

Original Paper

# Transdiagnostic Cognitive Control Training for Patients Waiting for Outpatient Psychotherapy: Randomized Clinical Trial

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## Abstract

**Background:** Various mental disorders are associated with impaired cognitive control, which is crucial for effective emotion regulation. Cognitive control training has demonstrated promise in enhancing emotion regulation and alleviating distress in disorders characterized by repetitive negative thinking, such as depression and anxiety.

**Objective:** Given the importance of cognitive control and emotion regulation across mental disorders, this study investigates the efficacy of a mobile cognitive control training in a transdiagnostic outpatient sample awaiting psychotherapy.

**Methods:** In this randomized clinical superiority trial with 2 parallel arms, 80 patients with various mental disorders from an outpatient waiting list received either 10 sessions of mobile cognitive control training using the Paced Auditory Serial Addition Test (PASAT) or an active control training using a speed of response task. The primary outcome was mental distress, measured by the Hopkins Symptom Checklist-11 (HSCL-11). Secondary outcomes included measures of cognitive control, rumination, repetitive negative thinking, difficulties in emotion regulation, cognitive emotion regulation, and disorder-specific symptoms. Outcomes were measured at baseline, post training, and at 3-month and 6-month follow-up.

**Results:** Contrary to our primary hypothesis, cognitive control training was not superior in improving global mental distress directly after training ( $B=-.03$ , 95% CI  $-0.21$ ,  $0.16$ ;  $t_{179.60}=-0.26$ ;  $P=.80$ ;  $d=-0.04$ , 95% CI  $-0.35$ ,  $0.28$ ); however, it led to greater improvements in cognitive control ( $B=-0.56$ , 95% CI  $-0.59$ ,  $-0.54$ ;  $z=-18.02$ ;  $P<.001$ ;  $d=-1.23$ , 95% CI  $-1.30$ ,  $-1.20$ ). This effect was similar at the 3-month and 6-month follow-up. Furthermore, at 3-month follow-up, cognitive control training resulted in fewer difficulties in emotion regulation ( $B=4.73$ , 95% CI  $0.52$ ,  $9.12$ ;  $t_{177.99}=2.09$ ;  $P=.04$ ;  $d=0.34$ , 95% CI  $0.04$ ,  $0.65$ ), and anxiety symptoms ( $B=2.94$ , 95% CI  $0.38$ ,  $5.82$ ;  $t_{66.51}=2.09$ ;  $P=.04$ ;  $d=0.70$ , 95% CI  $0.09$ ,  $1.38$ ), although the latter refers to a small subsample of patients with anxiety disorders. At 6-month follow-up, cognitive control training led to more adaptive cognitive emotion regulation ( $B=-5.18$ , 95% CI  $-9.74$ ,  $-0.41$ ;  $t_{180.90}=-2.16$ ;  $P=.03$ ;  $d=-0.40$ , 95% CI  $-0.75$ ,  $-0.03$ ), and less social anxiety ( $B=2.00$ , 95% CI  $0.14$ ,  $3.81$ ;  $t_{43.43}=2.08$ ;  $P=.04$ ;  $d=0.66$ , 95% CI  $0.05$ ,  $1.24$ ). The groups did not differ in any other outcome at any point in time.

**Conclusions:** This study is the first to assess the efficacy of a mobile cognitive control training using the PASAT in a transdiagnostic outpatient sample. There was no evidence for the training's efficacy on global mental distress and only weak evidence for the superiority in measures of emotion regulation and anxiety at follow-ups. Potential mediating pathways and moderating factors, such as the number of training sessions, should be investigated in larger studies.

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**Keywords:** cognitive control; emotion regulation; mHealth; eHealth; transdiagnostic approach

## Introduction

### Background

Mental disorders are a major contributor to global disability and are associated with increased mortality, somatic comorbidities, and substantial socioeconomic costs [1,2]. While meta-analyses confirm the efficacy of psychotherapy and pharmacotherapy [3,4], effect sizes remain modest and relapse rates are high [5], underscoring the need for more effective treatments.

Frameworks such as the Research Domain Criteria (RDoC) propose a transdiagnostic approach to investigating shared risk factors among mental disorders [6,7]. Emotion regulation is one such factor implicated in the onset, maintenance, and relapse of various disorders [8-10]. Emotion regulation refers to the process by which individuals modulate the quality, intensity, and timing of their emotional responses [11]. Crucially, emotion regulation skills predict symptom improvement across disorders and treatments [12-17], and emotion regulation is targeted by many psychotherapeutic interventions [18-20].

Since Ochsner and Gross's foundational work [21,22], research has increasingly focused on to what extent emotion regulation depends on cognitive control [23-25]. Cognitive control refers to a set of interrelated processes, largely mediated by the prefrontal cortex, that support goal-directed behavior [26,27]. These processes include shifting (flexibly switching between tasks or mental sets), inhibition (suppressing automatic or prepotent responses), and updating (maintaining and renewing relevant information in the working memory while discarding irrelevant material) [28]. A multitude of meta-analyses have shown that numerous mental disorders are associated with reduced cognitive control, covering all domains of shifting, inhibition, and updating (see [29] for a review of meta-analyses).

Notably, impairments in cognitive control, especially difficulties with inhibition and working memory updating, have been associated with maladaptive emotion regulation strategies [23,30-34]. As outlined in the impaired disengagement hypothesis [35], rumination serves as an illustrative case of how cognitive control impairments contribute to maladaptive emotion regulation. According to this hypothesis, individuals with impaired cognitive control struggle to divert their attention and focus from negative (self-referential) information in working memory. This prolongs a ruminative inward focus on the negative material that increases negative affect and thus lowers the threshold for additional negative information to enter working memory [35]. This cycle of increased rumination, qualified by impaired cognitive control, has been supported by meta-analytic findings on the links between rumination and difficulties in discarding no longer relevant material from working memory [36].

Given the links between impaired cognitive control and maladaptive emotion regulation, interventions that enhance

cognitive control may offer a promising avenue for alleviating mental distress rooted in such dysfunctional emotion regulation. One commonly used approach to improve cognitive control is based on training using the Paced Auditory Serial Addition Test (PASAT) [37,38]. In the PASAT, participants hear a continuous stream of single-digit numbers ranging from 1 to 9. They are instructed to continuously add and respond to the sum of the last 2 numbers they heard. For example, if they last heard the numbers 2 and 6, the correct answer is 8. If they hear the number 9 next, the correct answer then is 15 (because 6+9=15). This requires them to constantly update their working memory by replacing the next-to-last heard number with the most recently heard number to compute the updated sum. Furthermore, the PASAT requires inhibition, since participants have to suppress responses to the no longer relevant numbers and have to inhibit immediate responses to the current number (before summing the numbers up). As such, the PASAT engages cognitive control processes such as working memory updating and inhibition [28].

PASAT-based cognitive control training has shown efficacy in reducing symptoms of depression and maladaptive emotion regulation, such as rumination, in individuals with current depressive episodes [39-43], those at elevated risk [44], and patients in remission [45-47]. In these intervention studies, the PASAT has frequently been compared to an active control condition that focused on speed of responses (ie, respond immediately to the last heard number) and thus does not target core cognitive control processes such as updating of working memory [48]. However, the benefits of the training have not been observed in nonclinical samples of healthy students [49-51], older adults [52], or high trait ruminators [53]. Moreover, PASAT-based training has not shown an additive benefit when combined with behavioral activation [54] or when used as an adjunct to inpatient treatment [55]. Thus, while promising for clinical populations, its effectiveness appears limited in nonclinical samples and may be limited when combined with other treatments.

### Objectives

Most intervention studies using the PASAT have focused primarily on individuals with depression or an increased vulnerability for depression. This may have overlooked the broader applicability of cognitive control training across the spectrum of mental disorders. Given the well-documented impairments in cognitive control and maladaptive emotion regulation across various mental disorders [8,29], this study investigates the efficacy of PASAT-based cognitive control training as a transdiagnostic intervention in a heterogeneous clinical sample waiting for psychotherapy in an outpatient clinic. Our primary hypothesis is that participants receiving PASAT-based cognitive control training will show a significantly greater reduction in mental distress from baseline to post training (primary end point). This effect is expected to exceed that of an active control condition, which emphasizes response speed but does not target cognitive control processes. Based on previous evidence for the

intervention's efficacy in reducing rumination in individuals with depression [48], we further hypothesize that cognitive control training will lead to greater improvements in emotion regulation compared to the control condition. However, as a narrow focus on rumination may miss broader emotion regulation impairments across diagnoses [56], we will assess this outcome using multiple measures, including a repetitive negative thinking style, general difficulties in emotion regulation, and cognitive emotion regulation strategies. To explore the broader functional significance of training-induced improvements, we will also examine effects on work and social adjustment and quality of life. Finally, to capture potential improvements at the symptom level, we additionally examine changes in disorder-specific symptoms. To assess potential longer-term effects of the training, follow-up assessments are conducted not only post training but also at 3 and 6 months.

## Methods

### Design

Participants were randomly assigned (1:1 ratio) to receive either a cognitive control training or an active control training (ie, a training that focused on response speed). Randomization (without stratification) was carried out automatically with the *psych* package, version 2.2.9 [57] directly before the first training session. We used the online survey framework formr [58] throughout the entire study to conduct the assessments and deliver the interventions. The computerized allocation to training conditions was concealed from the study personnel. Only after a phone call at the time when half of the training sessions were completed (intended to enhance engagement following the first 5 training sessions, see section “Treatments”) could participants reveal their condition to the study personnel. Importantly, the study personnel never inquired about details of the participants’ training procedures that could disclose their conditions. Upon recruitment, participants were blind to the nature of the different training conditions. They were informed that during the training, they would engage in a task that demands their concentration. After randomization, they only received specific information about their respective condition. All instructions were written and standardized. All outcomes were either self-report measures or computerized tasks, so that no masking of assessors was required. Assessments of baseline, post training, 3-month, and 6-month follow-up data of all self-report questionnaires were conducted independently by the patients through formr. Training and assessment tasks were programmed using JsPsych [59] and JATOS [60]. This trial was conducted in line with the CONSORT (Consolidated Standards of Reporting Trials) guidelines (see Checklist 1).

### Participants

Between May 2021 and November 2023, a total of 80 patients were recruited from the waiting list of the outpatient psychotherapy clinic at the Institute of Psychology of the University of Göttingen, Germany, which offers cognitive behavioral therapy. Forty patients (50%) were randomly

assigned to the cognitive control training and 40 patients to the speed of response training (active control). Patients were initially made aware of the study with the help of leaflets upon registering for the waiting list of the treatment facility. Patients were eligible for the study if they were between 18 and 65 years old and met the diagnostic criteria for one or more of the following mental disorders that had been associated with cognitive control impairments [29]: major depressive disorder (including recurrent or persistent depressive disorder), agoraphobia, panic disorder, social anxiety disorder (SAD), specific phobia, generalized anxiety disorder, obsessive-compulsive disorder, posttraumatic stress disorder, somatic symptom disorder, eating disorder (anorexia nervosa, bulimia nervosa, and binge-eating disorder), substance use disorder (abstinent for at least 3 months), borderline personality disorder, or adjustment disorder. To maximize external validity, exclusion criteria were kept at a minimum: nonabstinent substance use disorder, severe psychiatric comorbidity (eg, schizophrenia), medical instability, dementia, acute suicidality, or insufficient knowledge of the German language to understand the questionnaires and instructions. Diagnostic eligibility was assessed according to the *DSM-5 (Diagnostic and Statistical Manual of Mental Disorders [Fifth Edition])* [61], using the *Diagnostic Interview for Mental Disorders* [62]. Screening of adjustment disorder was conducted using the relevant section from the *Structured Clinical Interview for DSM-5 Disorders* [63], since this diagnosis is not covered by the *Diagnostic Interview for Mental Disorders*. Interviews were conducted by clinical psychology graduate students with master’s degrees, trained and supervised by a licensed psychotherapist with extensive experience in both interview formats.

Sample size was determined by an a priori power analysis using G\*Power [64]. Based on previous findings in a similar study [45], this study was powered to detect a moderate within-between (time x training group) interaction effect of  $f=0.25$  in a repeated-measures design for the primary hypothesis that the cognitive control training would reduce global mental distress from baseline to posttraining assessment significantly more than the speed of response training. With  $\alpha=.05$ , statistical power  $1-\beta=.95$ , and while accounting for an approximate 25% of data loss, a minimum sample size of 72 would have been sufficient, which was conservatively rounded up to 80, following recommendations for pilot trials [65].

### Treatments

#### Overview

Participants performed 10 online training sessions of either the cognitive control training or the speed of response training over the course of 2 weeks. They conducted these sessions independently using their smartphones via a web browser, enabling a flexible and remote completion of the intervention. According to meta-analytic findings, conducting cognitive control training remotely instead of in a laboratory setting does not negatively affect its efficacy [66]. Additionally, remote training provides a more ecologically valid context. Immediately before the first training session, both

groups watched a standardized psychoeducative video that was specifically created for this study and that aimed to increase task engagement [41]. For both conditions, the video highlighted that participating in a cognitive training with a demanding task necessitates sustained attention and focus even if frustrating negative thoughts may emerge. The video and its transcript can be found on the project's Open Science Framework page [67]. After completing the first 5 training sessions, participants received a short phone call from the study personnel with the purpose of motivating participants to complete the remaining 5 sessions. During the call, participants' completion of the first half was positively acknowledged, and they were encouraged to finish the second half. The call also included a discussion on overcoming potential obstacles and improving conditions for training completion. For example, advice was given on training in a private, quiet environment and using headphones to minimize any distraction from the task.

