remitted depressives compared to healthy controls (Rock et al., 2014). Given the mixed results of previous research on patients in the euthymic phase of depression, it remains unclear which elements of episodic memory impairment persist into remission. To address this uncertainty, we considered current depression status as a moderator variable in the present meta-analysis.

Does Memory Improve With Depression Treatment?

As discussed, some studies have suggested that memory impairments in depression are dependent on the current depressive state, such that memory performance improves following successful treatment (Douglas & Porter, 2009; Russo et al., 2015). However, it is unclear whether these improvements vary as a function of the type of treatment. For example, while some have concluded that patients taking antidepressants experience greater cognitive control deficits than unmedicated patients (see Snyder, 2013, for review), the results are inconsistent with respect to memory performance. Some researchers have found poorer memory in unmedicated compared to medicated participants (Burt et al., 1995), others have found medication to be negatively associated with memory (Lee et al., 2012), and some have found no relationship between medication status and memory performance (Ahern & Semkovska, 2017; Kindermann & Brown, 1997). In the current meta-analysis, we investigate the moderating effect of antidepressant use on episodic memory performance.

Are There Factors That Protect Against the Negative Effects of Depression on Memory?

Some studies have found that education may serve as a protective factor: Higher educational attainment is associated with fewer memory deficits among patients with depression (Mackin et al., 2014; Murphy & O'Leary, 2010). However, some researchers have indicated that more years of education predict greater memory deficits in late-life depression (O'Shea et al., 2015), while others have found no significant relationship between education and general cognitive function (Bhalla et al., 2006; Siddarth et al., 2021). Some evidence has indicated an overall advantage for females on episodic memory tasks (see Asperholm et al., 2019 for meta-analysis and review); however, women are also twice as likely as men to experience MDD and report higher severity of symptoms (see Altemus et al., 2014, for review). Previous meta-analyses have not found a moderating effect of gender on memory in remitted depression (Bora et al., 2013) or on cognitive control in current depression (Snyder, 2013). Whether gender moderates depressive effects on episodic memory specifically has yet to be assessed in a meta-analysis. The current meta-analysis aims to determine whether factors like education and gender help to buffer against the negative effects of depression on episodic memory performance.

The Current Meta-Analysis

The last meta-analysis to explore memory impairments in depressed adults across a variety of memory tasks was published over two decades ago (Burt et al., 1995). Meta-analyses published since then have investigated deficits in cognitive functioning, not specifically episodic memory, across a narrow range of neuropsychological tests in specific patient populations, such as those in their first episode of clinically diagnosed MDD (Ahern & Semkovska, 2017; Bora et al., 2013; Burt et al., 1995; Lee et al., 2012; McDermott & Ebmeier, 2009; Rock et al., 2014). The goal of the current meta-analysis is to address the open questions outlined above by identifying both task-related (e.g., type of memory task, type of material used, stimuli valence) and participant (e.g., age,

diagnosis) variables that moderate the effects of depression on episodic memory. To do this, we conducted a three-level meta-analysis using 995 effect sizes from 205 studies with 236 unique comparisons between depressive and healthy-control groups. Our meta-analysis is the first to use a three-level model to assess episodic memory deficits in depression. One reason to prefer multilevel modeling of effect sizes is that this analysis allows for the inclusion of multiple effect sizes for each study (Cheung, 2014), as opposed to averaging these effects as is necessary in traditional meta-analysis. Episodic memory studies often contain multiple effect sizes (e.g., measures of immediate and delayed recall and recognition), and the use of multilevel modeling then improves the ability to examine our moderators of interest.

Method

The annotated data set and R code used in this meta-analysis are available on the Open Science Framework: https://osf.io/t8ynd/?view_only=05c342695faa476795218c6a31ecfec5

Selection of Studies

We conducted a systematic review and meta-analysis following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009). Two of the authors independently searched the electronic databases Pubmed and PsycINFO from the year 1980 to November 2020 for studies that investigated the association between depression and episodic memory. The year 1980 was selected because this was the year the DSM-III (American Psychiatric Association, 1980) introduced the term "major depressive disorder" and outlined the diagnostic criteria, which have remained essentially the same in newer editions. Limiting the search to human population, adults ages 18 and older, we used the terms (a) "depress*" or "MDD," and (b) "episodic" in combination with "memory," "encoding," or "retrieval"; "recollection"; "recall"; "recognition"; "source memory"; "subsequent memory"; or "associative memory" to search titles and abstracts of published studies. The asterisk allows for the inclusion of alternate endings (e.g., searching for "depress*" would return matches for "depression," "depressive," and "depressed"). A subsequent search was conducted by examining the references of the eligible articles to identify additional studies. Our database search was not limited to studies published in the English language.

Though we included clinical trials in our database searches, we also searched clinicaltrials.gov for completed studies with results of depression (or depressive disorders or depressed mood) in adults involving memory. To capture unpublished literature, we searched ProQuest Dissertations and Theses Global using the same search terms described above. Additionally, we made calls for unpublished data in APA Division 20: Adult Development and Aging, APA Division 40: Society for Clinical Neuropsychology, and the International Neuropsychological Society.

Eligibility was determined by examining first the title and abstract then the full text of each article. Reviews and meta-analyses were excluded. Inclusion criteria were as follows: (a) use of sample diagnosed with unipolar depression (either in a current or remitted state) or with depressive symptoms as measured by a standardized questionnaire; (b) inclusion of a healthy, control comparison group; and (c) reporting of at least one objective measure of episodic memory performance for both the depressed and control groups (means and standard deviations, or sufficient statistical data such as F or t statistics to be able to calculate effect sizes). Longitudinal studies were included if baseline assessments were reported for depressed and control groups; only these baseline assessments were used in the analyses. Because major medical or psychological comorbidities could have a confounding influence on memory performance, we excluded studies in which any member of the depressed or control sample was reported to have dementia, Alzheimer's disease, MCI, brain injury, Parkinson's disease, history of stroke, human immunodeficiency virus (HIV), cancer, diabetes, multiple sclerosis, attention deficit hyperactivity disorder (ADHD), schizophrenia, alcohol or substance abuse, personality disorders, posttraumatic stress disorder (PTSD), bipolar disorder, psychotic symptoms of depression, or postpartum depression. Generalized anxiety was not exclusionary due to the high comorbidity of depression and anxiety. Studies conducted in residential care facilities and inpatient settings were included if the sample met all other criteria. In cases where data from the same or overlapping groups of participants were reported in separate records, the most recently published study and/or that with the largest sample size was retained to prevent duplication of effect sizes. The interrater agreement for the inclusion/exclusion categorization of records was .96. Disagreements in decisions to include or exclude particular records were discussed and reconciled.

After deleting duplicate records, 2,938 records were screened (2,355 published and 583 unpublished). Of these, 533 were deemed eligible for the full-text search based on titles and abstracts. When full texts of potentially eligible records were not available online (72 in total), we attempted to contact the corresponding author; however, current author contact information could not be located for 49 of the records (particularly for dissertations, theses, and older articles). We received full texts from 15 of the 23 authors contacted. Corresponding authors were also contacted when records lacked sufficient detail to determine a record's eligibility (e.g., when samples from separate records were suspected to overlap). Five out of six authors responded to requests for this information. Through this search process we identified 205 records (176 published, 29 unpublished) that met all criteria (see Figure 1).

Data Extraction and Variable Coding

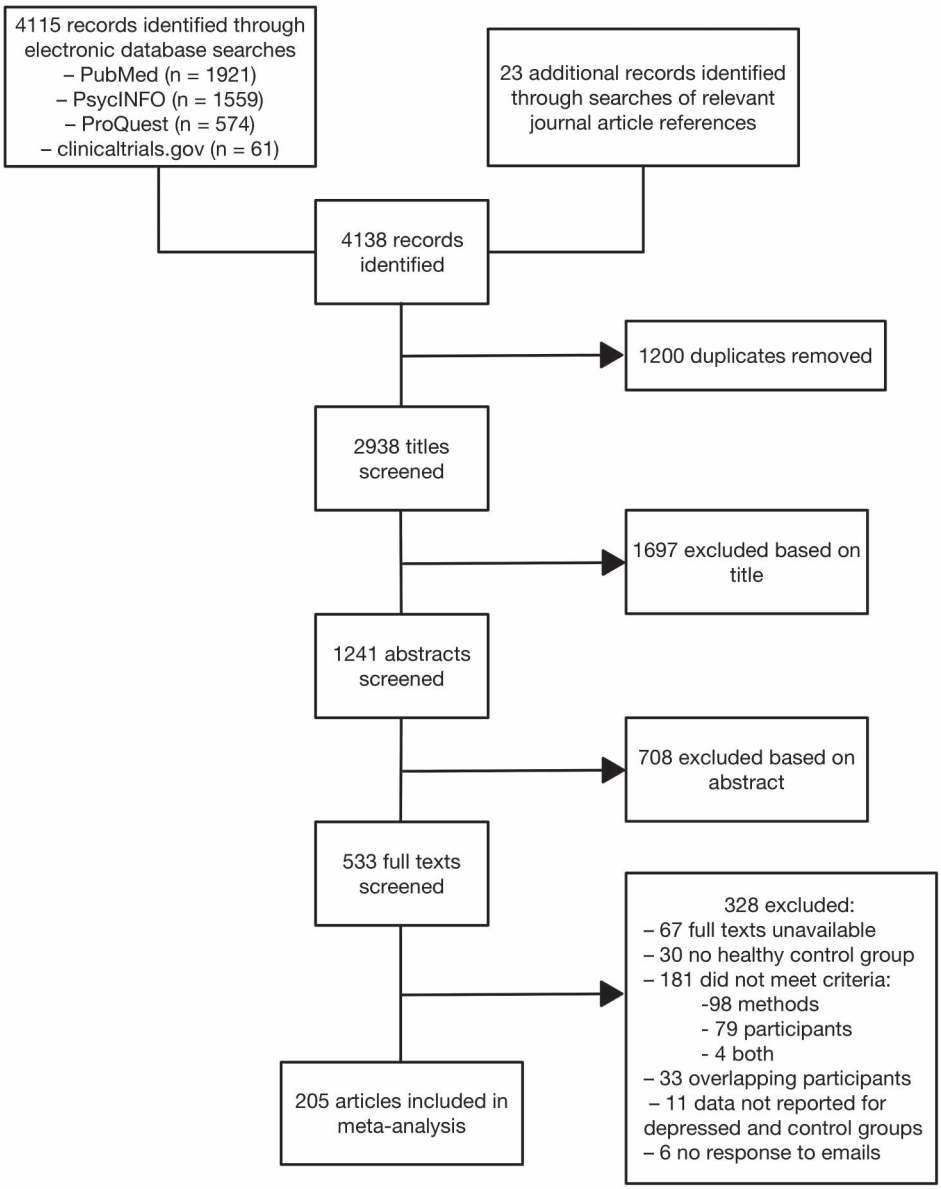
Two of the authors independently extracted data from eligible records and entered those into a spreadsheet created by the first author. All data were then compared and discrepancies were noted. The interrater agreement for data extraction and variable coding was .97. The first author referenced the full text of the records to rectify these discrepancies.

