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Computerized cognitive control training to reduce rumination in major depression: A randomized controlled trial

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ABSTRACT

Objective: Rumination is a major risk factor for the onset and recurrence of depressive episodes and has been associated with deficits in updating working memory content. This randomized controlled trial examines whether training updating-specific cognitive control processes reduces daily ruminative thoughts in clinically depressed individuals.

Methods: Sixty-five individuals with a current major depressive episode were randomized to 10 sessions of either cognitive control training (N = 31) or placebo training (N = 34). The frequency and negativity of individuals' daily ruminative thoughts were assessed for seven days before training, after training, and at a 3-month follow-up using experience sampling methodology. Secondary outcomes were depressive symptoms, depressed mood, and level of disability.

Results: Cognitive control training led to stronger improvements in the trained task than placebo training. However, cognitive control training did not lead to greater reductions in the frequency or negativity of daily ruminative thoughts than placebo training. There were no training-specific effects on participants' depressive symptoms or level of disability.

Conclusions: The robustness of the present null-findings, combined with the methodological strengths of the study, suggest that training currently depressed individuals to update emotional content in working memory does not affect the frequency or negativity of their daily ruminative thoughts.

1. Introduction

Major depression is the most prevalent of all mental disorders (Kessler et al., 2012) and involves substantial personal suffering and tremendous societal costs (Kessler, 2012). The main challenge in tackling depression is its high rate of recurrence and chronicity, ranging between 50% in population-based samples (Eaton et al., 2008) and 83% in clinical samples (Kennedy et al., 2003). Despite decades of research, psychological and pharmacological treatments have failed to improve this poor long-term outcome (Vittengl et al., 2007). It is thus of utmost importance to target processes increasing the risk for re-experiencing depressive episodes.

Rumination is a well-known risk factor for the onset and recurrence of depressive disorders (Buckman et al., 2018; Nolen-Hoeksema et al., 2008). It is characterized by repetitive negative thoughts (Ehring & Watkins, 2008), which in depressed people often focus on the causes and effects of their own symptoms (Nolen-Hoeksema et al., 2008). People typically ruminate in response to stressful events, with the majority being able to exit these negative thought loops after a while to continue with their daily activities. Some people, however, find it difficult to interrupt their ruminative thoughts, ending up in prolonged episodes of rumination. This has been related to a deterioration in mood over time and is known to interfere with effective problem solving, ultimately putting one at increased risk for (re-)experiencing depressive episodes (Buckman et al., 2018; Nolen-Hoeksema et al., 2008).

Evidence suggests that people with such a strong tendency to ruminate may have difficulties updating the contents of their working memory (for a review, see Whitmer & Gotlib, 2013). Specifically, they

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seem to be less able to *discard* no longer relevant emotional content from working memory, which is part of the updating-specific component of executive functions (for a meta-analysis, see Zetsche et al., 2018). As a result, it may be more difficult to let go of negative thoughts that arise.

It may thus be a promising avenue to train depressed individuals' ability to update the contents of working memory. This may enable them to exit their ruminative thoughts more quickly and ultimately make them more resilient to recurrent depression. Siegle et al. (2007, 2014) were the first to demonstrate that a computerized cognitive control training led to improved cognitive control (i.e., updating) and less rumination and depressive symptoms in a sample of severely depressed patients. These promising results spurred a larger number of studies examining the effectiveness of cognitive control trainings in reducing rumination. However, results of these studies have been mixed (for a review, see Koster et al., 2017). The variance in results may be explained by a large heterogeneity in samples, training material, dosage, and study design. Specifically, only one third of these training studies examined the effects of cognitive control training in clinically depressed samples (Ferrari et al., 2021; Iacoviello et al., 2014; Jopling et al., 2020; Moshier & Otto, 2017; Siegle et al., 2014; Vanderhasselt et al., 2015; Wanmaker et al., 2015). The remaining studies recruited unselected samples, individuals high in rumination, or remitted depressed individuals. Heightened levels of rumination and deficits in cognitive control are characteristic of individuals with current or remitted major depression (Joormann et al., 2006; Nolen-Hoeksema et al., 2008; Quigley et al., 2022). We thus assume that clinically depressed and remitted depressed individuals will benefit most from training cognitive control (Koster et al., 2017). However, it should be noted, that previous findings have been mixed across all types of samples and there was no sample type that was more prevalent in studies that found or did not find training effects on rumination. In addition, almost all studies used *neutral* stimuli to train cognitive control. However, evidence suggests that individuals with a tendency to ruminate show specific deficits in discarding *negative* content from working memory (e.g., Zetsche et al., 2012). It may thus be important to employ emotionally negative training stimuli to improve the ability to remove negative material from working memory. Indeed, some findings suggest that cognitive control training procedures relying on emotional stimuli may be more effective in reducing clinical symptoms (Iacoviello et al., 2014). Last but not least, almost all studies relied on retrospective questionnaires to assess training-specific changes in rumination. Depressed individuals, however, exhibit systematic biases in the recollection of emotional states (e.g., Zetsche et al., 2019). Thus, results from retrospective global self-reports should be interpreted with caution.

The present randomized controlled clinical trial set out to examine the effects of a computerized cognitive control training on rumination in clinical depression while overcoming the above-mentioned methodological constraints. Specifically, we recruited individuals experiencing a **current episode of major depression** as established by a structured clinical interview. Second, we used an adaptive n-back task with **emotional stimuli** as training task**,** which has already shown positive effects on rumination in a previous study (Iacoviello et al., 2014). Third, we compared the effects of this training task to the effects of a **placebo training** requiring minimal working memory resources. Last but maybe most importantly, we assessed **ruminative thoughts in the daily lives** of participants using experience sampling methodology (ESM) across seven consecutive days before to training, after training, and at 3-month follow-up. There have only been two previous studies assessing the effects of a cognitive control training on daily rumination using experience sampling methodology (Hoorelbeke et al., 2016, 2023). However, the focus of these studies was on modelling the *immediate* effects of a cognitive control training on subsequent emotion regulation in daily life. In addition, these studies relied on healthy (Hoorelbeke et al., 2016) or remitted depressed samples (Hoorelbeke et al., 2023). Note, that we also assessed rumination with a widely used retrospective self-report measure in order to compare our results with previous findings in additional exploratory analyses.

Primary outcome measures of the present RCT were (a) the frequency and negativity of daily ruminative thoughts and (b) the impact of these ruminative thoughts on subsequent affect. Secondary outcome measures were (c) the ability to update emotional content in working memory, (d) daily dysphoric affect, (e) depressive symptoms, and (f) levels of disability. We expected larger improvements in cognitive control from pre-to post-training (and to follow-up) in the training group as compared to the placebo group. In addition, we expected greater decreases in daily ruminative thoughts, daily dysphoric affect, depressive symptoms, and levels of disability from pre-to post-training (and to follow-up) in the training as compared to the placebo group.

2. Methods

2.1. Open science practice and ethical approval

The present randomized controlled clinical trial had been preregistered at ClinicalTrials.gov (https://clinicaltrials.gov/show/NCT03011216). We provide the anonymized and preprocessed dataset and the exact statistical code for all main and additional analyses at Open Science Framework (OSF): https://osf.io/7cdmw/?view_only=41de767b44a143e6937fea5e3219ab92.

The study was performed in accordance with the World Medical Association Declaration of Helsinki. The Ethics Committee at Freie Universität Berlin approved the study protocol (no. 137/2017). All participants gave written informed consent prior to participation and were debriefed and reimbursed at the end of their participation.