## Cognitive Control Training

Participants in the cognitive control training group underwent 10 training sessions, each lasting 20 minutes, using an adaptive version of the PASAT (aPASAT) that has been used in cognitive control training studies before [45,49]. During training, participants hear a continuous stream of single digits (1-9) and must calculate and respond with the sum of the 2 most recently presented digits, selecting the correct answer from options on the screen ranging from 1 to 18. To tailor the training to the individual's performance level, the speed of digit presentation was adjusted dynamically. The interstimulus interval (ISI) between 2 digits began at 3000 milliseconds for each training session and was decreased or increased by 100 milliseconds after every sequence of 4 consecutive correct or incorrect responses, respectively. Besides an initial set of 8 practice trials to familiarize participants with the task, each training session consisted of 400 trials. Consistent with prior research, training progress was monitored using the median ISI levels achieved in each session [45,49].

## Speed of Response Training

Participants in the speed of response training group received the same number of training sessions and trials as participants in the cognitive control training group. Contrary to the cognitive control training, patients receiving the control training based on the Speed of Response Task (SOR) [49], in which participants just respond as fast as possible to the last heard number. Therefore, this training does not target the same cognitive processes as the adaptive PASAT but captures response speed and sustained attention. The same criteria for the starting ISI and changes according to performance are applied.

## Outcomes

### Timeline for Assessments

All outcomes were assessed at baseline, directly following the training, and then again at 3-month and 6-month follow-up. In addition to assessing immediate training effects, we selected these follow-up time points commonly used in cognitive

control training studies, as they may capture longer-term effects that only emerge over time [48]. Unlike the other measures, quality of life and work and social adjustment were only assessed at baseline and at 3-month and 6-month follow-up to reduce the time burden for participants and because training was not expected to lead to meaningful changes in these outcomes within such a short period of time.

### Primary End Point: Global Mental Distress

The level of global mental symptom distress was assessed using the Hopkins Symptom Checklist-11 (HSCL-11) [68], a self-administered questionnaire with robust psychometric characteristics that measures a range of mental symptoms, such as anxiety and depression. Each of the 11 items is rated on a Likert scale from 1 to 4 with higher scores indicating greater symptom distress (mean scores for the total scale thus range between 1 and 4). At baseline assessment, Cronbach  $\alpha$  for the entire sample was 0.84. Given the transdiagnostic approach of the trial, the HSCL-11 was selected for its ability to assess mental distress that is not confined to the specific symptom pattern of a particular mental disorder but instead measures symptoms that are common across various mental disorders [68].

### Secondary End Points

#### Rumination

Rumination was assessed using the 10-item version of the Response Style Questionnaire (RSQ-10) [69]. The RSQ-10 is a self-report questionnaire that measures rumination on a 4-point Likert scale from 1 to 4, with higher scores indicating a stronger tendency to ruminate (total score range: 10-40). Reliability and validity of the German version have been demonstrated (RSQ-10D) [70]. Importantly, the brooding and reflection subscales of the RSQ-10 measure rumination above and beyond what can be explained by symptoms of depression [69]. At baseline, Cronbach  $\alpha$  was 0.73. Following suggestions by the authors of the RSQ-10, we also conducted exploratory subanalyses focusing on the 5-item brooding subscale of the questionnaire, since brooding depicts the more dysfunctional component of rumination that is associated with mental disorders [69,71].

#### Repetitive Negative Thinking

Given the transdiagnostic nature of repetitive negative thinking and difficulties with disengagement of (content- and disorder-independent) negative thoughts [72], repetitive negative thinking was measured alongside rumination to assess this specific style of thinking. We used the Perseverative Thinking Questionnaire (PTQ) [73]. Each of the 15 items of the PTQ (eg, The same thoughts keep going through my mind again and again.) is scored on a Likert scale from 0 to 4 with higher scores displaying more repetitive negative thinking (total score range: 0-60). At baseline, Cronbach  $\alpha$  was 0.93.

#### Difficulties in Emotion Regulation

Difficulties in emotion regulation were assessed using the 16-item version of the Difficulties in Emotion Regulation

Scale (DERS-16) [74]. The DERS-16 is a short form of the original 36-item DERS [75] which retains the good reliability and validity of the original scale. Difficulties in emotion regulation are assessed on a Likert scale from 1 to 5 with higher scores indicating greater difficulties in regulating emotions (total score range: 16-80). At baseline, Cronbach  $\alpha$  was 0.91.

### Cognitive Emotion Regulation

Due to the expected specific nature of the challenges in emotion regulation linked to impairments in cognitive control, we complemented the evaluation of general difficulties in emotion regulation with a more targeted assessment of habitual cognitive emotion regulation using the Cognitive Emotion Regulation Questionnaire (CERQ) [76]. With 36 items which are scored on a Likert scale from 1 to 5, the CERQ measures cognitive emotion regulation on 9 subscales. Consistent with an earlier study on cognitive control training [45], we calculated compound scores for adaptive cognitive emotion regulation using items from the subscales “positive refocusing,” “planning,” “positive reappraisal,” “putting into perspective,” and “acceptance” (total score range: 20-100) and for maladaptive cognitive emotion regulation using the items from the subscales “self-blame,” “other-blame,” “rumination,” “catastrophizing” (total score range: 16-80). A German translation has been psychometrically validated [77]. At baseline, Cronbach  $\alpha$  was 0.78 and 0.70 for the adaptive and maladaptive cognitive emotion regulation compound score, respectively.

### Cognitive Control

Cognitive control was measured using a nonadaptive assessment version of the PASAT [37] during which participants performed 3 blocks of increasing difficulty (ISI block 1=3000 ms; ISI block 2=2000 ms; ISI block 3=1500 ms). The assessment consisted of a total of 180 trials, with 60 trials per block. Additionally, participants performed 8 practice trials beforehand. As a performance indicator, we analyzed trial data and fitted a model using a binomial error distribution on the probability of a correct response for an individual trial (refer to the section “Statistical Analysis”).

**Table 1.** Sample characteristics.<sup>a</sup>

Variable	aPASAT <sup>b</sup> training group (n=40)	SOR <sup>c</sup> training group (n=40)	Total sample (N=80)
Age (years), mean (SD)	26.1 (6.3)	25.6 (3.9)	25.8 (5.2)
Women, n (%)	23 (57)	22 (55)	45 (56)
Psychotropic medication, n (%)	11 (28)	11 (28)	22 (28)
Education (years), mean (SD)	17.3 (3.4)	17.6 (3.3)	17.5 (3.3)
Diagnosis according to the DSM-5 <sup>d</sup> , n (%)			
Major depressive disorder	31 (78)	34 (85)	65 (81)
Anxiety disorder	15 (38)	16 (40)	31 (39)
Posttraumatic stress disorder	2 (5)	1 (2)	3 (4)
Somatic symptom disorder	0 (0)	3 (8)	3 (4)
Social anxiety disorder	10 (25)	11 (28)	21 (26)

This can also be interpreted as the expected ratio of correct trials per assessment (minimum 0, maximum 1).

### Quality of Life

Quality of life was assessed using the World Health Organization Quality of Life Brief Form (WHOQOL-BREF) [78]. The WHOQOL-BREF is a transdiagnostic self-report measure to assess the health condition and impact of illnesses on daily activities and behavior in 4 domains (physical, psychological, social, and environmental) on a Likert scale from 1 to 5. Scores for each domain are converted to a range between 0 (worst) and 100 (best quality of life). Psychometric quality and culture-sensitive validity have been shown for the WHOQOL-BREF in several studies (eg, [78]). Cronbach  $\alpha$  at baseline for the 4 domains ranged between 0.70 (social) and 0.83 (psychological).

### Work and Social Adjustment

The Work and Social Adjustment Scale (WSAS) [79] is a 5-item self-report measure of functional impairment in work-related and social domains attributable to an identified problem. Items are rated on a Likert scale from 0 (not at all impaired) to 8 (very severely impaired). Total scores range between 0 and 40. Psychometric quality and validity of the WSAS for various disorders have been reported [79]. At baseline, Cronbach  $\alpha$  was 0.74.

### Disorder-Specific End Points

Depending on their diagnoses, participants completed additional disorder-specific self-report questionnaires at baseline, post training, and at the 3- and 6-month follow-ups. Patients who met *DSM-5* criteria for major depressive disorder completed the Patient Health Questionnaire-9 (PHQ-9) [80]. Anxiety symptoms in patients with an anxiety disorder (excluding SAD) were assessed using the Generalized Anxiety Disorder Questionnaire (GAD-7) [81]. Participants diagnosed with SAD completed the Mini-Social Phobia Inventory (mini-SPIN) [82]. Other diagnoses were less common in our sample (each <5, see Table 1). Further details on disorder-specific outcome measures are provided in [Multimedia Appendix 1](#).

Variable	aPASAT <sup>b</sup> training group (n=40)	SOR <sup>c</sup> training group (n=40)	Total sample (N=80)
Borderline personality disorder	2 (5)	2 (5)	4 (5)
Eating disorder	0 (0)	0 (0)	0 (0)
Obsessive-compulsive disorder	2 (5)	0 (0)	2 (2)
Substance abuse disorder	0 (0)	0 (0)	0 (0)
Adjustment disorder	0 (0)	0 (0)	0 (0)

<sup>a</sup>Data are mean (SD) or n (%); anxiety disorder encompasses panic disorder, agoraphobia, specific phobia, and generalized anxiety disorder; eating disorder encompasses anorexia nervosa, bulimia nervosa, and binge eating disorder.

<sup>b</sup>aPASAT: adaptive Paced Auditory Serial Addition Test.

<sup>c</sup>SOR: Speed of Response Task.

<sup>d</sup>DSM-5: *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition).

## Additional Measures

We measured treatment credibility and expectancy using the Credibility Expectancy Questionnaire (CEQ) [83] at baseline and post training. Additionally, we assessed the intake of psychotropic medication and monitored the commencement of psychotherapy between baseline and 6-month follow-up.

## Procedure

Participants were instructed to complete all study assessments and training on their smartphones. After screening for eligibility, patients returned home and completed the baseline assessment on the next day. They were instructed to complete 10 online training sessions within a 2-week period. Patients could only complete 1 training session per day. To increase adherence, depending on their preferences, participants received automated daily reminders either via email or SMS. We monitored training adherence by regularly checking the completion of training sessions remotely and phoned participants in case of strong deviations from the training schedule (ie, multiple days without completing a training session). Participants returned to the laboratory for the posttraining assessment, where they conducted all self-report questionnaires and the PASAT assessment on their smartphones. At this time, they also received instructions for the upcoming follow-up assessments and engaged in a short qualitative interview on their experience with the training. The results of these qualitative interviews will be reported elsewhere since they are beyond the scope of this quantitative assessment of the cognitive control training. Follow-ups were conducted remotely, starting after the final training session. Patients were debriefed after the last follow-up.

## Statistical Analysis

Primary analyses followed an intention-to-treat approach. Outliers ( $|z|>3$ ) across outcomes were adjusted using winsorization to reduce the influence of extreme values on the analysis. However, results were cross-validated with unadjusted data to ensure robustness. All analyses were conducted using linear mixed-effects models [84] with the *lme4* package version 1.1-31 [85] in R (version 4.5.0; R Core Team) [86].

We compared the average outcome score trajectories between the 2 training conditions using random intercept models and fixed effects for time of assessment, condition,

and their interaction. Time of assessment was modeled as a repeated-measures factor with 4 levels (baseline, post training, 3-month, and 6-month follow-up), while training condition was a between-subjects factor (aPASAT vs SOR). For quality-of-life outcomes, time had 3 levels: baseline, 3-month follow-up, and 6-month follow-up. We fitted a model using a binomial error distribution to analyze the data from the PASAT assessment version to measure cognitive control, since PASAT performance is bound between 0 and 1 (portion of correct trials) and therefore not normally distributed. This yields a z-statistic. As performance is expected to change given different ISI, we included the current ISI as a covariate.

As a first step, we compared a full model including time of assessment, training condition, and their interaction to a reduced model with time of assessment as the sole predictor. This approach guards against “cryptic multiple testing,” that is, inflated false positives from interpreting multiple time point-specific effects without first establishing a significant overall interaction [87]. We then examined specific interaction effects between training condition and time of assessment for each interval: from baseline to post training, and to the 3- and 6-month follow-up (3 group-by-time contrasts in total).

