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Item-Method Directed Forgetting Is (Usually) Impaired in Clinical Populations: A Meta-Analysis

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The item-method directed forgetting paradigm is a common laboratory task used to measure memory control. While impaired memory control may contribute to the development and/or maintenance of a variety of psychological disorders, comparisons between clinical and nonclinical groups using this paradigm have been inconsistent—even within the same disorder. A systematic search for related articles utilizing clinical populations was conducted revealing 823 articles of which 36 met inclusion criteria. Raw mean differences were calculated and aggregated using Bayesian multilevel random-effects models. These models revealed a significant difference in the magnitude of directed forgetting between clinical and control populations, such that clinical populations (collapsing across all disorders or combining only the critical anxiety and depression clusters) exhibited a reduced directed forgetting effect. This difference tended to be larger in clinical (as opposed to clinical-analog) populations and in older samples. These results support the notion that item-method directed forgetting provides a suitable measure of memory control sensitive to real-world control deficits and further implies that memory control deficits may contribute to mental illness (although causality remains to be determined).

Public Significance Statement

Some people are better at controlling unwanted memories (e.g., a personal trauma) than others. The present meta-analysis demonstrates that those with mental disorders characterized by difficulty controlling unwanted thoughts often have trouble controlling unwanted memories in laboratory tasks, too. However, this is not always true, and more data are needed.

Keywords: directed forgetting, clinical psychology, memory suppression, cognition

Supplemental materials: <https://doi.org/10.1037/cep0000316.supp>

Forgetting plays an important, and often misunderstood, role in our lives. While commonly viewed as an inconvenience, forgetting actually facilitates peak cognitive function (Bjork, 1989). For example, forgetting allows us to unload outdated or useless information (e.g., an old license plate number) and prevents us from reliving painful events (see Fawcett & Hulbert, 2020; Nørby, 2015; Schacter, 1999, 2001, for reviews). For this reason, it is unsurprising that impairment in one's ability to forget is a characteristic of many psychological disorders. For example, recurrent, involuntary memories characterize posttraumatic stress disorder (PTSD); and intrusive thoughts and rumination are

characteristic of depression and obsessive-compulsive disorder (OCD; *Diagnostic and Statistical Manual of Mental Disorders 5 [DSM-5]*; American Psychiatric Association, 2013).

One manner in which the control of unwanted memories has been studied in the laboratory has been through the use of the item-method directed forgetting paradigm (see MacLeod, 1998, for a review). As depicted in Figure 1, participants are presented with a series of items, one at a time, each followed by an instruction to remember or forget the preceding item. During a subsequent test of all items, participants generally recall or recognize more of the items they were instructed to remember (R) than the items they were instructed to forget (F). This pattern is referred to as a directed forgetting effect (DFE) and has been attributed to either the selective rehearsal of the R items (and passive decay of the F items; e.g., Basden et al., 1993; Conway & Fthenaki, 2003; Hourihan & Taylor, 2006) or the application of one or more active (potentially inhibitory) mechanisms to prevent the F items from being adequately encoded or retrieved (e.g., Fawcett & Taylor, 2008; Zacks et al., 1996). Either account generally accepts that participants initially engage in maintenance rehearsal of the study item, awaiting the memory instruction, after which R items are rehearsed and F items receive minimal additional processing; however, they disagree with respect to the mechanism through which the cessation of rehearsal is implemented, with traditional selective rehearsal accounts adopting a “passive” perspective and alternate accounts positing one or more control processes (for a review of neural evidence, see Anderson & Hanslmayr, 2014).

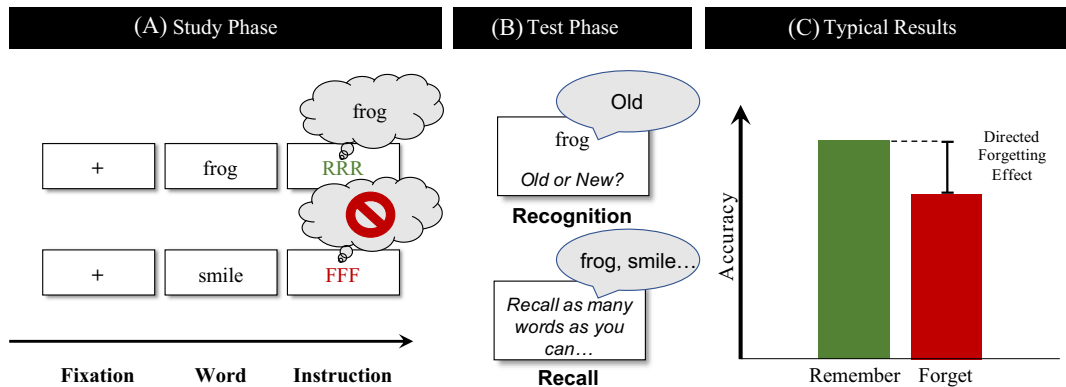
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The authors thank Michelle Swab, a research librarian at Memorial University of Newfoundland, for her assistance in their meta-analytic search. All data and code needed to conduct the reported analyses may be found on the project repository at <https://github.com/jmfawcett/imdfclinicalmeta>. Jonathan M. Fawcett received funding from the Natural Sciences and Engineering Council of Canada. The authors have no known conflicts of interest to disclose.

All data are available on GitHub at <https://github.com/jmfawcett/imdfclinicalmeta>. None of the analyses were preregistered.

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Figure 1
The Phases and Results of an Item-Method Directed Forgetting Task



Note. Panels A and B depict the study and test phases of a typical item-method directed forgetting procedure. During the study phase, participants learn a series of items, some of which they are instructed to remember (R) and others they are instructed to forget (F). During a later test phase, participants are typically tested for their recognition or recall memory of all items from the preceding study phase, regardless of the associated (R or F) instruction. The typical finding, depicted in Panel C, is defined as greater memory for the R than F items, which is referred to as the directed forgetting effect (DFE). See the online article for the color version of this figure.

Whatever the mechanism through which item-method directed forgetting operates, the DFE has often been used as a laboratory analog to explore control deficits in clinical populations characterized by an inability to control unwanted memories in everyday life. Such applications have included mood disorders (e.g., Kuehl et al., 2017; Wong & Moulds, 2008; Xie et al., 2018), anxiety and related disorders (e.g., McNally et al., 1999; Tolin et al., 2002), trauma and stress-related disorders (e.g., DePrince & Freyd, 2001; Moulds & Bryant, 2002; Moulds & Bryant, 2008), psychosis-related disorders (e.g., Patrick et al., 2015), personality disorders (e.g., Cloitre et al., 1996; Fleck et al., 2005), eating disorders (Tekcan et al., 2008; Woodard, 2004), and substance-abuse disorders (e.g., Fridrici et al., 2014; Todor, 2007). Due to the typical nature of real-world unwanted memories, such studies often focus on the control of negative or personally relevant material, which is thought to be more difficult to control even in nonclinical populations (see Hall et al., 2021, for a meta-analysis). The general prediction has been that participants with conditions characterized by disordered memory control should demonstrate similar impairments when controlling such material in the laboratory, resulting in a reduced DFE.¹ However, this literature has produced mixed results, even within the same disorder.