2.2. Participants

Participants were recruited through advertisements in online newspapers, social media, and at different sites within the community. After a telephone-screening, eligible participants completed a face-to-face Structured Clinical Interview for DSM-IV (SCID; Wittchen et al., 1997) with an adapted depression section to fit DSM-5 criteria. Interviewers were trained in applicating the SCID and were closely supervised for all interviews. All participants had to meet diagnostic criteria for a current major depressive episode according to DSM-5. Further, participants had to be between 18 and 65 years old, have a computer with access to the internet, be native German speakers (due to verbal demands in the cognitive tasks) and not receive psychotherapy for the duration of the main study. We included persons with psychotropic medication as long as it had been stable for four weeks prior to and throughout the study. Exclusion criteria were (a) substance abuse in the last 12 months or lifetime substance dependency, (b) lifetime bipolar or psychotic disorder, (c) current obsessive-compulsive disorder, (d) current borderline personality disorder, (e) reporting severe underweight (BMI<18), any neurological disease, severe head injury, or any brain damage.

The final sample comprised of 65 participants, who were randomized to either the cognitive control training (n = 31) or active placebo training (n = 34). Fig. 1 displays the detailed participant flow.

2.3. A priori power analysis

Prior to collecting data, we ran two simulation models to estimate the sample size needed to detect (a) a decrease in rumination frequency of 25% from pre-to post-training in the training as compared to the control condition, and (b) a change of $\beta = -0.20$ for the effect of rumination on dysphoric affect from pre-to post-training in the training as compared to the placebo group. Both estimates of change were based on effect sizes in prior studies (e.g., Hoorelbeke & Koster, 2017), see online supplements for details. The simulations resulted in an estimated total sample size of $n = 50$ ($n = 25$ per group) to generate a power of at least 90%. We slightly oversampled to compensate for expected attrition rates.

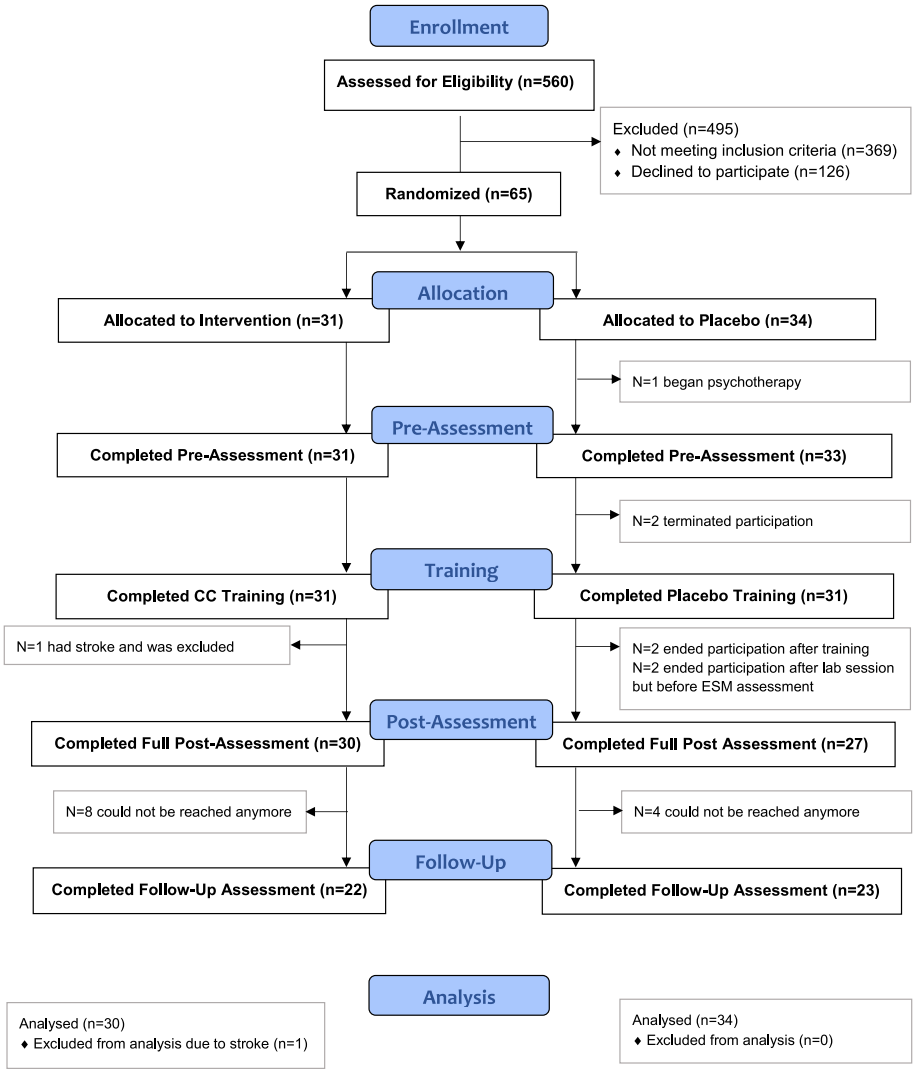

Fig. 1. CONSORT flow diagram.

2.4. Materials

2.4.1. Online training tasks

To ensure optimal training, task difficulty in both training tasks was adapted to individuals' performance by raising or lowering the level of difficulty at an accuracy rate of ≥90% or ≤60% of trials in the previous block, respectively. No feedback was provided. Both training tasks comprised of 15 blocks with 20 trials each per training session. Participants were asked to complete 10 sessions within 14 days. Training sessions could be distributed individually, but were limited to one session per day between 6 a.m. and midnight. The online training tasks ran on a secure university server.

2.4.1.1. Cognitive control training. The cognitive control training consisted of an adaptive emotional n-back task with emotional facial expressions (based on Iacoviello et al., 2014). On each trial, participants were presented with one emotional face expression and were asked to indicate whether or not the current facial expression displayed the same emotion as the facial expression *n* trials back (see Fig. S1 in online supplements). Participants have to update the sequence of items held in working memory continuously to accomplish this task. Thus, the n-back task is assumed to train updating-specific components of executive control. Each training session started at level $n = 2$, a stimulus presentation time of 2000ms, and a fixation cross presented for 2000ms. An increase in task difficulty was implemented by reducing the presentation time of the fixation cross to 1500ms in the subsequent block. For the next level of difficulty, *n* rose by one, and so on. To decrease task difficulty, the presentation time of the fixation cross was increased to 2000ms, and for the next decrease, *n* decreased by one, and so on (see Table S1 in online supplements for details). Every block of 20 trials contained seven match trials.

Stimuli were 80 pictures of facial expressions portraying happiness, surprise, sadness, or anger taken from three sets (i.e., Radboud Faces Database; Langner et al., 2010; the Warsaw set of emotional facial expression pictures; Olszanowski et al., 2015; The NimStim set of facial expressions; Tottenham et al., 2009). Every actor was used for only one emotion and presented only once per block to avoid unwanted interferences.

2.4.1.2. Placebo training. The placebo training task consisted of an adaptive non-emotional feature match task (based on Schweizer et al., 2011) requiring minimal working memory resources (Schweizer et al., 2011, 2013). On each trial, participants were presented with two frames containing 8–12 geometric shapes and had to indicate whether or not the content of the two frames was identical. Each training session started with 10 forms per frame and a stimulus presentation time of 3800ms. An increase in task difficulty was implemented by reducing the stimulus presentation time to 3100ms. For the next level of difficulty, the number

of forms per frame rose by two, and so on. A decrease in task difficulty was implemented by increasing the stimulus presentation time and the number of forms in an analogous manner (see Table S1 in online supplements). Stimuli were 14 different geometric shapes (e.g., circle, arrow).

