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Biased Cognitive Control of Emotional Information in Remitted Depression: A Meta-Analytic Review

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Cognitive theories of depression posit that maladaptive information processing increases the risk for depression recurrence. There is increasing theoretical and empirical support for the cognitive control of emotional information as a vulnerability factor for depression recurrence. In this investigation, findings from behavioral studies that compared the cognitive control of emotional information between participants with remitted major depressive disorder (rMDD) and healthy control (HC) participants were examined. Response times (RTs) and error rates were used as outcome variables, and aspects of clinical features, sample characteristics, and methodology and design were examined as moderating variables. The final review included 44 articles with a total of 2,081 rMDD participants and 2,285 HC participants. The two groups significantly differed in the difference score between RTs for negative and positive stimuli. Specifically, the difference in RTs between negative and positive stimuli was larger in participants with rMDD than in HC participants, indicating greater difficulty controlling irrelevant negative (vs. positive) stimuli in rMDD. Such cognitive control bias may be associated with preferential processing of negative over positive information in working memory. This imbalance may then be linked to other emotional information processing biases and emotion dysregulation, thereby increasing the risk for depression recurrence. Implications, limitations, and future directions are discussed.

General Scientific Summary

This systematic review suggests that people with a history of depression demonstrate greater difficulty in the cognitive control of negative than positive information. This imbalance in emotional information processing in working memory may increase the risk for depression recurrence.

Keywords: depression, recurrence, cognitive control, cognitive bias, emotional information processing

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Despite well-established interventions (e.g., Butler et al., 2006), relapse rates of major depressive disorder (MDD) remain high. Indeed, more than 50% of individuals with a first-time major depressive episode (MDE) will experience multiple subsequent episodes, often relapsing within 2 years of recovery (e.g., Boland et al., 2009). Thus, more insight is needed into the factors involved in MDD recurrence to improve treatment and relapse prevention efforts.

Cognitive theories posit that maladaptive information processing increases the risk for MDD onset and relapse (see Gotlib & Joormann, 2010, for review). Recently, there is growing interest in the role of cognitive control, a system that modulates cognitive processes, in the etiology of MDD (LeMoult & Gotlib, 2019). Cognitive control influences the maintenance of goal-directed behaviors, adaptation to changes in environment, and planning

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Deidentified data are available on the Open Science Framework: https://osf.io/5mhcb/

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The preregistered design and analysis plan is available on PROSPERO: https://www.crd.york.ac.uk/prospero/display_record.php?ID=CRD42021228081

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abilities (Dubois et al., 1994). One of the most influential frameworks for cognitive control is Miyake et al.'s (2000) three-component model, where cognitive control is conceptualized to involve inhibition, shifting, and updating contents in working memory.

Research on cognitive impairments broadly shows mixed evidence on cognitive deficits in MDD, but consistent evidence on the existence of cognitive biases in MDD (LeMoult & Gotlib, 2019). It is unclear if this pattern also applies to cognitive control. Meta-analytic reviews support the existence of cognitive control impairments in individuals with current and remitted MDD (rMDD; Dotson et al., 2020; Hasselbalch et al., 2011; Lee et al., 2012). More importantly, theoretical reviews highlight the problems in the cognitive control of emotional information in current MDD (Gotlib & Joormann, 2010; Joormann & Tanovic, 2015; LeMoult & Gotlib, 2019). However, no systematic review has evaluated the cognitive control of emotional information in rMDD. In addition, there is an underemphasis on positive information processing in this literature.

This meta-analytic review addresses these gaps by examining the cognitive control of emotional information in rMDD. First, we provide an overview of the key theoretical frameworks and empirical findings on the cognitive control of emotional information in depression. Then, we detail the methods for systematically reviewing articles on cognitive control of emotional information in rMDD and present the findings from the main and moderator analyses. Lastly, we discuss implications, limitations, and future directions. This meta-analysis focuses on Miyake et al.'s (2000) framework, as it is the most frequently adopted framework by studies that examined the cognitive control of emotional information (for reviews, see Joormann & Tanovic, 2015; Villalobos et al., 2021).

The Construct of Cognitive Control

Factor analyses of cognitive control have engendered several frameworks. For instance, Levin et al. (1996) conceptualized cognitive control to involve conceptualization, planning, schema, cluster, and inhibition, whereas Robbins (1996) conceptualized cognitive control to involve memory and learning, intelligence, response speed, and selective attention. Despite the existence of multiple frameworks, the depression literature has largely adopted the three-component model of cognitive control (Miyake et al., 2000), conceptualized as involving separate but related functions in working memory: (a) inhibiting information from entering, (b) shifting between multiple task-sets, and (c) monitoring and updating content.

The three-component model of cognitive control (Miyake et al., 2000) has been used to guide a plethora of empirical and theoretical work on depression (Grahek et al., 2018). Although it has played a key role in advancing research on cognitive control dysfunction in depression, the model is not free from conceptual and methodological issues. This includes the task impurity problem, issues in the replicability of the three-component structure, and questionable generalizability to emotional material (Grahek et al., 2018). The first two issues have been addressed by the authors' newer unity diversity framework (Miyake & Friedman, 2012), where cognitive control is conceptualized as having a common factor (i.e., general cognitive function) and specific factors (e.g., shifting specific function). However, there remains a paucity of research on the generalizability of these tasks to emotional information.

Theoretical and Empirical Support for Biased Cognitive Control and MDD

Theoretical frameworks propose cognitive control of emotional information processing as a key factor in MDD etiology. The impaired cognitive control account asserts that individuals with current MDD have difficulties in working memory update, that is, limiting the access to negative material in working memory (Joormann et al., 2007). These difficulties are conceptualized to underlie impaired disengagement from negative information, resulting in increased attention to, and enhanced elaboration and memory of, negative information. The impaired disengagement hypothesis postulates that difficulties with inhibition and shifting, that is, inhibiting irrelevant negative information and selectively focusing on task-relevant information, increases the risk for rumination (Koster et al., 2011). Persistent ruminative thinking, in turn, increases the risk for MDD onset and recurrence. Relatedly, the combined cognitive bias hypothesis argues that biased cognitive control and biases in attention, interpretation, and memory contribute to MDD vulnerability through their mutual interactions (Everaert et al., 2012). The cognitive control of emotional information, specifically, difficulties in inhibiting and updating negative information in working memory, is also proposed to underlie cognitive emotion regulation (LeMoult & Gotlib, 2019).

The literature on cognitive control—using both behavioral and neurobiological methods—suggests that individuals with current MDD have difficulty controlling the processing of negative information (for reviews, see Joormann & Gotlib, 2010; Pizzagalli, 2011; Stange et al., 2017). Specifically, individuals with current MDD have difficulty inhibiting (e.g., Joormann & Gotlib, 2010), shifting from (e.g., Murphy et al., 2012), and updating negative information in working memory (e.g., Joormann & Gotlib, 2008). Moreover, current MDD is associated with weaker activation in prefrontal cortical brain regions implicated in cognitive control when performing tasks on inhibiting, shifting from, and updating emotional stimuli (e.g., Beevers et al., 2010; Foland-Ross et al., 2013). There also is evidence for a link between cognitive control of emotional information and other depression-related processes (see LeMoult & Gotlib, 2019, for a review). For instance, difficulties in the cognitive control of negative information were associated with depressive symptoms through negative attentional and interpretation biases (e.g., Everaert et al., 2017). Moreover, increased activities in brain areas associated with cognitive control of emotional information are linked to maladaptive emotion regulation strategies such as brooding (e.g., Vanderhasselt et al., 2011, 2013).

There exist, however, three critical gaps in this literature. First, existing systematic reviews and meta-analyses have focused on current MDD, which, though valuable, offers limited inferences on the role of cognitive control in rMDD. Because risk factors for MDD onset and MDD recurrence may differ, there is a need for a systematic review on rMDD specifically. Second, the literature on the cognitive control of emotional information has predominantly focused on negative information processing and paid less attention to positive information processing. This is problematic given that low positive affect, which is related to impaired positive information processing, is a key feature of depression (e.g., Clark & Watson, 1991). Third, studies that investigated positive and negative information processing produced mixed findings in current MDD (see Joormann & Tanovic, 2015, for review). Moreover, in both empirical studies and systematic reviews, cognitive control of negative and

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positive material is typically only examined separately. This is problematic because it precludes the examination of the *relative* processing of positive versus negative stimuli, which may be important in recurrence vulnerability in addition to positive or negative information processing alone. Thus, there is a need for a comprehensive systematic review on the cognitive control in rMDD that includes the processing of negative, positive, and neutral information, both between and within individuals.