The following variables, when available, were extracted for each record: publication details, study characteristics, place of study, sample recruitment site (e.g., local communities, outpatient settings), study design (e.g., cross-sectional), diagnostic status (i.e., the proportion of individuals in the depressed group who had received a formal diagnosis of MDD[1]), depression status (the proportion of individuals in the depressed group who were currently depressed vs. remitted, see Footnote 1), symptom severity (name of depression questionnaire and

[1] When exact proportions could not be calculated, the mean value was entered. This was done for 8/236 entries for diagnostic status, 5/236 for depression status, 15/75 for episode, and 3/166 for medication status.

Figure 1

Flowchart Illustrating the Stages of Study Selection for Inclusion in the Meta-Analysis



score of individuals in the depressed group on that assessment), episode (the proportion of individuals in the depressed group with recurrent depression vs. first-episode depression, see Footnote 1), age of onset, time since diagnosis, medication status (the proportion of individuals in the depressed group who were taking medication for depression, see Footnote 1), sample size, age range, mean age for depressed and control groups, sex/gender[2] (the proportion of females in the sample), mean years of education, intelligence quotient (IQ) information (IQ test name and mean score for depressed and control groups), and whether groups were matched for age and education. When only age of onset or time since diagnosis was provided, one was calculated from the other using the mean age of individuals in the depression group. Table S1 (Supplemental Material) presents these details for each study included in the meta-analysis.

Episodic memory measures were coded to indicate the type of memory test (recall, recognition); the presence of a delay between study and test (immediate, short delay, long delay); for recall: whether the task was cued (free, cued); the stimulus materials (words, high verbalizable stimuli, low verbalizable stimuli); the valence of the stimuli (neutral, positive, negative); the memory outcome measure, accuracy (e.g., hits, items correctly recalled),

[2] The terms "sex" and "gender" are often used interchangeably in the literature. We used sex/gender as recorded by the original researchers: "sex" was reported for 70 of the depression samples, "gender" was reported for 133; for 22 samples, neither term was used (only number or proportion of females and males was reported); 11 samples did not report any sex/gender information. For simplicity, we use the term "sex" to refer to the sex/gender variable in the remainder of the article.

discrimination (*d′*, *Pr*), commission errors (e.g., false alarms, intrusions); and, when applicable, whether the measure assessed familiarity or recollection. For test delay, *immediate* was defined as memory tests that occurred right after encoding, with no break or intervening task, for example, list learning trials on the California Verbal Learning Test (CVLT). *Long delay* was defined as memory tests that occurred more than 20 min after encoding, consistent with neuropsychological tests (Delis et al., 2000; Wechsler, 2009). *Short delay* was defined as test delays that fell between immediate and long delay—after a brief intervening task (e.g., a mathematical distractor task as in MacQueen et al., 2003, or recall of a distractor word list like in the CVLT) or after a short break (the shortest being the 3-min delay in the Rey–Osterrieth complex figure task). For stimulus material, *high verbalizable stimuli* were defined as those that lend themselves toward verbal descriptions that are fairly consistent across participants, for example, pictures of objects, images from the International Affective Picture System (IAPS); *low verbalizable stimuli* were defined as those that cannot be easily described verbally, such as abstract designs or spatial locations (Golby et al., 2001). For valence, *positive*, *negative*, and *neutral* were defined using the classifications provided by the authors (see Supplemental Material, Table S2 for classification methods).

It should be noted that while Voyer et al. (2021) argue that Trial 1 (or List 1) on the CVLT reflects short-term or working memory rather than episodic memory, others do not (e.g., Chapman et al., 2006; Lundervold et al., 2019; Raffard et al., 2016). The main reason is that the list length for the CVLT far exceeds the typical short-term memory span, and it would tap primary or short-term memory only when participants elect to start their recall at the end of the list, which they are not instructed to do. Thus, while Trial 1 may be contaminated with short-term memory, it is unlikely to be a pure measure of this construct. We tested this contamination empirically by examining the 10 studies that provided details for both Trial 1 recall and final trial recall by dummy coding this variable and testing it as a moderator of the effect sizes. Results showed a nonsignificant effect of trial ($\beta = -0.10$, $p = .214$). That said, it is still important to note that the boundary between short-term/working memory and episodic memory is still unclear and not necessarily agreed upon, and the two may sometimes be difficult to distinguish (Asperholm et al., 2019).

The measures listed above were investigated as task moderators for the relationship between memory and depression. Note that for all moderator variables, effect sizes were coded for one level of the variable or the other (e.g., recall OR recognition for type of memory test, immediate OR short delay OR long delay for test delay). If a study reported just a single estimate for a battery that spanned multiple moderators of interest—for instance, a series of tests that included both recognition and recall but was reported as a single *z*-score rather than a separate score for recall and recognition—this estimate was not included in the analysis of that particular moderator, but it was included in all other relevant analyses.

All relevant effect sizes were included, but redundant measures were excluded (e.g., if a study reported performance on individual learning trials for a word-list memory test like the CVLT, the effect sizes for each learning trial were included; if a cumulative score was additionally reported, this measure was used in place of the individual learning trial scores). Estimates of memory bias or strategies (e.g., semantic clustering) were not included. If a study reported both index scores (e.g., verbal memory, immediate memory) and scores on individual subtests from the Wechsler Memory Test, effect sizes were calculated using the subtest scores to more precisely code the variables as outlined above (e.g., recall, immediate). Table S3 (Supplemental Material) presents effect sizes and associated task moderator categorizations for each study included in the meta-analysis.

Meta-Analytic Approach

Means and standard deviations were extracted for all episodic memory measures. Effect sizes for the memory measures reported in each study were calculated as Cohen's *d* to compare performance of depressed participants to controls. Cohen's *d* was calculated as $(M_1 - M_2)/SD_{pooled}$, where M_1 is the mean memory performance of the depressed group, M_2 is the mean memory performance of the control group, and SD_{pooled} is the pooled standard deviation of the two groups. For error measures in which higher scores indicate worse performance (e.g., false alarms), mean performance of the depressed group was subtracted from mean performance of the control group. Thus, more negative effect sizes reflect lower memory performance among depressed individuals relative to healthy controls. When means and standard deviations were not available, *F* or *t* statistics or *d* values were extracted. Effect sizes were estimated from *F* or *t* statistics using an online effect size calculator (Wilson, 2001; Wilson & Lipsey, 2001). In cases where data were presented only in a figure, the WebPlotDigitizer software (Rohatgi, 2019) was used to extract the relevant data. Corresponding authors were contacted to obtain missing information, particularly that which was necessary to calculate effect size. Fourteen of 38 authors responded to these requests. When such information could not be obtained, effect sizes were estimated (this was the case in 56 of the 995 included effect sizes, or 5.6%). In cases where the direction of the effect was known (e.g., when means but no measures of dispersion were provided), we estimated using conservative *p* values; $p = .05$ was used when the effect was labeled as significant, and $p = .5$ when it was labeled as nonsignificant, as in Coles et al. (2019; 27 of 56). If significance information was not provided but the direction of the effect was known, we assumed the effect was nonsignificant and used $p = .5$ for our calculations (15 of 56). In cases where the direction was not known, the effect size was entered as zero (14 of 56). Hedges' *g* correction for small-sample bias (Hedges & Olkin, 1985) was applied as $g = d[1 - (3/4\,df - 1)]$, where *df* represents the degrees of freedom for the entire sample (i.e., the total number of participants in both patient and healthy-control groups, minus two).

We conducted a three-level meta-analysis using the *metafor* package for R (Viechtbauer, 2010), augmented with the *clubSandwich* package (Pustejovsky, 2021). Additionally, the *dplyr* (Wickham et al., 2020) package was used for data manipulation. The three-level approach by itself (here implemented in the rma.mv() function) is more explicitly described in Cheung (2014). In a three-level random effects model, three sources of variance are modeled: sampling variance of the effect sizes, variance between effect sizes from the same study, and variance between effect sizes from different studies. A key advantage of this approach is that it allows one to fit models with dependent effect sizes nested within studies, allowing researchers to use all available information from studies with nonindependent effect sizes. This is in contrast to traditional two-level models, in which researchers may deal with dependencies by averaging effect sizes within a study, which may

lower the statistical power because some information in the effect sizes is lost through aggregation; alternatively, they may select only one effect size per study, which in turn limits the questions that can be addressed by the meta-analysis since some effect sizes are excluded. By analyzing individual effect sizes, the three-level model allows one to investigate variation between effect sizes and to test moderator variables to explain this variation while accounting for overlap in information that is contributed by effect sizes from the same study, therefore avoiding artificial inflation of power and an increase in Type I error. To deal with possible dependencies within studies, we deployed cluster-robust variance estimation with small-sample corrections through the coef_test() function in the *clubSandwich* package as described in Pustejovsky and Tipton (2021).

For each analysis, mean effect size and 95% confidence intervals were computed. Heterogeneity of effect sizes was assessed using the Q statistic, similar to traditional, two-level meta-analyses. When the Q statistic is not significant, it suggests studies differ only as a result of sampling error at the subject level; when the Q statistic is significant, it suggests factors in addition to sampling error are needed to account for variation. A second measure of heterogeneity, I^2, describes the percentage of variation across studies that is due to heterogeneity rather than chance, and is calculated as $100\% \times (Q - df)/Q$ (Higgins & Thompson, 2002). Unlike traditional methods, however, the I^2 statistic, which indexes the degree of heterogeneity, is split over levels. I^2 at Level 2 ($I_{(2)}^2$) and Level 3 ($I_{(3)}^2$) indicate the proportion of the total variation of the effect sizes due to Level 2 and Level 3 between-study heterogeneity. For multilevel regression models, τ^2 at Level 2 ($\tau I_{(2)}^2$) indicates the heterogeneity of effects due to differences between measures, while τ^2 at Level 3 ($\tau I_{(3)}^2$) indicates the heterogeneity of effect sizes across studies after controlling for the different types of measures at Level 2. R^2 indicates the proportion of estimated heterogeneity at Level 2 ($R_{(2)}^2$) and Level 3 ($R_{(3)}^2$) explained by the predictors.