For example, this line of thinking would predict a smaller DFE amongst patients with a diagnosed anxiety disorder as compared to those without. Whereas Wilhelm et al. (1996) observed this pattern for negatively valenced items amongst patients diagnosed with OCD, McNally et al. (1999) failed to replicate this deficit amongst patients with panic disorder. Indeed, articles have at different times associated anxiety with a smaller (Tudorache et al., 2019), larger (Cottencin et al., 2006; Liang et al., 2011), or equivalent DFE (Irak & Çapan, 2015; Tolin et al., 2002). The same has been found with respect to mood disorders, with some studies reporting no relation between depression and the DFE (Wingenfeld et al., 2013; Wong & Moulds, 2008), whereas others (e.g., Xie et al., 2018) have found a smaller DFE for negative items amongst those with depressive tendencies. In short, there is little consensus as to whether participants with disorders

characterized by impaired memory control exhibit similar impairments in laboratory tasks.

Our present goal is to provide a meta-analytic synthesis addressing whether clinical populations characterized by an inability to control unwanted thoughts or memories demonstrate similar deficits in the item-method directed forgetting paradigm. We have chosen to focus on item-method directed forgetting (as opposed to list-method directed forgetting; also see Sahakyan et al., 2013, for review) in particular because this paradigm focuses on our ability to “push” unwanted thoughts or memories from mind soon after they occur; this, in our view, provides a laboratory analog of how patients with unwanted recurrent thoughts must exert control in their everyday lives (for a similar analysis related to retrieval suppression, see Stramaccia et al., 2021 and for a nonquantitative review, see Delaney et al., 2020). Therefore, the purpose of the current meta-analysis was to (a) estimate (and compare) the magnitude of the DFE in clinical and control populations (particularly populations suffering from disorders characterized by unwanted thoughts or memories) using neutral and negative stimuli, and (b) identify factors influencing the magnitude of the DFE.

¹ Although recent evidence suggests a general memory control deficit in clinical populations using related paradigms (e.g., Stramaccia et al., 2021), attentional deficits have also been observed in these same populations, especially pertaining to symptom-relevant material (Delaney et al., 2020); for that reason, an alternate perspective might be that any observed deficits in the current paradigm might be attributable to attention dwelling on symptom relevant items, resulting in a reduced DFE. However, given the aforementioned evidence—as well as our own finding that such deficits are not limited to symptom relevant or even valenced materials—we have nonetheless chosen to frame our article with general deficits in mind. Further, although the DFE is sometimes thought to arise from attentional mechanisms (e.g., Fawcett et al., 2016), it is still traditionally viewed as a memory phenomenon (owing to the fact that items must be initially encoding preceding the memory instruction), and for that reason, we have retained the traditional framing of this phenomenon as forgetting. We thank an anonymous reviewer for pointing out these concerns.

Method

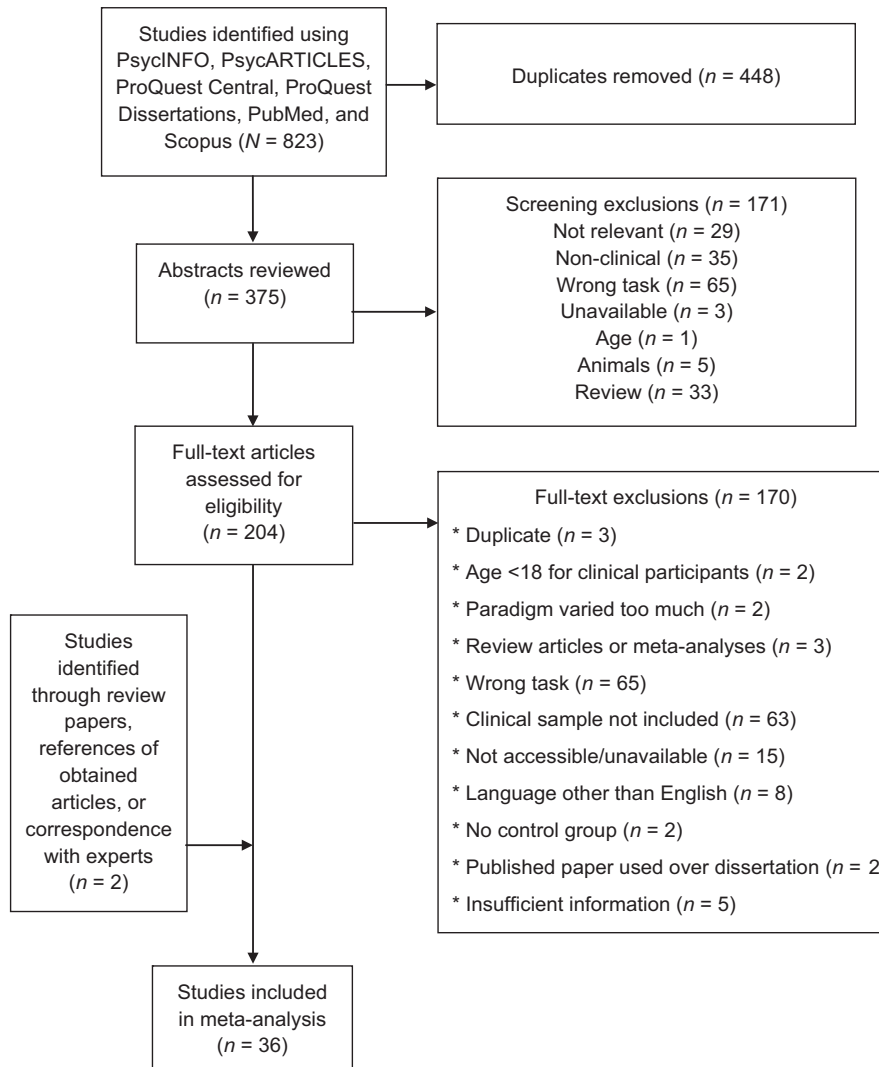
Literature Search

The search was conducted until April 2021 using the following online databases: *APA PsycInfo*, *PsychARTICLES*, *Pubmed*, *ProQuest*, *ProQuest Dissertations*, *ProQuest Theses*, and *Scopus*. The literature search consisted of both controlled and natural language Boolean search phrases (see the online Supplemental Material) conducted under the supervision of a research librarian. The controlled language search phrase was specific to the individual databases involved; however, *Scopus* does not use controlled terms, therefore only the natural search phrase was used. The natural language search phrase was applied to all databases. Only articles available in English were included. In addition to searching databases, all corresponding authors of included studies were contacted for raw or unpublished data, and advertisements were forwarded to the membership of relevant societies (e.g., Canadian Society for Brain, Behaviour, and Cognitive Science) for the same purpose.