To standardize interpretation across outcomes, where lower scores typically indicate improvement (eg, reduced mental distress), we set the aPASAT group as the reference. In this coding scheme, positive interaction effects indicate greater improvement in the aPASAT group relative to the SOR group. For outcomes where higher scores reflect improvement (eg, cognitive control and adaptive emotion regulation), the same reference coding applies, and negative interaction effects similarly indicate greater improvement in the aPASAT group. Two-tailed *P* values for fixed effects were calculated using the *lmerTest* package (version 3.1-3) [88], applying restricted maximum likelihood estimation and Satterthwaite approximation for degrees of freedom [89]. Effect sizes for fixed effects were computed by dividing the beta coefficient by the SD of outcome scores post training in the SOR condition [90]. CIs (95%) were derived using nonparametric bootstrapping with 1000 resamples [91].

To evaluate the robustness of results against missing data, we used a pattern-mixture model approach [92]. To this end, we compared pattern-mixture models including missingness

as an additional fixed effect to standard models via likelihood ratio tests.

To explore potential moderators, we tested 3-way interactions between each moderator, time of assessment, and condition. Moderators included training progress (measured as change in median ISI from first to last training session [41]), number of completed training sessions, and participant age (given known age-related decline in cognitive control; see [93]). All moderators were z-standardized.

Finally, we conducted a per-protocol analysis including only participants who completed at least 7 or more (ie,  $\geq 70\%$ ) training sessions and computed proportions of clinically significant change following the criteria by Jacobson and Truax [94].

## Ethical Considerations

This monocenter randomized controlled superiority trial with 2 parallel arms was approved by the ethics committee of the Georg-Elias-Müller-Institute of Psychology, University of Göttingen, Germany (case number #205) and registered at the German Clinical Trials Register [95]. The study procedures adhered to the ethical standards of the institutional and

national research committees and to the principles of the Declaration of Helsinki. All participants provided written informed consent and received financial compensation (€50 [approximately US \$55]) for their participation. All data were deidentified prior to analysis to ensure participant privacy and confidentiality. Deidentified data, analysis code, and source code of the mobile trainings and assessments are available on the project's Open Science Framework page [67].

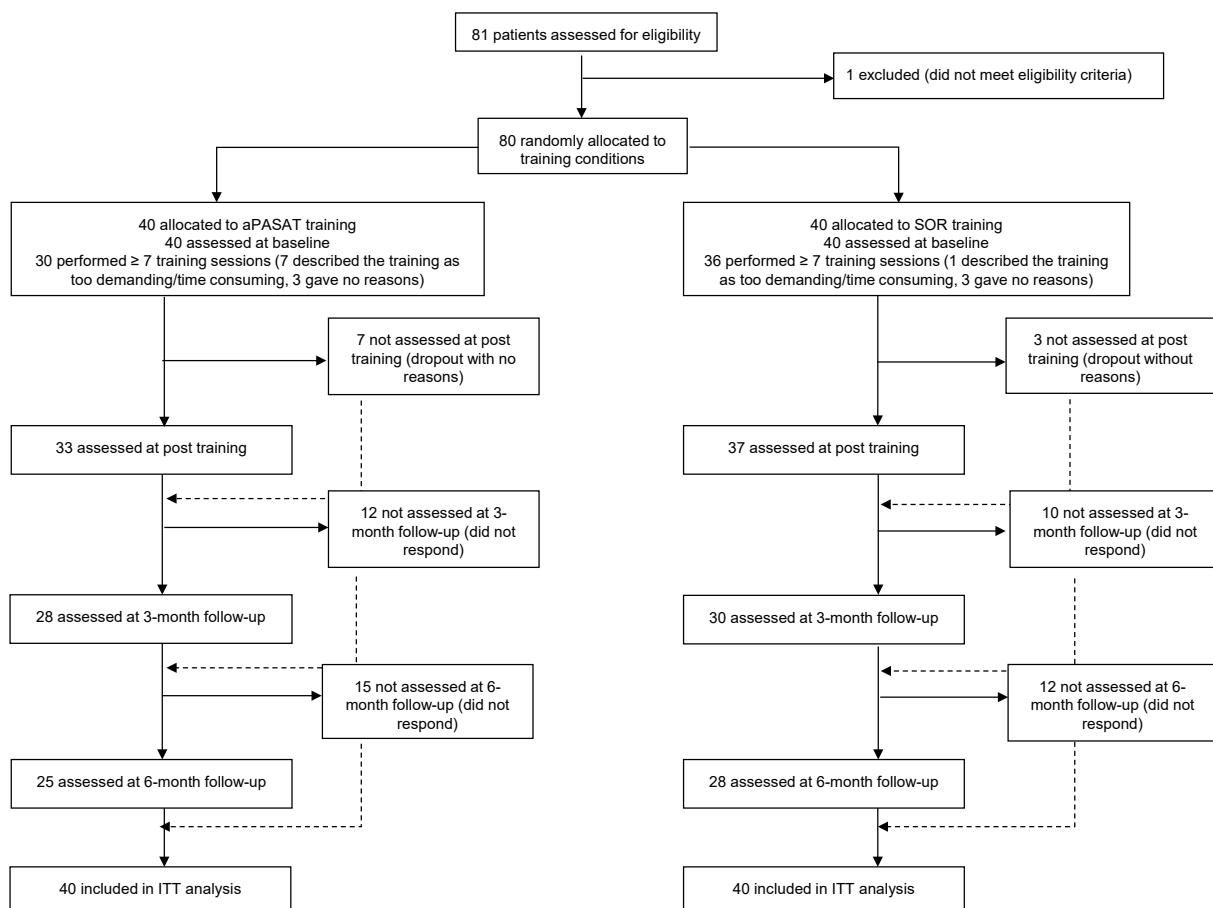
## Results

### Participant Overview

Sample characteristics including the distribution of diagnoses across conditions and the total sample can be seen in Table 1. Training groups did not differ regarding any of these characteristics (all  $P > .24$ ).

Participant flow is displayed in Figure 1. Fifteen (38%) patients in the cognitive control training group did not complete the 6-month follow-up, and 12 (30%) in the speed of response training group. Rates of complete and incomplete assessments did not differ ( $\chi^2_1 = 0.81$ ;  $P = .37$ ).

**Figure 1.** CONSORT (Consolidated Standards of Reporting Trials) flowchart. aPASAT: adaptive version of the Paced Auditory Serial Addition Test; ITT: intention-to-treat; SOR: Speed of Response Task.



Participants in the cognitive control training group performed 8.1 training sessions on average (SD 2.9). Participants in the speed of response training condition performed 9.1 (SD

1.8) training sessions on average. Training dosage did not differ between groups ( $t_{62.15} = 1.73$ ;  $P = .09$ ). Thirty (75%) participants in the cognitive control group completed the

training per protocol (ie, at least 7 training sessions), and 36 (90%) in the speed of response training group. Rates of training completion per protocol again did not differ ( $\chi^2_1=2.16$ ;  $P=.14$ ).

On average, participants in the cognitive control training group spent 14.0 (SD 9.9) days completing the training sessions, while participants in the speed of response training group spent 15.5 (SD 7.7) days. Training groups did not differ in this regard ( $t_{71.61}=0.76$ ;  $P=.45$ ).

At posttraining assessment, 8 patients in the aPASAT training condition had started outpatient therapy, while 5 did so in the SOR training group. Furthermore, patients in the aPASAT training group received, on average, 3.1 (SD 1.9) therapy sessions until posttraining assessment, and patients in the SOR group received 2.8 (SD 1.3) sessions. At the 3-month follow-up, 26 patients in the aPASAT and 35 in the SOR training condition commenced psychotherapy. Patients in the aPASAT group received, on average, 7.2 (SD 3.0) sessions until 3-month follow-up, while patients in the SOR group received 7.5 (SD 3.1) sessions. Finally, at the 6-month follow-up, nearly all participants have started the

awaited outpatient psychotherapy, with 29 in the aPASAT and 36 in the SOR training group. Patients in the aPASAT group received 11.0 (SD 6.4) therapy sessions until 6-month follow-up, while patients in the SOR group received 11.5 (SD 4.5) sessions. Training groups did not differ in therapy commencement (all  $P>.46$ ), nor in the number of therapy sessions at any assessment (all  $P>.72$ ).

Means, SDs, and number of participants for primary, secondary, and disorder-specific outcomes of depression and (social) anxiety across all assessments can be found in Table 2. Due to the small number of patients meeting criteria for a diagnosis of posttraumatic stress disorder, somatic symptom disorder, borderline personality disorder, an eating disorder, obsessive-compulsive disorder, substance use disorder, or adjustment disorder (each <5, see Table 1), no statistical models on disorder-specific measures have been conducted for these subsamples. Still, means, SDs, and number of participants for these outcomes together with quality of life and work and social adjustment across all assessments are available in Table S4 in [Multimedia Appendix 1](#).

**Table 2.** Means and SDs at baseline, post training, and follow-ups.

Outcome	Baseline		Post training		Three-month follow-up		Six-month follow-up	
	aPASAT <sup>a</sup>	SOR <sup>b</sup>	aPASAT	SOR	aPASAT	SOR	aPASAT	SOR
<b>HSCL-11<sup>c</sup></b>								
n	40	40	33	37	28	30	25	28
Mean (SD)	2.07 (0.57)	2.00 (0.50)	2.03 (0.56)	1.87 (0.59)	1.98 (0.53)	1.79 (0.51)	1.92 (0.51)	1.86 (0.52)
<b>RSQ-10D<sup>d</sup></b>								
n	40	40	32	37	28	30	25	28
Mean (SD)	23.27 (5.30)	23.30 (4.88)	23.50 (4.33)	21.95 (5.47)	23.36 (5.21)	20.77 (4.92)	22.48 (5.61)	21.11 (5.43)
<b>PTQ<sup>e</sup></b>								
n	40	40	32	37	28	30	25	28
Mean (SD)	39.55 (9.75)	36.17 (10.05)	39.19 (7.91)	31.78 (11.84)	35.37 (8.83)	31.30 (11.03)	35.40 (10.92)	32.50 (12.85)
<b>DERS-16<sup>f</sup></b>								
n	40	40	32	37	28	30	25	28
Mean (SD)	51.00 (13.50)	44.88 (11.61)	49.66 (11.24)	45.16 (14.00)	46.07 (11.13)	41.93 (12.54)	48.24 (12.29)	42.93 (14.90)
<b>CERQ<sup>g</sup> (adaptive)</b>								
n	40	40	32	37	28	30	25	28
Mean (SD)	49.55 (9.07)	55.90 (11.95)	51.47 (9.39)	56.19 (12.94)	51.14 (9.10)	53.67 (12.33)	50.56 (8.05)	51.82 (12.33)
<b>CERQ<sup>h</sup> (maladaptive)</b>								
n	40	40	32	37	28	30	25	28
Mean (SD)	39.64 (9.31)	38.80 (8.71)	40.44 (9.10)	37.78 (10.52)	39.11 (7.71)	36.67 (10.38)	39.60 (9.83)	36.75 (12.72)
<b>PASAT<sup>i</sup></b>								
n	37	36	33	36	25	27	24	23
Mean (SD)	0.58 (0.18)	0.56 (0.16)	0.88 (0.12)	0.71 (0.15)	0.87 (0.09)	0.73 (0.14)	0.90 (0.06)	0.79 (0.13)
<b>PHQ-9<sup>j</sup></b>								
n	31	34	25	31	22	26	18	24
Mean (SD)	12.94 (5.08)	11.24 (5.07)	11.08 (4.66)	9.50 (5.25)	11.32 (3.93)	8.92 (4.17)	10.78 (4.10)	9.50 (4.42)
<b>GAD-7<sup>k</sup></b>								
n	15	16	12	15	10	12	10	12

Outcome	Baseline		Post training		Three-month follow-up		Six-month follow-up	
	aPASAT <sup>a</sup>	SOR <sup>b</sup>	aPASAT	SOR	aPASAT	SOR	aPASAT	SOR
Mean (SD)	10.07 (5.13)	8.62 (4.81)	9.92 (4.19)	7.93 (4.22)	6.10 (3.75)	6.83 (4.09)	8.50 (4.28)	8.75 (5.12)
mini-SPIN <sup>c</sup>								
n	10	11	7	11	9	8	8	6
Mean (SD)	9.80 (2.39)	7.09 (2.81)	10.29 (1.80)	6.18 (3.06)	8.56 (2.79)	7.25 (2.60)	9.00 (2.73)	7.83 (3.06)
RSQ-10D <sup>d</sup> (brooding)								
n	40	40	32	37	28	30	25	28
Mean (SD)	11.15 (2.82)	11.07 (2.64)	11.16 (2.13)	10.62 (2.86)	11.04 (2.74)	10.10 (2.62)	10.84 (2.82)	10.04 (2.81)
RSQ-10D <sup>e</sup> (reflection)								
n	40	40	32	37	28	30	25	28
Mean (SD)	12.12 (3.17)	12.22 (2.84)	12.34 (2.60)	11.32 (3.00)	12.32 (2.87)	10.67 (2.71)	11.64 (2.97)	11.07 (2.96)

<sup>a</sup>aPASAT: adaptive version of Paced Auditory Serial Addition Test.

<sup>b</sup>SOR: Speed of Response Task.

<sup>c</sup>HSCL-11: Hopkins Symptom Checklist-11.

