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REVIEW

Treatment Response Following Adaptive PASAT Training for Depression Vulnerability: a Systematic Review and Meta-Analysis

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Abstract

In recent years, cognitive control training (CCT) has gained momentum as an intervention to remediate cognitive impairments and decrease depressive symptoms. One promising operationalization to train cognitive control is the adaptive Paced Auditory Serial Addition Task (aPASAT). In this systematic review and meta-analysis of aPASAT training, the efficacy of the intervention and potential moderators were examined. The PsycINFO, MEDLINE, Embase, Web of Science and Cochrane Library electronic databases were searched for studies examining aPASAT training for depressive symptomatology or rumination. Nineteen studies (n = 1255) were included, comprising of depressed patients, remitted depressed patients, at-risk, and healthy participants. We found small significant effects directly after training for both depressive symptomatology and rumination, with similar effect sizes at follow-up. Subgroup analyses suggest a significantly higher mean effect of aPASAT training in non-healthy populations for rumination immediately following training, but not for depressive symptomatology. The amount of training sessions did not moderate effects of CCT. aPASAT has a small but significant effect on depressive symptoms, with direct effects immediately after training, as well as sustained long-term effects. It is currently unclear how many sessions are required for sustained effects due to heterogeneity in training dosage and absence of sufficient trials. Our results suggest that aPASAT training may be most effective for at-risk, remitted- and clinically depressed populations. The effect sizes resulting from this meta-analysis could be used to adequately power future research, which could investigate a dose-response relationship and examine potential treatment gains when combining CCT with other antidepressant interventions.

Keywords Cognitive control training · Depression · Recurrence of depression · Meta-analysis · Working memory training · Cognitive remediation · Rumination

Introduction

Depression is known to be a highly common and severe psychiatric illness that has both detrimental individual consequences (e.g., suicide, substantial individual suffering), as well as a high societal cost (e.g., decreased school or work performance, high healthcare costs) (Kessler, 2012). Frequently used treatments for depression include pharmacological interventions, such as anti-depressant medication, and psychotherapy, such as cognitive behavioral therapy (CBT), which are moderately effective interventions in the acute phase (Cipriani et al., 2018; Gautam et al., 2020), but often fail to prevent the recurrence of depression later in life (Lorimer et al., 2019). It appears that these therapies insufficiently target mechanisms governing depression vulnerability, resulting in remaining residual symptoms and high relapse rates which can give rise to increasing psychosocial impairments (Bockting et al., 2015). Therefore, investigation of possible interventions to target the core vulnerabilities of depression is paramount for prevention of initial episodes of depression, as well as recurrent depression.

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Cognitive impairment is a known core factor of depression, shown through deficits in cognitive processing and emotion regulation (for review, see Gotlib & Joormann 2010; LeMoult & Gotlib, 2019). There is ample research showing impairments in working memory and cognitive inflexibility (Rose & Ebmeier, 2006), increased rumination (Nolen-Hoeksema, 2000) and increased repetitive negative thinking (RNT; Kertz et al., 2015; Lewis et al., 2019). RNT seems to be a key risk factor for cognitive impairments in the context of depression and also appears to be difficult to treat by interventions not specifically targeting RNT (Spinhoven et al., 2018). Interestingly, cognitive impairment (CI) has been linked with poor response to antidepressant treatment (Story et al., 2008) and with an unfavorable outcome of CBT (Porter et al., 2015).

One way of tackling these cognitive impairments is using cognitive control training (CCT), which has shown promising results on rumination (Hoorelbeke et al., 2015; Siegle et al., 2007) and on depressive symptomatology (Iacoviello et al., 2014; Siegle et al., 2007). CCT typically takes place through an automated computer program, often accessible via the internet. Therefore, the training can easily be disseminated. Other advantages of CCT over existing treatments for depression are relatively inexpensiveness and the absence of concern for side effects, as opposed to pharmacological interventions (Motter et al., 2016).