Study Inclusion Criteria

The inclusion criteria were modified from Hall et al. (2021) and required at least one measure of item-method directed forgetting in at least one clinical or clinical-analog sample (e.g., high self-reported anxiety) and one control population. The exclusion criteria for articles were as follows: (a) reported exclusively nonclinical samples, (b) reported no experimental data, (c) used a different task (e.g., list-method directed forgetting), (d) did not have a control group, (e) provided samples with a mean age <17 years old, (f) were written in a non-English language and an English version was not available, (g) the article was unavailable online and the corresponding author did not respond, (h) reported an animal model, and/or (i) reported duplicate information already included (e.g., a dissertation and published article reporting the same study). Exclusions can be seen in the flowchart in Figure 2. For studies using clinical-analog data (i.e., self-report measures) wherein participants were divided using a cutoff score,

Figure 2
Meta-Analysis Flowchart



participants who scored below the cutoff acted as the control group for participants who scored above the cutoff.

Although our focus was on disorders characterized by difficulty controlling unwanted thoughts or memories in everyday life (e.g., anxiety or depression), we still coded effects relating to conditions without these or related deficits (e.g., borderline personality disorder); whereas we predicted a smaller DFE in the former, we reserved judgement with respect to the latter and instead viewed our analyses of those data as exploratory. To that end, samples were categorized into five clusters, inspired broadly by the *Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV)*; American Psychiatric Association, 2000): addiction, anxiety, depression, psychosis, and mixed, with the latter containing conditions for which there were too few studies to form their own cohesive cluster (see also Stramaccia et al., 2021). The addiction cluster consisted of studies that dealt with addiction, whether that be substance abuse or otherwise (i.e., gaming addiction; Ko et al., 2015). The anxiety cluster consisted of generalized anxiety disorder, OCD, panic disorder, social anxiety, PTSD, and acute stress disorder. The depression cluster consisted of depression and one bipolar study (Fleck et al., 2005). This was done on the recommendation of a clinical psychologist (EF). Finally, the psychosis cluster consisted of studies on schizophrenia and psychosis. In addition to cluster-specific estimates, we also calculated aggregate estimates inclusive of all clusters as well as an analysis inclusive of only the anxiety and depression clusters, with the latter being our primary analysis. Given the small number of studies and their heterogeneous nature, we did not feel it appropriate to pursue estimates based on individual disorders at this time, although as more literature emerges, this would be an ideal target. During our search, we also came across a variety of studies measuring the DFE in neuropsychological populations. Although there were too few (and they were too heterogeneous) to properly meta-analyze, we provide a brief overview of those studies in the online Supplemental Material.

Data Extraction

The first author (Noah W. Pevie) completed the coding in correspondence with the remaining authors, with all studies coded by at least two coders: In cases of conflict, all coding decisions were discussed until an agreement was established, with deadlocks resolved by the senior author (Jonathan M. Fawcett). In addition to means, standard deviations, and sample sizes, we also coded several moderators in addition to cluster: This included (a) population (clinical or clinical-analog), (b) sample type (in/outpatients, students/community), (c) mean age of the clinical sample, (d) the proportion of patients on psychotropic medication, (e) the proportion of patients undergoing psychotherapy, and (f) mean Beck Depression Inventory scores.²

Effect Size Calculation and Analysis

Recall and recognition rates were converted to proportions for each memory instruction (R, F), valence condition (neutral, positive, negative, symptom-relevant), and group (clinical, control). Words considered neutral included “fountain, doorknob, stairs,” whereas negative words were “criticism, downcast, lonely” and positive words were “cheerful, healthy, celebrate.” Symptom-relevant words refer to words that relate directly to the disorder being studied and therefore varied. For example, in Fridrici et al. (2014), where the participants had alcohol abuse disorder, symptom-related words were “martini, whiskey, booze,” whereas for McNally et al. (1998),

symptom-related words were “assault, scream, rape” since the participants had experienced a form of sexual trauma. Although data pertaining to positive and symptom-relevant words were coded, there were far fewer estimates, and they were not our primary focus; for that reason, they are summarized in the online Supplemental Material. For cases where negative items were also clearly symptom-relevant or symptom-relevant items were clearly negative (e.g., threat words; Zoellner et al., 2003), they were coded as both. Effect sizes were calculated as a raw mean change score using the *escale* function in the *metafor* package (Viechtbauer, 2010) in *R v4.1.3* (R Core Team, 2022).³ To measure the magnitude of the DFE, the proportion of F words was subtracted from the proportion of R words). This was done individually for clinical and control groups, as well as for each valence category. In cases where only corrected recognition was reported (i.e., hits–false alarms), these values were substituted for the hit rate (and will algebraically produce the same difference so long as a common false alarm rate is used when calculating this metric). Where possible, a similar correction was applied for studies reporting separate false alarm rates for R and F items (Baumann et al., 2013; Zwissler et al., 2012), although our findings remained the same if we instead used raw hits for those studies (likely because response bias did not differ between conditions in their samples).⁴ Further differences were calculated contrasting the clinical and control groups within each valence

² Given our core research question dealt with whether clinical populations would exhibit impaired memory control relative to control populations, we opted to focus on moderators of clinical relevance rather than those more related to study design (as was the focus for Hall et al., 2021) and conducted these analyses only for the clinical-control comparisons.

³ Raw mean change scores were used rather than standardized mean change scores because all data were on the same scale and—when that is the case—the former measure is inherently more meaningful, allowing the reader to judge the nature and magnitude of the effect directly (for textbooks discussing unstandardized effect sizes, see Borenstein et al., 2010, Chapter 4; Borenstein, 2009). Further, not standardizing reduces (but does not eliminate) sources of contamination (e.g., standardizing a difference contaminated by a ceiling or floor effect could result in exaggerated effects as the standard deviation is artificially reduced). There are two primary benefits often discussed as a motivation to instead use standardized mean change scores. The first is that standardization permits the combination of effects measured on different scales (e.g., combining accuracy and reaction time data). In our case, this is not necessary, as all measures are already on the same scale. The second is that standardization permits the magnitude of the effect to be judged according to “norms”—in the case of Cohen’s *d* or Hedge’s *g*, small ($d \sim 0.2$), medium ($d \sim 0.5$), and large ($d \sim 0.8$); however, these “norms” are rarely themselves useful, and even Cohen both conceded that the values were at worst arbitrary (but chosen to “appear reasonable”; Cohen, 1962, p. 146) and at best required calibration to the specific area of research rather than direct application as is often done (Cohen, 1988; for a modern review criticizing such global norms, see Schäfer & Schwarz, 2019). Because we consider presenting the effect on its native scale (rather than comparing it to arbitrary, uncalibrated “norms”) to be more informative, we have opted to favour raw mean change scores over standardized ones.