Moderator Analyses

We investigated a number of moderators as possible sources of between-studies heterogeneity using metaregression analyses (using the maximum likelihood estimate method). The task moderators we examined were test type (recall, recognition), recall type (free, cued), test delay (immediate, short delay, long delay), material (words, high verbalizable stimuli, low verbalizable stimuli), valence (positive, neutral, negative), memory outcome (accuracy, discrimination, commission errors), and memory process (familiarity, recollection). The participant moderators were age, sex, years of education, IQ, diagnostic status, depression status, age of depression onset, time since diagnosis, episode (first episode vs. recurrent), early versus late-onset, symptom severity, medication status, matched age, matched education, and recruitment site. Only studies that reported education as a continuous variable (or provided enough information for years of education to be estimated) were included in the respective analysis. Recruitment site was assessed by comparing samples from patient settings (inpatient, outpatient, and randomized clinical trials) with those from community and university settings. Only studies that recruited depression groups exclusively from one of these two types of settings were included in the respective analysis (e.g., if a study recruited depressed participants from outpatient clinics *and* through community advertisements, it was not included in the analysis of recruitment site; however, if a study recruited from both inpatient and outpatient settings, it was included and coded for the "patient" category). Studies that did not report information about the moderator of interest were excluded from the respective analyses. Finally, to examine whether published studies had larger effects than unpublished studies, we included a moderator for publication status. Categorical moderators were dummy coded and entered into metaregression equations. For categorical moderators with more than two levels (e.g., valence), two of the levels were used as baselines in separate metaregression equations to allow for comparisons between all levels (e.g., neutral as baseline in one model to allow for comparisons with positive and negative; positive as baseline in another model to allow for comparison with negative).

Results

Descriptive Information

Effect sizes were calculated for each depression subsample with respect to a single healthy-control group, resulting in 236 unique comparisons from 205 articles with a total of 995 effect sizes. The included articles ranged in publication year from 1982 to 2020. These articles reflect research conducted across 30 countries: the United States (33.7%); the United Kingdom (9.8%); Germany (9.3%); Canada (8.3%); France (3.9%); Brazil and the Netherlands (3.4% each); Spain (2.9%); New Zealand, Sweden, and Switzerland (2.4% each); China, Japan, and Taiwan (2.0% each); Hungary and Norway (1.5% each); Belgium, Greece, Italy, and South Korea (1.0% each); Australia, Colombia, Czech Republic, Denmark, Hong Kong, Ireland, Israel, Portugal, Serbia, and Turkey (0.5% each). It was unclear where two studies were conducted. Included studies were published in English, Spanish, French, and Hungarian. The studies included a total of 7,900 depressed participants and 10,121 healthy controls. See Table S4 (Supplemental Material) for descriptive statistics for the sample and moderator variables and the effect size distribution across moderators; See Table 1 for descriptive statistics for continuous moderators across the levels of categorical moderators.

Overall Effect and Publication Bias

The need for a three-level model was assessed by comparing fit with that of a two-level model. The difference in fit was significant, $\chi^2 = 1{,}597.74$, $p < .0001$, indicating the three-level model was a better fit for the data. The three-level model yielded an average effect size of −0.36, 95% CI [−0.41 to −0.31]; $p < .0001$). Heterogeneity was significant, $Q(994) = 3{,}524.93$, $p < .0001$; $\tau_{(2)}^2 = 0.09$, $p < .0001$; $\tau_{(3)}^2 = 0.11$, $p < .0001$; $I_{(2)}^2 = 0.34$ and $I_{(3)}^2 = 0.41$), suggesting the effect sizes were highly variable and thus indicating the need for moderator analyses. Regression analysis on the funnel plot (Figure 2) was conducted using an Egger test appropriate for multilevel meta-analysis (Rodgers & Pustejovsky, 2020). Both the Egger sandwich test and the multi-level meta-analysis (MLMA) Egger test yielded nonsignificant slopes ($p = .66$ and .79, respectively, using the modified measure of precision described in Pustejovsky & Rodgers, 2019), indicating no evidence for selective reporting.

Task Moderators

Table 2 contains effect size estimates for each level of each task moderator and the accompanying moderator analyses. Our analyses

Table 1

Continuous Across Categorical Moderator Characteristics for Studies Included in the Meta-Analysis

Continuous moderator	*M*	Range	*j*	*M*	Range	*j*	*M*	Range	*j*
Test type		Recall			Recognition				
Age	47.6	18.2–92.3	180	47.5	18.5–92.3	106			
Sex	.680	.2–1	174	.655	.2–1	100			
Education	12.7	3.9–17.4	113	12.5	7.1–16.3	68			
Premorbid IQ	108.2	78.9–123.1	45	108.4	90.6–123.1	32			
Diagnostic status	.845	0–1	188	.820	0–1	110			
Depression status	.807	0–1	188	.822	0–1	110			
Medication status	.455	0–1	132	.380	0–1	81			
Symptom severity	15.5	.2–33	144	15.3	1.5–32.8	82			
Episode	.628	0–1	64	.608	0–1	36			
Time since diagnosis	135.6	1.7–477.6	57	137.7	7.1–477.6	35			
Age of depression onset	37.7	15.3–74.1	57	34.2	15.3–71.3	35			
Onset (LOD vs. EOD)	.572	0–1	28	.474	0–1	11			
Recall type		Cued			Free				
Age	46.3	21.1–83.3	37	47.9	18.2–92.3	168			
Sex	.674	.3–1	37	.676	.2–1	161			
Education	13.7	7.2–16.0	26	12.7	3.9–17.4	108			
Premorbid IQ	109.1	95.3–117.0	13	108.3	78.9–123.1	41			
Diagnostic status	.873	0–1	40	.850	0–1	173			
Depression status	.909	0–1	40	.791	0–1	173			
Medication status	.383	0–1	31	.438	0–1	122			
Symptom severity	17.7	2.4–28.5	31	15.2	.2–33.0	132			
Episode	.551	0–1	13	.614	0–1	60			
Time since diagnosis	164.1	1.7–477.6	18	132.2	1.7–477.6	52			
Age of depression onset	34.8	15.8–74.1	18	37.9	15.3–74.1	52			
Onset (LOD vs. EOD)	.530	0–1	7	.582	0–1	27			
Test delay		Immediate			Short delay			Long delay	
Age	49.2	18.2–92.3	135	44.9	18.5–84.9	88	49.5	19.5–76.5	104
Sex	.662	.2–1	127	.679	.2–1	87	.667	.2–1	102
Education	12.6	5.6–17.4	89	12.7	3.9–16.3	53	13.0	5.6–16.5	74
Premorbid IQ	107.9	78.9–123.1	37	109.7	90.6–119.9	20	107.8	78.9–123.1	34
Diagnostic status	.873	0–1	139	.777	0–1	93	.889	0–1	108
Depression status	.791	0–1	139	.883	0–1	93	.769	0–1	108
Medication status	.431	0–1	106	.408	0–1	58	.488	0–1	86
Symptom severity	15.6	.2–32.8	109	15.9	1.5–33.0	69	15.0	1.2–31.2	90
Episode	.646	0–1	50	.627	0–1	29	.602	0–1	47
Time since diagnosis	127.5	4.7–352.0	44	152.9	1.7–477.6	24	140.4	1.7–477.6	40
Age of depression onset	38.3	17.6–74.1	44	31.3	15.8–71.3	24	36.8	15.8–64.8	40
Onset (LOD vs. EOD)	.560	0–1	19	.496	0–1	11	.534	0–1	19
Stimulus material		Words			High verbalizable			Low verbalizable	
Age	46.7	18.2–92.3	190	50.7	19.9–92.3	42	55.0	32.0–76.4	44
Sex	.666	.2–1	186	.664	.2–1	40	.663	.2–1	42
Education	12.8	3.9–17.4	119	11.5	6.4–16.2	26	12.2	5.6–15.8	34
Premorbid IQ	108.5	78.9–123.1	48	105.2	78.9–123.1	12	104.9	78.9–119.9	19
Diagnostic status	.835	0–1	200	.736	0–1	44	.994	.812–1	44
Depression status	.824	0–1	200	.849	0–1	44	.796	0–1	44
Medication status	.428	0–1	139	.397	0–1	30	.527	0–1	37
Symptom severity	15.5	.2–33.0	152	16.7	3.1–32.8	30	17.2	1.2–32.8	36
Episode	.609	0–1	66	.564	0–1	12	.548	0–1	23
Time since diagnosis	136.0	1.7–477.6	59	63.1	7.1–160.0	7	118.9	4.7–316.8	21
Age of depression onset	37.1	15.3–74.1	59	38.7	23.2–71.3	7	41.4	25.0–64.8	21
Onset (LOD vs. EOD)	.572	0–1	28	.517	0–1	5	.561	0–1	13
Valence		Neutral			Positive			Negative	
Age	47.9	18.5–92.3	192	36.6	18.2–84.9	72	35.8	18.2–84.9	81
Sex	.665	.2–1	186	.724	.3–1	69	.709	.3–1	77
Education	12.7	3.9–17.4	129	13.2	8.1–17.4	27	13.2	8.1–17.4	28
Premorbid IQ	108.7	78.9–123.1	54	104.1	90.6–113.0	8	106.5	90.6–118.4	10

Table 1 (*continued*)