CCT can be operationalized in different ways, among which the Eriksen flanker task (Eriksen & Eriksen, 1974) and the n-back task (Kirchner, 1958) have been examined in multiple studies. Currently one of the most frequently used CCT procedures in depression is the Paced Auditory Serial Addition Task (PASAT), which was originally developed as a measure of recovery after head-injury (Gronwall, 1977) and is frequently used as a diagnostic tool for multiple sclerosis (Rogers & Panegyres, 2007). During the PASAT, participants are presented auditory stimuli (numbers ranging from 1 to 9) and are asked to continuously respond to the sum of the last two heard digits. This requires participants to keep these auditory stimuli in their working memory, perform the addition and discard previous (i.e., no longer relevant) stimuli, such as previously heard auditory stimuli, as well as the previous sums. In the context of depression, an adaptive version of the PASAT is proposed (Siegle et al., 2007) that is widely investigated (for review, see Koster et al., 2017). Where, in the regular PASAT, participants are presented with auditory stimuli at a fixed pace (e.g., 3000 ms), the adaptive PASAT (aPASAT) tailors the task to the performance of the individual by reducing the speed of stimulus presentation by 100 ms after four consecutive correct responses and increasing the speed by 100 ms after four consecutive incorrect responses.

A wide range of research has examined the effects of aPASAT training on depressive symptoms. Siegle et al. (2007) were the first to use a two-week aPASAT training in a sample of depressed individuals and found improvements in depressive symptomatology and decreases in rumination when compared to a treatment as usual (TAU) control condition. In later studies, the beneficial results of CCT on depressive symptomatology using an aPASAT training were replicated (Hoorelbeke & Koster, 2017; Siegle et al., 2014), where they also found decreases in clinical care needs after a one-year follow-up (Siegle et al., 2014; Hoorelbeke et al., 2021). These findings suggest that aPASAT training targets cognitive impairments associated with depression and that CCT is beneficial in the prevention of the recurrence of depression. To our knowledge, no previous meta-analysis examined the efficacy of an aPASAT training on depression vulnerability. The advantage of performing a meta-analysis on one specific form of CCT training is that task-specific heterogeneity is low, which sidesteps the uncertainty that comes with examining several forms of CCT in one meta-analysis and could potentially confound the origin of the treatment effects.

The mechanisms by which CCT influences depression vulnerability are not yet well understood. Models of rumination have argued that individuals exert less attentional control over processing of emotional, self-relevant information (Gotlib et al., 2004; Koster et al., 2011). When confronted with a stressor, negative thinking can occur and usually, after some time, most people have the ability to disengage from this type of negative thinking and realign with positive self-views. When attentional disengagement fails and an inward focus of prolonged rumination occurs, this can lead to the development of a depressive episode. Indeed, persistent negative thinking is a core factor of depression, and rumination has been shown to play an important mediating role linking cognitive control impairments with recurrence of depressive symptoms (Demeyer et al., 2012). By training cognitive control, one could potentially facilitate the use of attentional disengagement from repetitive negative thinking and reduce effects on depression. This model could be linked neuropsychologically to the structures involved with cognitive control. A cognitive neuroscience framework for increased risk for recurrence of depression proposed by De Raedt and Koster (2010) posits that biological factors (such as hypercortisolism) after prolonged periods of stress result in impaired serotonergic neurotransmission, which can lead to unadjusted patterns of prefrontal activity. The dorsolateral prefrontal cortex (DLPFC) has been repeatedly associated with executive functioning and has a central role in emotion regulation by regulating activity in the limbic regions. Unadjusted levels of prefrontal functioning can lead to increased amygdala reactivity, which has often been observed in depression (Pizzagalli & Roberts, 2021), and on a behavioral level has been linked with cognitive control impairments. CCT could potentially improve DLPFC's functioning to

regulate emotional activity and, as such, improve attentional disengagement of negative self-referent cognition.

Previous research provides support that corticolimbic connectivity could be significantly increased by CCT and that reduced amygdala reactivity following training was predictive of behavioral and clinical improvements (Cohen et al., 2016; Hoch et al., 2019). To help remediate reduced DLPFC function, transcranial direct-current stimulation (tDCS) in combination with CCT has also been used in the context of depression. Several studies looked at the combination of aPASAT training and tDCS (Brunoni et al., 2014; Segrave et al., 2014; Sommer & Plewnia, 2021; Vanderhasselt et al., 2015), after which participants scored lower on measures of depressive symptomatology. However, in these studies, the control groups (CCT + sham tDCS) also improved, indicating that both CCT alone and the combination of CCT and tDCS appeared effective. Importantly, Sommer and Plewnia (2021) suggest that a potential small effect of tDCS might be obscured by a more prominent CCT effect.