⁴ Because directed forgetting for recognition “hits” was calculated as the difference between the R and F items, which shared a common false alarm rate in all but two cases (i.e., Baumann et al., 2013; Zwissler et al., 2012), neither of which demonstrated differences in response bias, we expected differences between these conditions to be driven by sensitivity. All further subtractions operated on the magnitude of directed forgetting, meaning that we therefore believe the same of those metrics. Even so, it would be preferable for sufficient data to be provided to calculate metrics such as *d'* (which would require aggregate *d'* statistics to be reported or raw data to be available) or even more sophisticated metrics like area under the curve (which would require confidence judgments); we encourage future researchers to consider reporting these measures.

condition and the difference between neutral and negative items within each group, with a final contrast comparing the difference in the magnitude of the DFE for neutral and negative across the clinical and control groups.

Standard deviations (*SD*) were imputed for studies for which they were unavailable by taking the average of all available studies using the same measure, as is standard practice. Correlations used during calculation of our effects were taken from raw data when available, with the correlations imputed from the available studies when unavailable.

Data were analyzed (and dependencies accounted for) using a Bayesian three-level random-effects meta-analysis implemented within the *brms* package (Bürkner, 2017), with random effects for study and effect. Between-study heterogeneity was quantified using prediction intervals (IntHout et al., 2016), which reflect the range of probable “true” effects that would be expected should a new study be conducted like those included in the analysis. Priors for each model and further details pertaining to our modelling approach are provided by Hall et al. (2021; for another example, see Fawcett et al., 2023). Models were not conducted in cases for which we had less than three estimates. All data and code pertaining to our models may be found on the project repository (<https://github.com/jmfawcett/imdfclinicalmeta>).

Results

Directed Forgetting for Emotional and Neutral Items

We first analyzed the magnitude of the DFE as a function of cluster (addiction, anxiety, depression, psychosis, mixed), group (control, clinical), and valence (neutral, negative). As depicted in Figure 3, the DFE was robust in every case (with aggregate estimates ranging from ~7% to ~25%), with one exception: Within the psychosis cluster, the aggregate DFE was numerically in the expected direction but failed to exclude 0 as a credible value for any condition except for neutral items within the control groups.⁵ However, given the small number of studies, we urge caution in their interpretation.

Prediction intervals at times approached 0 but were generally exclusively positive (with the exception of the psychosis cluster), indicating that a DFE is almost always expected to occur in a given sample across each of our models. Notably, prediction intervals were tighter and more clearly positive for the combined anxiety and depression models, possibly due to the more cohesive nature of the samples—but also the aggregation of additional studies, improving our estimates.

Comparing Directed Forgetting for Emotional and Neutral Items

We next sought to replicate Hall et al. (2021) by comparing the DFE across neutral and negative items. As depicted in Figure 4, our findings provide a numerical replication but are limited in their statistical support (at least using uninformative priors). All models tended to favour a numerically smaller DFE for negative than neutral items. None of these effects excluded 0 as a credible value within the control groups; however, within the clinical groups, a credible effect was observed for the depression cluster, combined anxiety and depression clusters and the overall model. Prediction intervals included values close to 0 as well as high, positive values, supporting

Hall et al.’s (2021) conclusion that even under circumstances where a “typical” study (i.e., a study representative of the methods used by the included studies) might be expected to demonstrate a larger DFE for neutral than negative items, circumstances exist under which no such difference would be expected.

Given the apparent difference in the magnitude of this effect between control and clinical populations, a further exploratory meta-analytic comparison compared the difference in the DFE between the neutral and negative items for the clinical and control populations to determine if clinical populations exhibited a particularly reduced DFE for negative material. These models revealed a small but credible effect overall, $M = 3.55\%$, 95% CI [0.23%, 6.79%], and within the depression cluster itself, $M = 10.76\%$, 95% CI [5.52%, 15.88%], with a similar marginal trend in the combined anxiety and depression clusters, $M = 4.76\%$, 95% CI [−0.40%, 9.61%]; there was no such tendency within the anxiety cluster alone, $M = 0.09\%$, 95% CI [−4.63%, 4.79%], although this was driven in part by an apparent outlier (Zoellner et al., 2003).⁶ All other comparisons likewise failed to exclude 0, and prediction intervals were broad. This suggests that for clinical populations—at least those characterized by difficulty controlling unwanted thoughts (i.e., depression)—negative items may be particularly difficult to control, under certain circumstances. More data are required to explore moderating variables capable of explaining the demographic or methodological factors predictive of when clinical populations exhibit such deficits.

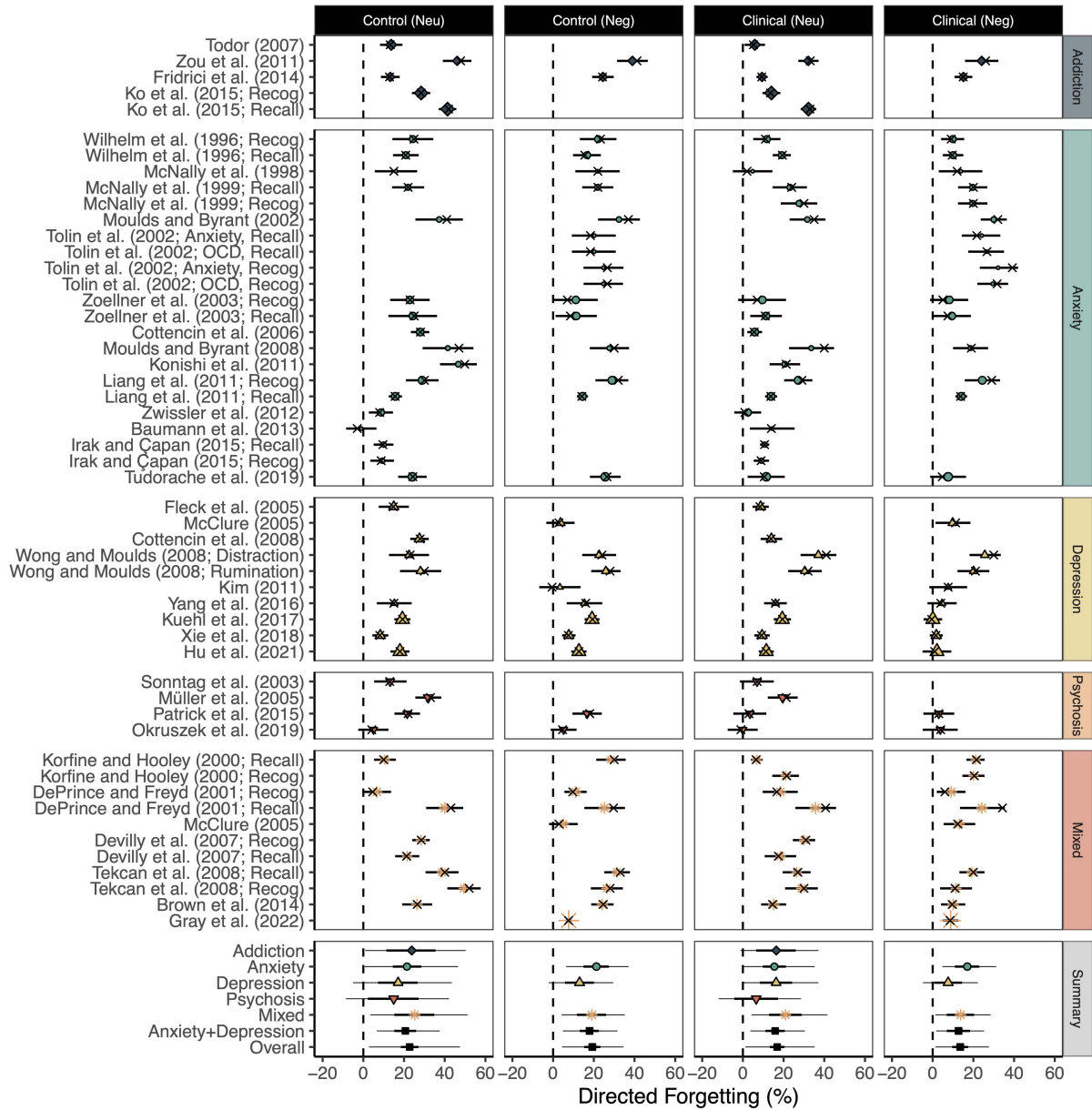
Comparing Directed Forgetting for Clinical and Control Populations