<sup>d</sup>RSQ-10D: Response Style Questionnaire (10-item version).

<sup>e</sup>PTQ: Perseverative Thinking Questionnaire.

<sup>f</sup>DERS-16: Difficulties in Emotion Regulation Questionnaire (16-item version).

<sup>g</sup>CERQ (adaptive): Cognitive Emotion Regulation Questionnaire (adaptive subscale).

<sup>h</sup>CERQ (maladaptive): Cognitive Emotion Regulation Questionnaire (maladaptive subscale).

<sup>i</sup>PASAT: Paced Auditory Serial Addition Test.

<sup>j</sup>PHQ-9: Patient Health Questionnaire 9.

<sup>k</sup>GAD-7: Generalized Anxiety Disorder Scale 7.

<sup>l</sup>mini-SPIN: Mini-form of the Social Phobia Inventory.

<sup>m</sup>RSQ-10D (brooding): Response Style Questionnaire (brooding subfacet).

<sup>n</sup>RSQ-10D (reflection): Response Style Questionnaire (reflection subfacet).

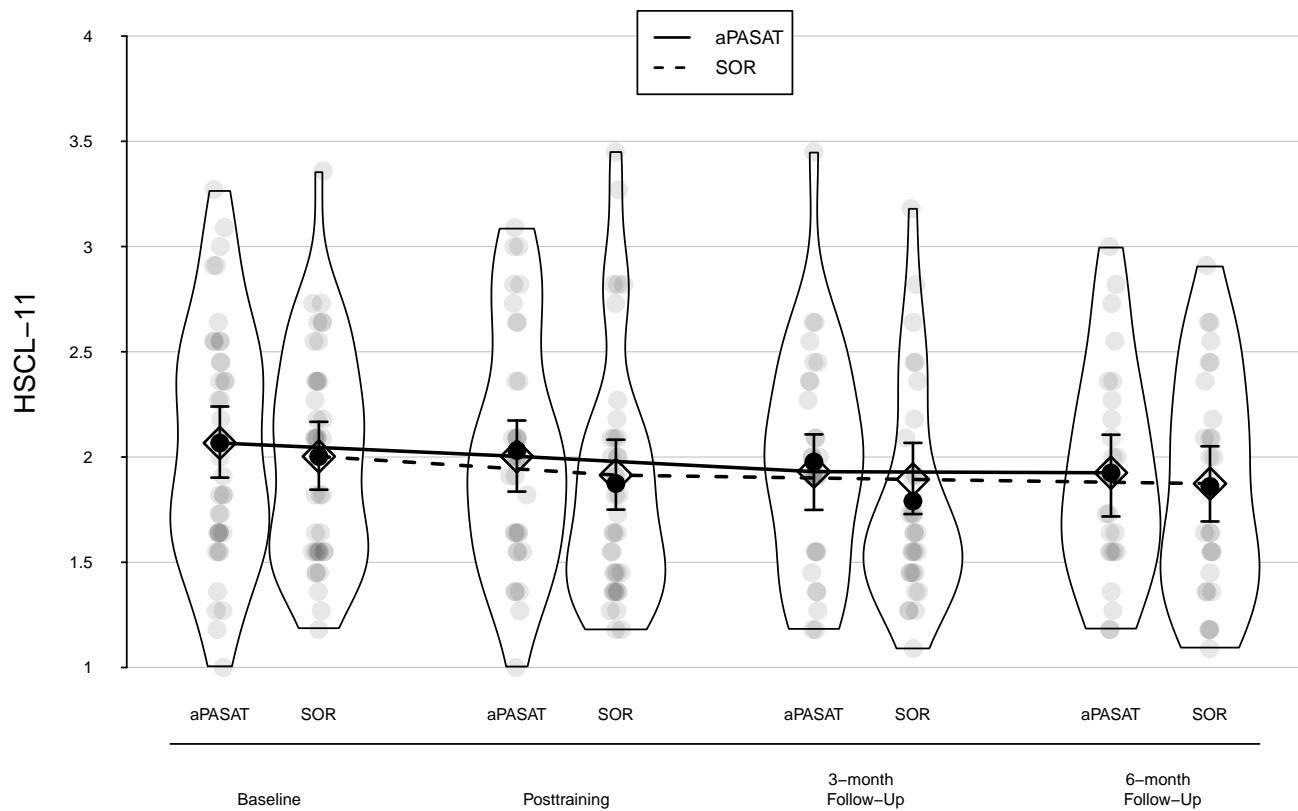
## Intention-to-Treat Analysis

### Global Mental Distress

The full-reduced model comparison for the primary outcome indicated that including training condition and its interaction with time of assessment did not significantly improve the model fit for global mental distress ( $\chi^2_4=0.60$ ;  $P=.96$ ). Consistent with the full-reduced model comparison, the individual effect for the interaction between training condition and time until postassessment (primary end point) was also not significant ( $B=-0.03$ , 95% CI  $-0.21$ ,  $0.16$ ;  $t_{179.60}=-0.26$ ;  $P=.80$ ;  $d=-0.04$ , 95% CI  $-0.35$ ,  $0.28$ ). The same applied to the interaction effect of training condition

and time between baseline and 3-month as well as 6-month follow-up assessment (secondary end point;  $B=0.03$ , 95% CI  $-0.17$ ,  $0.23$ ;  $t_{181.34}=0.25$ ;  $P=.80$ ;  $d=0.05$ , 95% CI  $-0.28$ ,  $0.39$ ; and  $B=0.01$ , 95% CI  $-0.21$ ,  $0.22$ ;  $t_{181.69}=0.11$ ;  $P=.91$ ;  $d=0.02$ , 95% CI  $-0.35$ ,  $0.37$ , respectively). Taken together, this indicates that the change in global mental distress over time was not different between training groups, neither in the short term nor in the long term. Figure 2 displays the change trajectories for HSCL-11 scores by group, including both raw data and model-based estimates. In addition, Figure S3 in the [Multimedia Appendix 1](#) depicts individual patient-level changes in global mental distress as measured with the HSCL-11 from baseline to 6-month follow-up.

**Figure 2.** Model-based change trajectories in global mental distress. Gray dots = individual data points; black dots = empirical means; diamonds = model-based estimates (95% CIs). Bean width reflects distribution. aPASAT: adaptive Paced Auditory Serial Addition Test training; HSCL-11: Hopkins symptom checklist 11; SOR: Speed of response training.



## Cognitive Control

The full-reduced model comparison indicated that including training condition and its interaction with time of assessment significantly improved model fit for the PASAT assessment outcome ( $\chi^2_{4}=389.04$ ;  $P<.001$ ). Consistent with this, the individual effect for the interaction between training condition and time until posttraining assessment was significant for PASAT performance ( $B=-0.56$ , 95% CI  $-0.59$ ,  $-0.54$ ;  $z=-18.02$ ;  $P<.001$ ;  $d=-1.23$ , 95% CI  $-1.30$ ,  $-1.20$ ). The same was true for the interaction between training condition and time until 3- and 6-month follow-up ( $B=-0.42$ , 95% CI  $-0.48$ ,  $-0.40$ ;  $z=-11.62$ ;  $P<.001$ ;  $d=-0.92$ , 95% CI  $-1.06$ ,  $-0.88$ ; and  $B=-0.44$ , 95% CI  $-0.50$ ,  $-0.42$ ;  $z=-11.22$ ;  $P<.001$ ;  $d=-0.97$ , 95% CI  $-1.10$ ,  $-0.92$ , respectively). In summary, the improvement in cognitive controls as measured by the assessment version of the PASAT was more pronounced in the cognitive control training group across all points in time.

## Emotion Regulation

None of the full-reduced model comparisons for the outcomes measuring rumination, repetitive negative thinking, difficulties in emotion regulation, or cognitive emotion regulation were significant (all  $P > .06$ ). This indicates that overall changes over time were not different between training conditions for these measures. Consequently, significant effects at the level of individual interactions (eg, the interaction between training condition and time from baseline

to posttraining assessment) should be interpreted with caution, as we did not specify hypotheses for every outcome at each assessment, and the effects were not strong enough to substantially improve model fit beyond what was explained by time alone considering all assessments.

However, the analysis of time point-specific interaction effects indicated that cognitive control training was superior to speed of response training in improving difficulties in emotion regulation ( $B=4.73$ , 95% CI  $0.52$ ,  $9.12$ ;  $t_{177.99}=2.09$ ;  $P=.04$ ;  $d=0.34$ , 95% CI  $0.04$ ,  $0.65$ ) and adaptive cognitive emotion regulation ( $B=-3.81$ , 95% CI  $-8.55$ ,  $0.67$ ;  $t_{180.51}=-1.64$ ;  $P=.10$ ;  $d=-0.29$ , 95% CI  $-0.66$ ,  $0.05$ ) from baseline to 6-month follow-up. In contrast, cognitive control training appeared less efficacious than the speed of response training in reducing rumination over the same period ( $B=-2.35$ , 95% CI  $-4.49$ ,  $-0.19$ ;  $t_{182.49}=-2.16$ ;  $P=.03$ ;  $d=-0.43$ , 95% CI  $-0.82$ ,  $-0.03$ ). Details on estimates and effect sizes for other nonsignificant comparisons as well as conditional main effects of time (effects of time in the cognitive control training group) can be found in Table S3 in [Multimedia Appendix 1](#).

## Disorder-Specific Outcomes

### Depression

Most patients (81%) met criteria for a diagnosis of major depressive disorder (see [Table 1](#)). As indicated by the nonsignificant full-reduced model comparison, training

groups did not differ regarding changes in symptoms of depression as measured with the PHQ-9 ( $\chi^2_4=3.08$ ;  $P=.54$ ). Consistent with that, symptoms of depression did not change differently between training groups from baseline to posttraining assessment ( $B=0.85$ , 95% CI  $-0.92$ ,  $2.74$ ;  $t_{142.00}=0.87$ ;  $P=.39$ ;  $d=0.16$ , 95% CI  $-0.18$ ,  $0.52$ ), 3-month follow-up ( $B=1.26$ , 95% CI  $-0.53$ ,  $3.14$ ;  $t_{143.30}=1.21$ ;  $P=.23$ ;  $d=0.24$ , 95% CI  $-0.10$ ,  $0.60$ ), or 6-month follow-up ( $B=1.39$ , 95% CI  $-0.65$ ,  $3.57$ ;  $t_{143.72}=1.27$ ;  $P=.21$ ;  $d=0.26$ , 95% CI  $-0.12$ ,  $0.68$ ).

### Social Anxiety

The full-reduced model comparisons revealed a significant interaction of time and training condition for symptoms of social anxiety as measured by the mini-SPIN ( $\chi^2_4=13.32$ ;  $P=.01$ ). While symptoms of social anxiety did not change differently between training groups from baseline to posttraining assessment ( $B=-0.32$ , 95% CI  $-1.93$ ,  $1.24$ ;  $t_{43.04}=-0.37$ ;  $P=.72$ ;  $d=-0.10$ , 95% CI  $-0.63$ ,  $0.41$ ), the interaction was significant from baseline to 3-month follow-up ( $B=1.93$ , 95% CI  $0.28$ ,  $3.72$ ;  $t_{43.26}=2.16$ ;  $P=.04$ ;  $d=0.63$ , 95% CI  $0.09$ ,  $1.21$ ), and from baseline to 6-month follow-up ( $B=2.00$ , 95% CI  $0.14$ ,  $3.81$ ;  $t_{43.43}=2.08$ ;  $P=.04$ ;  $d=0.66$ , 95% CI  $0.05$ ,  $1.24$ ). However, these findings are based on the small number of patients who met the diagnostic criteria for SAD and who had also completed the 3-month follow-up ( $n=17$ ) or 6-month follow-up ( $n=14$ ), respectively (see Table S6 in [Multimedia Appendix 1](#) for the number of measurements per outcome and assessment). As such, they should be interpreted with caution.

### Anxiety

The full-reduced model comparison showed that the interaction between training condition and time did not improve the model fit for symptoms of anxiety other than social phobia beyond effects of time ( $\chi^2_4=5.43$ ;  $P=.25$ ). In line with the full-reduced model comparison, symptoms of anxiety did not change differently between groups from baseline to posttraining assessment ( $B=0.45$ , 95% CI  $-1.90$ ,  $2.99$ ;  $t_{66.00}=0.34$ ;  $P=.73$ ;  $d=0.11$ , 95% CI  $-0.45$ ,  $0.71$ ). However, the interaction of training condition and time was significant between baseline and 3-month follow-up ( $B=2.94$ , 95% CI  $0.38$ ,  $5.82$ ;  $t_{66.51}=2.09$ ;  $P=.04$ ;  $d=0.70$ , 95% CI  $0.09$ ,  $1.38$ ), but this effect was not maintained to the 6-month follow-up ( $B=1.65$ , 95% CI  $-0.91$ ,  $4.32$ ;  $t_{66.46}=1.17$ ;  $P=.25$ ;  $d=0.39$ , 95% CI  $-0.22$ ,  $1.03$ ). Like the analysis for symptoms of social anxiety, it was only a small number of patients ( $n=22$ ) who met the diagnostic criteria for an anxiety disorder other than SAD and who also completed the 3-month follow-up (see Table S6 in [Multimedia Appendix 1](#)). As with the analysis of emotion regulation, the nonsignificant full-reduced model comparison advises caution in interpreting single interaction effects.