2.4.2. Assessment of cognitive control

2.4.2.1. Close transfer. Close transfer was assessed by an emotional n-back task almost identical to the training task, except that the assessment task was non-adaptive. It included 12 trial blocks following a predefined order of difficulty (see Table S1 in online supplements). There were 10 match trials per block of 20 trials, i.e., the probability of a correct answer when guessing was 0.5. We used a different set of emotional face stimuli (i.e. 56 facial expressions from the Karolinska Directed Emotional Faces, KDEF; Lundqvist et al., 1998).

2.4.2.2. Far transfer. We employed the Working Memory Selection Task, which is a modified Sternberg task based on Joormann and Gotlib (2008) to assess far training transfer. On each trial, participants had to memorize one line of three positive words and one line of three negative words. Next, a cue indicated which line of words remained relevant. Participants were asked to keep the relevant words in mind and discard the other three words. Finally, a probe word was displayed and participants had to indicate whether or not the probe was part of the relevant line. The probe was either a word from the relevant line, a word from the to-be-discarded line (irrelevant probe), or a novel word not presented before. Joormann and Gotlib (2008) proposed that correctly rejecting irrelevant probes takes longer than rejecting novel probes because of a residual activation of the formerly relevant word. The ability to discard no longer relevant information from working memory was thus assessed as the difference in response latencies to novel and irrelevant probes. To test our hypothesis, we only analyzed response latencies to negative probes, because we were interested in individual differences in the ability to discard *negative* material from working memory. Results of additional analyses on positive probes can be found in the html markdown file at OSF.

The task consisted of 90 trials, including 24 irrelevant probe trials and 24 novel probe trials. Words were presented for 6000 ms and the cue for 1000 ms. There was no time limit for responding to the probe. Stimuli were 150 positive and 150 negative words from the Berlin Affective Word List – Reloaded (BAWL-R; Võ et al., 2009). Positive and negative words were matched for arousal, word length, and frequency of use.

2.4.3. Clinical measures

2.4.3.1. Primary clinical outcome: rumination. We used an experience sampling design to assess participants' level of rumination and its impact on affect in their daily lives. Participants received a smartphone with a pre-installed experience sampling application (movisensXS; movisens GmbH, Karlsruhe, Germany). The application beeped eight times a day for seven consecutive days at each assessment period (baseline, post-training, 3-months follow-up). Beeps occurred pseudo-randomly between 9 a.m. and 9 p.m. with >30 min and no more than 2 h between consecutive beeps. Participants could postpone prompts twice for 5 min.

At each prompt, participants were asked to rate on a scale from 0 ("*never*") to 6 ("*very often*") how often they had ruminated since the last prompt ("*I thought over and over again about a situation or my feelings.*"; formulation taken verbatim from Heiy & Cheavens, 2014). They also rated how negative their thoughts had been ("*How negative were these thoughts?*"; 0 = "*not at all negative*", 6 = "*very negative*"). Note, that these items showed good external validity in a previous study by the authors (Zetsche et al., 2024). Specifically, both items were highly correlated with the Ruminative Responses Scale ($r = 0.57–0.68$) and were able to detect (a) elevated levels of daily rumination in two previous clinical samples and (b) a large negative effect of rumination on subsequent affect. This mirrors the findings from a comprehensive body of research on depressive rumination as assessed by trait questionnaires. At each prompt, participants also rated on a scale from 0 ("*not at all*") to 6 ("*very much*") how sad, depressed, lonely, angry, anxious, ashamed, cheerful, and happy they felt right at the prompt. Participants also rated the use of five other emotion regulation strategies not part of this study (suppression, reappraisal, distraction, acceptance, social sharing; see online supplements). The experimenter explained each experience sampling item to participants and also handed over a document with detailed instructions to take home (see online supplements). Participants received an extra incentive of 5€ per assessment period if responding to more than 90% of beeps.

2.4.3.2. Secondary clinical outcome measures. The German Version of the 20-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977; German version: Hautzinger & Bailer, 1993) assessed the *severity of depressive symptoms* at baseline, post- and follow-up assessment. Internal consistencies at the three assessment times in the present sample were good, baseline: $\Omega_{total} = 0.84$; post: $\Omega_{total} = 0.89$; follow-up: $\Omega_{total} = 0.91$. The WHO Disability Assessment Schedule (WHODAS; World Health Organisation, 2010) assessed the *level of disability* in cognition, mobility, self-care, getting along, life activities, and participation. Its 12-item sum score was found to be reliable and valid (Pösl et al., 2007). It showed good internal consistency in this sample, baseline: $\Omega_{total} = 0.84$; post: $\Omega_{total} = 0.88$; follow-up: $\Omega_{total} = 0.88$. For exploratory analyses, participants also completed the 10-item Ruminative Responses Scale – Short Form (Treynor et al., 2003) at baseline, post- and follow-up assessment. Note, that we modified the time frame of the questionnaire (i.e., *Please indicate what you did or felt during the past week* (instead of in general)), in order to be sensitive to change following the intervention. Internal consistencies at the three assessment times in the present sample were good, baseline: $\Omega_{total} = 0.84$; post: $\Omega_{total} = 0.81$; follow-up: $\Omega_{total} = 0.81$.

2.5. General procedure

After a telephone-screening, eligible participants completed a face-to-face clinical interview and were then randomized to either cognitive control or placebo training (parallel group design). Participants were blind to their condition. Randomization was stratified by gender (male, female) and BDI-II sum score, which was assessed once at the end of the clinical interview (BDI-II intervals: <24, 24–28, 29–33, 34–38, 39–43, >44). Next, participants completed a seven-day experience sampling procedure assessing daily rumination and affect, and then returned to the lab for pretesting (Working Memory Selection Task (WMST), non-adaptive n-back task, and clinical questionnaires). Within the following 14 days, participants had to complete 10 online training sessions from home. They returned to the lab for post-testing (WMST, n-back task, questionnaires) and subsequently completed a second seven-day experience sampling procedure. At 3-months follow-up, participants completed a third 7-day experience sampling procedure and online questionnaires (WHO Disability Scale, Center for Epidemiologic Studies-Depression Scale). At the end of the follow-up assessment, participants were debriefed and reimbursed. Data was collected between July 2017 and September 2020 at Freie Universität Berlin.

2.6. Statistical analysis

To examine our hypotheses, we estimated Bayesian hierarchical models using the R package brms (Bürkner, 2017), which is based on Stan (Carpenter et al., 2017). We always used the default priors of brms, which are chosen to be non- or weakly informative (Bürkner, 2017,

2018). Specifically, the priors on the overall regression coefficients were flat and the priors on the random effects SDs were data-scaled half--student-t with three degrees of freedom. For all models, we ran 4 MCMC chains, each with 1000 warmup and 4000 post-warmup samples, resulting in a total of 16,000 post-warmup samples. All computed models converged with Rhat = 1 (Vehtari et al., 2021) and estimated effective sample sizes of at least 1200. There were no divergent transitions. All independent variables are unstandardized. The exact R code of all models can be found in the online supplements.