Finally, our primary analysis of interest compared the magnitude of the DFE in clinical and control populations. As depicted in Figure 5, all conditions tended to be numerically in the same direction, favouring a smaller DFE for clinical populations as compared to their matched controls. The critical comparisons—namely those aggregating across all clusters or only the anxiety and depression clusters—demonstrated a credible pattern favouring greater memory control for control populations. However, as with earlier models, the prediction interval—although not particularly broad—crossed 0 in each case. This means that although individuals experiencing difficulty controlling unwanted thoughts in everyday life generally exhibit similar deficits in laboratory analog tasks intended to measure such deficits, this is not universally true, and under certain circumstances, this difference may not emerge. However, although some studies might be expected to fail to replicate these differences, such deficits are expected in 95% and 97% of all samples for the critical comparison of the combined anxiety and depression group for neutral and negative stimuli, respectively.

⁵ The failure to exclude 0 as a credible value is equivalent to stating that a difference is not significant in Frequentist terms; Bayesian statistics do not use the term significant in the same way and instead permit interpretation of the confidence intervals as a demarcation of the most likely values of a given parameter.

⁶ We conducted an exploratory contrast of negative < neutral comparison for the anxiety and depression clusters. Although the depression cluster exhibited a numerically large effect, this was only marginal, difference = 7.3, 95% CI [−1.4, 16.0]. More data are required to resolve whether such a difference exists, although it is also worth keeping in mind that items in the negative condition of the included experiments were also more likely to be symptom relevant (in addition to being negative) for the depression as compared to the anxiety cluster.

Figure 3
Mean Directed Forgetting Effect (%) as a Function of Disorder Cluster (Addiction, Anxiety, Depression, Psychosis, Mixed), Group (Control, Clinical), and Valence (Neutral, Negative)



Note. Off-green diamonds: addiction cluster; green circles: anxiety cluster; yellow upward triangles: depression cluster; orange downward triangles: psychosis cluster; and peach stars: mixed cluster. Symbols and error bars represent posterior estimates and their corresponding 95% confidence intervals. Xs represent the empirical values reported in the relevant article. Symbol size is scaled to reflect the relative sample size. Estimates provided in the bottom panel represent aggregate effects; in this panel, thick lines reflect 95% confidence intervals and thin lines reflect 95% prediction intervals. Data are sorted in descending order of their publication date. OCD = obsessive-compulsive disorder. See the online article for the color version of this figure.

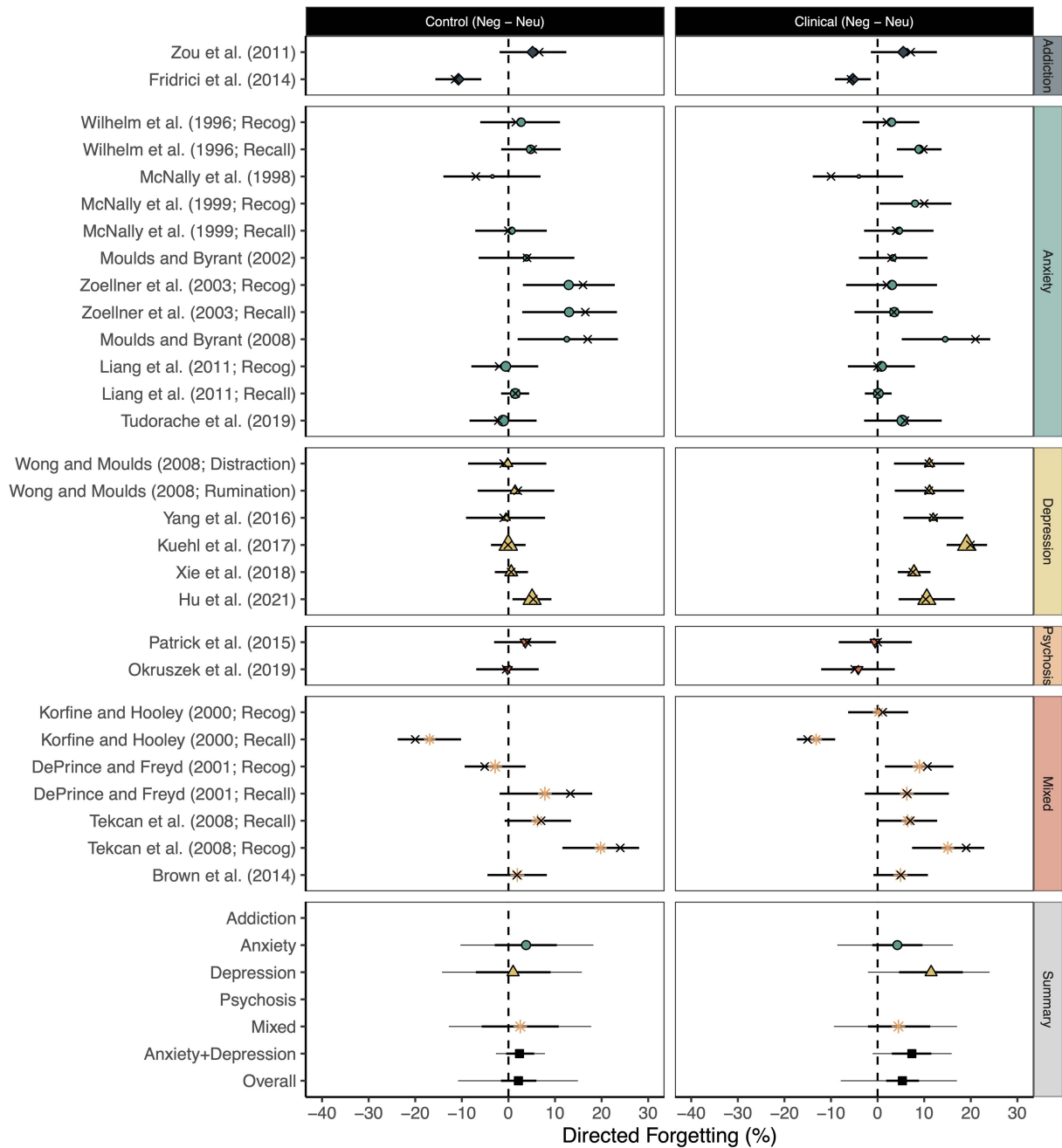
Moderators

Due to our focus on comparing clinical and control populations, moderator analyses were conducted only for the combined anxiety and depression clusters and only for the models comparing those populations.