Continuous moderator	*M*	Range	*j*	*M*	Range	*j*	*M*	Range	*j*
Diagnostic status	.850	0–1	200	.758	0–1	75	.696	0–1	86
Depression status	.829	0–1	200	.853	0–1	75	.868	0–1	86
Medication status	.440	0–1	150	.436	0–1	42	.417	0–1	44
Symptom severity	15.9	.2–33.0	149	15.4	1.5–29.5	60	14.9	1.5–29.5	69
Episode	.635	0–1	70	.600	0–1	14	.560	0–1	15
Time since diagnosis	133.8	1.7–477.6	65	99.3	22.0–230.6	17	99.3	22.0–230.6	17
Age of depression onset	36.2	15.3–74.1	65	28.1	15.3–56.0	17	28.1	15.3–56.0	17
Onset (LOD vs. EOD)	.554	0–1	28	1	—	2	1	—	2
Memory outcome		Accuracy			Discrimination			Commission errors	
Age	46.6	18.1–92.3	208	41.3	19.4–84.9	32	44.3	18.2–92.3	32
Sex	.665	.2–1	186	.718	.5–1	30	.652	.3–1	34
Education	12.7	3.9–17.4	129	13.3	10.2–15.9	19	12.5	8.4–16.0	15
Premorbid IQ	108.7	78.9–123.1	54	108.8	93.5–118.4	14	106.7	95.3–117.0	9
Diagnostic status	.850	0–1	200	.713	0–1	32	.699	0–1	38
Depression status	.829	0–1	200	.843	0–1	32	.958	0–1	38
Medication status	.440	0–1	150	.363	0–1	21	.375	0–1	23
Symptom severity	15.9	.2–33.0	149	15.2	2.4–27.5	27	17.6	7.0–28.5	24
Episode	.635	0–1	70	.467	0–1	11	.677	.5–1	4
Time since diagnosis	133.8	1.7–477.6	65	118.1	4.7–477.6	13	140.6	30.7–205.3	12
Age of depression onset	36.2	15.3–74.1	65	28.9	15.8–42.3	13	29.9	18.9–41.8	12
Onset (LOD vs. EOD)	.554	0–1	28	0	—	1	—	—	0
Memory process		Familiarity			Recollection				
Age	34.2	19.9–47.1	9	35.5	19.9–47.3	10			
Sex	.685	.571–.813	10	.677	.571–.813	11			
Education	14.7	13.8–15.5	4	14.7	13.8–15.5	4			
Premorbid IQ	113.6	110.5–118.4	6	114.0	110.5–118.4	7			
Diagnostic status	.5	0–1	10	.545	0–1	11			
Depression status	.884	0–1	10	.894	0–1	11			
Medication status	.536	0–1	6	.459	0–1	7			
Symptom severity	13.9	10.3–19.1	9	14.7	10.3–21.6	10			
Episode	.750	0–1	4	.726	0–1	5			
Time since diagnosis	133.3	25.2–241.2	4	133.3	25.2–241.2	4			
Age of depression onset	28.0	24.8–35.8	4	28.0	24.8–35.8	4			
Onset (LOD vs. EOD)	—	—	0	—	—	0			
Publication status		Unpublished			Published				
Age	37.5	18.2–84.9	34	47.4	18.5–92.3	190			
Sex	.739	.3–1	30	.663	.2–1	190			
Education	13.1	10.7–16.0	9	12.7	3.9–17.4	127			
Premorbid IQ	113.6	106.0–120.8	6	108.2	78.9–123.1	49			
Diagnostic status	.518	0–1	37	.867	0–1	199			
Depression status	.915	0–1	37	.824	0–1	199			
Medication status	.245	0–.706	16	.463	0–1	150			
Symptom severity	15.0	1.5–20.3	27	15.9	.2–33.0	152			
Episode	.587	.3–1	7	.632	0–1	68			
Time since diagnosis	85.3	74.6–95.9	2	133.9	1.7–477.6	65			
Age of depression onset	23.3	22.4–24.2	2	36.7	15.3–74.1	65			
Onset (LOD vs. EOD)	.320	—	1	.578	0–1	28			
Recruitment site		Community/university setting			Patient setting				
Age	36.9	18.2–83.3	58	48.3	22.6–76.5	114			
Sex	.686	.3–1	61	.666	.2–1	109			
Education	13.8	8.7–16.5	20	12.1	3.9–16.1	82			
Premorbid IQ	113.2	104.1–123.1	9	106.8	78.9–117.0	31			
Diagnostic status	.450	0–1	69	.975	0–1	114			
Depression status	.841	0–1	69	.838	0–1	114			
Medication status	.201	0–1	34	.585	0–1	92			
Symptom severity	13.4	1.5–33.0	49	17.5	1.2–31.2	90			
Episode	.699	.5–1	17	.577	0–1	37			

(*table continues*)

Table 1 (*continued*)

Continuous moderator	*M*	Range	*j*	*M*	Range	*j*	*M*	Range	*j*
Time since diagnosis	146.0	63.6–330.0	10	110.7	1.7–352.0	40			
Age of depression onset	35.1	15.3–56.4	10	38.2	18.9–74.1	40			
Onset (LOD vs. EOD)	.570	—[a]	4	.621	0–1	17			

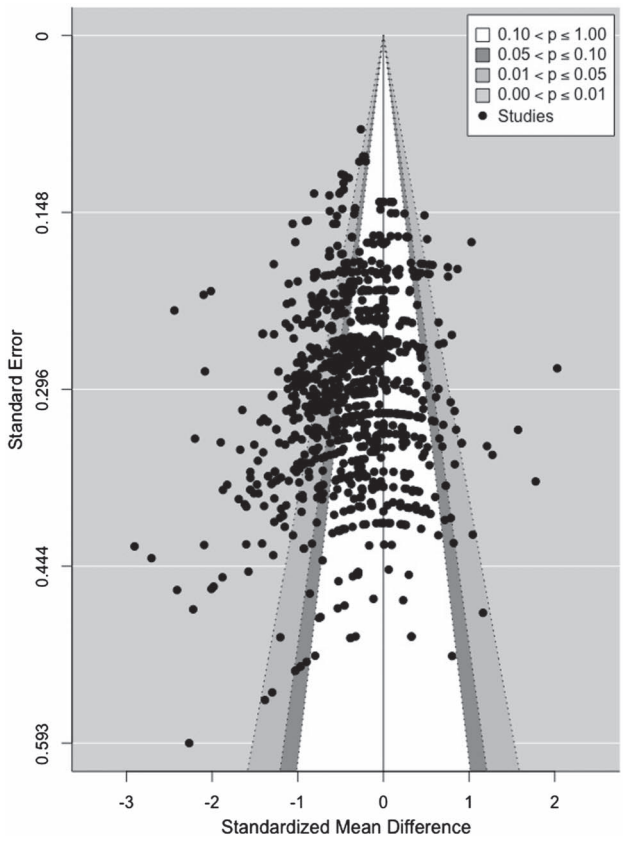
Note. *j* = number of unique depression-control group comparisons. All continuous moderators are based on information from depression groups. Diagnostic status = proportion diagnosed with major depressive disorder. Depression status = proportion experiencing current (as opposed to remitted) symptoms of depression. Episode = proportion with recurrent episodes of depression (as opposed to first-episode depression). Onset = proportion with late-onset depression (LOD; onset after age ~50) relative to early-onset depression (EOD). Medication = proportion taking medication for depression. If a study only reported Age of Depression Onset, Time Since Diagnosis was calculated using the mean age (and vice versa).
[a] Range is not reported because the estimated onset was used in all four studies. That is, the exact proportion of participants with LOD versus EOD could not be calculated for these studies, so the proportion was estimated using the mean proportion from all other studies that reported data on this variable.

show that depressed individuals performed worse than controls across all measures except for familiarity and negatively valenced stimuli.

Test Type

There was a significant effect of type of memory test, where the difference between depressed individuals and normal controls was significantly larger in recall than in recognition ($\beta = -0.17$, $p = .002$).

Figure 2
Funnel Plot of Standard Error as a Function of Effect Size (Standardized Mean Difference Between Depression and Control Groups)



Stimulus Material

Group differences were larger for low verbalizable stimuli than for high verbalizable stimuli ($\beta = -0.17$, $p = .024$). However, differences were not significant between words and high verbalizable stimuli ($\beta = 0.06$, $p = .266$) or between words and low verbalizable stimuli ($\beta = -0.10$, $p = .074$).

Memory Outcome

Poorer performance in the depressed group compared to control was due more to lower accuracy (e.g., hits, items correctly recalled) than to a greater number of commission errors (e.g., false alarms, intrusions). While accuracy did not significantly differ from discrimination ($\beta = 0.08$, $p = .553$), and discrimination did not significantly differ from commission errors ($\beta = 0.13$, $p = .375$), accuracy did significantly differ from commission errors ($\beta = 0.22$, $p = .004$). That is, those with depression were more likely to make errors of omission than errors of commission, indicative of a conservative response bias.

Valence

The effect of valence was significant. While memory performance between positive and neutral stimuli did not differ between groups ($\beta = 0.03$, $p = .516$), memory for negative stimuli was significantly better than that for both neutral and positive stimuli in depressed participants relative to controls ($\beta > 0.30$, $p < .001$).

Some evidence suggests that the manner in which events are encoded (e.g., with reference to the self vs. others) may be an important factor influencing emotional memory in depression (see Wisco, 2009, for review). To examine this, we compared memory performance for positive and negative material in self-referencing tasks (e.g., "Does this adjective describe me?") versus other kinds of tasks where attention at encoding is externally focused (e.g., "Is this caption compatible with the image?"). Only in self-referencing tasks ($\beta = 0.71$, $p < .001$) but not externally focused tasks ($\beta = 0.12$, $p = .064$) did valence moderate the relationship between depression and memory. As can be seen in the last six rows of Table 2, those with depression showed worse memory compared to controls for positive material in self-referencing tasks ($g = -0.45$), and for both positive and negative material in externally focused tasks ($g = -0.23$ and -0.11, respectively). However, in self-referencing tasks, those with depression showed superior memory for negative material relative to controls ($g = 0.26$).