2.6.1. Training effects on cognitive control

2.6.1.1. N-back task (close transfer). All trials with response latencies <300ms were excluded from analyses, assuming insufficient time for cognitive processing. This resulted in an average of 0.43 (*sd* = 1.03) deleted trials per person and session (i.e., 0.18 % (*sd* = 0.43%)).

To test whether the CCT group showed larger improvements in n-back accuracy from pre to post training than the placebo group, we computed a two-level (trials nested in persons) Bayesian hierarchical model with accuracy (0,1) as dependent variable. Predictor variables were Group (intervention, placebo) and Session (pre-, post-training) as well as their interaction. The intercept and the slope for Session were allowed to vary across persons with varying between-person variances allowed for the two training groups (given that we assumed the between-person variances of the slope and intercept at post training to be larger in the intervention than in the placebo group). We used a Bernoulli distribution equipped with a probit link as likelihood to model the dichotomous outcome variable.

2.6.1.2. Working memory selection task (far transfer). All trials with response latencies <300ms or longer than individuals' mean per experimental condition plus three times their interquartile range were excluded from analyses. This resulted in an average of 2.71 (*sd* = 2.05) deleted trials per person and session (i.e., 3.01% (*sd* = 2.27%)). Accuracy rates in the remaining trials were high and ranged between 89.4% and 92.5% per group and session (for details, see Table S2 in online supplements). In line with previous literature, we only included trials with correct responses for all reaction times analyses (e.g., Joormann & Gotlib, 2008).

We tested the hypothesis that the CCT group will show larger improvements in discarding negative material from working memory from pre-to post-training than the placebo group. Thus, we computed a two-level (trials nested in persons) Bayesian hierarchical model with response latencies in trials with negative probes as dependent variable. Predictor variables were Group (intervention, placebo), Session (pre-, post-training), and Experimental Condition (irrelevant, new probes), as well as their three-way interaction. The intercept and slopes for Session and Experimental Condition were allowed to vary across persons with varying between-person variances allowed for the two training groups. We used a shifted lognormal distribution as likelihood family.

2.6.2. Training effects on rumination

2.6.2.1. Rumination frequency. To test whether the CCT group showed a larger decrease in daily rumination from pre to post training (and to follow-up) as compared to the placebo group, we computed a Bayesian hierarchical model with rumination frequency as dependent variable. The three-level model accounted for the structure of the ESM data, i.e. prompts nested in days, nested in persons. Predictor variables were Group (intervention, placebo) and Session (pre-training, post-training, follow-up), and their interaction. The intercept was allowed to vary across persons and across the assessment days within persons. The slope for Session was allowed to vary across persons with different between-person variances allowed for the two training groups. Finally, we allowed a serial correlation of successive prompts within each assessment day of a person. We used a gaussian distribution as likelihood.

2.6.2.2. Valence of ruminative thoughts. This model was identical to the model for rumination frequency except for the dependent variable (valence rating instead of frequency rating).

2.6.2.3. Effect of rumination on affect. To measure the effect of rumination on subsequent affect, we calculated a dysphoric affect score (mean across items *depressed* and *sad*) and a positive affect score (mean across items *happy* and *cheerful*). Both scales showed excellent internal consistency, dysphoric affect: $\text{omega}_{\text{between-person}}$ = 0.94 (95% *CI* = [0.90, 0.97]), positive affect: $\text{omega}_{\text{between-person}}$ = 0.95 (95% *CI* = [0.93, 0.98]).

We expected that the negative effect of rumination on subsequent affect (i.e., increase in dysphoric and decrease in positive affect from time *t-1* to time *t*) will decrease more strongly from pre-to post-training (and to follow-up) in the CCT group as compared to the placebo group. Thus, we computed two three-level (prompts nested in days nested in persons) Bayesian hierarchical models with the respective affect score (dysphoric, positive) at time *t* as dependent variable. Predictor variables were the respective Affect score at time *t-1*, Group (intervention, placebo), Session (pre-training, post-training, follow-up), Rumination frequency between time *t-1* and time *t*, and the interaction between Rumination x Group x Session. We included the actual time difference between time *t-1* and *t* as predictor to control for varying time intervals between successive prompts. Affect at time *t-1* and the actual time difference between *t-1* and *t* were entered within a flexible, two-dimensional, non-linear function realized via a thin-plate spline (Wood, 2003). The intercept and slope for Affect at time *t-1* were allowed to vary across persons and across assessment days within persons. The slope for Rumination and Session and their interaction was allowed to vary across persons with different between-person variances allowed for the two training groups. We used a gaussian distribution as likelihood family.

2.6.3. Training effects on secondary clinical outcomes

2.6.3.1. Depression symptoms, level of disability, rumination[2]. To test whether the CCT group showed a larger decrease in *depression symptoms, level of disability, or RRS-SF rumination scores* from pre-to post-training, and to follow-up as compared to the placebo group, we computed four two-level (assessment sessions nested in persons) Bayesian hierarchical models. The dependent variable was the CES-D sum score, the WHODAS sum score, or the RRS-SF sum or brooding score, respectively. Predictor variables were Group (intervention, placebo) and Session (pre-training, post-training, follow-up), and their interaction. The intercept and slope for Session were allowed to vary across persons with different between-person variances allowed for the two training groups. We used a gaussian distribution as likelihood family.

2.6.3.2. Daily dysphoric affect. To test whether the CCT group showed a larger decrease in daily dysphoric affect from pre-to post-training, and to follow-up as compared to the placebo group, we computed a three-level (prompts nested in days, nested in persons) Bayesian hierarchical model with dysphoric affect as dependent variable. Predictor variables were Group (intervention, placebo) and Session (pre-training, post-training, follow-up), and their interaction. The intercept was allowed to vary across persons and across the assessment days within persons. The slope for Session was allowed to vary across persons with different between-person variances allowed for the two training groups. Finally, we

[2] Note, that the analysis on the RRS-SF scores are exploratory because the RRS-SF was not pre-registered as a secondary outcome measure

Table 1
Demographic statistics and baseline clinical characteristics.

	Total Sample (n = 64)	CCT (n = 30)	Placebo (n = 34)
Demographics			
N (%) female[1]	45 (70.31)	22 (73.33)	23 (67.65)
Age (*mean* (*SD*))	38.83 (12.74)	39.23 (13.06)	38.47 (12.65)
Education (*N* (%))			
No degree	2 (3.17)	1 (3.33)	1 (3.03)
Secondary school diploma	11 (17.46)	4 (13.33)	7 (21.21)
High school diploma	14 (22.22)	5 (16.67)	9 (27.27)
Vocational training	10 (15.87)	5 (16.67)	5 (15.15)
University degree	26 (41.27)	15 (50.00)	11 (33.33)
Baseline clinical characteristics			
BDI-II (*mean* (*sd*))	30.88 (7.14)	30.87 (7.11)	30.88 (7.28)
WHODAS (*mean* (*sd*))	33.28 (6.70)	34.05 (6.71)	32.52 (6.73)
N (%) taking antidepressants	9 (13.85)	6 (9.23)	3 (4.62)
% Comorbid Disorders (0/1/>1)	64.1/18.8/17.2	66.7/16.7/16.7	61.8/20.6/17.7
Form of Depression[2] (N single mde/recurrent/chronic)	7/31/26	4/15/11	3/16/15

Note. The range of the BDI score was 11–47. According to the BDI-II thresholds, there was one participant with low symptom severity (0–13), two participants with mild symptom severity (14–19), 21 participants with moderate symptom severity (20–28), and 40 participants with severe symptom severity (29–63). 1 = participants were asked how they identify themselves with the response options female and male; 2 = chronic depression includes full MDE symptoms for ≥2 years or current full MDE symptoms and dysphoria for ≥2 years; WHODAS = WHO Disability Assessment Schedule.

allowed a serial correlation of successive prompts within each assessment day of a person. We used a gaussian distribution as likelihood.