Of our moderators, we were unable to evaluate the proportion of patients on relevant medication due to this information being available for too few studies. The remaining moderator analyses were undertaken for both the neutral and negative conditions and are summarized in Table 1.

Figure 4

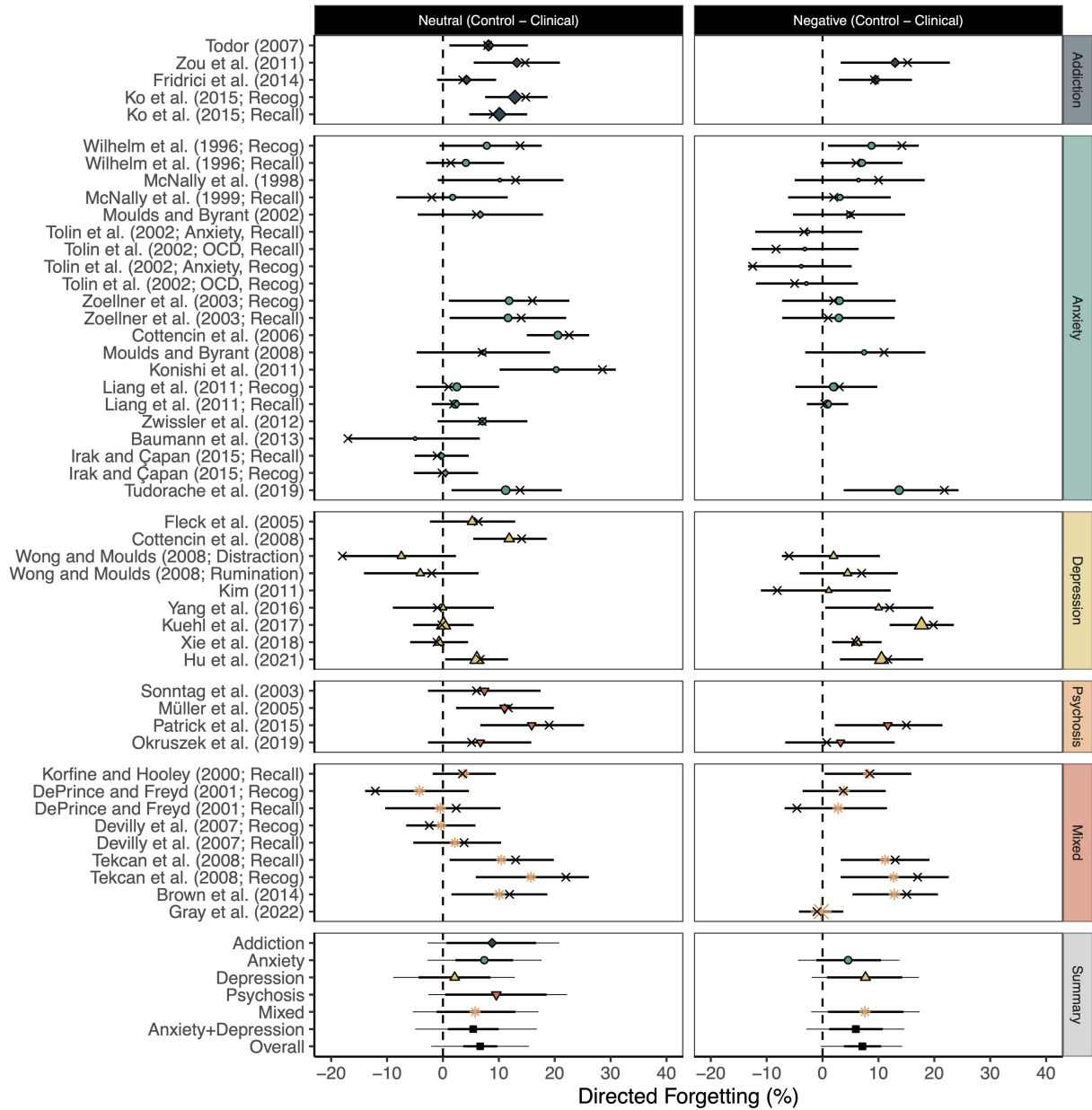
Mean Difference in Directed Forgetting Effect (%) Between Negative and Neutral Items as a Function of Disorder Cluster (Addiction, Anxiety, Depression, Psychosis, Mixed) and Group (Control, Clinical)



Note. Off-green diamonds: addiction cluster; green circles: anxiety cluster; yellow upward triangles: depression cluster; orange downward triangles: psychosis cluster; and peach stars: mixed cluster. Symbols and error bars represent posterior estimates and their corresponding 95% confidence intervals. Xs represent the empirical values reported in the relevant article. Symbol size is scaled to reflect relative sample size. Estimates provided in the bottom panel represent aggregate effects; in this panel, thick lines reflect 95% confidence intervals and thin lines reflect 95% prediction intervals. Data are sorted in descending order of their publication date. See the online article for the color version of this figure.

Figure 5

Mean Difference in Directed Forgetting Effect (%) Between Clinical and Control Groups as a Function of Disorder Cluster (Addiction, Anxiety, Depression, Psychosis, Mixed) and Valence (Neutral, Negative)



Note. Off-green diamonds: addiction cluster; green circles: anxiety cluster; yellow upward triangles: depression cluster; orange downward triangles: psychosis cluster; and peach stars: mixed cluster. Symbols and error bars represent posterior estimates and their corresponding 95% confidence intervals. Xs represent the empirical values reported in the relevant article. Symbol size is scaled to reflect relative sample size. Estimates provided in the bottom panel represent aggregate effects; in this panel, thick lines reflect 95% confidence intervals and thin lines reflect 95% prediction intervals. Data are sorted in descending order of their publication date. OCD = obsessive-compulsive disorder. See the online article for the color version of this figure.

In short, only population and age had an impact on the difference between clinical and control populations, and only for neutral stimuli, although the numerical pattern was the same for negative stimuli. With respect to the former, clinical populations (for which participants had a clinical diagnosis) demonstrated a larger difference than clinical-

analog populations (often based on self-report questionnaires); notably, this difference just failed to exclude 0 as a credible value. Furthermore, the age of the clinical sample was a credible predictor, with larger differences observed for older samples (the same was observed if average age across the groups was used).