In CCT studies, participants show substantial heterogeneity in treatment response. Several factors might play a role, such as the context of how CCT is administered. Furthermore, it is currently unclear for whom an aPASAT training is beneficial in reducing depression vulnerability. Previous research with the aPASAT has been done in several population types, such as patients with major depressive disorder (MDD), remitted depressed patients (RMD), at-risk (e.g., people scoring high on trait rumination), and healthy participants. It is possible that the observed heterogeneity in the literature might in part be attributable to differences in population type. We hypothesize that aPASAT training could have more pronounced effects on depression vulnerability in at-risk, RMD and MDD populations compared to healthy populations, with most pronounced effects to be expected in clinical populations, considering that people with the highest cognitive impairments could have the most to gain. Another area that has not yet been explored is the required amount of CCT sessions to observe a reduction in depression vulnerability. Siegle et al. (2007) were the first to use the aPASAT as a CCT in combination with an attention training and used six sessions, but more recent studies have mostly used a higher aPASAT dose (usually ten sessions). The effects of a higher CCT dose on depression vulnerability is currently unclear as no dose-response studies have yet been conducted. This meta-analysis aims to investigate the overall effectiveness of the aPASAT as a CCT procedure for depressive symptomatology and rumination, immediately after the training, as well as examine long-term effects. In addition, follow-up analyses will be conducted to determine effects of study population and training dosage. Shedding light on these key concepts is imperative to the consideration of the use of an aPASAT training as a preventative intervention for depression.

Method

For this study, PRISMA guidelines for reporting systematic reviews were followed (Page et al., 2021). Preregistered information of the design of this study can be found on PROSPERO with identifier CRD42021245971. Data and script to replicate this meta-analysis can be found on Open Science Framework (OSF): https://osf.io/dhqfk.

Literature Search Strategy

Searches were first performed on 29/03/2021 and later repeated on 28/09/2021 in the PsycINFO, MEDLINE, Embase, Cochrane Library and Web of Science electronic databases, where the following string was used to search at the title and abstract level: *("cognitive control therapy" OR CCT OR "cognitive control training" OR "cognitive control task" OR "neurocognitive training" OR "cognitive training" OR "executive control training" OR "working memory training" OR "cognitive emotional training" OR "cognitive remediation" OR "neurobehavioral therapy" OR PASAT OR "Paced Auditory Serial Addition Task") AND (depress* OR "negative repetitive thinking" OR "repetitive negative thinking" OR ruminat* OR "negative mood" OR brooding)*. Reference lists of included articles were screened for other relevant studies and a snowballing approach was used on the first authors of the selected studies to identify other potential articles. To reduce the impact of publication bias, grey literature such as PhD theses were also considered. In addition, first authors of the selected studies were contacted and additional unpublished manuscripts in which effects of aPASAT training were evaluated on depressive symptomatology or repetitive negative thinking were considered for inclusion.

Inclusion and Exclusion Criteria

Studies were included if they: (i) were written in English, (ii) included an experimental manipulation of cognitive control based on the adaptive Paced Auditory Serial Addition Task (aPASAT) and (iii) evaluated the effects of aPASAT training on repetitive negative thinking or depressive symptomatology in a healthy, at-risk (e.g. showing subclinical levels of depressive symptomatology, elevated trait rumination scores, children of parents with MDD, etc.), clinically depressed, or remitted depressed (RMD) sample. Studies with a primary focus on anxiety, substance abuse, neurological and psychotic disorders were excluded.

Study Selection and Data Extraction

The systematic search resulted in 3632 initial hits, from which titles and abstracts were exported from the electronic databases and imported into systematic review manager software Covidence (Veritas Health Innovation, Melbourne, n.d.) for review. After removal of duplicate articles ($n = 1720$), 1912 studies remained. Two researchers (YVZ & EL) then examined the titles and abstracts against the inclusion and exclusion criteria. After the selection on title and abstract level, 91 studies were chosen for a full-text review, of which 17 studies were selected to be included in our meta-analysis. After contacting authors of the included studies, two unpublished studies at the time of the literature search were uncovered. This grey literature was then added to the meta-analysis. Of all 19 included studies, 18 were randomized controlled trials (RCT). One study was categorized as a single-arm trial (Hoorelbeke et al., 2022b). The search and selection process is depicted in a PRISMA flow diagram (see Fig. 1). An inter-rater reliability analysis using Cohen's Kappa was performed to determine consistency between the two independent raters.