Biased Cognitive Control and MDD Recurrence Risk

Biased cognitive control of emotional information may be crucial in MDD recurrence for two reasons. First, compared to healthy individuals, individuals with rMDD demonstrate preferential processing of negative over positive material in attention (e.g., fixating longer on negative images than positive images), self-referential processing (e.g., endorsing more negative than positive self-descriptive adjectives), and memory (e.g., recalling more negative than positive words; see LeMoult & Gotlib, 2019, for review). These findings suggest that MDD vulnerability is not only characterized by prolonged processing of negative information, but also by reduced processing of positive information. Given the associations between cognitive control and other processes such as attention, memory, and emotion regulation (e.g., Everaert et al., 2017), biased cognitive control characterized by sustained processing of irrelevant negative versus positive information may also be present in rMDD.

Second, biased cognitive control that reduces the processing of irrelevant positive, relative to negative, material may maintain or exacerbate a low positive mood, thereby elevating the risk for future MDD. Reductions in the inhibition of positive relative to negative distractors, the shift from positive relative to negative stimuli, and the removal of positive material relative to negative material from working memory all could increase the processing of positive relative to negative material that is no longer relevant. For healthy individuals, this sustained processing of positive relative to negative material in working memory can facilitate the recruitment of positive emotions during emotion regulation (Tugade et al., 2004), which is linked to lower rates of mood disorders (Southwick et al., 2005). In contrast, biased cognitive control that leads to reduced processing of positive relative to negative material can hinder the use of positive emotions at times of stress (Fredrickson et al., 2003). Thus, biased cognitive control may confer risk for MDD recurrence.

The Current Investigation

This meta-analytic review examined whether biased cognitive control characterized by sustained processing of negative relative to positive material in working memory is a plausible vulnerability factor for MDD recurrence. Meta-analytic techniques were used to synthesize findings from behavioral experiments that examined the cognitive control of negative and positive information in rMDD. The current study also investigated potential moderators of the effects of interest, including clinical features, participant characteristics, and study design.

Operationalization of Group Differences in Biased Cognitive Control

Given that there is relatively less information on cognitive control of emotional versus nonemotional information, it is important to clearly define cognitive control bias. In the current investigation, we define biased cognitive control as the *preferential processing of material of one valence over another in working memory*. Specifically, *negative bias* refers to preferential processing of negative over neutral material; *positive bias* refers to preferential processing of positive over neutral material; and *negative–positive bias* refers to preferential processing of negative over positive material.

Because cognitive control is assessed by various tasks (see Table 1 for tasks used in the articles reviewed), it is important to operationalize cognitive control bias. Cognitive control is generally assessed by response times (RTs) and/or error rates. In the current investigation, we comprehensively examine group differences in RTs and error rates for negative, positive, and neutral stimuli, the relative processing of emotional and nonemotional stimuli, reflecting negative and positive biases, and the relative processing of positive and negative stimuli, reflecting differences in the negative–positive bias, the last in which was of particular interest to us. A difference score in RTs and/or error rates between negative and positive trials (i.e., negative–positive bias) that is greater in the rMDD group than in the healthy control (HC) group would indicate biased cognitive control characterized by greater difficulty in the cognitive control of negative relative to positive material in working memory.

Hypotheses for the Main and Moderator Analyses

Participants with rMDD were predicted to demonstrate greater biased cognitive control characterized by difficulty controlling irrelevant negative (vs. positive) material in working memory compared to healthy participants. Specifically, the rMDD group was expected to have significantly longer RTs, and, to a lesser extent, higher error rates in inhibiting, shifting from, and updating negative than positive stimuli compared to the HC group, resulting in positive effect sizes. The group differences were expected to be larger for RTs than error rates, paralleling the findings in current MDD (e.g., Levens & Gotlib, 2015; Quigley et al., 2020).

We expected clinical features, participant characteristics, and aspects of study design to moderate these group differences. Regarding the clinical features, more MDEs and shorter time elapsed since the last MDE are linked to higher recurrence risk (e.g., Ten Doesschate et al., 2010), and, therefore, were expected to be associated with larger group differences. Relatedly, larger group differences were expected with more depressive symptoms in the rMDD group because higher symptom levels are associated with greater biased cognitive control (e.g., de Lissnyder et al., 2011; Quinn & Joormann, 2015; Wen & Yoon, 2019) and may reflect a higher recurrence risk. Moreover, because antidepressant use has been linked to reduced depression-related cognitive impairments (e.g., Tranter et al., 2009; Wells et al., 2014), larger effect sizes were expected with lower proportions of rMDD participants currently on antidepressants. Lastly, effects were expected to be larger with more rMDD participants having comorbid disorders, because comorbidity is linked to greater depressive symptom severity (e.g., Avenevoli et al., 2015; Melartin et al., 2004) and greater biased cognitive control (e.g., Malooly et al., 2013; Najmi et al., 2010; Wen et al., 2019).

Regarding participant characteristics, the group differences were expected to be larger in older samples because depression-related cognitive impairments tend to be more severe with increased age (e.g., Austin et al., 2001). Given that women are more likely to develop MDD than men (Kessler, 2003) and report

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Table 1
Experimental Tasks Used by the Studies in the Current Meta-Analysis

Task	Domain	Description	Example study
Affective Flexibility	SI	Switch between sorting stimuli based on an affective rule (valence) or a nonaffective rule (type of word, number of humans in image).	Rnic (2019)
Affective Go/No-Go	IN	Respond to the target stimuli (e.g., positive words) while withholding response to distractor stimuli (e.g., negative words) presented one at a time. Targets vary across blocks.	Roiser et al. (2009)
Color Emotion	SI	Switch between indicating the emotional expression of facial stimuli as happy or sad and the color of the frame around the image as red or blue.	Quigley et al. (2020)
Cued Emotional Conflict	GEN	Respond to the actual or opposite emotional expression of the target face.	Vanderhasselt et al. (2011)
Emotional Card Sort	SI	Switch between sorting cards based on color, form, or number of times the emotional word appears on the cards.	Cerny et al. (2019)
Emotional Flanker	IN	Categorize target stimuli as positive or negative while ignoring adjacent distractor stimuli.	Quigley et al. (2020)
Emotional Stroop	IN	Name the colors of emotional words presented one at a time while ignoring the meaning of the words.	Hsu and Davison (2017)
Exogenous Cueing	GEN	Respond to targets (i.e., emotional faces) that are in the same or opposite spatial location of the peripheral cues that precede the targets.	Figueroa et al. (2019)
N-Back	UP	Indicate whether the valence of the stimulus is the same or different as the stimulus presented N trials ago.	Levens and Gotlib (2010)
Negative Affective Priming	IN	Categorize target words based on valence while ignoring distractor words presented simultaneously. In the control condition, the prime-trial distractors are neutral; in the negative priming condition, prime trial distractors are test trial targets.	Joormann (2004)
Visual Oddball	GEN	Respond to rarely presented target stimuli and ignore rarely presented distracter stimuli in the context of frequently presented stimuli of a third type.	Bistricky et al. (2014)

Note. IN = inhibition; SI = shifting; UP = updating; GEN = general cognitive control.

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more depression-related impairments in cognition and emotion regulation (e.g., Calvete & Cardeñoso, 2005; McRae et al., 2008), the effects were predicted to be larger in samples with a greater percentage of female participants.

Regarding study design, the group differences were expected to be more pronounced in studies that used facial pictures due to their ability to elicit heightened vigilance for interpersonal rejection (e.g., Joiner et al., 1999) than in studies that used words. The effects were also expected to be larger with the inclusion of an induction procedure that may enhance depression-related biases (e.g., Beck & Bredemeier, 2016). We also examined the moderation effect of experimental task, but no a priori hypotheses were made.[1] Lastly, we explored possible moderation effects of sample size, publication year, and study quality.