Table 2
Task Moderator Analyses

Moderator and levels	*j*	*k*	*g*	β	95% CI	*p*	$R_{(2)}^2$	$R_{(3)}^2$	*Q*	$\tau_{(2)}^2$	$\tau_{(3)}^2$
Test type (*recognition vs. recall*)	233	988	—	−0.17	[−0.27, −0.06]	.002	.058	.007	3504.2*	0.08*	0.11*
Recognition	110	342	−0.24	—	[−0.32, −0.16]	—	—	—	—	—	—
Recall	188	646	−0.41	—	[−0.47, −0.34]	—	—	—	—	—	—
Recall type (*cued vs. free*)	186	643	—	−0.04	[−0.15, 0.06]	.427	.009	.000	2429.9*	0.09*	0.13*
Cued	40	80	−0.37	—	[−0.48, −0.25]	—	—	—	—	—	—
Free	173	563	−0.41	—	[−0.48, −0.35]	—	—	—	—	—	—
Test delay	217	921	—	—	—	—	.000	.004	3238.9*	0.09*	0.10*
Immediate vs. short delay	194	660	—	0.02	[−0.06, 0.09]	.679	—	—	—	—	—
Immediate vs. long delay	170	644	—	0.01	[−0.04, 0.07]	.665	—	—	—	—	—
Short delay vs. long delay	168	538	—	−0.004	[−0.08, 0.07]	.922	—	—	—	—	—
Immediate	140	380	−0.38	—	[−0.43, −0.31]	—	—	—	—	—	—
Short delay	92	277	−0.35	—	[−0.43, −0.28]	—	—	—	—	—	—
Long delay	108	261	−0.36	—	[−0.42, −0.30]	—	—	—	—	—	—
Stimili material	229	978	—	—	—	—	.000	.044	3482.7*	0.09*	0.10*
Words vs. high verbal	223	898	—	0.06	[−0.05, 0.17]	.266	—	—	—	—	—
Words vs. low verbal	206	827	—	−0.10	[−0.21, 0.01]	.074	—	—	—	—	—
High vs. low verbal	78	231	—	−0.17	[−0.30, −0.03]	.024	—	—	—	—	—
Verbal words	200	747	−0.36	—	[−0.41, −0.30]	—	—	—	—	—	—
Verbal pictures	44	151	−0.29	—	[−0.40, −0.19]	—	—	—	—	—	—
Nonverbal	44	80	−0.46	—	[−0.57, −0.35]	—	—	—	—	—	—
Valence	232	984	—	—	—	—	.071	.186	3470.7*	0.08*	0.09*
Neutral vs. positive	231	815	—	0.03	[−0.06, 0.12]	.516	—	—	—	—	—
Neutral vs. negative	231	840	—	0.33	[0.23, 0.43]	<.001	—	—	—	—	—
Positive vs. negative	87	313	—	0.30	[0.17, 0.44]	<.001	—	—	—	—	—
Neutral	200	671	−0.42	—	[−0.47, −0.36]	—	—	—	—	—	—
Positive	75	144	−0.39	—	[−0.48, −0.30]	—	—	—	—	—	—
Negative	86	169	−0.08	—	[−0.18, 0.01]	—	—	—	—	—	—
Memory outcome	236	995	—	—	—	—	.037	.029	3524.9*	0.09*	0.10*
Accuracy vs. discrimination	234	904	—	0.08	[−0.19, 0.36]	.553	—	—	—	—	—
Accuracy vs. com errors	222	897	—	0.22	[0.08, 0.36]	.004	—	—	—	—	—
Discrimination vs. com errors	69	189	—	0.13	[−0.16, 0.43]	.375	—	—	—	—	—
Accuracy	220	806	−0.38	—	[−0.45, −0.32]	—	—	—	—	—	—
Discrimination	32	98	−0.30	—	[−0.55, −0.05]	—	—	—	—	—	—
Errors	38	91	−0.16	—	[−0.30, −0.03]	—	—	—	—	—	—
Memory process (*Fam. vs. Rec.*)	11	50	—	−0.25	[−0.66, 0.15]	.274	.146	.000	146.6*	0.07*	0.10
Familiarity	10	23	−0.18	—	[−0.45, 0.09]	—	—	—	—	—	—
Recollection	11	27	−0.44	—	[−0.79, −0.09]	—	—	—	—	—	—
Publication status (*unpub vs. pub*)	236	995	—	−0.19	[−0.33, −0.06]	.008	.000	.050	3524.9*	0.09*	0.10*
Unpublished	37	210	−0.20	—	[−0.32, −0.08]	—	—	—	—	—	—
Published	199	785	−0.39	—	[−0.45, −0.34]	—	—	—	—	—	—
Valence in self-reference tasks (*positive vs. negative)*	34	99	—	0.71	[0.38, 1.04]	<.001	.293	0	574.4*	0.03*	[a]
Positive	31	47	−0.45	—	[−0.66, −0.24]	—	—	—	—	—	—
Negative	34	52	0.26	—	[0.10, 0.43]	—	—	—	—	—	—
Valence in external focus tasks (*positive vs. negative)*	57	204	—	0.12	[−0.003, 0.24]	.064	.027	.002	718.4*	0.11*	0.06*
Positive	46	92	−0.23	—	[−0.35, −0.11]	—	—	—	—	—	—
Negative	56	112	−0.11	—	[−0.21, −0.01]	—	—	—	—	—	—

Note. The moderator name is in boldface, the specific comparison between levels is in boldface italics. The first variable in the comparisons serves as the baseline (e.g., recognition is the baseline). *j* = number of unique depression-control group comparisons. *k* = number of effect sizes. *g* = Hedges' *g*. β coefficients are from metaregression analyses where categorical moderators with two or three levels were dummy coded and entered into the models as predictors. 95% CI corresponds to the β for moderators or *g* values for individual levels of moderators. *p* corresponds to the β for moderators. $R_{(2)}^2$ = proportion of estimated heterogeneity explained by the predictors at Level 2. $R_{(3)}^2$ = proportion of estimated heterogeneity explained by the predictors at Level 3. *Q* = *Q* statistic on the homogeneity of effect sizes. $\tau_{(2)}^2$ = heterogeneity of effects due to differences between measures. $\tau_{(3)}^2$ = heterogeneity of effect sizes across studies after controlling for the different types of measures at Level 2. High verbal = high verbalizable stimuli; Low verbal = low verbalizable stimuli; Com errors = commission errors; Fam. = familiarity; Rec. = recollection; Unpub = unpublished; Pub = published. For the levels of the moderators, *j* does not necessarily add up to the total *j* for the moderator (nor do the percentages add up to 100) because depression and control group performance may be compared on both recall and recognition, for example, within a single study.

[a] The three-level model indicated there was not enough variance at Level 3, creating a convergence issue. However, rerunning this analysis with a two-level model yielded the same result as the three-level model.

* $p < .05$ for *Q* and τ^2 statistics.

Nonsignificant Task Moderators

Neither test delay ($\beta < 0.02$, $p > .665$), recall type ($\beta = -0.04$, $p = .427$), nor memory process ($\beta = -0.25$, $p = .274$) significantly moderated the relationship between depression and memory.

Participant Moderators

Table 3 presents the moderator analyses for each of the participant variables.

Age

Age showed a significant linear relationship with effect size ($\beta = -0.007$, $p < .001$), such that each year of age was associated with an increase in the depression effect of 0.007 *SD* units (see Figure 3A). Adding a quadratic component for age to test for nonlinear effects yielded a significant result ($\beta = 0.0002$, $p = .041$), indicating that memory performance in depression decreases from early to midlife, then improves somewhat in later life, yet not back to the level observed in young ages.

Education

As shown in Figure 3B, for each additional year of education, individuals in the depressed group performed 0.05 *SD* better relative to controls ($p < .001$). Since some studies reported very low estimates for years of education (e.g., Tam & Lam, 2012, in which the duration of education was reported as 3.9 years in the depression group), we wanted to ensure these studies were not biasing the relationship between education and effect size. We conducted an additional analysis limited to those studies in which depressed participants had at least 10 years of education. Again, this yielded a significant effect, $\beta = 0.05$, 95% CI [0.01, 0.09], $j = 117$, $k = 465$, $p = .009$, $R_{(2)}^2 = .000$ and $R_{(3)}^2 = .082$.

Diagnostic Status: Formal MDD Diagnosis Versus Subthreshold Depression

Diagnostic status was found to have a significant effect ($\beta = -0.30$, $p < .001$): The larger the proportion of MDD diagnoses in the