All other disorder-specific outcomes were not analyzed in detail due to the small number of patients (each  $n<5$ ) meeting diagnostic criteria of the respective disorder. However, means and SDs for these outcomes are provided in Table S4 in [Multimedia Appendix 1](#).

### Quality of Life and Work and Social Adjustment

All full-reduced model comparisons showed that adding training condition and its interaction with time did not change the model fit for any quality of life and work and social adjustment outcome, including all WHOQOL-BREF subscales and the WSAS, all  $P>.22$ . Estimates and effect sizes can be found in Table S3 in [Multimedia Appendix 1](#).

### Additional Tables and Figures

The number of measurements and participants in the linear mixed-effect models can be found in Table S5 in [Multimedia Appendix 1](#). Change trajectories for other outcomes, including disorder-specific outcomes and quality of life and work and social adjustment, are illustrated in Figures S4-S17 in [Multimedia Appendix 1](#).

### Credibility and Expectancy

Credibility and expectancy for the training's efficacy both decreased between baseline and posttraining assessment ( $B=-0.82$ ,  $t_{77.75}=-2.79$ ,  $P=.007$ ; and  $B=-0.63$ ,  $t_{74.68}=-2.34$ ,  $P=.02$ , respectively). Groups did not differ in this regard (all  $P>.34$ ).

### Clinical Significance

Only 1 patient experienced a clinically significant change in the primary outcome (decrease in HSCL-11 total score of at least 0.62 and final HSCL-11 score lower than 1.91) [68] from baseline to 6-month follow-up. No clinically significant changes occurred from baseline to the posttraining or 3-month follow-up assessment. Additionally, there were no patients who experienced a clinically significant change in symptoms of depression (decrease in PHQ-9 of at least 6.76 and final PHQ-9 score below 8.60) [80] from baseline to post training, 3-month, or 6-month follow-up assessment.

### Missing Data Analysis

For all measures including global mental distress, emotion regulation, and cognitive control, likelihood ratio tests of the pattern mixture models indicated that the results did not change when including missingness into the models (all  $P>.28$ , see Table S6 in [Multimedia Appendix 1](#)). This indicates that missing values had no influence on the above-reported results.

### Per-Protocol Analysis

The analysis of the data of participants that finished at least two-thirds of the training sessions revealed the same pattern of results with one exception: for repetitive negative thinking, the full-reduced model comparison was significant,  $P=.05$ . However, none of the individual interaction effects between training condition and time of assessment between baseline, post training, 3-, or 6-month follow-up were significant (all  $P>.19$ ). Therefore, the significant full-reduced model comparison likely reveals a general difference between training conditions in repetitive negative thinking, which did not change across time of assessments.

For detailed estimates and effect sizes for direct comparisons between training groups at all assessments in the per-protocol analysis, see Table S7 and Table S8 for the number of measurements and participants in [Multimedia Appendix 1](#). Figures S18-S24 in [Multimedia Appendix 1](#) depict change trajectories for global mental distress, emotion regulation, and cognitive control across conditions in the per-protocol analysis.

## Moderation Analysis

### Training Progress

Training performance as measured by the median ISI in a training session improved in the cognitive control training condition ( $B=-64.70$ ;  $t_{69.28}=-7.60$ ;  $P<.001$ ). A significant interaction effect showed that the training progress was stronger in the cognitive control compared to the speed of response training group ( $B=57.31$ ;  $t_{68.56}=4.94$ ;  $P<.001$ ).

Training progress as measured by change in median ISI significantly moderated the interaction effect between training condition and time for the performance in the assessment version of the PASAT ( $B=-0.08$ ;  $z=-5.65$ ;  $P<.001$ ). This simply indicates that cognitive control training was more efficacious in improving near-transfer cognitive control for those patients with more training progress. All other moderation effects by training progress were not significant (all  $P>.12$ ).

### Age

The 3-way interaction between age, training condition, and assessment was not significant for any primary or secondary outcome (all  $P>.09$ ), indicating that differing changes in outcomes between training groups over time were not moderated by the age of participants.

### Number of Training Sessions

Although the number of completed training sessions was not different between training conditions (see above), the number of completed training sessions significantly moderated the interaction effect of time and training condition. Specifically, completing more training sessions led to greater reductions in global mental distress in the speed of response compared to the cognitive control training ( $B=-0.16$ ;  $t_{204.86}=-3.01$ ;  $P=.003$ ). The same was true for improvements in rumination ( $B=-1.64$ ;  $t_{208.79}=-3.02$ ;  $P=.003$ ) and cognitive control ( $B=-0.18$ ;  $z=-7.68$ ;  $P<.001$ ). In other words, participants who received the speed of response training showed greater improvements in these outcomes than those in the cognitive control group when they completed more training sessions.

## Discussion

### Principal Results

To our knowledge, this study is the first to examine the efficacy of mobile cognitive control training across a broad spectrum of mental disorders in individuals waiting for outpatient psychotherapy. Contrary to our primary hypothesis,

cognitive control training, using the adaptive PASAT, was not superior to speed of response training in reducing global mental distress, neither in the short nor in the long term.

### Comparison With Prior Work

These findings diverge from earlier research on adaptive PASAT training in individuals with major depression [39-43]. Four of these 5 studies focusing on patients with current major depression combined cognitive control training with transcranial direct current stimulation [39,40,42,43]. Notably, control groups in these studies receiving cognitive control training paired with sham transcranial direct current stimulation improved as well, suggesting that cognitive control training might be similarly effective without any parallel brain stimulation. However, these studies were characterized by relatively small sample sizes, with the largest  $n=43$  [41]. This limitation is notable, as only large effects could be detected in such small samples, unlike the smaller overall effect (Hedges  $g=0.29$ ) reported for PASAT-based cognitive control training in a meta-analysis [48]. Consequently, studies [39] and [42], which reported large postassessment training effects greater than 1.00, may be considered outliers given their small sample sizes. When planning our study, we based our sample size on the most comparable study using the same intervention protocol, which reported a medium-sized effect on the primary outcome [45]. However, considering the meta-analysis published after our data collection was completed, future studies should aim for larger sample sizes. Our results are consistent with studies that examined cognitive control training as an adjunct to inpatient treatment [55] or in combination with behavioral activation for outpatients [54], which found no added benefit from the training. Given the efficacy of cognitive control training in studies with larger samples of individuals with vulnerability to or in remission from major depression [45, 96], it can be tentatively concluded that the intervention may be more suitable as a (relapse) prevention for depression and less for people with an acute full-syndrome mental disorder.

### Implications

The cognitive control training led to significant improvements in the nonadaptive assessment version of the PASAT, showing a near-transfer effect. This outcome is expected given that the training and the assessment version of the task are almost identical. Cognitive control training often involves assessment tasks closely related to those used in the intervention, and studies typically report limited far-transfer effects to other cognitive tasks [53,55,97]. Conversely, one study found that adaptive PASAT training enhanced performance in an n-back task, which targets similar cognitive functions, highlighting some degree of transfer [49]. Given the already challenging nature of the PASAT [98,99] and the number of outcome measures used to cover a broad spectrum of clinical parameters and emotion regulation, we did not assess transfer effects to other cognitive control tasks. Despite several months without further training, the cognitive control training group maintained a higher performance on the assessment version of the PASAT compared to the speed of response group at follow-ups, indicating the potential for a

lasting impact of the training. Future research should explore far-transfer effects using various cognitive measures, ideally with minimal overlap in processes not related to cognitive control, such as auditory or spatial processing.

The analysis of individual effects indicated superiority of the cognitive control training in improving difficulties in emotion regulation from baseline to 3-month follow-up and in cognitive emotion regulation from baseline to 6-month follow-up, respectively. These results align with prior research on patients remitted from depression [45]. However, contrary to the findings in the previous study that also used the CERQ, our results showed an interaction effect in the compound score for adaptive cognitive emotion regulation. We did not find any differences for the compound score of maladaptive cognition and emotion regulation. Additionally, a significant interaction effect for rumination from baseline to 3-month follow-up showed that the improvement was stronger for the speed of response training group. This result, which indicates the inferiority of the PASAT training condition regarding rumination, is in stark contrast to the meta-analytic findings in clinically depressed samples [48]. However, for all outcomes related to emotion regulation, the full-reduced model comparison was not significant. Therefore, these single effects were not substantial enough considering all assessments. As we did not formulate hypotheses for each of these secondary outcomes for each given time interval, there is a risk for these individual effects to be statistical artifacts, and they should be interpreted with caution.

The findings on disorder-specific symptoms tentatively suggest that the training may be able to reduce anxiety symptoms, especially social anxiety, in the long term. However, it is important to consider the limited number of patients with the respective diagnosis who provided data at follow-up assessments (see Table S5 in [Multimedia Appendix 1](#)). Thus, these findings should be treated with caution until replication in larger samples.

Any improvements in outcomes that were not significantly different between training conditions may be attributed to factors that applied to both groups equally, such as the commencement of or number of psychotherapy sessions during follow-up periods (which were not different between training groups), spontaneous symptom improvements, regression to the mean, demand effects, or a combination of those. It also cannot be ruled out that shared but unknown mechanisms of both cognitive control and speed of response training contributed to these improvements. Although the speed of response task may not train the updating of working memory content, it does necessitate sustained attention to effectively perform the task, thus making it an active control condition [45].

Finally, the optimal dosage and frequency of cognitive control training need to be established. A recent study suggests that at least 10 training sessions are necessary to achieve a medium-sized effect on symptoms of depression [100]. Consistent with our approach, most studies using the PASAT have implemented approximately 10 training sessions

[48]. Contrary to our expectations, the moderation analysis suggests that cognitive control training was relatively less efficacious than speed of response training in alleviating global mental distress, reducing rumination, and improving cognitive control among participants who completed a high number of training sessions. However, this was an exploratory analysis, and identifying moderators in 3-way interactions typically requires sample sizes in the several hundred [101], far exceeding that of this study. Moreover, the number of training sessions was limited, as participants could complete 10 training sessions at most. Additionally, most participants completed 7 or more training sessions (see “Participant Overview”). This limits the variability needed to meaningfully assess the moderating role of differing numbers of training sessions.

Beyond determining the optimal dosage for posttraining effects, future research should address whether booster sessions during follow-up periods could maintain or enhance long-term outcomes. Furthermore, it would be similarly important to investigate the ideal timing of training sessions, for instance, in individuals at risk for depression, training during periods of increased rumination may amplify its efficacy [102]. In this regard, mobile health interventions, such as the mobile cognitive control training studied here, might serve as suitable just-in-time adaptive interventions [103].

## Limitations

The findings of this study should be interpreted considering its methodological strengths and limitations. Despite our efforts to reach out to patients to encourage the completion of assessments, nearly half of the participants had missing data at one of the 4 assessments, primarily the 3-month and 6-month follow-up (see [Figure 1](#)). Notably, however, attrition rates did not differ between groups, and the vast number of participants finished the training per protocol, that is, they completed at least 7 of the 10 scheduled training sessions. This indicates that factors related to attrition at follow-up are likely unrelated to the specifics of the training protocol. Moreover, our follow-up sample sizes are comparable with those in other cognitive control training studies [48].

While a meta-analysis suggests that PASAT-based cognitive control training is more effective in reducing depressive symptoms immediately after the training than at follow-up [48], the lack of follow-up assessments in most studies limits conclusions about long-term effects. Small samples and highly variable follow-up intervals (ranging from weeks to a year) further reduce power and comparability. Some evidence points to longer-term benefits for outcomes such as rumination and emotion regulation [45]. These effects may unfold gradually, as participants need time to apply trained strategies such as filtering irrelevant (negative) information from working memory in daily life. Future studies should extend follow-up periods up to 12 months, as done in cognitive bias modification research [104], and explore whether training-induced gains in cognitive control enhance responsiveness to subsequent psychotherapy [105].

Our sample, with a mean age of 25.8 (SD 5.2) years, was relatively young compared to other cognitive control training studies in depression [48]. This might be since the treatment facility is situated on a university campus, and therefore often frequented by students with mental health problems. Cognitive control tends to decline with age [93], but we found no evidence that age moderated training effects in our relatively young sample. Future studies should include more diverse community samples in terms of age and education. Since mental disorders frequently co-occur, some participants met criteria for more than one disorder (see Table 1). This raises the possibility of overlap in disorder-specific analyses, as individuals may influence multiple outcomes. However, our primary outcome was global mental distress, not tied to any single diagnosis. Given the high prevalence of comorbidity in clinical populations [106], we chose to include patients with multiple diagnoses to enhance ecological validity. Finally, due to the monocentric design and recruitment from a Western European outpatient setting, the generalizability of our findings to other clinical or cultural contexts may be limited.