Method

Transparency and Openness

The current meta-analysis followed the preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P; Shamseer et al., 2015). Data were analyzed using Meta-Essentials (Suurmond et al., 2017). The protocol for the current meta-analysis has been preregistered and published on PROSPERO (Wen et al., 2021). Deidentified data are available on the Open Science Framework (Wen, 2023)

Literature Search

Systematic searches were conducted for articles published either online or in print before March 2023 using PsycInfo and Medline. The following keywords along with their truncated versions and synonyms were searched across all fields (i.e., title, abstract, keywords): depression, remission, cognitive control, and bias. Specifically, the following terms were searched:

Construct A: depress*
Construct B: remit* or recover* or euthymic or previously
Construct C: cognitive control or executive function or executive control or shift* or updat* or inhibit* or working memory
Construct D: emotional or bias

The searches were supplemented by examining relevant citations in the references section of the articles and relevant articles that cited these

[1] We had planned to examine the moderation effects of cognitive control domains (e.g., shifting, updating) as well as the type of emotion within each valence category (e.g., sad vs. angry for negative valence). However, we were unable to do so due to the small number of studies that examined shifting and updating or used stimuli other than sad or happy emotions. Relatedly, the moderation effects of experimental task were examined only for the inhibition domain. Moreover, the moderation analyses for the effect of stimulus type only included words and faces due to the small number of studies that used non-facial pictures.

articles. Furthermore, authors who have published substantially in this area were contacted to obtain any unpublished work. Dissertations, master's theses, and conference presentations were included in the search criteria on the PsycInfo and Medline databases.

Study Selection Criteria and Procedure

A study was selected if it met the following criteria: (a) reported RTs or error rates on cognitive control (e.g., inhibition, shifting, updating), (b) utilized and reported data on emotional (positive and/or negative) stimuli, (c) diagnosed rMDD using a clinical interview based on established diagnostic criteria (e.g., DSM, ICD), (d) included a sample of $n > 10$ participants in each of the rMDD and HC groups, (e) recruited participants who are older adolescents or adults (age $\geq$ 15; Sawyer et al., 2018; World Health Organization, 2023), and (f) were written in English.

Using these criteria, relevant articles were identified by screening the titles and abstracts by a trained research assistant (Ethan Ray Fischer). For articles deemed to be eligible for inclusion, the full articles were retrieved for further assessment. Final inclusion was determined after review of the full article by a doctoral student (Alainna Wen). A total of 703 articles were identified from the search engines, and an additional 235 articles of interest were identified through hand-searches (see Figure 1). Of these, 44 articles met the criteria for inclusion. Three of the included articles each contained two independent samples, resulting in 47 samples (2,081 rMDD participants and 2,285 HC participants) being included in the current review. If an article did not report data or the data were presented in a nonusable format for this meta-analysis, the author(s) was contacted. The authors of all the selected articles were contacted as none provided data on all variables.

Quality Assessment

Study quality was assessed using a modified version of the Joanna Briggs Institute Critical Appraisal Checklist for Analytical Cross-Sectional Studies (Moola et al., 2019). In addition to the items on the checklist, two items were added to assess whether interrater reliability of the clinical diagnosis was computed and whether a rationale for the sample size was presented, resulting in nine items total.[2] Each item was given a score of 1 = Yes or 0 = No, and the number of items rated "Yes" were summed and multiplied by 10.

Coding Procedure

The main study variables included RTs and error rates on tasks that assessed the cognitive control of negative, positive, and neutral stimuli. Moderator variables included clinical features (depressive symptom level, number of MDEs, time since the last MDE, percentage of participants with antidepressant use, and percentage of participants with comorbid disorders), participant characteristics (mean age and percentage of female participants), and study design (cognitive control domain, experimental task, stimulus type, inclusion of induction procedure). We also examined the moderating effects of sample size, publication year, and study quality. If a study investigated multiple cognitive control domains (e.g., shifting, updating) or utilized multiple emotions within the same valence (e.g., angry and sad faces for the negative valence), coding was done separately for each domain or emotion. See coding manual in the online supplemental materials (S1) for details. One coder (Alainna Wen) coded the variables in all the studies and a secondary coder (K. Lira Yoon) coded 10 randomly selected studies (i.e., 23% of the included studies). The two coders had good inter-rater reliability, with a Cohen's κ of .91 for categorical variables and an ICC of .99 for continuous variables.

Effect Size Calculation

Effect sizes of the *d*-family (Lipsey & Wilson, 2007) were computed based on the RTs and/or error rates. Specifically, standardized mean differences were calculated with Cohen's *d* followed by applying Hedges' correction for bias in small samples (Hedges' *g*; Hedges & Olkin, 1985). Hedges' *g* is interpreted in the same fashion as Cohen's *d*: below .2 as small, around .5 as medium, and above .8 as large (Cohen, 1988). The values of Hedges' *g* and standard error for each study as well as aggregated across studies were calculated with Meta-Essentials (Suurmond et al., 2017).

Separate effect sizes were computed using the RTs and error rates for negative, positive, and neutral stimuli for the between-group comparisons. In addition, effect sizes for the difference scores (negative–positive, negative–neutral, positive–neutral) were also computed using the RTs and error rates. Correlations between the RTs and error rates were obtained from the data reported and were used for the within-group comparisons. When correlations were not reported, a recommended value of .7 was used (Rosenthal, 1991).

Finally, when a study examined multiple cognitive control domains, the effect size for each domain was first computed, and a combined effect and its variance were then calculated. This approach was also utilized whenever a study reported data using multiple emotions of the same valence (e.g., sad and angry faces for negative valence). Because only two studies examined multiple domains of cognitive control and three studies included multiple emotions of stimulus valence, only one effect size from each sample was included in the analyses.

Analytic Approach

Main Analyses

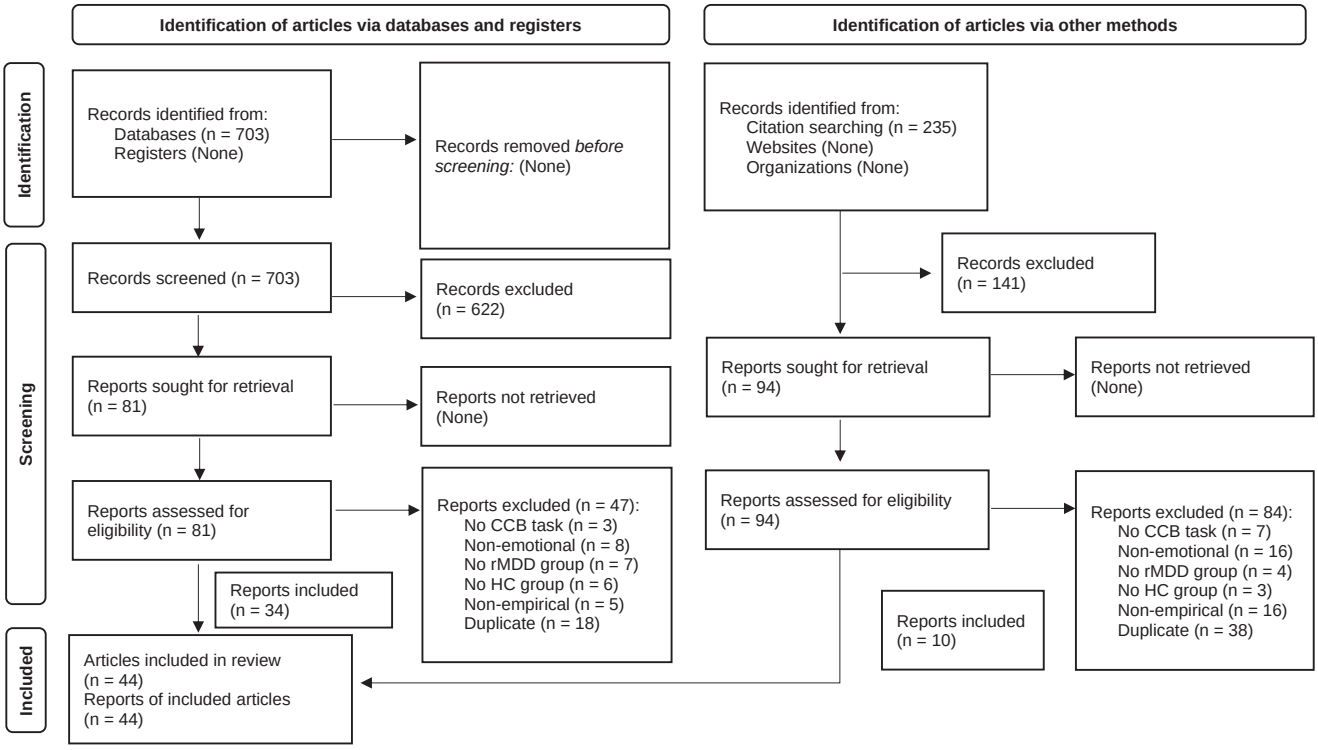
Random-effects models assume that the effect sizes differ from the population by sampling error and by variability among the studies (e.g., Field, 2003; Lipsey & Wilson, 2007) and therefore permit inferences that generalize beyond the sample effect sizes (e.g., Hedges & Olkin, 1985; Hedges & Vevea, 1998). Larger standard errors are typically produced due to modeling both between- and within-study variability (Field, 2003). In turn, the random-effects models reduce the risk of type I errors. Therefore, random-effects models were conducted.