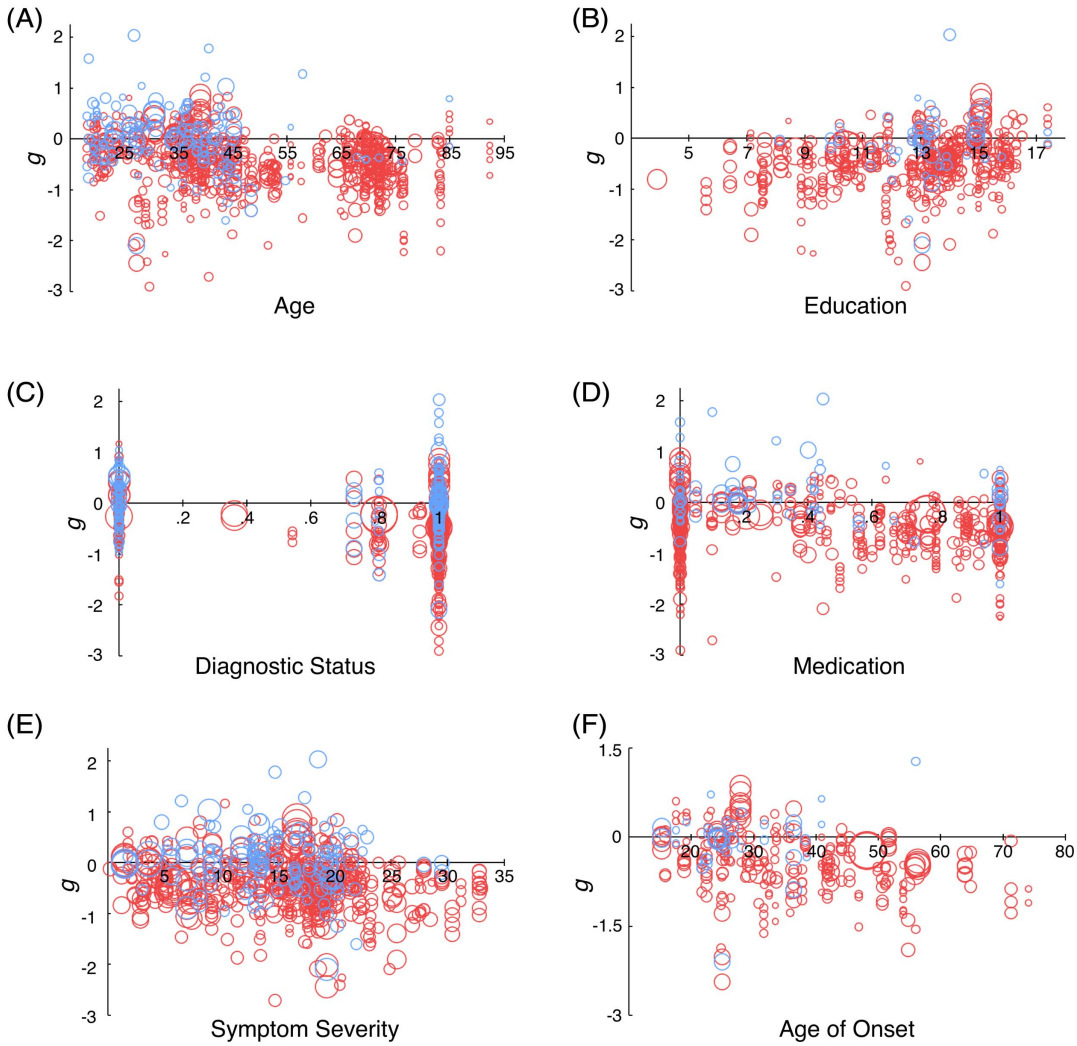
Table 3
Participant Moderator Analyses

Moderator	*j*	*k*	β	95% CI	*P*	$R_{(2)}^2$	$R_{(3)}^2$	*Q*	$\tau_{(2)}^2$	$\tau_{(3)}^2$
Age (linear)	224	943	−0.007	[−0.01, −0.005]	<.0001	.000	.179	3368.9*	0.08*	0.09*
Age (quadratic)	—	—	0.0002	[0.00001, 0.0003]	.041	.000	.200	—	0.08*	0.09*
Sex	220	901	−0.17	[−0.51, 0.17]	.324	.002	.000	3289.5*	0.09*	0.11*
Education	136	563	0.05	[0.03, 0.07]	<.001	.000	.163	2049.1*	0.08*	0.10*
Premorbid IQ	55	243	0.01	[−0.005, 0.02]	.333	.001	.024	841.3*	0.08*	0.07*
Diagnostic status	236	995	−0.30	[−0.40, −0.21]	<.001	.000	.134	3524.9*	0.09*	0.09*
Depression status	236	995	−0.06	[−0.18, 0.07]	.387	.000	.002	3524.9*	0.09*	0.11*
Matched age	216	911	−0.13	[−0.27, 0.02]	.096	.000	.022	3226.7*	0.09*	0.11*
Matched education	177	713	0.06	[−0.11, 0.22]	.504	.001	.001	2682.5*	0.10*	0.11*
Medication status	166	661	−0.24	[−0.39, −0.09]	.002	.001	.091	2340.7*	0.08*	0.11*
Symptom severity—MADRS	27	115	−0.01	[−0.02, 0.001]	.101	.000	.213	307.6*	0.0003	0.05*
Symptom severity—HDRS	83	327	−0.01	[−0.02, 0.0002]	.053	.001	.046	972.8*	0.04*	0.09*
Symptom severity—BDI	102	438	−0.01	[−0.02, −0.004]	.009	.000	.191	1735.8*	0.18*	0.05*
Symptom severity (HDRS scale)	179	752	−0.01	[−0.02, −0.01]	<.001	.000	.097	2683.6*	0.10*	0.07*
Episode (first vs. recurrent)	75	298	−0.03	[−0.32, 0.26]	.844	.0003	.0002	1147.7*	0.06*	0.11*
Time since diagnosis	67	296	0.0001	[−0.001, 0.001]	.851	.000	.002	1278.3*	0.08*	0.12*
Age of depression onset	67	296	−0.01	[−0.02, −0.01]	<.001	.000	.228	1278.3*	0.08*	0.09*
Early- vs. late-onset	29	116	−0.25	[−0.71, 0.21]	.301	.005	.057	357.8*	0.01	0.14*
Recruitment site	183	772	−0.36	[−0.46, −0.25]	<.001	.004	.269	3068.0*	0.11*	0.08*
Mood-congruency effects (interactions with valence)										
Age	221	936	—	—	—	.075	.255	3320.2*	0.08*	0.08*
Age			−0.01	[−0.01, −0.003]	<.001					
Valence			0.33	[0.05, 0.61]	.029					
Age × Valence			−0.001	[−0.01, 0.01]	.722					
Depression status	232	984	—	—	—	.076	.173	3470.7*	0.08*	0.09*
Depression status			−0.08	[−0.21, 0.05]	.251					
Valence			0.30	[−0.07, 0.66]	.153					
Depression status × Valence			0.03	[−0.36, 0.41]	.901					
Diagnostic status	232	984	—	—	—	.083	.256	3470.7*	0.08*	0.08*
Diagnostic status			−0.29	[−0.39, −0.19]	<.001					
Valence			0.18	[0.04, 0.31]	.020					
Diagnostic status × Valence			0.19	[0.01, 0.40]	.071					

Note. *j* = number of studies/clusters. *k* = number of effect sizes. β coefficients are from metaregression analyses where continuous moderators were entered in the models as predictors, or categorical moderators (in the case of matched age and matched education) with two levels were dummy coded and entered in the models as predictors. 95% CI corresponds to the β for moderators. *p* corresponds to the β for moderators. $R_{(2)}^2$ = proportion of estimated heterogeneity explained by the predictors at Level 2. $R_{(3)}^2$ = proportion of estimated heterogeneity explained by the predictors at Level 3. $Q = Q$ statistic on the homogeneity of effect sizes. $\tau_{(2)}^2$ = heterogeneity of effects due to differences between measures. $\tau_{(3)}^2$ = heterogeneity of effect sizes across studies after controlling for the different types of measures at Level 2. MADRS = Montgomery–Åsberg Depression Rating Scale; HDRS = Hamilton Depression Rating Scale; BDI = Beck Depression Inventory.
* $p < .05$.

Figure 3

Effect Size as a Function of Age (A), Education (B), Diagnostic Status (C), Medication Status (D), Symptom Severity (E), and Age of Onset (F)



Note. Bubble size corresponds to the sample size of the depression group. Blue = negative stimuli, red = positive and neutral stimuli. All presented variables significantly moderated the relationship between depression and episodic memory. See the online article for the color version of this figure.

depressed sample, the larger the difference between depressed and control groups (see Figure 3C).

Medication Status

Participants in the depression group who were taking medication performed significantly worse than those who were not taking medication, relative to controls ($\beta = -0.24$, $p = .002$; see Figure 3D). To ensure this effect was not confounded by including individuals without diagnoses, we conducted an additional analysis only in those who were diagnosed with MDD. The effect remained significant, $\beta = -0.23$, 95% CI [−0.40 to −0.07], $j = 149$, $k = 583$, $p = .006$, $R_{(2)}^2 = .001$ and $R_{(3)}^2 = .082$).

Symptom Severity

The three most widely used inventories to measure depressive symptom severity in the present sample of studies were the Montgomery–Åsberg Depression Rating Scale (MADRS), the Hamilton Depression Rating Scale (HDRS), and the Beck Depression Inventory (BDI). Higher scores on the BDI ($\beta = -0.013$, $p = .009$), but not on the MADRS ($\beta = -0.012$, $p = .101$) or HDRS ($\beta = -0.009$, $p = .053$), were associated with greater memory deficits in the depressed groups compared to the controls. An additional analysis was conducted after transforming all severity scores to the HDRS scale using the formulas described in Heo et al. (2007; HDRS = −1.58 + 0.86 [MADRS]) and Vittengl et al. (2005;

HDRS = 0.65 + 0.67 [BDI]).[3] This analysis revealed that higher symptom severity was associated with a larger deficit in depressed versus control groups (β = −0.13, $p < .001$; see Figure 3E).

Age of Depression Onset

Later age of onset was associated with significantly larger differences between depressed individuals and controls (β = −0.01, $p < .001$; see Figure 3F).

Recruitment Site

The settings from which participants were recruited did significantly moderate the relationship between depression and memory (β = −0.36, $p < .001$; see Table 3). While those in depression groups showed reduced memory relative to controls across the board, these differences were smaller in groups recruited from community and university settings, $g = -0.17$, 95% CI [−0.24 to −0.10] than in groups recruited from patient settings, $g = -0.53$, 95% CI [−0.61 to −0.45].

Nonsignificant Participant Moderators

Depression status (β = −0.06, $p = .387$),[4] sex (β = −0.17, $p = .324$), premorbid IQ (β = 0.01, $p = .333$), depressive episode (first vs. recurrent; β = −0.03, $p = .844$), late versus early-onset (β = −0.25, $p = .301$), time since diagnosis (β = 0.0001, $p = .851$), matched age (β = −0.13, $p = .096$), and matched education (β = 0.06, $p = .504$) did not significantly influence effect size. Note that for the analysis of premorbid IQ, only studies that used standardized measures of IQ were included. One study (Ladegaard et al., 2014) reported scores on the Danish Adult Reading Test (DART); these scores were converted using the formula described in Hjorthøj et al. (2013): IQ = 128.5 − 0.84 × (DART errors).

Publication Status

A concern in meta-analyses is that effect size estimates may be inflated if unpublished materials are excluded, as effects are often smaller than those found in published literature. In the current meta-analysis, 21% of the effect size estimates were from unpublished material. Our analyses showed that effects from published sources were in fact larger than those from unpublished sources, $g = -0.39$ 95% CI [−0.45, −0.34] and $g = -0.20$ [−0.32, −0.08], respectively; β = −0.19, $p = .008$; see Table 2.

Unique Predictive Power of Moderators

Ideally, we would be able to explore the unique predictive power of each moderator variable by simultaneously entering all moderators into a regression equation. Although our sample is relatively large, not all studies reported information for all moderators. However, because some moderators were correlated across studies (correlation tables presented in Supplemental Material, Table S5), we wanted to investigate whether they were accounting for unique variance in effect sizes. To avoid overfitting the model and to ensure sample size was sufficiently large, we only considered moderators with information reported for >75% of the effect sizes (see Table S4, Supplemental Material). Each of these moderators (age, diagnostic status, depression status, symptom severity, publication status, and recruitment site) was analyzed in a model with the other moderators with which it was correlated. For the sake of brevity, select results are presented here, with the statistics for all models presented in Table S6 (Supplemental Material).

When included in a model with age, depression status, publication status, and recruitment site, diagnostic status was no longer a significant moderator (β = −0.14, $p = .122$) of the relationship between depression and memory (age and recruitment site, however, remained significant; β = −0.01, $p = .002$ and β = −0.23, $p = .002$, respectively), Diagnostic status likely accounts for shared variance with other moderators. For instance, patient samples are more frequently diagnosed with depression relative to community and university samples. However, diagnostic status and symptom severity were both significant (β = −0.23, $p = .002$ and β = −0.02, $p = .015$, respectively) when entered into a model with depression status (β = 0.10, $p = .385$), suggesting that diagnostic status and symptom severity account for unique variance in the relationship between depression and memory performance.

While medication status was only reported for 66.4% of the effect sizes, a reviewer suggested we assess its unique predictive power given it was significantly correlated with onset age, episode, and diagnostic status (see Table S5, Supplemental Material); episode and diagnostic status may be particularly necessary to examine with medication status as they can be seen as measures of severity (though notably, medication status was not found to correlate with symptom severity). We therefore conducted three additional models with multiple moderators:

1. Medication status, episode, and diagnostic status.
2. Medication status, episode, diagnostic status, and age of onset (age of onset was not added to the initial model since it decreased the number of effect sizes from 255 to 167).
3. Medication status and symptom severity (though these variables were not correlated, we felt this may be an important model to examine).

In the first two models, medication status was the only significant moderator (β = −0.429, $p < .001$ in Model 1; β = −0.318, $p = .033$ in Model 2). In the third model, both medication status and symptom severity were significant (β = −0.237, $p = .003$ and β = −0.013, $p = .003$, respectively).