We consider effects clearly different from zero if the estimate's 95% credible-interval does not include zero. For directed hypotheses, we estimated the posterior probability (*PP*) that the respective effect is in the expected direction. *PP* values range from 0 to 1 with higher values implying more support for the effect going into the expected direction.

3. Results

3.1. Descriptive statistics

3.1.1. Participant characteristics

Table 1 displays demographic statistics and baseline clinical variables for the total sample and by group. Groups were similar in all demographic and baseline clinical variables. The majority of the sample was severely depressed (62% had a BDI-II ≥ 29) and showed high rumination scores (90% had an RRS-SF > 20).

3.1.2. Training adherence and compliance to experience sampling protocol

Participants completed an average of 9.84 (*SD* = 1.20) training sessions, which was similar across both groups, (M_{CCT} = 9.80 (*SD* = 1.06), $M_{placebo}$ = 9.87 (*SD* = 1.34).

Due to technical problems with the movisensXS application, an average of 1.57 percent of beeps were not presented. Participants' compliance with presented beeps was high and ranged between 82% and 87% per group and assessment period (see Table 3 for details). Three participants from the placebo group only completed six days of experience sampling. We did not impute missing data.

3.2. Cognitive control

Table 2 depicts median values and interquartile ranges for accuracy rates in the emotional n-back task and response latencies in the WMS task by session and group.

3.2.1. Close transfer

The placebo group showed a clear increase in n-back accuracy rates from pre to post training, $b_{Session\_inPlacebo} = 0.25$ (95% *CI* = [0.17, 0.33]). The CCT group also showed a clear increase in n-back accuracy rates from pre to post training, $b_{Session\_inCCT} = 0.60$ (95% *CI* = [0.44, 0.77]). In accordance with our hypothesis, the CCT group showed a clearly stronger increase in accuracy rates from pre to post training than the placebo group, $b_{SessionXGroup} = 0.35$ (95% *CI* = [0.18, 0.53], $PP(b > 0) > 0.99$).

3.2.2. Far transfer

The placebo group showed no clear increase in WMST performance (i.e., response latencies to irrelevant negative versus new negative probes) from pre to post training, $b_{SessionXRelevance\_inPlacebo} = -0.08$ (95% *CI* = [−0.15, 0.00]. Similarly, the CCT group showed no clear increase in WMST performance from pre to post training, $b_{SessionXRelevance\_inCCT} = -0.07$ (95% *CI* = [−0.14, 0.00]. Thus, contrary to our predictions, the CCT group did not show stronger improvements in WMST performance from pre to post training than the placebo group, $b_{SessionXRelevanceXGroup} = 0.00$ (95% *CI* = [−0.10, 0.10], $PP(b < 0) = 0.48$).

Additional exploratory analyses showed that a training specific effect on WMST performance was also not present, when using (a) response latencies to irrelevant and new positive probes as outcome variable, or (b) accuracy rates instead of response latencies (for exact results see html markdown file at OSF).

Table 2
Median and IQR for n-back accuracy rates and WMST response latencies for the total sample and by group and session.

	CCT		Placebo	
	Pre-training N = 30	Post-training N = 29	Pre-training N = 33	Post-training N = 29
Accuracy rates n-back task (%)	78.4 (7.29)	92.5 (8.33)	80.4 (15.9)	84.6 (15.4)
Response latencies WMST (ms)				
Irrelevant negative probes	1475 (706)	1100 (433)	1450 (317)	1283 (383)
New negative probes	1008 (397)	767 (308)	1042 (434)	900 (283)
Irrelevant positive probes	1666 (560)	1150 (334)	1383 (542)	1283 (333)
New positive probes	1075 (426)	866 (283)	1033 (424)	975 (284)

Table 3
Descriptive statistics for primary and secondary clinical outcomes by group and session.

	Placebo Group			CCT Group		
	Pre-training	Post-training	Follow-up	Pre-training	Post-training	Follow-up
Rumination frequency	2.74 (95% *CI* [2.37, 3.11])	2.73 (95% *CI* [2.32, 3.15])	2.47 (95% *CI* [2.03, 2.93])	2.73 (95% *CI* [2.25, 3.22])	2.54 (95% *CI* [2.08, 3.03])	2.50 (95% *CI* [1.87, 3.14])
Rumination valence	2.94 (95% *CI* [2.52, 3.34])	2.77 (95% *CI* [2.31, 3.22])	2.53 (95% *CI* [2.07, 3.02])	2.92 (95% *CI* [2.48, 3.37])	2.53 (95% *CI* [2.05, 3.01])	2.40 (95% *CI* [1.85, 2.95])
Dysphoric affect	3.79 (95% *CI* [3.38, 4.18])	3.67 (95% *CI* [3.24, 4.10])	3.42 (95% *CI* [2.98, 3.89])	3.87 (95% *CI* [3.48, 4.27])	3.48 (95% *CI* [3.07, 3.89])	3.21 (95% *CI* [2.68, 3.74])
Technical loss[1] in % (*SD*)	1.37 (4.74)	3.30 (8.79)	1.56 (4.13)	0.48 (2.52)	1.37 (3.41)	1.71 (3.00)
Compliance in % (*SD*)	85.59 (16.98)	86.90 (10.46)	82.17 (15.62)	84.96 (12.13)	81.89 (15.28)	84.08 (13.29)
CES-D scores *M* (*SD*)	33.99 (7.63)	30.97 (8.98)	27.84 (8.08)	36.42 (7.29)	31.77 (8.75)	29.48 (11.22)
WHODAS scores *M* (*SD*)	32.52 (6.73)	28.27 (6.54)	27.99 (7.62)	34.05 (6.71)	32.17 (8.11)	28.32 (8.24)
RRS-SF sum scores *M* (*SD*)	25.70 (4.90)	25.10 (3.99)	25.20 (4.12)	26.17 (5.40)	25.00 (4.92)	25.50 (5.23)

1 = Some beeps were not presented due to technical problems with the movisensXS App. Note, that values for rumination frequency, rumination valence, and dysphoric affect represent estimated means from the Bayesian hierarchical models. The rating scales for rumination frequency and valence of ruminative thoughts ranged from 0 to 6; 95% *CI* = 95% credible interval; CES-D = Center for Epidemiological Studies Depression Scale; WHODAS = World Health Organization Disability Assessment Schedule, RRS-SF = Ruminative Responses Scale – Short Form.

3.3. Rumination

Table 3 and Fig. 2a/b display estimated mean values for rumination frequency and valence of ruminative thoughts during the 7-day ESM period by group and session.

3.3.1. Rumination frequency

In the placebo group, there was no change in rumination frequency from pre to post training, $b_{SessionPost\_inPlacebo} = -0.01$ (95% *CI* = [−0.30, 0.29]) and only a slight decrease from pre to follow-up, which was not clearly different from zero, $b_{SessionFU\_inPlacebo} = -0.27$ (95% *CI* = [−0.56, 0.03]). The CCT group experienced a slight decrease in rumination frequency from pre to post training and almost no change from post to follow-up. However, neither the decrease in rumination from pre to post training, $b_{SessionPost\_inCCT} = -0.18$ (95% *CI* = [−0.47, 0.09]), nor from pre to follow-up, $b_{SessionFU\_inCCT} = -0.23$ (95% *CI* = [−0.62, 0.17]), was clearly different from zero.