Between-group comparisons were conducted on the RTs and error rates separately for negative, positive, and neutral stimuli. Difference scores on RTs and error rates (negative–positive, negative–neutral, positive–neutral) were also compared between groups to investigate the biased cognitive control of emotional information. Lastly, RTs and error rates for negative versus positive stimuli were compared within each group to further examine the relative processing of negative and positive information in cognitive control in each group.

[2] The Exposure Measure and Outcome Measure items assess the same aspect in experimental studies (i.e., the construct of cognitive control); therefore, the latter item was removed from the checklist for the current investigation.

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Figure 1
Information Flow on Article Selection



Note. CCB = cognitive control bias; rMDD = remitted major depressive disorder; HC = healthy control.

Moderator Analyses

The *Q* statistic (Hedges & Olkin, 1985) was used to determine the presence of heterogeneity in the effect sizes (e.g., Huedo-Medina et al., 2006). The degree of heterogeneity was quantified using the I^2 statistic, where scores of 25, 50, and 75 indicate low, moderate, and high heterogeneity, respectively (Higgins et al., 2003). Because heterogeneity statistics typically lack power in studies with smaller samples and therefore are more susceptible to Type II errors (Huedo-Medina et al., 2006), moderators were evaluated even in the absence of significant heterogeneity (e.g., Rosenthal & DiMatteo, 2001).

Results

Study Characteristics

Demographic characteristics of the participants and clinical features of the rMDD participants in the included articles are presented in Table 2. The methodology and design of the included articles are presented in Table 3. Of the 44 articles reviewed, 32 examined inhibition, three examined shifting, three examined updating, one examined all domains, one examined shifting and updating, and four examined broad cognitive control. Regarding outcome measures, 22 articles assessed RTs only, 18 assessed RTs and error rates, and four assessed error rates only. Of the 11 articles that utilized induction procedures, three utilized a repeated measures design, four randomly assigned participants into stress and no-stress conditions, and four assessed cognitive control after all participants completed the induction procedure.

Main Analyses Findings

Between-Group Differences in Negative, Positive, and Neutral Trial Performance

Of the 44 articles that met the criteria for the meta-analysis, 27 studies reported data on performance in emotional (negative and/or positive) trials, and 17 studies reported data on emotional and neutral trial performance. There were no significant group differences in error rates: positive stimuli, $g = .17$, $SE = .11$, $p = .118$, 95% CI [−0.066, 0.40], negative stimuli, $g = .19$, $SE = .13$, $p = .149$, [−0.098, 0.48], neutral stimuli, $g = .042$, $SE = .047$, $p = .929$, [−0.10, 0.11].[3] In contrast, the rMDD group (vs. control group) had significantly longer RTs in positive trials, $g = .21$, $SE = .060$, $p = .001$, [0.085, 0.33], negative trials, $g = .28$, $SE = .074$, $p < .001$, [0.12, 0.43], and neutral trials, $g = .38$, $SE = .066$, $p < .001$, [0.24, 0.52] (Figures 2 and 3). That is, individuals with rMDD exhibited slower cognitive control for all three valences of stimuli compared to healthy individuals. Heterogeneity was not significant for RTs in positive trials, $Q = 32.81$, $p = .168$, $I^2 = 20.75$, or neutral trials, $Q = 17.96$, $p = .326$, $I^2 = 10.93$, but was significant for RTs in negative trials, $Q = 45.11$, $p = .011$, $I^2 = 42.37$.

[3] An outlier (i.e., Joormann & Gotlib, 2010) was removed for the analyses on positive and negative trials. Prior to the outlier removal, error rates marginally differed across the groups for positive trials, $g = .40$, $SE = .20$, $p = .051$, 95% CI [−.046, .84], and did not differ for negative trials, $g = .99$, $SE = 1.32$, $p = .452$, 95% CI [−1.88, 3.86]. There was no outlier for neutral trials.

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Table 2

Demographic and Clinical Characteristics of the Participants in the Studies

	Sample size		Age			% Females			BDI							
Study	rMDD	CTL	rMDD	CTL	Total	rMDD	CTL	Total	rMDD	CTL	Num. of MDEs	Weeks Last MDE	% ATD	% AD	% OCD	% PTSD
Aker et al. (2014)	85	62	38.2	37.2	37.8	100.0	100.0	100.0	10.1	4.0			0.0	23.5	3.5	1.2
Alders et al. (2020)	16	30	33.0	33.0	33.0	75.0	73.0	74.2			4.0					
Beevers (2002)	23	54	*18.7*[a]	*18.3*[a]	*18.5*[a]	73.9	81.5	79.2	7.0	3.4						
Bistricky et al. (2014)	14	14	21.8	18.9	20.3	71.4	57.1	64.3	3.8	2.1			*7.1*[a]			
Cerny et al. (2019)	93	35	23.2	22.4	23.0	73.0	57.0	68.6			4.9		12.9			
Dai and Feng (2011)	17	17	27.5	25.7	26.6	64.7	58.8	61.8	8.1	1.4			17.6			
Dai et al. (2011) S1	17	17	27.4	26.3	26.8	52.9	58.8	55.9	7.8	2.4		22.3	11.8			
Dai et al. (2011) S2	17	17	27.3	25.9	26.6	58.8	52.9	55.9	8.3	1.7		20.3				
De Nooij et al. (2022)	282	784	60.0	62.0	61.5	70.9	53.2	64.4					30.1			
Fang et al. (2022)	18	20	32.1	29.5	30.7	77.8	75.0	76.3	5.8	2.0	3.5		44.4			
Figueroa et al. (2019)	69	43	53.7	51.5	52.6	65.8	69.8	67.3			4.0		0.0			
Fritzsche et al. (2010)	20	20	40.0	38.5	39.2	50.0	50.0	50.0	8.8	2.5			30.0	0.0		
Gilboa and Gotlib (1997) S1	34	28							5.7	5.4						
Gilboa and Gotlib (1997) S2	38	53							5.3	3.7	1.7					
Gleave (2014)	13	30	20.6	19.5	20.0	100.0	100.0	100.0	10.5	5.5						
Goeleven et al. (2006)	20	20	44.6	38.9	41.8	60.0	70.0	65.0	11.7	1.1						
Guha et al., (2021)[b]	34	50	35.6	34.3	34.9	60.0	58.0	58.9						29.0	5.3	5.3
Gupta and Kar (2012)	10	10	43.0	43.6	43.3	50.0	50.0	50.0				17.4				
Hamilton (2004) no stress	23	19	40.4	31.0	35.7	100.0	100.0	100.0	9.7	3.0	3.9		47.8	*4.3*[a]	*4.3*[a]	*4.3*[a]
Hamilton (2004) stress	25	19	36.0	30.5	33.2	100.0	100.0	100.0	10.1	3.4	3.3		56.0	8.0	0.0	0.0
Harfmann (2013)	30	30	19.0	19.5	19.2	33.3	53.3	43.3	6.3	1.9	6.3					
Hedlund and Rude (1995)	15	20	31.2	30.6	30.9	73.3	77.8	75.9	4.7	2.6		43.4	73.3	60.0	0.0	0.0
Hsu and Davison (2017)	30	29	23.9	24.1	24.0	66.7	62.1	64.4	7.6	5.9	4.3	115.2				
Ingram and Ritter (2000)	35	38				74.3	55.3	64.4	3.5	2.1						
Joormann (2004)	11	15	32.0	25.0	28.5	90.9	73.3	80.8	3.8	4.6						
Joormann and Gotlib (2010)	77	47	35.9	38.9	37.4	61.0	68.1	63.7	4.7	3.1	3.6		22.0			
Keedwell et al. (2016)	21	25	22.1	21.9	22.0	100.0	100.0	100.0	11.3	2.6	2.8		0.0			
Kerestes et al. (2012)	19	20	33.6	35.8	34.7	78.0	50.0	63.6			4.4		0.0			
Kyte et al. (2005)[b]	22	49	15.3	15.2	15.3	60.0	59.2	59.4			1.0		10.0	36.7	23.3	3.3
Lange et al. (2012)	69	76	38.2	38.4	38.3	52.2	50.0	51.0	2.8	2.2	3.4	136.5	55.1			
Levens and Gotlib (2015)	23	24	41.0	37.0	39.0	82.6	54.2	68.1						21.7	8.7	0.0
Liu et al. (2017)	25	27	32.7	30.4	31.6	68.0	66.7	67.3	7.4	6.3		31.8				
Loeffler et al. (2019)[b]	16	26	35.3	35.3	35.3	53.8	53.8	53.8	17.6	2.42	3.3	31.1	84.6			
Maalouf et al. (2012)	20	17	15.4	15.2	15.3	75.0	52.9	64.9			1.2		68.0	50.0	0.0	0.0
Matsubara et al. (2014)	16	20	45.4	41.4	43.4	100.0	100.0	100.0			2.8		93.8	0.0	0.0	0.0
Merens et al. (2008)	20	21	48.7	44.1	46.4	45.0	57.1	51.2	12.9	5.2	4.8	25.6	100.0	20.0	0.0	0.0
Munafò et al., (2006)	48	24	36.8	38.6	37.4	58.3	58.3	58.3	2.6	1.8			50.0			
Niemegeers et al. (2019)	21	18	33.9	32.7	33.3	100.0	100.0	100.0					66.7	0.0	0.0	0.0
Quigley et al. (2020)	55	51	34.9	32.7	33.8	87.3	80.4	84.0	8.6	2.7	*5.2*[a]	*85.8*[a]	49.1	*21.8*[a]	*3.6*[a]	*0.0*[a]
Rnic (2019)	69	57	26.0	24.4	25.2	81.2	24.4	55.5	12.8	4.5	2.9		23.2			
Roiser et al. (2009)	23	20	32.4	30.5	31.5	73.9	65.0	69.8			2.7	78.2	0.0	0.0	0.0	0.0
Strand et al. (2013)	20	20											0.0			
van Oostrom et al. (2013)	23	21	45.2	42.1	43.7	100.0	100.0	100.0			4.5		73.9			
Vanderhasselt et al. (2011)	15	18	27.9	27.2	27.5	53.0	50.0	51.4	4.8	2.0	4.1	104.8	0.0	0.0	0.0	0.0
Vergara-Lopez et al. (2016)	90	95	19.2	18.7	18.9	60.0	52.6	56.2								
Vrijsen et al. (2014)	337	83	48.0	43.0	45.5	66.0	67.0	66.2	14.0	4.0	*3.5*[a]		*46.0*[a]			
Wang et al. (2012)	18	20				38.9	60.0	50.0					100.0			
Sample Mean	44.8	48.6	33.3	31.7	32.9	71.3	67.4	69.4	7.9	3.2	3.6	59.4	36.9	17.6	3.3	0.7