Notably, almost no patients showed clinically significant improvements in global mental distress or depressive symptoms at the 6-month follow-up, which is unexpected given that most began psychotherapy during this period. However, our sample reported relatively low baseline distress, leaving limited room for substantial improvement. For example, patients with major depression had an average baseline PHQ-9 of 12.0 (see Table 2). Given the PHQ-9's

reliable change index of 6.8 points [80], a clinically significant improvement would require a follow-up score below 6, which is typical of healthy individuals [107]. Moreover, such changes generally do not occur within the first few sessions of outpatient therapy [108]. That most participants began psychotherapy during the follow-up is unsurprising given the recruitment from a waiting list. While this could confound observed improvements, clinically significant changes were rare overall, and psychotherapy uptake did not differ between training groups, nor did the number of therapy sessions between groups. Thus, any confounding effect by psychotherapy is either likely minimal or evenly distributed between groups.

## Conclusions

This study examined the efficacy of mobile cognitive control training as a transdiagnostic intervention for individuals awaiting outpatient psychotherapy. Despite its limitations, the study's design allowed for an ecologically valid training context. In summary, we did not find evidence for the superiority of the cognitive control training over an active control in reducing symptoms of mental distress. Based on our results, we cannot generally suggest cognitive control training in its current form as an intervention for patients with an acute, full-blown mental disorder. Furthermore, examining the phase of illness, potential working mechanisms, and far-transfer effects in large samples is necessary to further assess the potential of this intervention.

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## Authors' Contributions

TB and EK contributed to the conceptualization of the study. MB was responsible for data curation and formal analysis. TB acquired funding for the project. MB and LO conducted the investigation. TB, EK, and MB contributed to the development of the methodology. TB and MB were responsible for project administration and resource provision, with support from LO. MB developed the software. TB and EK supervised the research activity. MB and TB contributed to validation and visualization. MB prepared the original draft of the manuscript. TB, EK, MB, and LO reviewed and edited the manuscript.

## Conflicts of Interest

None declared.

## Multimedia Appendix 1

Additional material including tables, figures, and details for disorder-specific outcomes.

[[PDF File \(Adobe File\), 1963 KB](#) [Multimedia Appendix 1](#)]

## Checklist 1

CONSORT checklist.

[[PDF File \(Adobe File\), 246 KB](#) [Checklist 1](#)]

## References

1. Vigo D, Thornicroft G, Atun R. Estimating the true global burden of mental illness. *Lancet Psychiatry*. Feb 2016;3(2):171-178. [doi: [10.1016/S2215-0366\(15\)00505-2](https://doi.org/10.1016/S2215-0366(15)00505-2)]
2. Smith K. Trillion-dollar brain drain. *Nature New Biol*. Oct 6, 2011;478(7367):15-15. [doi: [10.1038/478015a](https://doi.org/10.1038/478015a)]
3. Huhn M, Tardy M, Spineli LM, et al. Efficacy of pharmacotherapy and psychotherapy for adult psychiatric disorders: a systematic overview of meta-analyses. *JAMA Psychiatry*. Jun 2014;71(6):706-715. [doi: [10.1001/jamapsychiatry.2014.112](https://doi.org/10.1001/jamapsychiatry.2014.112)] [Medline: [24789675](#)]
4. Leichsenring F, Steinert C, Rabung S, Ioannidis JPA. The efficacy of psychotherapies and pharmacotherapies for mental disorders in adults: an umbrella review and meta-analytic evaluation of recent meta-analyses. *World Psychiatry*. Feb 2022;21(1):133-145. [doi: [10.1002/wps.20941](https://doi.org/10.1002/wps.20941)] [Medline: [35015359](#)]
5. Steinert C, Hofmann M, Kruse J, Leichsenring F. Relapse rates after psychotherapy for depression – stable long-term effects? A meta-analysis. *J Affect Disord*. Oct 2014;168:107-118. [doi: [10.1016/j.jad.2014.06.043](https://doi.org/10.1016/j.jad.2014.06.043)]
6. Insel TR. The NIMH Research Domain Criteria (RDoC) Project: precision medicine for psychiatry. *Am J Psychiatry*. Apr 2014;171(4):395-397. [doi: [10.1176/appi.ajp.2014.14020138](https://doi.org/10.1176/appi.ajp.2014.14020138)] [Medline: [24687194](#)]
7. Insel T, Cuthbert B, Garvey M, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *Am J Psychiatry*. Jul 2010;167(7):748-751. [doi: [10.1176/appi.ajp.2010.09091379](https://doi.org/10.1176/appi.ajp.2010.09091379)] [Medline: [20595427](#)]
8. Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across psychopathology: a meta-analytic review. *Clin Psychol Rev*. Mar 2010;30(2):217-237. [doi: [10.1016/j.cpr.2009.11.004](https://doi.org/10.1016/j.cpr.2009.11.004)] [Medline: [20015584](#)]
9. Hu T, Zhang D, Wang J, Mistry R, Ran G, Wang X. Relation between emotion regulation and mental health: a meta-analysis review. *Psychol Rep*. Apr 2014;114(2):341-362. [doi: [10.2466/03.20.PR0.114k22w4](https://doi.org/10.2466/03.20.PR0.114k22w4)] [Medline: [24897894](#)]
10. Sloan E, Hall K, Moulding R, Bryce S, Mildred H, Staiger PK. Emotion regulation as a transdiagnostic treatment construct across anxiety, depression, substance, eating and borderline personality disorders: a systematic review. *Clin Psychol Rev*. Nov 2017;57:141-163. [doi: [10.1016/j.cpr.2017.09.002](https://doi.org/10.1016/j.cpr.2017.09.002)] [Medline: [28941927](#)]
11. Gross JJ. The emerging field of emotion regulation: an integrative review. *Rev Gen Psychol*. Sep 1998;2(3):271-299. [doi: [10.1037/1089-2680.2.3.271](https://doi.org/10.1037/1089-2680.2.3.271)]
12. Berking M, Ebert D, Cuijpers P, Hofmann SG. Emotion regulation skills training enhances the efficacy of inpatient cognitive behavioral therapy for major depressive disorder: a randomized controlled trial. *Psychother Psychosom*. 2013;82(4):234-245. [doi: [10.1159/000348448](https://doi.org/10.1159/000348448)] [Medline: [23712210](#)]
13. Fehlinger T, Stumpenhorst M, Stenzel N, Rief W. Emotion regulation is the essential skill for improving depressive symptoms. *J Affect Disord*. Jan 10, 2013;144(1-2):116-122. [doi: [10.1016/j.jad.2012.06.015](https://doi.org/10.1016/j.jad.2012.06.015)] [Medline: [22939389](#)]
14. Kleinstäuber M, Allwang C, Bailer J, et al. Cognitive behaviour therapy complemented with emotion regulation training for patients with persistent physical symptoms: a randomised clinical trial. *Psychother Psychosom*. 2019;88(5):287-299. [doi: [10.1159/000501621](https://doi.org/10.1159/000501621)] [Medline: [31430755](#)]
15. Mennin DS, Fresco DM, O'Toole MS, Heimberg RG. A randomized controlled trial of emotion regulation therapy for generalized anxiety disorder with and without co-occurring depression. *J Consult Clin Psychol*. Mar 2018;86(3):268-281. [doi: [10.1037/ccp0000289](https://doi.org/10.1037/ccp0000289)] [Medline: [29504794](#)]
16. O'Toole MS, Renna ME, Mennin DS, Fresco DM. Changes in decentering and reappraisal temporally precede symptom reduction during emotion regulation therapy for generalized anxiety disorder with and without co-occurring depression. *Behav Ther*. Nov 2019;50(6):1042-1052. [doi: [10.1016/j.beth.2018.12.005](https://doi.org/10.1016/j.beth.2018.12.005)] [Medline: [31735240](#)]
17. Radkovsky A, McArdle JJ, Bockting CLH, Berking M. Successful emotion regulation skills application predicts subsequent reduction of symptom severity during treatment of major depressive disorder. *J Consult Clin Psychol*. Apr 2014;82(2):248-262. [doi: [10.1037/a0035828](https://doi.org/10.1037/a0035828)] [Medline: [24564219](#)]
18. Berking M, Wupperman P, Reichardt A, Pejic T, Dippel A, Znoj H. Emotion-regulation skills as a treatment target in psychotherapy. *Behav Res Ther*. Nov 2008;46(11):1230-1237. [doi: [10.1016/j.brat.2008.08.005](https://doi.org/10.1016/j.brat.2008.08.005)] [Medline: [18835479](#)]
19. Gratz KL, Weiss NH, Tull MT. Examining emotion regulation as an outcome, mechanism, or target of psychological treatments. *Curr Opin Psychol*. Jun 1, 2015;3:85-90. [doi: [10.1016/j.copsyc.2015.02.010](https://doi.org/10.1016/j.copsyc.2015.02.010)] [Medline: [25859561](#)]
20. Rottenberg J, Gross JJ. Emotion and emotion regulation: a map for psychotherapy researchers. *Clin Psychol Sci Pract*. 2007;14(4):323-328. [doi: [10.1111/j.1468-2850.2007.00093.x](https://doi.org/10.1111/j.1468-2850.2007.00093.x)]
21. Ochsner K, Gross J. The cognitive control of emotion. *Trends Cogn Sci (Regul Ed)*. May 2005;9(5):242-249. [doi: [10.1016/j.tics.2005.03.010](https://doi.org/10.1016/j.tics.2005.03.010)]
22. Ochsner KN, Silvers JA, Buhle JT. Functional imaging studies of emotion regulation: a synthetic review and evolving model of the cognitive control of emotion. *Ann N Y Acad Sci*. Mar 2012;1251(1). [doi: [10.1111/j.1749-6632.2012.06751.x](https://doi.org/10.1111/j.1749-6632.2012.06751.x)]

23. Joormann J, Yoon KL, Zetsche U. Cognitive inhibition in depression. *Appl Prev Psychol*. Dec 2007;12(3):128-139. [doi: [10.1016/j.appsy.2007.09.002](https://doi.org/10.1016/j.appsy.2007.09.002)]
24. Joormann J, D'Avanzato C. Emotion regulation in depression: examining the role of cognitive processes. *Cogn Emot*. Sep 2010;24(6):913-939. [doi: [10.1080/02699931003784939](https://doi.org/10.1080/02699931003784939)]
25. Pruessner L, Barnow S, Holt DV, Joormann J, Schulze K. A cognitive control framework for understanding emotion regulation flexibility. *Emotion*. Feb 2020;20(1):21-29. [doi: [10.1037/emo0000658](https://doi.org/10.1037/emo0000658)] [Medline: [31961173](#)]
26. Miller EK. The prefrontal cortex and cognitive control. *Nat Rev Neurosci*. Oct 2000;1(1):59-65. [doi: [10.1038/35036228](https://doi.org/10.1038/35036228)] [Medline: [11252769](#)]
27. Miyake A, Friedman NP. The nature and organization of individual differences in executive functions: four general conclusions. *Curr Dir Psychol Sci*. Feb 2012;21(1):8-14. [doi: [10.1177/0963721411429458](https://doi.org/10.1177/0963721411429458)] [Medline: [22773897](#)]
28. Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD. The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. *Cogn Psychol*. Aug 2000;41(1):49-100. [doi: [10.1006/cogp.1999.0734](https://doi.org/10.1006/cogp.1999.0734)] [Medline: [10945922](#)]
29. Snyder HR, Miyake A, Hankin BL. Advancing understanding of executive function impairments and psychopathology: bridging the gap between clinical and cognitive approaches. *Front Psychol*. 2015;6:328. [doi: [10.3389/fpsyg.2015.00328](https://doi.org/10.3389/fpsyg.2015.00328)] [Medline: [25859234](#)]
30. De Lissnyder E, Koster EHW, Derakshan N, De Raedt R. The association between depressive symptoms and executive control impairments in response to emotional and non-emotional information. *Cogn Emot*. Feb 2010;24(2):264-280. [doi: [10.1080/02699930903378354](https://doi.org/10.1080/02699930903378354)]
31. De Lissnyder E, Koster EHW, De Raedt R. Emotional interference in working memory is related to rumination. *Cogn Ther Res*. Aug 2012;36(4):348-357. [doi: [10.1007/s10608-011-9352-4](https://doi.org/10.1007/s10608-011-9352-4)]
32. Joormann J. Cognitive inhibition and emotion regulation in depression. *Curr Dir Psychol Sci*. Jun 2010;19(3):161-166. [doi: [10.1177/0963721410370293](https://doi.org/10.1177/0963721410370293)]
33. Whitmer AJ, Gotlib IH. An attentional scope model of rumination. *Psychol Bull*. Sep 2013;139(5):1036-1061. [doi: [10.1037/a0030923](https://doi.org/10.1037/a0030923)] [Medline: [23244316](#)]
34. Zetsche U, D'Avanzato C, Joormann J. Depression and rumination: relation to components of inhibition. *Cogn Emot*. Jun 2012;26(4):758-767. [doi: [10.1080/02699931.2011.613919](https://doi.org/10.1080/02699931.2011.613919)]
35. Koster EHW, De Lissnyder E, Derakshan N, De Raedt R. Understanding depressive rumination from a cognitive science perspective: the impaired disengagement hypothesis. *Clin Psychol Rev*. Feb 2011;31(1):138-145. [doi: [10.1016/j.cpr.2010.08.005](https://doi.org/10.1016/j.cpr.2010.08.005)] [Medline: [20817334](#)]
36. Zetsche U, Bürkner PC, Schulze L. Shedding light on the association between repetitive negative thinking and deficits in cognitive control - a meta-analysis. *Clin Psychol Rev*. Jul 2018;63:56-65. [doi: [10.1016/j.cpr.2018.06.001](https://doi.org/10.1016/j.cpr.2018.06.001)] [Medline: [29913351](#)]
37. Gronwall DMA. Paced auditory serial-addition task: a measure of recovery from concussion. *Percept Mot Skills*. Apr 1977;44(2):367-373. [doi: [10.2466/pms.1977.44.2.367](https://doi.org/10.2466/pms.1977.44.2.367)]
38. Siegle GJ, Ghinassi F, Thase ME. Neurobehavioral therapies in the 21st century: summary of an emerging field and an extended example of cognitive control training for depression. *Cogn Ther Res*. Apr 2007;31(2):235-262. [doi: [10.1007/s10608-006-9118-6](https://doi.org/10.1007/s10608-006-9118-6)]
39. Brunoni AR, Boggio PS, De Raedt R, et al. Cognitive control therapy and transcranial direct current stimulation for depression: a randomized, double-blinded, controlled trial. *J Affect Disord*. Jun 2014;162:43-49. [doi: [10.1016/j.jad.2014.03.026](https://doi.org/10.1016/j.jad.2014.03.026)] [Medline: [24767004](#)]
40. Segrave RA, Arnold S, Hoy K, Fitzgerald PB. Concurrent cognitive control training augments the antidepressant efficacy of tDCS: a pilot study. *Brain Stimul*. 2014;7(2):325-331. [doi: [10.1016/j.brs.2013.12.008](https://doi.org/10.1016/j.brs.2013.12.008)] [Medline: [24486425](#)]
41. Siegle GJ, Price RB, Jones NP, Ghinassi F, Painter T, Thase ME. You gotta work at it: pupillary indices of task focus are prognostic for response to a neurocognitive intervention for rumination in depression. *Clin Psychol Sci*. Jul 2014;2(4):455-471. [doi: [10.1177/2167702614536160](https://doi.org/10.1177/2167702614536160)]
42. Sommer A, Plewnia C. Depression treatment by tDCS-enhanced cognitive control training: a test of two stimulation intensities. *Brain Stimul*. 2021;14(4):987-989. [doi: [10.1016/j.brs.2021.06.004](https://doi.org/10.1016/j.brs.2021.06.004)] [Medline: [34147374](#)]
43. Vanderhasselt MA, De Raedt R, Namur V, et al. Transcranial electric stimulation and neurocognitive training in clinically depressed patients: a pilot study of the effects on rumination. *Prog Neuropsychopharmacol Biol Psychiatry*. Mar 3, 2015;57:93-99. [doi: [10.1016/j.pnpbp.2014.09.015](https://doi.org/10.1016/j.pnpbp.2014.09.015)] [Medline: [25455589](#)]
44. Calkins AW, McMorran KE, Siegle GJ, Otto MW. The effects of computerized cognitive control training on community adults with depressed mood. *Behav Cogn Psychother*. Sep 2015;43(5):578-589. [doi: [10.1017/S1352465814000046](https://doi.org/10.1017/S1352465814000046)] [Medline: [24589123](#)]