Contrary to our hypothesis, the decrease in rumination frequency from pre to post training was not clearly larger in the CCT group as compared to the placebo group, $b_{SessionPostXGroup} = -0.18$ (95% *CI* = [−0.59, 0.22], *PP*($b < 0$) = 0.81)[3]. Similarly, the decrease in rumination frequency from pre-training to follow-up was not clearly larger in the CCT group than in the placebo group, $b_{SessionFUXGroup} = 0.04$ (95% *CI* = [−0.47, 0.54], *PP*($b < 0$) = 0.44).

Please note, that we ran an additional exploratory analysis to examine whether there was a training specific-effect on ruminative thoughts rated as at least moderately negative. For this purpose, we included only those prompts where participants rated their ruminative thoughts as at least moderately negative (i.e., negativity rating ≥2). However, results did not change (see online supplements for details). We also computed an exploratory analysis on the combined frequency and negativity item (rumination frequency x negativity). High scores on this outcome measure reflect a high frequency of very negative ruminative thoughts. Again, results did not change (see online supplements for details).

3.3.2. Valence of ruminative thoughts

Participants in the placebo group rated their ruminative thoughts at post training slightly less negative than at pre-training, but this decrease was not clearly different from zero, $b_{SessionPost\_inPlacebo} = -0.17$ (95% *CI* = [−0.41, 0.08]). Participants in the placebo group experienced a clear decrease in the negative valence of ruminative thoughts from pre-training to follow-up, $b_{SessionFU\_inPlacebo} = -0.41$ (95% *CI* = [−0.73, −0.09]). The CCT group rated their ruminative thoughts clearly less negative at post-training, $b_{SessionPost\_inCCT} = -0.39$ (95% *CI* = [−0.61, −0.17]), and follow-up, $b_{SessionFU\_inCCT} = -0.52$ (95% *CI* = [−0.79, 0.26]), compared to pre-training.

Nevertheless, and contrary to our hypothesis, the decrease in the negative valence of ruminative thoughts from pre to post training was not clearly larger in the CCT group as compared to the placebo group, $b_{SessionPostXGroup} = -0.22$ (95% *CI* = [−0.55, 0.10], *PP*($b < 0$) = 0.91). Similarly, the decrease in the negative valence of ruminative thoughts from pre-training to 3-months follow-up was not clearly larger in the CCT group as compared to the placebo group, $b_{SessionPostXGroup} = -0.11$ (95% *CI* = [−0.53, 0.30], *PP*($b < 0$) = 0.71).

3.3.3. Effect of rumination on subsequent dysphoric affect

Prior to the intervention, rumination clearly increased subsequent dysphoric affect in the placebo group, $b_{Rum\_inPlacebo} = 0.23$ (95% *CI* = [0.16, 0.30]), as well as in the CCT group, $b_{Rum\_inCCT} = 0.24$ (95% *CI* = [0.18, 0.31]). After the intervention, rumination still clearly increased subsequent dysphoric affect in both, the placebo group, $b_{RumxSessionPost\_inPlacebo} = 0.27$ (95% *CI* = [0.19, 0.35]), and the CCT group, $b_{RumxSessionPost\_inCCT} = 0.27$ (95% *CI* = [0.20, 0.34]). Similarly, at 3-month follow-up, rumination still clearly increased subsequent dysphoric affect in both, the placebo group, $b_{RumxSessionFU\_inPlacebo} = 0.14$ (95% *CI* = [0.04, 0.23]), and the CCT group, $b_{RumxSessionFU\_inCCT} = 0.25$ (95% *CI* = [0.16, 0.33]). Thus, contrary to our hypothesis, the negative effect of rumination on subsequent dysphoric affect did not decrease more strongly from pre to post training in the CCT group as compared to the placebo group, $b_{RumXSessionPostXGroup} = -0.01$ (95% *CI* = [−0.11, 0.09], *PP*($b < 0$) = 0.58). It also did not decrease more strongly from pre-training to follow-up in the CCT group as compared to the placebo group, $b_{RumXSessionFUXGroup} = 0.01$ (95% *CI* = [−0.03, 0.23], *PP*($b < 0$) = 0.07).

3.3.4. Effect of rumination on subsequent positive affect

Prior to the intervention, rumination clearly decreased subsequent

[3] Please note that the results remained the same when excluding six participants with low RRS-SF scores (<20) at baseline (see online supplements for details)