Note. Empty cells reflect values that were unavailable. rMDD = remitted major depressive disorder; CTL = healthy control; BDI = Beck Depression Inventory; MDE = major depressive disorder; ATD = current antidepressant treatment; AD = comorbid anxiety disorder; OCD = comorbid obsessive-compulsive disorder; PTSD = comorbid post-traumatic stress disorder. S1 = study 1, S2 = study 2.
[a] Values were obtained from correspondence with authors. [b] Sample consisted mostly of participants in remission and a small portion of participants experiencing a current episode.

Table 3
Characteristics of the Methodology and Design of the Included Studies

Study	Domain	Experimental task	Stimulus type	Outcome measure	Induction procedure
Aker et al. (2014)	SI	Emotional Picture Sorting Task	Pictures	Error	N
Alders et al. (2020)	IN	Emotional Conflict Task	Words, Faces	Both	N
Beevers (2002)	IN	Emotional Stroop Task	Words	RT	Imagery
Bistricky et al. (2014)	GEN	Visual Oddball Task	Faces	Both	N
Cerny et al. (2019)	SI	Emotion Card Sort Test	Words	Error	N
Dai and Feng (2011)	IN	Emotional Stroop Task	Words	RT	N
Dai et al. (2011) S1	IN	Negative Affective Priming Task	Faces	RT	N
Dai et al. (2011) S2	IN	Negative Affective Priming Task	Faces	RT	N
De Nooij et al. (2022)	IN	Affective Go/No-Go Task	Faces	RT	N
Fang et al. (2022)	IN	Emotional Stroop Task	Words	Both	N
Figueroa et al. (2019)	GEN	Exogenous Cueing Task	Faces	RT	N
Fritzsche et al. (2010)	IN	Emotional Stroop Task	Words	RT	Picture
Gilboa and Gotlib (1997) S1	IN	Emotional Stroop Task	Words	RT	Recall
Gilboa and Gotlib (1997) S2	IN	Emotional Stroop Task	Words	RT	Recall
Gleave (2014)	IN	Sternberg Go/No-Go Task	Words	Both	N
Goeleven et al. (2006)	IN	Negative Affective Priming Task	Faces	RT	N
Guha et al. (2021)	IN	Emotional Stroop Task	Words	RT	N
Gupta and Kar (2012)	IN	Emotional Stroop Task	Words	RT	N
Hamilton (2004)	IN	Emotional Stroop Task	Words	RT	Recall
Harfmann (2013)	IN	Affective Go/No-Go Task	Words	Both	N
Hedlund and Rude (1995)	IN	Emotional Stroop Task	Words	RT	N
Hsu and Davison (2017)	IN	Emotional Stroop Task	Words	RT	N
Ingram and Ritter (2000)	IN	Dichotic Listening Task	Audio	Error	Music, recall
Joormann and Gotlib (2010)	IN	Negative Affective Priming Task	Words	Both	N
Joormann (2004)	IN	Negative Affective Priming Task	Words	RT	N
Keedwell et al. (2016)	IN	Emotional Stroop Task	Words, Faces	Both	N
Kerestes et al. (2012)	UP	EFNBACK Task	Faces	Both	N
Kyte et al. (2005)	IN	Affective Go/No-Go Task	Words	RT	N
Lange et al. (2012)	IN	Affective Shifting Task	Pictures	Both	N
Levens and Gotlib (2015)	UP	N-Back Task	Faces	Both	N
Liu et al. (2017)	UP	WM Manipulation Task	Pictures	Both	N
Loeffler et al. (2019)	IN	Emotional Stroop Task	Words, Faces	RT	N
Maalouf et al. (2012)	IN	Affective Go/No-Go Task	Words	Both	N
Matsubara et al. (2014)	IN	Emotional Stroop Task	Words	Both	N
Merens et al. (2008)	IN	Emotional Stroop Task	Words	RT	N
Munafò et al. (2006)	IN	Emotional Stroop Task	Words	RT	Bio.
Niemegeers et al. (2019)	IN	Emotional Stroop Task	Words	RT	TSST, bio.
Quigley et al. (2020)	IN	Emotional Flanker Task	Faces	Both	N
Quigley et al. (2020)	SI	Color Emotion Task	Faces	Both	N
Quigley et al. (2020)	UP	N-Back Task	Faces	Both	N
Rnic (2019)	SI	Affective Flexibility Task	Pictures	Both	N
Rnic (2019)	UP	N-Back Task	Words	Both	TSST
Roiser et al. (2009)	IN	Affective Go/No-Go Task	Words	Both	Bio.
Strand et al. (2013)	IN	Emotional Stroop Task	Words, Faces	Both	N
van Oostrom et al. (2013)	IN	Emotional Stroop Task	Words	RT	N
Vanderhasselt et al. (2012)	GEN	Cued Emotional Conflict Task	Faces	Both	N
Vergara-Lopez et al. (2016)	SI	Emotional Card Sort Task	Words	Error	N
Vrijsen et al. (2014)	IN	Emotional Stroop Task	Words	RT	Film clip
Wang et al. (2012)	GEN	Visual Oddball Task	Pictures	RT	N

Note. Music, recall, imagery, picture, and film clip used in the induction procedure all contained sad content. IN = inhibition; SI = shifting; UP = updating; GEN = general cognitive control; WM = working memory; bio. = biological stressor; RT = response time; TSST = Trier Social Stress Task. S1 = study 1, S2 = study 2.