45. Hoorelbeke K, Koster EHW. Internet-delivered cognitive control training as a preventive intervention for remitted depressed patients: evidence from a double-blind randomized controlled trial study. *J Consult Clin Psychol*. Feb 2017;85(2):135-146. [doi: [10.1037/ccp0000128](https://doi.org/10.1037/ccp0000128)] [Medline: [27362792](https://pubmed.ncbi.nlm.nih.gov/27362792/)]
46. Hoorelbeke K, Van den Bergh N, De Raedt R, Wichers M, Koster EHW. Preventing recurrence of depression: long-term effects of a randomized controlled trial on cognitive control training for remitted depressed patients. *Clin Psychol Sci*. Jul 2021;9(4):615-633. [doi: [10.1177/2167702620979775](https://doi.org/10.1177/2167702620979775)]
47. Hoorelbeke K, Van den Bergh N, De Raedt R, Wichers M, Albers CJ, Koster EHW. Regaining control of your emotions? Investigating the mechanisms underlying effects of cognitive control training for remitted depressed patients. *Emotion*. Feb 2023;23(1):194-213. [doi: [10.1037/emo0001067](https://doi.org/10.1037/emo0001067)]
48. Vander Zwalmen Y, Liebaert E, Hoorelbeke K, et al. Treatment response following adaptive PASAT training for depression vulnerability: a systematic review and meta-analysis. *Neuropsychol Rev*. Mar 2024;34(1):232-249. [doi: [10.1007/s11065-023-09581-8](https://doi.org/10.1007/s11065-023-09581-8)] [Medline: [36853526](https://pubmed.ncbi.nlm.nih.gov/36853526/)]
49. Hoorelbeke K, Koster EHW, Demeyer I, Loey T, Vanderhasselt MA. Effects of cognitive control training on the dynamics of (mal)adaptive emotion regulation in daily life. *Emotion*. Oct 2016;16(7):945-956. [doi: [10.1037/emo0000169](https://doi.org/10.1037/emo0000169)]
50. Van den Bergh N, Vermeersch S, Hoorelbeke K, Vervaeke J, De Raedt R, Koster EHW. Cognitive control training as an augmentation strategy to CBT in the treatment of fear of failure in undergraduates. *Cogn Ther Res*. Dec 2020;44(6):1199-1212. [doi: [10.1007/s10608-020-10129-w](https://doi.org/10.1007/s10608-020-10129-w)]
51. Vervaeke J, Hoorelbeke K, Baeken C, Van Looy J, Koster EHW. Transfer and motivation after cognitive control training for remitted depression in healthy sample. *J Cogn Enhanc*. Mar 2020;4(1):49-61. [doi: [10.1007/s41465-019-00135-6](https://doi.org/10.1007/s41465-019-00135-6)]
52. Vanderhasselt MA, Demeyer I, Van Imschoot L, Hoorelbeke K, De Raedt R. Cognitive control training in healthy older adults: a proof of concept study on the effects on cognitive functioning, emotion regulation and affect. *Cogn Ther Res*. Oct 2021;45(5):959-968. [doi: [10.1007/s10608-020-10154-9](https://doi.org/10.1007/s10608-020-10154-9)]
53. Hoorelbeke K, Koster EHW, Vanderhasselt MA, Callewaert S, Demeyer I. The influence of cognitive control training on stress reactivity and rumination in response to a lab stressor and naturalistic stress. *Behav Res Ther*. Jun 2015;69:1-10. [doi: [10.1016/j.brat.2015.03.010](https://doi.org/10.1016/j.brat.2015.03.010)] [Medline: [25841177](https://pubmed.ncbi.nlm.nih.gov/25841177/)]
54. Moshier SJ, Otto MW. Behavioral activation treatment for major depression: a randomized trial of the efficacy of augmentation with cognitive control training. *J Affect Disord*. Mar 1, 2017;210:265-268. [doi: [10.1016/j.jad.2017.01.003](https://doi.org/10.1016/j.jad.2017.01.003)] [Medline: [28068613](https://pubmed.ncbi.nlm.nih.gov/28068613/)]
55. Ferrari GRA, Vanderhasselt MA, Rinck M, et al. A cognitive control training as add-on treatment to usual care for depressed inpatients. *Cogn Ther Res*. Oct 2021;45(5):929-943. [doi: [10.1007/s10608-020-10197-y](https://doi.org/10.1007/s10608-020-10197-y)]
56. Aldao A, Nolen-Hoeksema S. Specificity of cognitive emotion regulation strategies: a transdiagnostic examination. *Behav Res Ther*. Oct 2010;48(10):974-983. [doi: [10.1016/j.brat.2010.06.002](https://doi.org/10.1016/j.brat.2010.06.002)] [Medline: [20591413](https://pubmed.ncbi.nlm.nih.gov/20591413/)]
57. Revelle W. Psych: procedures for psychological, psychometric, and personality research. Comprehensive R Archive Network (CRAN). 2015. URL: <https://doi.org/10.32614/CRAN.package.psych> [Accessed 2025-11-16]
58. Arslan RC, Walther MP, Tata CS. formr: a study framework allowing for automated feedback generation and complex longitudinal experience-sampling studies using R. *Behav Res Methods*. Feb 2020;52(1):376-387. [doi: [10.3758/s13428-019-01236-y](https://doi.org/10.3758/s13428-019-01236-y)] [Medline: [30937847](https://pubmed.ncbi.nlm.nih.gov/30937847/)]
59. de Leeuw JR. jsPsych: a JavaScript library for creating behavioral experiments in a Web browser. *Behav Res Methods*. Mar 2015;47(1):1-12. [doi: [10.3758/s13428-014-0458-y](https://doi.org/10.3758/s13428-014-0458-y)] [Medline: [24683129](https://pubmed.ncbi.nlm.nih.gov/24683129/)]
60. Lange K, Kühn S, Filevich E. “Just Another Tool for Online Studies” (JATOS): an easy solution for setup and management of web servers supporting online studies. *PLoS One*. 2015;10(6):e0130834. [doi: [10.1371/journal.pone.0130834](https://doi.org/10.1371/journal.pone.0130834)] [Medline: [26114751](https://pubmed.ncbi.nlm.nih.gov/26114751/)]
61. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. American Psychiatric Association; 2013.
62. Margraf J, Cwik JC, von BR, Suppiger A, Schneider S. DIPS Open Access 12: Diagnostisches Interview Bei Psychischen Störungen. OMP Ruhr-Universität Bochum; 2022.
63. First MB. Structured clinical interview for the DSM (SCID). In: The Encyclopedia of Clinical Psychology. John Wiley & Sons, Ltd; 2015:1-6. [doi: [10.1002/9781118625392.wbecp351](https://doi.org/10.1002/9781118625392.wbecp351)]
64. Faul F, Erdfelder E, Buchner A, Lang AG. Statistical power analyses using G\*Power 3.1: tests for correlation and regression analyses. *Behav Res Methods*. Nov 2009;41(4):1149-1160. [doi: [10.3758/BRM.41.4.1149](https://doi.org/10.3758/BRM.41.4.1149)] [Medline: [19897823](https://pubmed.ncbi.nlm.nih.gov/19897823/)]
65. Whitehead AL, Julius SA, Cooper CL, Campbell MJ. Estimating the sample size for a pilot randomised trial to minimise the overall trial sample size for the external pilot and main trial for a continuous outcome variable. *Stat Methods Med Res*. Jun 2016;25(3):1057-1073. [doi: [10.1177/0962280215588241](https://doi.org/10.1177/0962280215588241)] [Medline: [26092476](https://pubmed.ncbi.nlm.nih.gov/26092476/)]

66. Au J, Sheehan E, Tsai N, Duncan GJ, Buschkuhl M, Jaeggi SM. Improving fluid intelligence with training on working memory: a meta-analysis. *Psychon Bull Rev*. Apr 2015;22(2):366-377. [doi: [10.3758/s13423-014-0699-x](https://doi.org/10.3758/s13423-014-0699-x)]

67. Transdiagnostic cognitive control training. OSF. URL: <https://osf.io/5cah4/> [Accessed 2025-11-09]

68. Lutz W, Tholen S, Schürch E, Berking M. The development, validation, and reliability of short-forms of current instruments for the evaluation of therapeutic progress in psychotherapy and psychiatry.. *Diagnostica*. Jan 2006;52(1):11-25. [doi: [10.1026/0012-1924.52.1.11](https://doi.org/10.1026/0012-1924.52.1.11)]

69. Treynor W, Gonzalez R, Nolen-Hoeksema S. Rumination reconsidered: a psychometric analysis. *Cognit Ther Res*. Jun 2003;27(3):247-259. [doi: [10.1023/A:1023910315561](https://doi.org/10.1023/A:1023910315561)]

70. Huffziger S, Kühner C. The rumination components broadening and reflection. *Zeitschrift für Klinische Psychologie und Psychotherapie*. Jan 2012;41(1):38-46. [doi: [10.1026/1616-3443/a000118](https://doi.org/10.1026/1616-3443/a000118)]

71. Watkins ER. Constructive and unconstructive repetitive thought. *Psychol Bull*. Mar 2008;134(2):163-206. [doi: [10.1037/0033-2909.134.2.163](https://doi.org/10.1037/0033-2909.134.2.163)] [Medline: [18298268](https://pubmed.ncbi.nlm.nih.gov/18298268/)]

72. Ehring T, Watkins ER. Repetitive negative thinking as a transdiagnostic process. *Int J Cogn Ther*. Sep 2008;1(3):192-205. [doi: [10.1521/ijct.2008.1.3.192](https://doi.org/10.1521/ijct.2008.1.3.192)]

73. Ehring T, Zetsche U, Weidacker K, Wahl K, Schönfeld S, Ehlers A. The Perseverative Thinking Questionnaire (PTQ): Validation of a content-independent measure of repetitive negative thinking. *J Behav Ther Exp Psychiatry*. Jun 2011;42(2):225-232. [doi: [10.1016/j.jbtep.2010.12.003](https://doi.org/10.1016/j.jbtep.2010.12.003)]