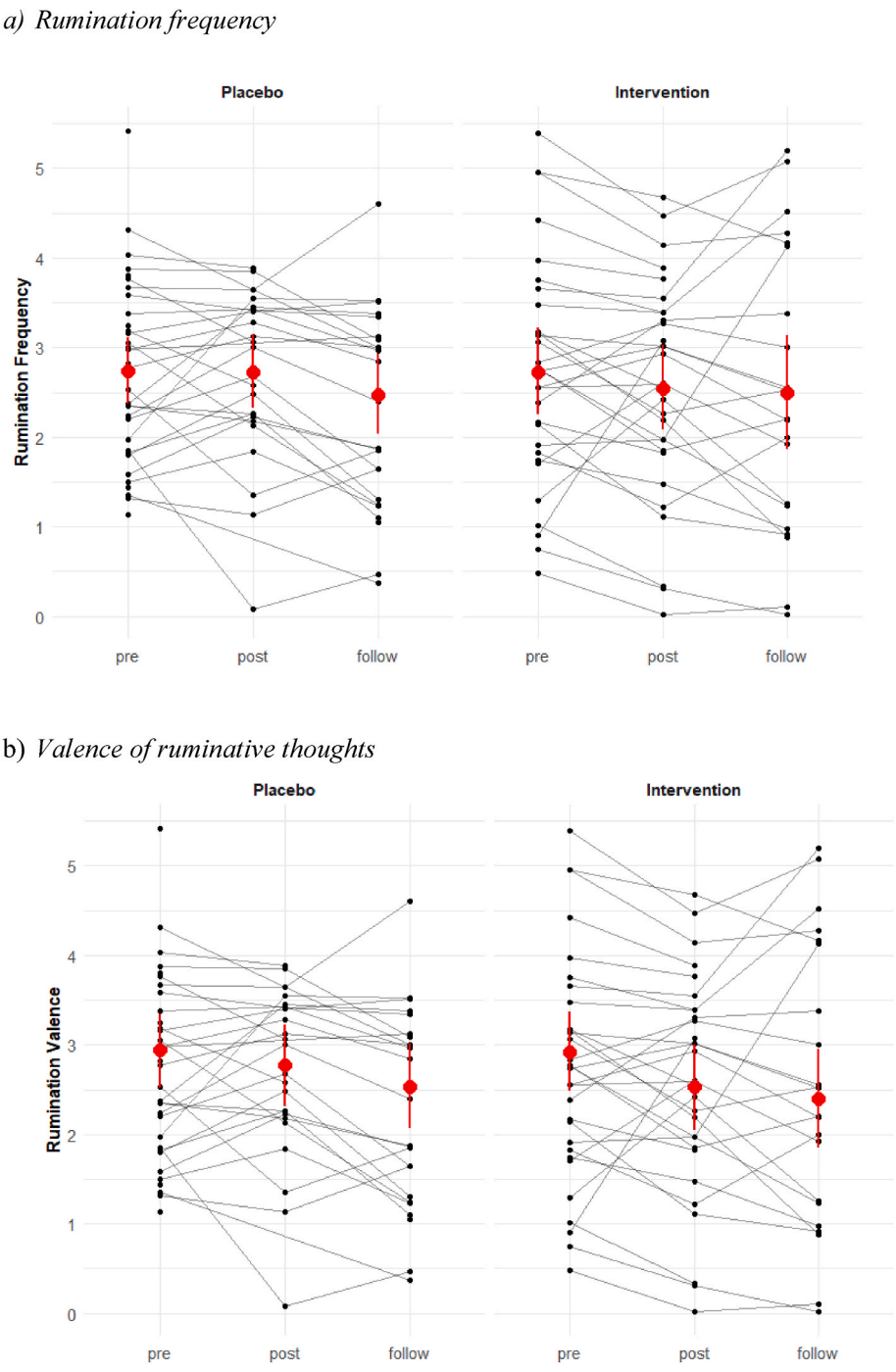

Fig. 2. Rumination frequency and valence at pre, post, and follow-up assessment.

positive affect in the placebo group, $b_{Rum\_inPlacebo} = -0.10$ (95% $CI = [-0.16, -0.05]$), as well as in the CCT group, $b_{Rum\_inCCT} = -0.12$ (95% $CI = [-0.16, -0.07]$). After the intervention, rumination still clearly decreased subsequent positive affect in both, the placebo group, $b_{RumxSessionPost\_inPlacebo} = -0.08$ (95% $CI = [-0.15, -0.01]$), and the CCT group, $b_{RumxSessionPost\_inCCT} = -0.14$ (95% $CI = [-0.02, -0.08]$). Similarly, at 3-month follow-up, rumination clearly decreased subsequent positive affect in both the placebo group, $b_{RumxSessionFU\_inPlacebo} = -0.10$ (95% $CI = [-0.18, -0.04]$), and the CCT group, $b_{RumxSessionFU\_inCCT} = -0.14$ (95% $CI = [-0.21, -0.08]$). Thus, contrary to our hypothesis, the negative effect of rumination on subsequent positive affect did not decrease more strongly from pre to post training in the CCT group as compared to the placebo group, $b_{RumxSessionPostXGroup} = -0.05$ (95% $CI = [-0.14, 0.04]$, $PP(b > 0) = 0.15$). It also did not decrease more strongly from pre-training to 3-month follow-up in the CCT group as compared to the placebo group, $b_{RumxSessionFUXGroup} = -0.02$ (95% $CI = [-0.12, 0.07]$, $PP(b > 0) = 0.07$).

3.4. Secondary clinical outcomes and exploratory analyses

Table 3 displays means and standard deviations for depressive symptoms (CES-D scores), level of disability (WHODAS scores), RRS-SF rumination scores, and daily dysphoric affect by group and session.

3.4.1. Depressive symptoms

In the placebo group, depressive symptoms decreased clearly from

pre to post training, $b_{SessionPost\_inPlacebo} = -3.20$ (95% $CI = [-6.18, -0.17]$), and from pre-training to follow-up, $b_{SessionFU\_inPlacebo} = -5.73$ (95% $CI = [-8.91, -2.60]$). In the CCT group, depressive symptoms also decreased clearly from pre to post training, $b_{SessionPost\_inCCT} = -4.64$ (95% $CI = [-7.54, -1.73]$), and from pre-training to follow-up, $b_{SessionFU\_inCCT} = -7.03$ (95% $CI = [-11.33, -2.76]$). Contrary to our hypothesis, depressive symptoms did not decrease more strongly in the CCT than the placebo group, pre to post: $b_{SessionPostXGroup} = -1.44$ (95% $CI = [-5.66, 2.75]$, $PP(b < 0) = 0.75$), pre to follow-up: $b_{SessionFUXGroup} = -1.31$ (95% $CI = [-6.61, 4.02]$, $PP(b < 0) = 0.69$).

3.4.2. Level of disability

In the placebo group, the level of disability decreased clearly from pre to post training, $b_{SessionPost\_inPlacebo} = -4.60$ (95% $CI = [-7.40, -1.71]$), and from pre-training to follow-up, $b_{SessionFU\_inPlacebo} = -4.43$ (95% $CI = [-7.37, -1.41]$). In the CCT group, level of disability did not decrease clearly from pre to post training, $b_{SessionPost\_inCCT} = -1.87$ (95% $CI = [-5.16, 1.39]$), but from pre-training to follow-up, $b_{SessionFU\_inCCT} = -5.01$ (95% $CI = [-8.56, -1.44]$). Contrary to our hypothesis, the level of disability did not decrease more strongly in the CCT group than in the placebo group, pre to post: $b_{SessionPostXGRoup} = 2.73$ (95% $CI = [-1.60, 7.06]$, $PP(b < 0) = 0.11$), pre to follow-up: $b_{SessionFUXGroup} = -0.58$ (95% $CI = [-5.27, 4.07]$, $PP(b < 0) = 0.60$).

3.4.3. Daily dysphoric affect

In the placebo group, daily dysphoric affect did not decrease clearly from pre-to post-training, $b_{SessionPost\_inPlacebo} = -0.12$ (95% $CI = [-0.37, 0.14]$), but from pre-training to follow-up, $b_{SessionFU\text{-}inPlacebo} = -0.37$ (95% $CI = [-0.73, -0.01]$). In the CCT group, dysphoric affect decreased clearly from pre-to post-training, $b_{SessionPost\_inCCT} = -0.40$ (95% $CI = [-0.66, -0.13]$), and from pre-training to follow-up, $b_{SessionFU\_inCCT} = -0.66$ (95% $CI = [-1.07, -0.26]$). However, contrary to our hypothesis, dysphoric affect did not decrease more strongly in the CCT than in the placebo group, pre to post: $b_{SessionPostXGroup} = -0.28$ (95% $CI = [-0.64, 0.09]$, $PP(b < 0) = 0.93$), pre to follow-up: $b_{SessionFUXGroup} = -0.29$ (95% $CI = [-0.83, 0.26]$, $PP(b < 0) = 0.86$).

3.4.4. RRS-SF rumination scores (exploratory)

RRS-SF sum scores and RRS-SF brooding scores did not decrease clearly from pre-to post-training nor from pre-training to follow-up in either group (see online supplements for detailed results). Furthermore, RRS-SF sum scores did not decrease more strongly in the CCT than the placebo group, pre to post: $b_{SessionPostXGroup} = -0.38$ (95% $CI = [-2.80, 1.98]$), pre to follow-up: $b_{SessionFUXGroup} = 0.22$ (95% $CI = [-1.91, 2.34]$). Similarly, RRS-SF brooding scores did not decrease more strongly in the CCT than the placebo group, pre to post: $b_{SessionPostXGroup} = 0.04$ (95% $CI = [-1.23, 1.30]$), pre to follow-up: $b_{SessionFUXGroup} = 0.47$ (95% $CI = [-0.82, 1.75]$).