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Between-Group Differences in Negative and Positive Trial Difference Scores

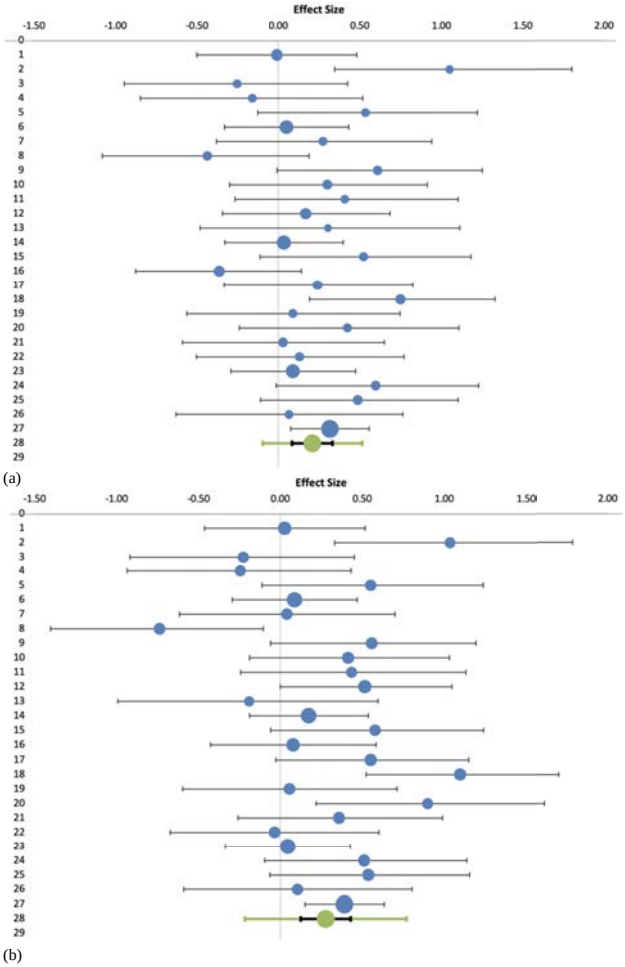
Negative–positive, positive–neutral, and negative–neutral difference scores were computed separately for RTs and error rates. For error rates, there were no significant group differences: negative–positive error difference score, $g = -.11$, $SE = .061$, $p = .065$, 95% CI [−0.25, 0.020], negative–neutral error difference score, $g = .128$, $SE = .16$, $p = .420$, [−0.24, 0.49], positive–neutral error difference score, $g = .11$, $SE = .070$, $p = .126$, [−0.054, 0.27].[4] For RTs, biased cognitive control characterized by reduced cognitive control (i.e., sustained processing) for negative relative to positive information is reflected by longer RTs for negative relative to positive stimuli. This would result in a larger negative trial RT–positive trial RT difference score. Compared to the HC group, the rMDD group had a

[4] Prior to outlier removal (i.e., Joormann & Gotlib, 2010), the between-group difference in the negative-positive error difference score was also non-significant, $g = .55$, $SE = 1.35$, $p = .684$, 95% CI [−2.40, 3.50]. There was no outlier for the negative-neutral and positive-neutral difference scores.

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Figure 2

Forest Plot for the Between-Group RT Comparisons for (a) Positive Stimuli and (b) Negative Stimuli

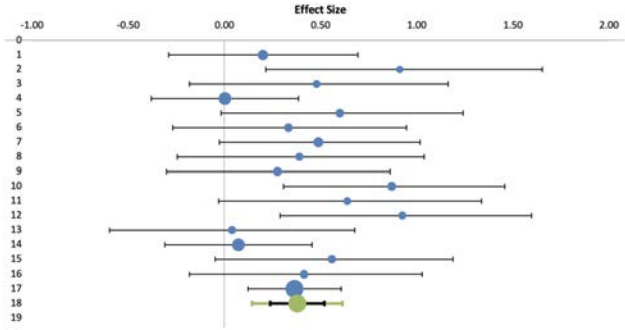


Note. Larger effect sizes reflect longer RTs for (a) positive stimuli and (b) negative stimuli in the rMDD group relative to the HC group. Blue circles represent effect sizes of individual studies; larger circles reflect larger sample sizes. The combined effect size is represented by the green circle with its confidence interval in black and its prediction interval in green. Numbers on the *Y*-axis correspond to studies listed as follows: [1]Beevers (2002); [2]Dai and Feng (2011); [3]Dai et al. (2011, S1); [4]Dai et al. (2011, S2); [5]Fang et al. (2022); [6]Figueroa et al. (2019); [7]Gleave (2014); [8]Goeleven et al. (2006); [9]Hamilton (2004, S1); [10]Hamilton (2004, S2); [11]Hedlund and Rude, (1995); [12]Hsu and Davison (2017); [13]Joormann (2004); [14]Joormann and Gotlib (2010); [15]Kerestes et al. (2012); [16]Kyte et al. (2005); [17]Levens and Gotlib (2015); [18]Liu et al. (2017); [19]Maalouf et al. (2012); [20]Matsubara et al. (2014); [21]Merens et al. (2008); [22]Niemegeers et al. (2019); [23]Quigley et al. (2020); [24]Roiser et al. (2009); [25]van Oostrom et al. (2013); [26]Vanderhasselt et al. (2011); [27]Vrijsen et al. (2014). HC = healthy control. See the online article for the color version of this figure.

significantly larger negative–positive RT difference score, $g = .12$, $SE = .050$, $p = .012$, 95% CI [0.022, 0.23] (Figure 4). However, there were no significant group differences in the negative–neutral RT difference score, $g = .061$, $SE = .054$, $p = .266$, 95% CI [−0.055, 0.18], or in the positive–neutral RT difference score, $g = -.086$, $SE = .058$, $p = .143$, [−0.21, 0.038]. Thus, compared to healthy individuals, individuals with rMDD exhibited biased cognitive control characterized specifically by sustained processing of negative relative to positive material in working memory. Heterogeneity was not significant for the negative–positive RT difference score, $Q = 25.30$, $p = .502$, $I^2 = 0$.

Figure 3

Forest Plot for the Between-Group RT Comparisons for Neutral Stimuli



Note. Larger effect sizes reflect longer RTs for neutral stimuli in the rMDD group relative to the HC group. Blue circles represent effect sizes of individual studies; larger circles reflect larger sample sizes. The combined effect size is represented by the green circle with its confidence interval in black and its prediction interval in green. Numbers on the *Y*-axis correspond to studies listed as follows: [1]Beevers (2002); [2]Dai and Feng (2011); [3]Fang et al. (2022), [4]Figueroa et al. (2019); [5]Hamilton (2004, S1); [6]Hamilton (2004, S2); [7]Hsu and Davison (2017); [8]Kerestes et al. (2012); [9]Levens and Gotlib (2015); [10]Liu et al. (2017); [11]Matsubara et al. (2014); [12]Merens et al. (2008); [13]Niemegeers et al. (2019); [14]Quigley et al. (2020); [15]Roiser et al. (2009); [16]van Oostrom et al. (2013); [17]Vrijsen et al. (2014). HC = healthy control. See the online article for the color version of this figure.

Within-Group Differences in Negative and Positive Trial Performance

To further delineate the association between the cognitive control of negative relative to positive stimuli and depression status, RTs and error rates were examined within each group. The RTs for negative stimuli did not significantly differ from the RTs for positive stimuli in either the rMDD group, $g = .033$, $SE = .045$, $p = .471$, 95% CI [−0.061, 0.13], or in the HC group, $g = -.016$, $SE = .034$, $p = .649$, [−0.086, 0.055]. Similarly, the difference in the error rates between negative and positive trials was not significant in the rMDD group, $g = .0016$, $SE = .063$, $p = .980$, 95% CI [−0.14, 0.14] or in the HC group, $g = .046$, $SE = .061$, $p = .449$, [−0.087, 0.18].[5] Thus, cognitive control for negative and positive information did not differ significantly in either group.

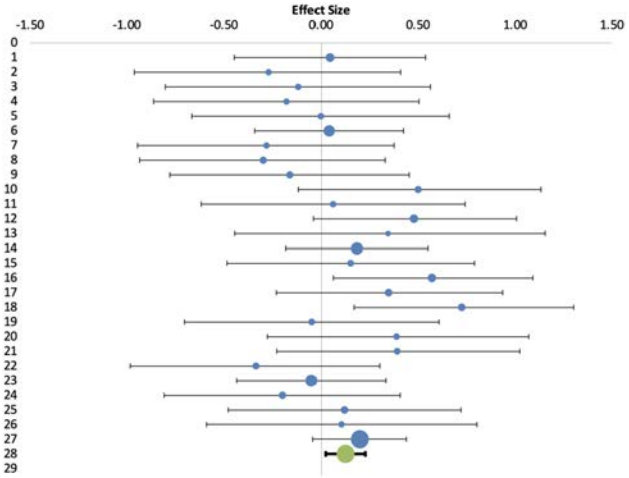
Moderator Analyses Findings

The hypothesized moderator variables were examined for the significant overall effects: the between-group comparisons in the

[5] Prior to outlier removal (i.e., Joormann & Gotlib, 2010), this difference was also not significant in the rMDD group, $g = .46$, $SE = .85$, $p = .585$, 95% CI [−1.40, 2.32], or in the healthy control group, $g = -.17$, $SE = .36$, $p = .634$, 95% CI [−.97, .63].