Methodological Quality Assessment

The Downs and Black checklist (Downs & Black, 1998) for risk of bias was used independently by two reviewers (YVZ & EL) to assess the quality of the included studies. When the two reviewers had different opinions about risk of bias, a third reviewer (KH) served as arbitrator, as an unbiased reconciliation method (conform recommendations of the Agency for Healthcare Research and Quality; Viswanathan et al., 2018).

To check for indications of the presence of publication bias, a funnel plot was created for the primary outcome (i.e., depressive symptomatology) at post measure. Egger's test (Egger et al., 1997) was conducted to examine the asymmetry of the funnel plot and Duval and Tweedie's trim and fill procedure (Duval & Tweedie, 2000) was performed to check the estimate of the effect size after taking possible publication bias into account. To grade the quality of the evidence, the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system was used (Brożek et al., 2009).

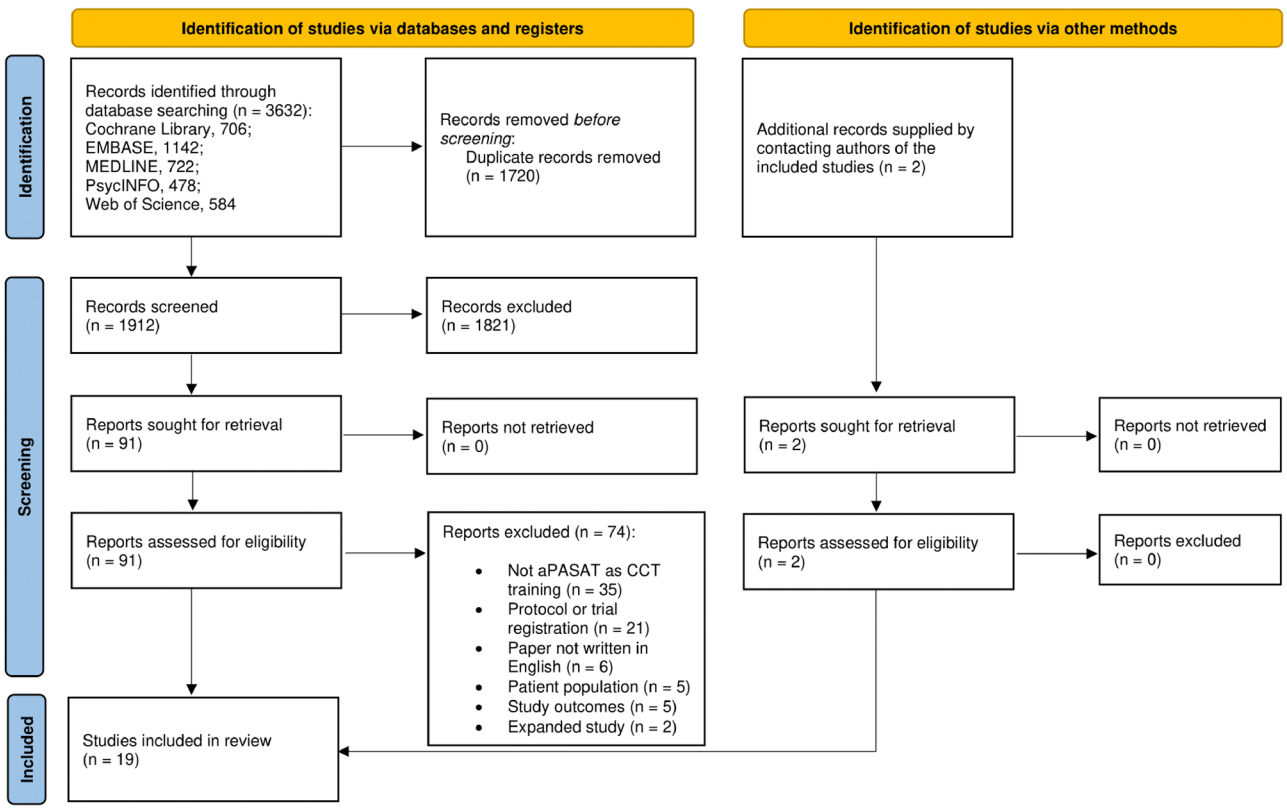

Fig. 1 PRISMA flow diagram

Statistical Analysis

Analyses were conducted in R, version 4.0.5 (R Core Team, 2021). A list of the used R packages and their version numbers can be found in the supplementary material (Table A1). For each of the selected studies, Hedges' g effect sizes were calculated for depressive symptomatology and rumination. Hedges' g was chosen over Cohen's d, because it provides a correction for positive bias in small samples. The effect sizes were interpreted according to Cohen's recommendations (i.e., 0.2 as 'small', 0.5 as 'medium' and 0.8 as 'large'; Cohen, 1988). For studies incorporating multiple conditions, the CCT group was compared to the most comparable control group. For these between-subject designs, effect sizes were calculated using post measure means and their standard deviations, with the esc_mean_sd function from the esc package in R (Lüdecke, 2019). For four studies, where no suitable control group was available (Brunoni et al., 2014; Sommer & Plewnia, 2021; Vanderhasselt et al., 2015; Hoorelbeke et al., 2022b), individual participant data was requested from the authors to calculate pre-post correlations (see supplementary Table A2). Effect sizes were then calculated from pre-post measures, taking their correlations into account (Harrer et al., 2021; Hedges et al., 2009). The search strategy also returned two studies which utilized an Experience Sampling Method (ESM; Hoorelbeke et al., 2016, 2021) design, for which aggregated means were calculated to calculate the standardized mean differences. When a study employed more than one measure to assess the same outcome, effect sizes were first pooled within studies before effect sizes were pooled across studies, by using the mean of the effect sizes.[1] This was the case for Brunoni et al., 2014, Moshier & Otto, 2017, Sommer & Plewnia, 2021 and Vervaeke et al., 2021.