74. Bjureberg J, Ljótsson B, Tull MT, et al. Development and Validation of a Brief Version of the Difficulties in Emotion Regulation Scale: the DERS-16. *J Psychopathol Behav Assess*. Jun 2016;38(2):284-296. [doi: [10.1007/s10862-015-9514-x](https://doi.org/10.1007/s10862-015-9514-x)] [Medline: [27239096](https://pubmed.ncbi.nlm.nih.gov/27239096/)]

75. Gratz KL, Roemer L. Multidimensional assessment of emotion regulation and dysregulation: development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. *J Psychopathol Behav Assess*. Mar 2004;26(1):41-54. [doi: [10.1023/B:JOBA.0000007455.08539.94](https://doi.org/10.1023/B:JOBA.0000007455.08539.94)]

76. Garnefski N, Kraaij V, Spinhoven P. Negative life events, cognitive emotion regulation and emotional problems. *Pers Individ Dif*. Jun 2001;30(8):1311-1327. [doi: [10.1016/S0191-8869\(00\)00113-6](https://doi.org/10.1016/S0191-8869(00)00113-6)]

77. Loch N, Hiller W, Withköft M. The Cognitive Emotion Regulation Questionnaire (CERQ): psychometric evaluation of a German adaptation. *Zeitschrift für Klinische Psychologie und Psychotherapie*. Apr 2011;40(2):94-106. [doi: [10.1026/1616-3443/a000079](https://doi.org/10.1026/1616-3443/a000079)]

78. Skevington SM, Lotfy M, O'Connell KA, WHOQOL Group. The World Health Organization's WHOQOL-BREF quality of life assessment: psychometric properties and results of the international field trial. A report from the WHOQOL group. *Qual Life Res*. Mar 2004;13(2):299-310. [doi: [10.1023/B:QURE.0000018486.91360.00](https://doi.org/10.1023/B:QURE.0000018486.91360.00)] [Medline: [15085902](https://pubmed.ncbi.nlm.nih.gov/15085902/)]

79. Mundt JC, Marks IM, Shear MK, Greist JH. The Work and Social Adjustment Scale: a simple measure of impairment in functioning. *Br J Psychiatry*. May 2002;180(5):461-464. [doi: [10.1192/bjp.180.5.461](https://doi.org/10.1192/bjp.180.5.461)] [Medline: [11983645](https://pubmed.ncbi.nlm.nih.gov/11983645/)]

80. Kroenke K, Spitzer RL, Williams JBW. The PHQ-9. *J Gen Intern Med*. Sep 2001;16(9):606-613. [doi: [10.1046/j.1525-1497.2001.016009606.x](https://doi.org/10.1046/j.1525-1497.2001.016009606.x)]

81. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing Generalized Anxiety Disorder: the GAD-7. *Arch Intern Med*. May 22, 2006;166(10):1092-1097. [doi: [10.1001/archinte.166.10.1092](https://doi.org/10.1001/archinte.166.10.1092)] [Medline: [16717171](https://pubmed.ncbi.nlm.nih.gov/16717171/)]

82. Wiltink J, Kliem S, Michal M, et al. Mini - Social Phobia Inventory (mini-SPIN): psychometric properties and population based norms of the German version. *BMC Psychiatry*. Nov 25, 2017;17(1):377. [doi: [10.1186/s12888-017-1545-2](https://doi.org/10.1186/s12888-017-1545-2)] [Medline: [29178857](https://pubmed.ncbi.nlm.nih.gov/29178857/)]

83. Devilly GJ, Borkovec TD. Psychometric properties of the credibility/expectancy questionnaire. *J Behav Ther Exp Psychiatry*. Jun 2000;31(2):73-86. [doi: [10.1016/s0005-7916\(00\)00012-4](https://doi.org/10.1016/s0005-7916(00)00012-4)] [Medline: [11132119](https://pubmed.ncbi.nlm.nih.gov/11132119/)]

84. Baayen RH, Davidson DJ, Bates DM. Mixed-effects modeling with crossed random effects for subjects and items. *J Mem Lang*. Nov 2008;59(4):390-412. [doi: [10.1016/j.jml.2007.12.005](https://doi.org/10.1016/j.jml.2007.12.005)]

85. Bates D, Mächler M, Bolker B, Walker S. Fitting linear mixed-effects models using lme4. *J Stat Softw*. 2015;67:1-48. [doi: [10.18637/jss.v067.i01](https://doi.org/10.18637/jss.v067.i01)]

86. Team RC. R: a language and environment for statistical computing. R Foundation for Statistical Computing; 2020.

87. Forstmeier W, Schielzeth H. Cryptic multiple hypotheses testing in linear models: overestimated effect sizes and the winner's curse. *Behav Ecol Sociobiol*. Jan 2011;65(1):47-55. [doi: [10.1007/s00265-010-1038-5](https://doi.org/10.1007/s00265-010-1038-5)]

88. Kuznetsova A, Brockhoff PB, Christensen RHB. lmerTest package: tests in linear mixed effects models. *J Stat Soft*. Dec 2017;82(13):1-26. [doi: [10.18637/jss.v082.i13](https://doi.org/10.18637/jss.v082.i13)]

89. Luke SG. Evaluating significance in linear mixed-effects models in R. *Behav Res Methods*. Aug 2017;49(4):1494-1502. [doi: [10.3758/s13428-016-0809-y](https://doi.org/10.3758/s13428-016-0809-y)] [Medline: [27620283](https://pubmed.ncbi.nlm.nih.gov/27620283/)]

90. Feingold A. Effect sizes for growth-modeling analysis for controlled clinical trials in the same metric as for classical analysis. *Psychol Methods*. Mar 2009;14(1):43-53. [doi: [10.1037/a0014699](https://doi.org/10.1037/a0014699)] [Medline: [19271847](https://pubmed.ncbi.nlm.nih.gov/19271847/)]
91. Davison AC, Hinkley DV. *Bootstrap Methods and Their Application*. Cambridge University Press; 1997.
92. Hedeker D, Gibbons RD. Application of random-effects pattern-mixture models for missing data in longitudinal studies. *Psychol Methods*. Mar 1997;2(1):64-78. [doi: [10.1037/1082-989X.2.1.64](https://doi.org/10.1037/1082-989X.2.1.64)]
93. Campbell KL, Grady CL, Ng C, Hasher L. Age differences in the frontoparietal cognitive control network: implications for distractibility. *Neuropsychologia*. Jul 2012;50(9):2212-2223. [doi: [10.1016/j.neuropsychologia.2012.05.025](https://doi.org/10.1016/j.neuropsychologia.2012.05.025)] [Medline: [22659108](https://pubmed.ncbi.nlm.nih.gov/22659108/)]
94. Jacobson NS, Truax P. Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. *J Consult Clin Psychol*. 1991;59(1):12-19. [doi: [10.1037/0022-006X.59.1.12](https://doi.org/10.1037/0022-006X.59.1.12)]
95. International clinical trials registry platform. World Health Organization. URL: <https://trialsearch.who.int/Trial2.aspx?TrialID=DRKS00024408> [Accessed 2025-11-09]
96. Hoorelbeke K, Vervaeke J, Siegle GJ, Baeken C, Koster EHW. Individual differences associated with treatment adherence and transfer effects following gamified web-based cognitive control training for repetitive negative thinking. *Internet Interv*. Mar 2022;27:100507. [doi: [10.1016/j.invent.2022.100507](https://doi.org/10.1016/j.invent.2022.100507)] [Medline: [35242588](https://pubmed.ncbi.nlm.nih.gov/35242588/)]
97. Koster EHW, Hoorelbeke K, Onraedt T, Owens M, Derakshan N. Cognitive control interventions for depression: a systematic review of findings from training studies. *Clin Psychol Rev*. Apr 2017;53:79-92. [doi: [10.1016/j.cpr.2017.02.002](https://doi.org/10.1016/j.cpr.2017.02.002)] [Medline: [28273486](https://pubmed.ncbi.nlm.nih.gov/28273486/)]
98. Lejeuz CW, Kahler CW, Brown RA. A modified computer version of the Paced Auditory Serial Addition Task (PASAT) as a laboratory-based stressor. *Behav Ther*. 2003;26(4):290-293. URL: <https://psycnet.apa.org/record/2003-06608-006> [Accessed 2025-11-19]
99. Tombaugh TN. A comprehensive review of the Paced Auditory Serial Addition Test (PASAT). *Arch Clin Neuropsychol*. Jan 2006;21(1):53-76. [doi: [10.1016/j.acn.2005.07.006](https://doi.org/10.1016/j.acn.2005.07.006)] [Medline: [16290063](https://pubmed.ncbi.nlm.nih.gov/16290063/)]
100. Vander Zwalm Y, Demeester D, Hoorelbeke K, Verhaeghe N, Baeken C, Koster EHW. The more, the merrier? Establishing a dose-response relationship for the effects of cognitive control training on depressive symptomatology. *J Consult Clin Psychol*. Mar 2025;93(3):161-175. [doi: [10.1037/ccp0000945](https://doi.org/10.1037/ccp0000945)] [Medline: [40014506](https://pubmed.ncbi.nlm.nih.gov/40014506/)]
101. Dawson JF, Richter AW. Probing three-way interactions in moderated multiple regression: development and application of a slope difference test. *J Appl Psychol*. Jul 2006;91(4):917-926. [doi: [10.1037/0021-9010.91.4.917](https://doi.org/10.1037/0021-9010.91.4.917)] [Medline: [16834514](https://pubmed.ncbi.nlm.nih.gov/16834514/)]
102. Michalak J, Hölz A, Teismann T. Rumination as a predictor of relapse in mindfulness-based cognitive therapy for depression. *Psychol Psychother*. Jun 2011;84(2):230-236. [doi: [10.1348/147608310X520166](https://doi.org/10.1348/147608310X520166)]
103. Nahum-Shani I, Smith SN, Spring BJ, et al. Just-in-Time Adaptive Interventions (JITAIs) in mobile health: key components and design principles for ongoing health behavior support. *Ann Behav Med*. May 18, 2018;52(6):446-462. [doi: [10.1007/s12160-016-9830-8](https://doi.org/10.1007/s12160-016-9830-8)]
104. Wiers RW, Eberl C, Rinck M, Becker ES, Lindenmeyer J. Retraining automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychol Sci*. Apr 2011;22(4):490-497. [doi: [10.1177/0956797611400615](https://doi.org/10.1177/0956797611400615)] [Medline: [21389338](https://pubmed.ncbi.nlm.nih.gov/21389338/)]
105. Mohlman J, Gorman JM. The role of executive functioning in CBT: a pilot study with anxious older adults. *Behav Res Ther*. Apr 2005;43(4):447-465. [doi: [10.1016/j.brat.2004.03.007](https://doi.org/10.1016/j.brat.2004.03.007)]
106. Kessler RC, Ormel J, Petukhova M, et al. Development of lifetime comorbidity in the World Health Organization world mental health surveys. *Arch Gen Psychiatry*. Jan 2011;68(1):90-100. [doi: [10.1001/archgenpsychiatry.2010.180](https://doi.org/10.1001/archgenpsychiatry.2010.180)] [Medline: [21199968](https://pubmed.ncbi.nlm.nih.gov/21199968/)]
107. Gräfe K, Zipfel S, Herzog W, Löwe B. Screening for psychiatric disorders with the Patient Health Questionnaire (PHQ-D)“. *Diagnostica*. Oct 2004;50(4):171-181. [doi: [10.1026/0012-1924.50.4.171](https://doi.org/10.1026/0012-1924.50.4.171)]
108. Anderson EM, Lambert MJ. A survival analysis of clinically significant change in outpatient psychotherapy. *J Clin Psychol*. Jul 2001;57(7):875-888. [doi: [10.1002/jclp.1056](https://doi.org/10.1002/jclp.1056)] [Medline: [11406801](https://pubmed.ncbi.nlm.nih.gov/11406801/)]

## Abbreviations

**aPASAT:** adaptive version of the PASA  
**CEQ:** Credibility Expectancy Questionnaire  
**CERQ:** Cognitive Emotion Regulation Questionnaire  
 **CONSORT:** Consolidated Standards of Reporting Trials  
**DERS-16:** Difficulties in Emotion Regulation Scale 16  
**DSM-5:** *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition)  
**GAD-7:** Generalized Anxiety Disorder Questionnaire  
**HSCL-11:** Hopkins Symptom Checklist-11  
**ISI:** interstimulus interval

**mini-SPIN:** Mini-Social Phobia Inventory

**PASAT:** Paced Auditory Serial Addition Test

**PHQ-9:** Patient Health Questionnaire 9

**PTQ:** Perseverative Thinking Questionnaire

**RDoC:** Research Domain Criteria

**RSQ:** Response Style Questionnaire

**SAD:** Social anxiety disorder

**SOR:** Speed of Response Task

**WHOQOL-BREF:** World Health Organization Quality of Life Brief Form

**WSAS:** Work and Social Adjustment Scale

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