Table 1
Moderators Influencing Differences in the Magnitude of the Directed Forgetting Effect (%) Between Clinical and Control Populations

Moderator	<i>k</i>	<i>M</i> (%)	Difference (%)	<i>p</i>
Neutral				
Population				
Clinical	17	8.18 [3.04, 13.19]	8.60 [−0.59, 17.35]	.97
Clinical-analog	8	−0.42 [−7.88, 7.00]		
Sample type				
Patient	13	7.41 [1.24, 13.34]	4.88 [−4.94, 14.10]	.85
Nonpatient	11	2.52 [−4.65, 9.94]		
Age	25	2.55 [1.22, 8.95]		.99
% in therapy	9	3.17 [−6.00, 11.78]		.79
Mean BDI		−0.09 [−3.37, 3.28]		.53
Negative				
Population				
Clinical	16	6.86 [0.89, 12.66]	2.46 [−8.13, 12.45]	.70
Clinical-analog	6	4.39 [−3.81, 12.82]		
Sample type				
Patient	11	8.29 [1.06, 15.33]	4.98 [−5.00, 14.82]	.85
Nonpatient	10	3.31 [−3.56, 10.14]		
Age	22	2.80 [−1.69, 7.35]		.90
% in therapy	8	−0.89 [−8.95, 7.27]		.59
Mean BDI	18	2.43 [−3.02, 7.48]		.84

Note. *k* = number of effects; *M* = mean estimate of directed forgetting for each level of the moderator, in the case of (scaled) continuous moderators it indicates the slope, 95% confidence interval presented in brackets; difference (%) = difference in magnitude of directed forgetting between levels of the moderator, 95% confidence interval presented in brackets; *p* = Bayesian *p* value reflecting confidence in the direction of the effect (e.g., *p* = .95, for a positive effect means 95% confidence the effect is positive); BDI = Beck Depression Inventory.

Publication Bias

To evaluate the possibility of publication bias, a series of multilevel regression models were undertaken using the (scaled) standard error or sample size of each study as a moderator. This approach is comparable to the *regtest* function of the *metafor* package (Viechtbauer, 2010) and was undertaken for the combined anxiety and depression models for each analysis depicted in our figures. A relation between standard error and/or sample size and the effect of interest was observed only for analyses of the basic DFE within negative items. This was observed for either predictor and in both clinical and control populations, suggesting—ironically—that some effects may be excluded showing small or absent DFEs for negative items. Because our hypotheses predicted smaller or absent DFEs for negative than neutral items, this might imply that the magnitude of these deficits is slightly larger than observed. Importantly, similar publication bias was observed in both the clinical and control populations, as evidenced by the fact that no such relation between standard error and/or sample size was observed for the difference between these populations.

Discussion

The present meta-analysis addressed whether clinical populations demonstrate impaired memory control for neutral and negative material in a common laboratory measure of intentional forgetting. A DFE was observed across conditions overall and within each clinical cluster (with the exception of psychosis, which had few estimates). Furthermore, our comparison of the magnitude of the DFE across neutral and negative items replicated the numerical pattern of a larger DFE for neutral than negative items as observed by Hall et al. (2021), albeit weakly, with a tendency for this difference to be larger in clinical populations. Owing

to differences in the number of included effects (Hall et al., 2021, had almost twice as many effects as the present effort) and greater variation in study design across the present experiments, we reserve interpretation of this outcome.⁷ Most importantly, we demonstrated a reduced DFE in clinical populations as compared to healthy populations for both neutral and negative material although heterogeneity was observed suggesting that this is true of “typical” studies, it is not the case for all studies.

These findings are well aligned with our hypothesis that populations characterized by an inability to control unwanted thoughts and memories in everyday life would demonstrate similar impairments in a laboratory task measuring memory control.⁸ Such control deficits may

⁷ Another observation of potential relevance to our comparison of negative and neutral items is that the present studies included far more measures of recall than did the sample used by Hall et al. (2021). In some cases, this may have resulted in a floor effect for the less memorable neutral items, with the more memorable negative and symptom relevant items elevating performance above floor and allowing the difference to emerge (e.g., Fridrici et al., 2014). Supporting this speculation, the neutral–emotional comparison was numerically smaller for recall than recognition in Hall et al.’s (2021) sample.

⁸ The causality of the relationship between deficits observed in the DFE and those observed in real life remains to be definitively proven, and critics might fairly point out that control deficits in a laboratory measure such as ours might be driven by the burden imposed by the mental illness itself. However, it is our view that such a perspective lacks parsimony: Specifically, the disorders in question are themselves characterized by an inability to push unwanted thoughts from mind. As such, arguing that reduced DFE in such populations arises from disease burden requires additional mechanisms (a deficit resulting in the illness-causing burden producing the laboratory deficit) than viewing the two as arising from common cause. Although we recognize that we are unable to resolve this debate in the context of the present meta-analysis, we would suggest that the burden of supporting such an argument be placed on those critics.

well facilitate the development and maintenance of these disorders. Concerning the tendency for clinical populations to tend toward having a larger negative < neutral DFE, one possibility is that the negative items are more relevant to those participants, thwarting control efforts and encouraging deep encoding of F items (Cloitre et al., 1996; Rogers et al., 1977; Wilhelm et al., 1996). Related to this point, negative word lists are often more interrelated than neutral lists, often confounding valence and symptom relevance (Gray et al., 2022; Tolin et al., 2002). Alternately, it is possible that clinical populations avoid rehearsal of such material, reducing memory for R items rather than increasing memory for F items (for discussion of avoidant encoding strategies, see McNally et al., 1998); without a suitable baseline (i.e., a neutral condition that is neither R nor F), it is impossible to differentiate between these explanations, but the fact that similar deficits exist for other memory control paradigms where baselines are more common might argue against the latter interpretation (e.g., the think/no-think paradigm demonstrates worse memory for items that are suppressed compared to baseline items not subject to control processes and such deficits are observed in this paradigm; Stramaccia et al., 2021).