Valence × Participant Moderator Interactions

Age-related changes in emotional memory are well-documented in the literature (Reed et al., 2014), but it is unclear whether these changes are impacted in depression. Furthermore, given our interest in exploring whether depressive deficits are tied to one's current mood state, it is also worth exploring mood-congruency effects in particular. That is, does memory for emotional material differ as a function of diagnosis or current symptoms? To investigate these questions, we conducted additional analyses with age, depression

[3] In cases where articles reported both BDI and MADRS scores, we transformed both values then took the average.

[4] Because those with current symptoms included individuals with and without diagnoses, we also assessed depression status just in those who were diagnosed; however, the effect remained nonsignificant, β = −0.14, 95% CI [−0.27 to −0.002], $j = 180$, $k = 736$, $p = .052$, $R_{(2)}^2 = .000$, and $R_{(3)}^2 = .023$.

status, and diagnostic status with the addition of valence interaction terms. Because memory did not differ for positive and neutral valence, we used the dummy-coded variable for negative valence, with 1 indicating a negative stimulus and 0 indicating a positive or neutral stimulus. Each model included terms for the participant moderator (e.g., age), valence, and the Moderator × Valence interaction. The lower section of Table 3 presents the results of these analyses. The interactions with age ($\beta = -0.001$, $p = .722$), depression status ($\beta = 0.03$, $p = .901$), and diagnostic status ($\beta = 0.19$, $p = .071$) were all nonsignificant.

Discussion

MDD is not simply a condition of disordered mood, but rather is characterized by wide-ranging symptoms that disrupt daily functioning. Particularly important for daily functioning is episodic memory—the ability to accurately remember personally experienced events. Consistent with previous meta-analyses that have investigated memory, but not episodic memory specifically (Ahern & Semkovska, 2017; Bora et al., 2013; Burt et al., 1995; Lee et al., 2012; McDermott & Ebmeier, 2009; Rock et al., 2014), we found individuals with depression showed an overall episodic memory deficit compared to healthy controls, with a Cohen's *d* of 0.36—a small to medium effect. Our large sample of studies and the use of a three-level model allowed for powerful, novel explorations of task and participant variables as moderators of the relationship between depression and memory impairments. We set out to answer four main questions regarding the nature of these impairments. These are described in detail below.

Is the Pattern of Episodic Memory Deficits in Depression Consistent Across the Adult Lifespan?

The finding that participant age predicted greater episodic memory deficits in depression suggests that depression-related disruptions in memory are not uniform across the adult lifespan, but are in fact magnified in later years. It should be noted that the addition of a quadratic component in this analysis was significant, suggesting that studies with middle-aged participants showed the worst performance in depressed groups relative to controls. As can be seen in Figure 3A, however, there was a paucity of studies with midlife individuals. Thus, further work is needed to assess this midlife pattern.

The linear effect of age, however, was quite robust, indicating that age is a significant moderator of the relationship between depression and memory. Our results suggest that at age 20, an individual with depression is expected to perform 0.14 *SD* below controls; at age 70 the group difference grows to 0.49 *SD*. As Dotson et al. (2020) noted, age- and depression-related changes may interact to produce a sort of "double jeopardy" for cognitive dysfunction. It is therefore possible that depression accelerates the cognitive declines often observed in aging. The present findings support this idea and are consistent with previous research suggesting that memory impairment is closely linked to functional disability in depressed older adults (Gallo et al., 2003; Yen et al., 2011).

One possibility for this finding of worse performance with increased age is that older individuals may have spent a greater total amount of time in depressive episodes. However, many (Albert et al., 2018; Bearden et al., 2006; Delaloye et al., 2010; Dresler et al., 2010) but not all (Behnken et al., 2010) studies have failed to find correlations between duration of depressive symptoms and memory performance, consistent with the current meta-analysis. It should be noted, however, that "time since diagnosis" does not capture the time spent in depressive episodes, as individuals with depression may spend time in and out of episodes over the course of many years (Möller, 2008). At the same time, the extent to which the "time since diagnosis" measure reflects actual illness duration may be informed by cohort effects. For instance, as social stigma surrounding mental health has been lessening in the relatively recent past, it could be that older adults, relative to their younger counterparts, might underreport depressive symptoms or avoid treatment entirely (Conner et al., 2010). In other words, it could be that at least for some studies, older adults are potentially underreporting their depressive symptoms relative to young adults and/or only those older adults with severe depression are willing to seek treatment, relative to younger adults. Such cohort effects could contribute, at least in part, to the finding of lower performance with increasing age.

Another indicator of longer symptom duration is recurrent depression; however, episode (recurrent vs. first episode) did not significantly moderate the relationship between memory and depression, in samples where all individuals had first-episode depression, $g = -0.46$, 95% CI [−0.62, −0.31]; in samples where all individuals had recurrent depression, $g = -0.44$, 95% CI [−0.71, −0.17]. It is possible these effects were obscured by the age of onset effect, in which later age of onset was associated with significantly worse memory performance. This age of onset effect seems relevant to the idea that late-life depression may be an early symptom or risk factor of dementia (Barnes et al., 2012; Byers et al., 2012; Dotson et al., 2008, 2010; Saczynski et al., 2010). Depressive symptoms may also develop in reaction to perceived declines in cognitive functioning (see Byers & Yaffe, 2011, for review). Longitudinal assessments with careful consideration of the timing of symptom development are needed to elucidate this relationship. While we cannot rule out the possibility that some of the cases of late-onset depression in the current meta-analysis include individuals with prodromal dementia, the age of onset effect was reliable across age, suggesting this explanation is unlikely. Collectively, these results support the idea that the age at which an individual is diagnosed with depression is more predictive of memory performance than is symptom duration or recurrence.

Across age, depression was associated with worse performance on almost all episodic memory tasks—recall (both free and cued) and recognition; immediate, short, and long-delayed tests; verbalizable words and pictures and nonverbalizable material; positive and neutral material; and recollection. Recollection, but not familiarity-based memory, was significantly affected in depression. These results support the idea that, like aging, depression has the largest impacts on cognition under conditions in which demands on frontally mediated cognitive control processes are high (see Dotson et al., 2020; Grahek et al., 2019; Snyder, 2013, for reviews), such as strategic retrieval of source-specifying details and subjective recollection of episodic information (e.g., temporal, spatial, internally generated associations; Cohn et al., 2008; Duarte & Dulas, 2020; Duarte et al., 2008; Duarte & Kensinger, 2019).

However, the nonsignificant difference between free and cued recall tasks challenges this idea: free recall requires self-initiated reconstructive processes, whereas in cued recall, the cue provides more environmental support for recovery of the original event

details (Craik, 1983). It is worth noting, however, the heterogeneity in the cued recall tasks. In some tasks, like the CVLT, semantic categories (e.g., fruits, tools) are used as cues. In others, such as associative memory tasks, participants are given pairs of items, and one of the items later serves as a retrieval cue for the other; the semantic similarity between cue and item can vary. This is important because age-related associative memory impairments are typically reduced when items are semantically related or can be integrated or unitized (Ahmad et al., 2015; Badham et al., 2012; Naveh-Benjamin et al., 2003). However, an analysis comparing these two types of cued recall tasks showed no significant differences, $\beta = -0.14$, 95% CI [−0.40 to 0.12], $j = 38$, $k = 75$, $p = .299$. While we were not able to investigate the benefit of unitization in depression due to few studies reporting such information, this is a question future studies may wish to explore.

It should be noted that the lack of a significant test delay effect in our results suggests that depression is associated with episodic memory impairments as well as impairments in other abilities. As previously mentioned, delayed retrieval tasks are the optimal measures for assessing episodic memory. Immediate retrieval tasks recruit some of the same processes as those used for delayed retrieval, in addition to other processes, including those governing executive functioning and processing speed (Casaletto et al., 2017). Other meta-analyses have shown depression-related deficits in these domains (Dotson et al., 2020; Snyder, 2013), therefore our findings are consistent with the idea that depressive deficits are observed in a variety of cognitive domains.

What Are the Effects of Valence on Episodic Memory in Depression?

While memory performance for positive and neutral material was impaired in those with depression relative to controls, this was not the case with negative stimuli. These findings indicate mood-congruency effects (Bower, 1981; Marchetti et al., 2018; Van Vleet et al., 2019), such that material which is consistent with one's mood state, or depressive self-schema, is better remembered than that which is inconsistent (Beck, 1967; Disner et al., 2011). Some evidence suggests that mood-congruent effects may be more prevalent in self-referencing tasks (e.g., Bradley et al., 1995; Romero et al., 2014, 2016) compared to externally focused encoding tasks (see Wisco, 2009, for review). This was supported by the current findings: Although individuals with depression exhibited poorer memory than controls for positive material across the two types of tasks, only in self-referencing tasks were their memory better than that of controls for negative material. The self-reference effect has consistently been demonstrated to improve memory performance, as self-referential processing encourages greater elaboration that taps into well-established schemas (Klein & Nelson, 2014; Symons & Johnson, 1997). In individuals with depression, the self-reference effect is stronger for negative material because it is more consistent with their self-schema (LeMoult & Gotlib, 2019).

It is also worth considering the potential confounding effects of stimulus traits besides valence. (Bireta et al., 2021) recently suggested that factors such as word length, arousal, and frequency of use might be responsible for some of the significant results typically attributed to valence in verbal memory tasks. After equating lists of positively and negatively valenced words on a host of linguistic factors, the authors failed to find immediate recall performance differences between the two stimulus sets in a healthy sample. As for the present work, 58 of the studies included in the valence analyses used verbal stimuli. Most, but not all of these studies controlled for at least word length (53.4%) and frequency of use (55.2%; see Table S2, Supplemental Material for details), suggesting that our findings are somewhat robust to stimulus noise. Unfortunately, information on other word qualities was not commonly provided in the included studies. Although beyond the scope of this work, further defining the interaction of depression, valence, and linguistic factors remains an interesting line of inquiry.

One intriguing question for future studies is whether the so-called age-related "positivity effect" is minimized in depression. The positivity effect is illustrated in memory studies when older adults remember more positive than negative information while younger adults tend to show the opposite pattern (Carstensen & Mikels, 2005; Mather, 2012; see Reed et al., 2014, for meta-analysis and review). Older adults engage in emotion regulation strategies to prioritize processing of positive information; however, when these regulatory processes are disrupted, the positivity effect in memory is eliminated (Mather & Knight, 2005). As chronic stressors have been shown to decrease the ability to use these regulation strategies (see Knight & Durbin, 2015, for review), it is feasible that depression later in life may similarly limit their implementation. We were unable to show that mood congruency was affected by age, however, this analysis was likely underpowered as a result of only eight effect sizes associated with negative valence in studies where the mean age was over 55. Thus, the interaction between age and depression on episodic emotional memory biases remains unclear and is a question ripe for future investigation.