To calculate pooled effect sizes, random-effects models using the Hartung-Knapp-Sidik-Jonkman method were performed in R using the metagen package (Balduzzi et al., 2019). Two sensitivity analyses were conducted on the primary outcome at post measure: one to check if excluding the within-subject studies would influence the results and another excluding the studies using less than five CCT sessions. Heterogeneity in the models is reported using τ^2 for the between-study variance and using the I^2 statistic, which tests the homogeneity of effect sizes (i.e., the percentage of the variance that is not attributed to random error or chance). I^2 is always reported with its 95% confidence intervals (Cuijpers, 2016; Ioannidis et al., 2007). An $I^2 < 30\%$ was considered as low heterogeneity, between 31 and 60% as moderate, between 61 and 75% as substantial and between 76 and 100% as considerable. Heterogeneity for the primary outcome at post measure was visualized with a Baujat plot (Baujat et al., 2002) to visually inspect which studies contributed the most to the heterogeneity.

To examine the heterogeneity introduced by the different population types, a subgroup analysis was performed on the study population: healthy, at-risk (e.g., showing subclinical levels of depressive symptomatology), depressed (MDD), or remitted depressed (RMD). Classification by study population was done by two independent raters (YVZ & EL) and a third reviewer (KH) served as arbitrator. A meta-regression analysis was done to examine the association between the number of aPASAT sessions and depression vulnerability.

To summarize the meta-analysis, forest plots with the effect sizes and 95% confidence intervals of the individual studies, as well as an overall pooled effect size and its 95% confidence interval, were created for both the primary and secondary outcomes, for post and follow-up measures. Follow-up measures ranged widely in time, from 2 weeks to 12 months ($M = 3.6$ months, $SD = 4.0$ months). To conclude, clinical significance was elucidated by converting the pooled effect size of the primary outcome at post measure into the Number Needed to Treat (NNT). This was calculated with the R package dmetar (Harrer et al., 2019), using the Kraemer & Kupfer method (Kraemer & Kupfer, 2006).