4. Discussion

Rumination is one of the most important risk factors for the onset and recurrence of depressive episodes and hard to tackle in psychotherapy. The present study was the first randomized-controlled trial examining whether cognitive control training reduces ruminative thoughts in the daily lives of individuals with a current major depressive disorder. Results show that cognitive control training did lead to a clearly stronger increase in cognitive control ability than the placebo training, however only in a task similar to the training (i.e., task-specific transfer). There was no transfer of the training effect to a training-unlike cognitive control task. In addition, cognitive control training did not lead to a greater reduction in daily rumination frequency, negativity of ruminative thoughts, or the negative influence of rumination on subsequent affect than placebo training. There was also no training-specific effect on participants' depressive symptomatology or health impairment.

The training-specific effect on the non-adaptive n-back task in the current study is in accordance with previous findings. Most studies examining the effect of cognitive control training on depression or rumination found training-specific improvements in cognitive control tasks similar to the trained task (e.g., Hoorelbeke et al., 2021; Hoorelbeke & Koster, 2017; Vervaeke et al., 2021). Importantly, however, the absence of a transfer effect on an untrained cognitive control task is also consistent with previous findings. Indeed, hardly any previous study examining the effect of diverse cognitive control trainings on rumination or depression was able to find considerable training-specific improvements in training-unlike cognitive control tasks (e.g., Ferrari et al., 2021; Hoorelbeke et al., 2015; Iacoviello et al., 2014, 2018; Vervaeke et al., 2020; Wanmaker et al., 2015). Thus, it is unclear whether cognitive control training is able to improve cognitive control beyond task-specific training effects. It is important to note that, nonetheless, several of these studies did find training-specific effects on rumination or depression (Hoorelbeke et al., 2015; Iacoviello et al., 2014, 2018). This raises key questions about the mechanisms of action of the implemented cognitive control training. Are other skills being trained that have a positive impact on rumination and depression? Or did an improvement in cognitive control mechanisms take place, but could not be detected in the selected transfer tasks? Indeed, different tasks assessing cognitive control functions often do not correlate well because of low reliability or task impurity. Friedman and Miyake (2017) hence advice to measure cognitive control ability by a latent variable approach. Latent variables reflect common variance across multiple assessment tasks. As such, they are free of random measurement error or variance due to task-specific skills. Thus, one way to overcome the task impurity problem is by assessing cognitive control ability by multiple tasks and using latent growth analyses to detect training-specific improvements in cognitive control.

The present study did not find training-specific effects on the frequency or negativity of ruminative thoughts, or the negative influence of rumination on subsequent affect. Importantly, the present null findings were very robust. Specifically, there was also no training-specific effect on the frequency of daily ruminative thoughts when only those ruminative thoughts rated as negative were considered. Thus, the possibility that the present ESM rumination frequency item may have also captured non-depressive rumination did not account for the present null findings. In addition, there was no training-specific effect on daily ruminative thoughts when six participants with rumination scores below the usual cut-off for high ruminators were excluded. Finally, exploratory analyses on the RRS-SF sum and brooding score mirrored the null findings on the ESM rumination items. The robustness of the present null findings, combined with the methodological strengths of the present study (i.e., clinical sample, assessment of rumination in participants' daily lives, emotional training stimuli), suggest, that training currently depressed individuals high in rumination to update emotional content in working memory does not have any effect on the frequency or negativity of their daily ruminative thoughts. This finding joins numerous other studies failing to find an effect of different types of cognitive control training on rumination in different samples (Ferrari et al., 2021; Hoorelbeke et al., 2021; Jopling et al., 2020; Lass et al., 2021; Moshier & Otto, 2017; Onraedt & Koster, 2014; Van den Bergh et al., 2020; Vanderhasselt et al., 2021; Vervaeke et al., 2020, 2021; Wanmaker et al., 2015).

There are some potential limitations of the present study design that need to be discussed. First, the present training task relied on emotional face pictures whereas rumination is a verbal process. Thus, it is possible that a training task using verbal material may have been able to reduce rumination. Interestingly, Jopling et al. (2020) recently employed an adaptive version of the modified emotional Sternberg task as cognitive control training. This task is an adaptive version of the far transfer task used in the present study and uses emotional words as training stimuli. However, this task also failed to produce an effect on participants' level of rumination as assessed by the RRS (Jopling et al., 2020). This speaks against the argument that a mismatch between the pictorial training stimuli and the verbal outcome measure is responsible for the present null results.

Second, one might argue that severely depressed individuals may not be able to benefit from cognitive control training and that cognitive control training may be more effective as a preventive intervention for non-depressed individuals high in rumination. However, the evidence that we have so far does not support this hypothesis. Specifically, training studies with non-depressed high ruminators produced positive and null results in a similar ratio (Daches & Mor, 2014; Hoorelbeke et al., 2015; versus Onraedt & Koster, 2014 (two samples)) as training studies with clinically depressed samples (Iacoviello et al., 2014; Siegle et al., 2007, 2014; versus Ferrari et al., 2021; Jopling et al., 2020; Moshier & Otto, 2017).

Finally, the a priori sample size determination was based on expected effect sizes that seemed realistic at the time the study was designed. Given the increasing number of studies that have since found no effect of cognitive control training on rumination, our estimate of change at that time may have been too optimistic. As a result, the present sample size may be too small to detect smaller effects. However, it is important to keep in mind that cognitive control training showing very small effects on rumination is not clinically useful.

Finally, the present study did not find greater reductions in dysphoric affect or depressive symptoms in the training as compared to the placebo group. Earlier studies on the effect of cognitive control training on depressive symptoms also produced mixed results (e.g., Calkins et al., 2015; Hoorelbeke et al., 2022; Iacoviello et al., 2018; versus e.g., Ferrari et al., 2021; Hoorelbeke et al., 2023; Vervaeke et al., 2021; Jopling et al., 2020). Interestingly, the present null results are at odds with training-specific effects on depressive symptoms in two studies applying the same training task in clinically depressed individuals (Iacoviello et al., 2014, 2018). It is important to note, however, that the latter studies were based on relatively small samples. In addition, the present sample included a high percentage of individuals with chronic forms of depression (41%). Chronic depression was an exclusion criteria in at least one of the studies by Iacoviello et al. (2014). Although the symptom severity in the current sample and both Iacoviello samples was comparable, it is possible that individuals with chronic depression suffer from cognitive impairments that are more resistant to change, potentially requiring more intensive training procedures. Thus, the generalizability of these results is restricted to severely depressed individuals.

In sum, the present randomized controlled trial found no evidence for the effectiveness of a two-week online cognitive control training on ruminative thoughts or depressive symptoms in the daily lives of clinically depressed individuals. Due to its high-quality design including daily assessment of outcome variables, a follow-up assessment, and a clinical sample, the current study makes an important contribution to this lively area of research.

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Ulrike Zetsche: Conceptualization, Formal analysis, Funding acquisition, Supervision, Validation, Writing – original draft, Writing – review & editing. **Pauline Neumann:** Formal analysis, Investigation, Project administration, Writing – original draft, Writing – review & editing. **Paul-Christian Bürkner:** Formal analysis, Supervision, Validation, Writing – review & editing. **Babette Renneberg:** Conceptualization, Funding acquisition, Writing – review & editing. **Ernst H.W. Koster:** Conceptualization, Supervision, Writing – review & editing. **Kristof Hoorelbeke:** Conceptualization, Supervision, Writing – review & editing.

Declaration of competing interest

None. None of the authors have any financial or personal interest that could affect their objectivity.

Data availability

We provide the de-individualized and preprocessed dataset and the statistical code for all analyses at Open Science Framework (OSF): https://osf.io/7cdmw/?view_only=41de767b44a143e6937fea5e3219ab92.

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Appendix A. Supplementary data

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