Figure 4

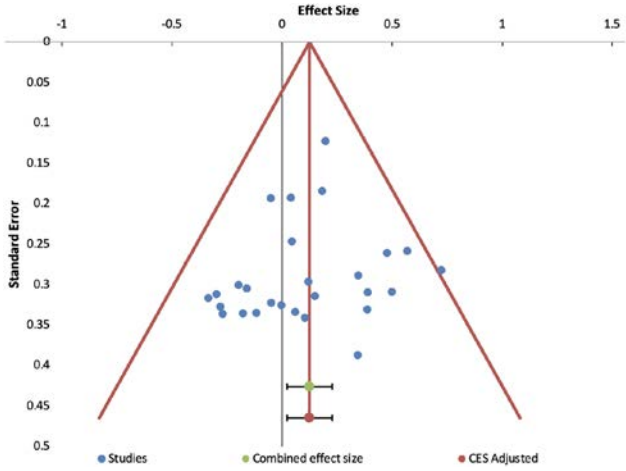
Forest Plot for the Between-Group Comparisons in the Negative–Positive RT Difference Scores



Note. Larger effect sizes reflect greater differences in the negative-positive RT difference score in the rMDD group relative to the HC group. Blue circles represent effect sizes of individual studies; larger circles reflect larger sample sizes. The combined effect size is represented by the green circle with its confidence interval in black and its prediction interval in green. Numbers on the *Y*-axis correspond to studies listed as follows: [1]Beevers (2002); [2]Dai and Feng (2011); [3]Dai et al. (2011, S1); [4]Dai et al. (2011, S2); [5]Fang et al. (2022), [6]Figueroa et al. (2019); [7]Gleave (2014); [8]Goeleven et al. (2006); [8]Hamilton (2004, S1); [10]Hamilton (2004, S2); [11]Hedlund and Rude (1995); [12]Hsu and Davison (2017); [13]Joormann (2004); [14]Joormann and Gotlib (2010); [15]Kerestes et al. (2012); [16]Kyte et al. (2005); [17]Levens and Gotlib (2015); [18]Liu et al. (2017); [19]Maalouf et al. (2012); [20]Matsubara et al. (2014); [21]Merens et al. (2008); [22]Niemegeers et al. (2019); [23]Quigley et al. (2020); [24]Roiser et al. (2009); [25]van Oostrom et al. (2013); [26]Vanderhasselt et al. (2011); [27]Vrijsen et al. (2014). HC = healthy control. See the online article for the color version of this figure.

Figure 5

Funnel Plot of the Between-Group Comparisons on the Negative–Positive RT Difference Scores



Note. CES = combined effect size. See the online article for the color version of this figure.

negative–positive RT difference score, negative RTs, positive RTs, and neutral RTs. Experimental task and stimulus type moderated the significant effects of interest. Other moderators related to clinical features (depressive symptom level, number of MDEs, time since last MDE, % antidepressant use, % comorbid disorders), participant characteristics (age, % females), and study design (induction procedure), as well as sample size, publication year, and study quality did not moderate the significant effects of interest. Only the results of significant moderator analyses are presented; detailed results of the nonsignificant moderator analyses are presented in the online supplemental materials (S2).

The effect of the experimental task was examined for the inhibition domain ($n = 23$) using subgroup analysis. The experimental task significantly moderated the between-group difference for the negative RTs, $F(3, 18) = 11.28$, $p = .010$, and positive RTs, $F(3, 18) = 8.73$, $p = .033$. Specifically, positive effect sizes were observed for the Stroop task, the AGNG task, and tasks in the Other category, whereas a negative effect size was observed for the NAP task.[6] Subgroup analysis for stimulus type (face vs. word) revealed a significant moderation effect on the between-group difference for neutral RTs, $F(1, 14) = 5.40$, $p = .020$, and for negative RTs, $F(1, 24) = 4.06$, $p = .044$. Specifically, effect sizes were larger for word stimuli than facial stimuli.[7]

Publication Bias

The funnel plot, Egger's test, and Rosenthal's fail-safe N were investigated for the between-group difference in the negative-positive RT difference score. As displayed in Figure 5, the funnel plot appears relatively symmetrical, suggesting little evidence for substantial publication bias. Likewise, the Egger's test was not significant, $t(26) = -.83$, $p = .413$. Lastly, Rosenthal's fail-safe N analysis indicates that the number of additional studies with an effect size of 0 required to nullify this significant result would be 18. In addition, publication bias is unlikely to account for the significant group differences in the negative, positive, and neutral trial RTs (see the online supplemental materials, S3). Overall, these results indicate that the significant findings in the current investigation are unlikely to be due to publication bias.

Discussion

The current study is the first comprehensive meta-analysis that investigated the cognitive control of both negative and positive information in rMDD. We compared performance on behavioral tasks with emotional stimuli in individuals with rMDD and healthy individuals. To this end, we examined the between-group comparisons in RTs, error rates, and difference scores (negative–positive, negative–neutral, positive–neutral) of RTs and error rates. We also

[6] The following are the effect sizes for the significant moderation effect of inhibition task: negative stimuli (NAP, $g = -.19$; Stroop, $g = .42$; AGNG, $g = .20$; Other, $g = .32$), and positive stimuli (NAP, $g = -.08$; Stroop, $g = .33$; AGNG, $g = .09$; Other, $g = .37$).

[7] The following are the effect sizes for the significant moderation effect of stimulus type: negative stimuli (words, $g = .34$; faces, $g = .03$), and neutral stimuli (words, $g = .44$; faces, $g = .12$).

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examined the within-group comparisons in RTs and error rates for negative versus positive, negative versus neutral, and positive versus neutral stimuli.

Cognitive Control of Emotional Information Across Groups

Individuals with rMDD responded significantly slower to negative, positive, and neutral stimuli compared to healthy individuals, suggesting that individuals with rMDD had greater difficulty in the cognitive control of emotional and nonemotional stimuli. Importantly, the extent of this difficulty varied across the valence of the stimuli. Specifically, the groups differed when the relative cognitive control of negative and positive stimuli was examined together via a difference score (i.e., negative RT–positive RT), with a greater difference in the rMDD group. That is, negative–positive bias (i.e., a positive value for the negative-positive RT difference score) was greater in the rMDD group than in the HC group. These findings were not accounted for by publication bias. Furthermore, no group differences were observed for the negative–neutral or positive–neutral RT difference scores. That is, only the processing of negative versus positive information in working memory differentiated individuals with rMDD from healthy individuals. Thus, the common practice of examining the between-group differences in negative and positive cognitive control separately might not provide a complete picture. A further examination of the between-group difference in negative–positive bias may help us better understand MDD recurrence.

Moderating Effects of Clinical Features, Participant Characteristics, and Study Design

Most of the moderators examined did not influence the association between the biased cognitive control of emotional stimuli and depression status. This includes moderators related to clinical features (depressive symptom level, number of MDEs, time since last MDE, antidepressant use, comorbid disorders), participant characteristics (age, gender distribution), and study design (induction procedure, study quality), as well as sample size and publication year. Heterogeneity was low, which likely contributed to these null moderation findings.

Some moderators, however, did influence the association between depression status and the cognitive control of emotional and nonemotional material individually. Specifically, stimulus type influenced the between-group differences in RTs for negative, positive, and neutral stimuli. Moreover, the type of inhibition task moderated the between-group differences in RTs for negative and positive stimuli. These significant moderator effects may be due to the greater heterogeneity observed for the between-group comparisons of the raw RTs, particularly for negative stimuli. Contrary to expectations, stronger effects were observed using words than faces, which may be because more than twice as many studies used words compared to faces. This finding may also reflect the difference in how words versus facial expressions are processed in working memory. Research suggests that facial expressions are processed more automatically than emotional words, with less involvement of cognitive resources (see Palermo & Rhodes, 2007, for review). Thus, it is possible that cognitive control plays a greater role in the processing of emotional words than facial expressions, resulting in a more pronounced effect of cognitive bias observed for words than faces. Future studies are needed to further compare the cognitive control of emotional information across stimulus types. The observation of different magnitudes of effects across inhibition tasks is consistent with the fact that the NAP, AGNG, and Stroop tasks were designed to assess different aspects of inhibitory control (e.g., Miyake et al., 2000; Schulz et al., 2007). Overall, however, more studies that utilize picturial stimuli and examine shifting and updating are needed for a more comprehensive examination of the moderator effects of stimulus type and experimental task on the cognitive control of emotional information in rMDD.