Are Depression-Related Memory Deficits Tied to One's Current Depressive State?

Given our combined findings of greater episodic memory impairments in diagnosed MDD than in subthreshold depression and a nonsignificant difference between those with current and remitted symptoms, we believe memory deficits are not tied to an individual's depressed mood. It is, however, possible that remitted participants were still depressed relative to controls, as evidenced by significantly higher severity scores on depression inventories (see Table S7, Supplemental Material). Though the scores for all remitted groups were in the range considered "normal" for the various inventories, questions about these cutoff scores have been raised (see Romera et al., 2011; Zimmerman et al., 2004, 2012, for reviews). Furthermore, longitudinal studies have found that episodic memory impairments persist following recovery from depressive symptoms (Airaksinen et al., 2006). It is therefore likely that remission may represent lessening of affective symptoms but not cognitive symptoms. Together, these findings suggest episodic memory impairments in depression are independent of depressed mood.

Consistent with the idea that treated depression may not confer better episodic memory performance, use of depression medication was associated with poorer memory performance. While some meta-analyses have identified positive effects for certain antidepressants on hippocampal neurogenesis (Boldrini et al., 2009; Micheli et al., 2018) and certain memory functions (Prado et al., 2018; Rosenblat et al., 2015), others have not (Deuschle et al., 2004; Nagane et al., 2014;

Shilyansky et al., 2016). For example, Neu et al. (2005) found that while depressive symptoms as measured by the HDRS disappeared after treatment with antidepressants; verbal memory impairments persisted even after a 6-month remission period. The persistence of memory difficulties in remitted depression highlights a blind spot in treatment interventions. To date, most pharmacological treatments focus only on alleviating mood-related symptoms. Our findings underscore the necessity for interventions that target both mood and cognitive symptoms in depression.

Antidepressant medications may sometimes contribute to episodic memory deficits in depression, perhaps due to anticholinergic side effects, which have been shown to reduce long-term memory recall, even in healthy, nondepressed participants (Schmitt et al., 2001; see Serretti et al., 2010, for review). Indeed, some of the most commonly reported cognitive side effects of antidepressants include memory impairments (Popovic et al., 2015). Thus, although the current meta-analysis cannot address this question directly, it is possible that memory impairments may be worsened or even induced following antidepressant use in depression.

We would be remiss if we did not consider other potential explanations for the negative association between depression medication use and memory performance. The metaregression approach is only able to identify associations between variables across studies, but it is unable to determine causal relationships or directionality. It is certainly possible that memory disturbances are a particularly motivating factor for individuals to seek treatment. Relatedly, these symptoms may contribute to physician's perceptions of patients' depression severity, thereby making it more likely for those individuals who exhibit memory problems to be prescribed medication for depression. Notably, when assessing the unique predictive power of moderators, medication remained a significant predictor of memory deficits in depression when controlling on diagnostic status, episode, onset age, and symptom severity. That said, the data we used necessarily represent a snapshot that cannot speak to other factors relevant to antidepressant treatment. For instance, medication may have been prescribed when subjects had more severe symptoms that had lessened by the experiment date. Because we only had information on present symptom severity available, it was not possible to additionally test the predictive power of depressive symptoms pretreatment. Further, the analyses conducted herein could not consider the specific details of patients' antidepressant regimens (e.g., length of treatment, types of medications administered, dosage, polypharmacy). Consequently, we do not believe physicians should change their recommendations for medication based purely on these results. Although past work has suggested that acute treatment with antidepressants does not alleviate (nor worsen) verbal memory deficits in depression, the impact of chronic treatment could be different (Shilyansky et al., 2016). Additional experimental work over a longer time period is needed to more precisely define the relationship between depression medication and memory performance.

Are There Factors That Protect Against Memory Impairments in Depression?

In the current meta-analysis, higher educational attainment was associated with less memory impairment. Education is often used as a proxy for cognitive reserve (Anthony & Lin, 2018; Stern, 2002; Yochim & Woodhead, 2018). Cognitive reserve can be viewed as an active process in which the brain attempts to leverage preexisting cognitive processes or new compensatory techniques to counteract the effects of pathology (Stern, 2002; Stern et al., 2018). That is, two individuals with depression may have similar pathology (e.g., reduced hippocampal volume), but the individual with higher cognitive reserve will show fewer cognitive deficits than the one with lower cognitive reserve (Barulli & Stern, 2013).

Similar moderating effects of cognitive reserve have been reported in previous studies (Murphy & O'Leary, 2010; Venezia et al., 2018) and in systematic reviews and meta-analyses (Lee et al., 2012; Opdebeeck et al., 2015), with differences between MDD and control groups diminishing with higher levels of reserve. Similar effects have also been reported in subthreshold depression (McLaren et al., 2015). The protective effects of cognitive reserve have been linked to cognitive networks being (a) more efficient, in that less activation is needed to perform at a similar level as less efficient networks, and (b) having a higher capacity, in that networks can ramp up activation to a higher level in response to increasing task difficulty (Barulli & Stern, 2013; Cabeza et al., 2018). Although IQ correlates with education (see Ritchie & Tucker-Drob, 2018, for review), we did not find IQ to have the same moderating effect as education on memory performance in depression. However, fewer studies reported information about IQ (54 vs. 136 that reported years of education), likely limiting our ability to detect robust effects. More consistent reporting of IQ estimates in future studies is needed. Furthermore, a number of different tests were used to measure IQ. Some, like the National Adult Reading Test, simply measure reading ability, while others like the Wechsler Adult Intelligence Scale measure verbal and nonverbal abilities. The variety of tests, in combination with the relatively small-sample size, was a major limitation of this analysis.

It should be noted that correlates of education such as socioeconomic status (SES), occupation, health-care access, diet, lifestyle factors, etc. could also moderate the relationship between depression and memory impairments. Though the studies included in the current meta-analysis did not provide sufficient detail for us to explore these factors, future research should investigate how they may interactively serve as protective or harmful influences on memory impairments. For example, high education could buffer the impact of poor diet on memory, while low education and poor access to health care could magnify impairments.

Limitations

Like other meta-analyses, some of our results may have been influenced by publication bias and outcome reporting bias (Dwan et al., 2013; John et al., 2012), however, analysis on the funnel plot did not reveal significant evidence for publication bias in the current sample. Selection bias in participant samples is another potential concern, in that individuals selected for depression groups may differ in substantial ways from the population. One concern in particular is that individuals with higher levels of depressive symptomology might elect to not participate in studies but might once remitted; this effect would then reduce the effect sizes. Additionally, the majority of the records included in this work were published in English. Because depression etiology and expression are known to vary based on cultural and social factors (Chentsova-Dutton et al., 2014; Juhasz et al., 2012), it is possible that our results are not entirely generalizable to cultures where English is less dominant.

Nonetheless, we hope that by including reports from a wide range of countries we were able to mitigate this issue to some extent.

We were unable to determine whether the quadratic effect of age is indicative of a true dip in memory performance in midlife depression, or if the trend is simply attributable to fewer studies with middle-aged individuals. An additional limitation for the age analysis is that the included studies varied greatly in the age ranges of their samples. For example, while some recruited only young or older adults, others recruited participants across the adult lifespan. By using the mean age of the depressed groups as we did in the current meta-analysis, those studies with wider age range tended to have mean ages that fell into the midlife range (e.g., in Sheline et al., 1999, age range = 23–86, M_{age} = 52.8). More studies with middle-age and older adults, particularly those that assess emotional memory, are needed to address outstanding questions regarding interactive influences of age and depression on episodic memory.

A limitation in our analyses of participant moderators is that a number of these variables were defined as the proportion of individuals in the samples with particular characteristics (e.g., the proportion of females, the proportion taking medication for depression). To explore, say, sex effects, ideally one would have memory performance means for females and for males. However, it is not common practice for researchers to report performance estimates in this manner unless sex differences are an explicit focus on the study. Additionally, some of our moderators may have not fully captured informative details about the variables. For example, our education moderator represented duration of schooling, which may be less informative than the quality of educational attainment (see Jones et al., 2011, for review). Lastly, while we explored the effects of pharmacological treatment, we were unable to analyze specific classes of antidepressants, polypharmacy, or whether therapy or other nonpharmacological interventions moderate the relationship between depression and memory due to insufficient data.

Potential confounds identified in Table 1 must be taken into consideration. For example, diagnostic status and medication status—variables shown to significantly influence memory performance in depression groups—were much lower in unpublished studies than in published studies and in community/university settings than in patient settings. This is perhaps unsurprising given that most unpublished studies were theses and dissertations that used convenience samples of university students who were more likely to be undiagnosed and thus unmedicated compared to patient samples. It is certainly possible that the differences in unpublished versus published studies and in community/university versus patient samples were driven by diagnostic and medication status variables.

The limitations for laboratory tasks to predict everyday memory functioning (Salthouse, 2012) must also be acknowledged. While we have provided some evidence of memory disruptions in depression, the real-world implications of these disruptions are beyond the scope of the current meta-analysis but are certainly worthy of future investigations. Another drawback, as mentioned in the Results, is that we were limited in our ability to simultaneously investigate sets of moderator variables. Finally, given the cross-sectional nature of the data included in the current meta-analysis, in addition to the limitations of the metaregression approach to determine directionality, we are unable to draw conclusions about the causal links between depression and memory impairments.

Conclusions

Depression has long been recognized as a burdensome mood disorder but only recently have researchers begun appreciating the full toll depression takes on a wide range of cognitive functions. Until now, major questions regarding episodic memory impairments across different types of tasks and materials, as well as the influence of demographic and other participant-related variables, have remained unresolved. We believe our meta-analysis has helped to clarify and paint a more comprehensive picture of the memory disruptions in depression. Based on 995 effect sizes from 205 studies with 236 unique depressed-control group comparisons, we definitively show clear evidence of depression-related deficits in episodic memory across the adult lifespan. These deficits were most pronounced in older age, in diagnosed MDD, in those taking medication for depression, and in those with fewer years of education. Depression status did not influence depressive memory deficits. Memory for positive and neutral material was particularly impacted in depression, while memory for negative material was not. Educational attainment was associated with lower levels of depressive deficits in memory.

We believe our meta-analysis provides a much-needed update to the literature on episodic memory in depression, providing considerable evidence for what is and is not impaired, and what factors moderate those impairments. While some questions were beyond what we were able to investigate, we hope our meta-analysis inspires continued investigations of the challenges faced by those with depression to inform novel treatments that can target the cognitive symptoms, in addition to hallmark affective symptoms, that cause severe disruptions in everyday life.

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