The observed reduction in the DFE for clinical populations could also be interpreted as support for an active or even inhibitory interpretation of item-method directed forgetting. As noted in the introduction, contemporary theories of item-method directed forgetting ascribe the phenomenon to either an active control process elicited following F instructions (e.g., Fawcett & Taylor, 2008; Zacks et al., 1996) or the selective rehearsal of R items and passive exclusion of F items (e.g., Basden et al., 1993; Conway & Fthenaki, 2003; Hourihan & Taylor, 2006). Those supporting the active perspective vary as to whether they view the mechanisms involved as primarily attentional (e.g., Fawcett et al., 2016) or inhibitory in nature (e.g., Zacks et al., 1996). Because there is little reason to expect clinical populations to preferentially attend to or rehearse F items to a greater degree than nonclinical populations, present findings would appear at odds with a selective rehearsal account that views forgetting as a passive process. This is especially true that given deficits are observed regardless of the valence or symptom relevance of the study material. This perspective also suggests strategy as a plausible source of heterogeneity in the observed effects: Recent evidence suggests that item-method directed forgetting may be accomplished via either attempting to forget the item directly or via substitution of the F item with some other thought or idea (Hubbard & Sahakyan, 2021, 2023; see also Experiment 2 of Fawcett & Taylor, 2008, who admonished participants against thought substitution), as has also been observed in other memory control paradigms (e.g., think/no-think; Benoit & Anderson, 2012; Stramaccia et al., 2021). It could be that whereas control processes are deficient in these populations, thought substitution remains a viable strategy, for example. However, this claim remains speculative, and further work is required for evaluating the DFE using different strategies.

Importantly, we observed between-study variation amongst the included effects, indicating that although clinical populations are expected to demonstrate reduced DFEs (relative to controls) in a “typical” study (that is to say, on average, in a study using methods similar to the included studies, such a deficit will most often emerge), this is not true of all studies, and there exist circumstances (albeit rare) under which this would not be expected or the effect would be quite small. Two factors that may contribute to the emergence of disorder-related deficits in some studies but not others are differences between clinical and clinical-analog samples and the mean age of the sample.

With respect to the former, insofar as one accepts that those with greater clinical impairments related to memory or thought control ought to demonstrate increasing impairments with respect to the DFE, it is sensible that those diagnosed with a relevant disorder ought to demonstrate greater impairments than those scoring moderately high on a self-report measure. As such, we attribute any association between our population variable and the clinical < control difference to the severity of the clinical impairments. However, this remains to be tested.

With respect to the mean age of the sample, it is possible that clinically relevant control deficits exacerbate the typical decline observed in memory control abilities as one ages. In a meta-analysis of the ageing literature, Titz and Verhaeghen (2010) observed a smaller DFE in older as opposed to younger populations, which they attributed to either differences in encoding efficiency or inhibitory control. It is plausible that the natural decline in these control abilities occurs earlier or more rapidly in clinical populations, either due to a predisposition to such decline or disease burden speeding the natural ageing process. Additional studies ought to test the impact of age on memory control impairments in clinical populations.

Another probable source of heterogeneity is the fact that any disorder-related deficits observed in the present analyses are unlikely to be “process pure” owing to our inability to account for comorbidities. For example, McNally et al. (1998) putatively studied patients with PTSD, but this same group exhibited elevated depression relative to controls, and some qualified for secondary diagnoses (e.g., Generalized Anxiety Disorder). Sonntag et al. (2003) made a similar observation pertaining to differences in standardized intelligence. Likewise, McNally et al. (1998) and Zou et al. (2011) point out the difficulties inherent in dissociating deficits related to a disorder from other factors, such as trauma history or disease burden.

Limitations and Future Directions

It would have been desirable to evaluate directed forgetting within specific disorders, as their etiology and core symptomatology varied greatly even within our clusters. However, there was not enough literature to justify disorder-specific analyses. As such, our conclusions are limited to disorders characterized by uncontrollable thoughts or memories (i.e., our combined anxiety and depression clusters) rather than being linked to a specific condition. However, this also indicates a greater need for studies using this paradigm in clinical populations. Similarly, although our analyses provide an interim resolution to disagreement in the field, we are unable to isolate the locus of those deficits: Although suggestive of impaired neurocognitive control, the DFE (lacking a suitable baseline condition) actually reflects a combination of rehearsal (which improves memory for R items) and forgetting processes (which impair memory for F items). It therefore remains tenable that the observed differences reflect variation in overall memory performance, with clinical populations exhibiting disproportionately worse memory for R items rather than improved memory for F items.⁹

⁹ As noted earlier, it also remains possible that any observed deficits arise from attentional rather than memory control processes, as item-method directed forgetting is thought to occur at encoding. However, such a concern reflects a general criticism of the field—rather than our specific work—and we have chosen to discuss our findings in terms of memory (rather than attentional) control in keeping with convention within this paradigm.

These limitations also point to areas of possible improvement. First, many studies failed to report their full results, at times excluding entire dependent measures (e.g., McNally et al., 1998; Moulds & Bryant, 2002; Tolin et al., 2002), reporting data aggregated over a subset of variables making it impossible to explore that study's full design (e.g., Cloitre et al., 1996; Tudorache et al., 2019; Woodard, 2004) or excluding details such as the false alarms from a recognition task (e.g., Liang et al., 2011). Many studies also exhibited unusual features, such as uneven items per cell (e.g., 30 drug-related and 20 unrelated items; Zou et al., 2011) or alternating between the reporting of proportions and the number of items recalled. We believe these issues have improved over time, but we encourage those conducting research in this area to clearly report all data relevant to all dependent measures without aggregation.

Conclusion

In conclusion, the present results suggest item-method directed forgetting provides a suitable measure of memory control sensitive to real-world control deficits. Specifically, the fact that item-method directed forgetting is associated with psychological well-being suggests that it may contribute to adaptive coping mechanisms similar to other forms of control, such as retrieval suppression (e.g., Stramaccia et al., 2021). Although controlling our memories may not always be adaptive, it is our view that—when wielded effectively—it aids in maintaining our cognitive health and efficiency.

Résumé

Le paradigme de l'oubli dirigé par la méthode des items est une tâche couramment utilisée en laboratoire pour mesurer le contrôle de la mémoire. Bien que le contrôle altéré de la mémoire puisse contribuer au développement et (ou) au maintien de divers troubles psychologiques, les comparaisons entre des groupes cliniques et non cliniques faisant appel à ce paradigme se sont révélées incohérentes, même lorsque les sujets étaient atteints du même trouble. Une recherche systématique d'articles connexes mettant en cause des populations cliniques a été menée et a permis de relever 823 articles, parmi lesquels 36 satisfaisaient aux critères d'inclusion. Les écarts moyens bruts ont été calculés et cumulés en utilisant des modèles bayésiens à effets aléatoires à niveaux multiples. Ces modèles ont mis en lumière une différence considérable dans la magnitude de l'oubli dirigé entre les populations cliniques et de contrôle, si considérable en fait que l'oubli dirigé présentait un effet moins important chez les populations cliniques (parmi l'ensemble des troubles, ou combinant uniquement les groupes de l'anxiété profonde et de la dépression). Cette différence était généralement plus marquée chez les populations cliniques (par opposition aux populations cliniques analogues) et parmi les groupes plus âgés. Ces résultats soutiennent l'idée selon laquelle l'oubli dirigé par la méthode des items est une mesure convenable du contrôle de la mémoire – sensible aux insuffisances du contrôle du monde réel – et suppose en outre que ces insuffisances peuvent être un facteur de trouble mental.

Mots-clés : oubli dirigé, psychologie clinique, suppression de la mémoire, cognition

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