In addition to the low heterogeneity, there may be theoretical explanations for the null effects of some moderators. A recent theory posits that people with rMDD may belong to either a depression-capable (few recurrences) group or a recurrence-prone (many recurrences) group (dual pathway model; Monroe et al., 2019). Life events are proposed to provoke the onset of MDE in the depression capable subtype but not in the recurrence-prone subtype. Over time, people who had a first MDE form a bimodal distribution for recurrences, with depression-capable people at the low end and recurrence-prone people at the high end. Individuals with rMDD in the current meta-analysis experienced an average of four episodes and an average of one year since the last MDE. Therefore, the sample in the current investigation may have a relatively low risk for recurrence. There is a need for studies to include clinical samples with a wider range of these moderator variables to empirically examine this theory.

The null moderation effect of gender is contrary to expectations, given that girls and women report higher levels of negative cognition in the form of self-attributions and rumination compared to boys and men (e.g., Hankin & Abramson, 2002; Nolen-Hoeksema & Jackson, 2001). Some evidence suggests that cognitive biases in women require activation by stressors more so than cognitive biases in men (e.g., Carr et al., 2016). Thus, gender differences in biased cognitive control may be more pronounced with the inclusion of a stress induction procedure. The small number of studies in the current meta-analysis that included an induction procedure precludes the examination of the interaction effect between gender and stress induction. Future studies are needed to investigate this proposition.

Implications of the Current Findings

Overall, findings in this meta-analysis suggest that individuals with rMDD have biased cognitive control characterized by sustained processing of negative relative to positive irrelevant information compared to healthy individuals. This bias can prolong the presence of negative material while shortening the presence of positive material in working memory, which may lead to a cycle of higher levels of negative (vs. positive) mood and thoughts. Furthermore, biased cognitive control can reduce disengagement from negative stimuli (e.g., Joormann et al., 2007) and enhance disengagement from positive stimuli, leading to a cascade of biased information processing in attention, self-referential processing, interpretation, and memory that prefer negative information (LeMoult & Gotlib, 2019). Thus, this biased cognitive control that is present during remission may confer risk for future MDD by influencing emotional information processing broadly.

Biased cognitive control may also increase recurrence risk through impairing emotion regulation. Sustained processing of negative versus positive material in working memory can hinder the use of effective regulatory strategies under stress, such as positive

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reappraisal (e.g., thinking about what one has learned from the situation). Other information processing biases resulting from biased cognitive control, such as attention and memory, can further hinder emotion regulation (Gotlib & Joormann, 2010). For instance, biased memory characterized by a greater recall of negative than positive experiences would hinder the ability to use pleasant memories to cope with stress (Kim & Yoon, 2020). Difficulties in emotion regulation can, in turn, lead to dysregulated mood and undermine goal-directed behaviors (Joormann & Stanton, 2016), increasing the risk for future MDD.

Interestingly, significant group differences were observed with RTs but not with error rates, which is consistent with the literature on cognitive bias in current MDD (e.g., Levens & Gotlib, 2010). Researchers have distinguished performance effectiveness (measured by error rates) from performance efficiency (measured by RTs; Eysenck et al., 2007). The current findings suggest cognitive control efficiency, rather than effectiveness, is biased in rMDD. Individuals with rMDD may have preserved effectiveness through increasing effort and allocating more resources to the task at the cost of reduced efficiency. The null findings, however, may also be due to the floor effect that resulted from low error rates. Future studies using more challenging tasks—resulting in higher error rates—are needed to clarify emotional cognitive control effectiveness in rMDD.

Lastly, the current findings highlight the importance of examining the processing of negative and positive information collectively when studying biased cognitive control in rMDD. When responses to negative and positive stimuli were examined together, rMDD participants responded slower to negative than positive stimuli compared to HC participants. In contrast, the group differences were not present for the differences in the responses to negative versus neutral stimuli or to positive versus neutral stimuli. This suggests that prolonged processing of negative information alone may not be associated with remission from MDD. Instead, prolonged processing of negative material in combination with shortened processing of positive information may be more relevant in rMDD. Future studies on cognitive biases, particularly during MDD remission, may benefit from examining the *relative* processing of negative, positive, and neutral stimuli for a more comprehensive picture of emotional information processing.

Strengths, Limitations, and Future Directions

The current investigation has several strengths. First, in addition to examining between-group differences in biased cognitive control like most meta-analytic reviews of similar topics, we also examined and presented on within-group differences as well as between-group differences of within-group differences. Second, we tested a comprehensive list of moderators predicted to influence the biased cognitive control in rMDD, including aspects related to clinical features (e.g., number of MDEs), methodological design (e.g., stimulus type), sample characteristics (e.g., gender), as well as sample size and publication year. Lastly, we comprehensively assessed for and examined the effect of study quality on the effects of interest.

Several limitations in the literature on biased cognitive control and rMDD that restricted the current investigation should also be noted. First, no domain-specific analyses were conducted due to the small number of studies that examined shifting and updating. This is particularly important given that recent latent variable work suggests that whereas inhibition reflects general cognitive control, updating and shifting remain specific factors of cognitive control (Miyake & Friedman, 2012). Thus, in both the original and updated models of cognitive control by Miyake et al. (2000) and Miyake and Friedman (2012), shifting and updating contain aspects that are related to but distinct from that of inhibition, or cognitive control broadly. Therefore, the link between depression status and biased cognitive control may differ across these domains. More studies that examine biases in shifting and updating are needed to better understand the role of different aspects of cognitive control in rMDD etiology.

Relatedly, correlations among the RTs and error rates were estimated for multiple studies due to the unavailability of such data from their respective experimental studies. This may reduce the precision of the effect sizes for the within-group comparisons. The unavailability of descriptive and correlational data creates problems in replicating effects, conducting comprehensive reviews, and practicing open science more broadly. Thus, there is a need for experimental studies to report such data in text or as supplemental materials.

Second, there exist limitations regarding the samples. This includes the low variability in the number of MDEs (range = one to six episodes) and the preponderance of younger samples ($M_{age} = 32.9$). The focus on younger samples in the literature is problematic given that any aging effects that differentially impact depression-related cognitive impairments (e.g., Austin et al., 2001) may not be prominent in younger samples. Moreover, younger participants are less likely to have experienced numerous episodes, thereby restricting the moderator analyses of both age and the number of MDEs. Future studies with more diverse age groups would allow for a more comprehensive examination of the effects of age and the number of MDEs on the link between biased cognitive control and rMDD.

Third, the measures of cognitive control likely have psychometric issues. Reliability is generally unknown or low for the RT-based tasks that assess cognitive control (e.g., Strauss et al., 2005). Low reliability limits the power to detect significant effects. Moreover, subtractions can further reduce the reliability of the difference score (Hedge et al., 2018). Therefore, future studies should both report and improve the reliability of these measures. Lastly, this meta-analysis used Miyake et al.'s (2000) framework of cognitive control because it has been widely adopted in cognitive bias research. However, other frameworks (e.g., Levin et al., 1996; Robbins, 1996) proposed subdomains that are different from those in the Miyake et al. model. Moreover, Miyake and Friedman (2012) reconceptualized their model to be composed of a general cognitive control factor and shifting and updating specific factors. Thus, there is a need for studies to examine and compare the validity of different frameworks. Relatedly, Miyake et al.'s framework (2000, 2012) is based on latent factor analyses using neutral stimuli (e.g., Friedman & Miyake, 2004), and it remains unclear whether similar results would be found with emotional stimuli. Future studies should extend the latent variable work by using tasks with emotional stimuli.

Conclusion

To summarize, the present results showed that individuals with rMDD demonstrated reduced cognitive control for emotional and non-emotional stimuli compared to healthy individuals. Importantly, compared to healthy individuals, individuals with rMDD had greater biased cognitive control characterized by sustained processing of negative over positive irrelevant information in working memory. This imbalance might prolong the presence of negative relative to positive

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material in working memory, leading to more negative (vs. positive) mood and thoughts that are characteristic of MDD. Thus, biased cognitive control may be a recurrence risk factor for MDD. Moreover, examining negative and positive information processing together allowed for the identification of biased cognitive control not otherwise observed when examined separately. To comprehensively examine the link between cognitive bias and depression, studies may benefit from examining relative processing of negative versus positive information.

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