Results

Study Characteristics

This meta-analysis examined 19 studies, comprising a total of 1255 participants. For the determination of the inclusion of studies, a high degree of inter-rater reliability between the two independent researchers was obtained ($\kappa = 0.86$). One study (Siegle et al., 2007) was relevant and passed inclusion criteria, but was not included in the meta-analysis, because a later study (Siegle et al., 2014) expanded the original sample in an extension study and thus, this later study was included. In addition, Brunoni et al. (2014) and Vanderhasselt et al. (2015) reported results of one RCT, each focusing on different outcomes. As such, Brunoni et al. (2014) was included in the meta-analysis for the primary outcome measure (depressive symptomatology), whereas for the analysis pertaining the secondary outcome measure (rumination) Vanderhasselt et al. (2015) was used. Furthermore, one study (Moshier et al., 2015) passed inclusion criteria, but insufficient data was available to accurately calculate effect sizes and was thus not included in our meta-analysis. Finally, Hoorelbeke et al. (2022a) presents immediate effects of aPASAT training

[1] To evaluate the impact of this analytical strategy, the standardized mean differences of these studies were also analyzed without first pooling the same outcomes within studies before pooling across studies. This did not meaningfully change the pooled effect size. As such, first pooling within studies was kept as this uses all available data.

in daily life, relying on ESM measures, whereas Hoorelbeke et al. (2021) presents effects of aPASAT training at one year follow-up. For this purpose, Hoorelbeke et al. (2022a) has been included for post measure data, while follow-up data was retrieved from Hoorelbeke et al. (2021).

Across all incorporated studies, around 72% of participants were female, with a mean age of 37.9 (SD = 12.7). The sample sizes which were used for the calculation of effect sizes ranged from n = 9 (Segrave et al., 2014) to n = 213 (Hoorelbeke et al., 2022b), with a median of n = 49 (IQR = 50). Of the 19 included studies, 7 (36.8%) focused on a major depressed population, 4 (21.1%) on remitted depressed patients, 5 on an at-risk population (26.3%) and 3 examined effects of aPASAT training in a healthy sample (15.8%). For the determination of the sample population, a high degree of inter-rater reliability between the two independent researchers was obtained ($\kappa = 0.93$). Most studies (n = 14) used a general age group between the ages 18 and 65 (74%), while four studies examined a younger population (21%) and one study used an older sample (5%). Table 1 presents the characteristics of the included studies and the extracted outcomes. Included N in the table refers to number of participants for which data was reported and available for the calculation of effect sizes, only taking comparable groups into account.

A total of 57 effect sizes were extracted: 34 for the primary outcome depressive symptomatology (18 for post measure, 16 for follow-up), 23 for the secondary outcome rumination (14 for post measure, 9 for follow-up). When studies had multiple groups, the most comparable or most conservative control group was used. For instance, Lass et al. (2021) had three groups: CCT, active control and waitlist control: CCT and active control groups were used for the calculation of effect sizes. The study from Sommer and Plewnia (2021) had three groups: CCT + sham tDCS, CCT + 1mA tDCS and CCT + 2mA tDCS: only the CCT and sham tDCS groups were used. Table 2 summarizes the extracted data and the calculated effect sizes.

Risk of Bias

Assessment of risk of bias between the two independent raters (YVZ & EL) was very similar. Of the 19 examined studies, only three resulted in a different assessment category, each only differing by one point on the Downs and Black checklist. A third reviewer (KH) provided the final decision on these three items. The risk of bias in the examined studies was overall adequate. Reporting, blinding and randomization were mostly satisfactory. However, only 12 out of the 19 studies performed a statistical power analysis, usually assuming a moderate to high effect size in their power calculations. Possible adverse effects were not widely addressed in the aPASAT literature. It is possible that because very few adverse effects are expected for a CCT procedure, this has not received much attention in previous research. The resulting assessment concluded that no studies scored in the "poor" category, 3 studies scored "fair" (15.8%), 14 studies scored "good" (73.7%) and 2 scored "excellent" (10.5%). See Fig. 2 for a summary of the risk of bias assessment.

Effect of aPASAT Training on Depressive Symptomatology

15 studies examined depressive symptomatology at post, resulting in a pooled effect size of g = 0.29 with a 95% confidence interval [0.06, 0.52]. The pooled effect size was significant ($p = .018$) and heterogeneity was moderate ($\tau^2 = 0.13$, $I^2 = 54.7\%$, 95% CI [18.8%, 74.7%]). At follow-up, 11 studies resulted in a pooled effect size of g = 0.44 with a 95% confidence interval [0.04, 0.83]. The pooled effect size for follow-up was also significant ($p = .033$) and heterogeneity was substantial ($\tau^2 = 0.28$, $I^2 = 69.4\%$, 95% CI [42.9%, 83.6%]). For both measures, the Sommer and Plewnia (2021) study was a small outlier. Using a "leave-one-out" analysis and excluding this study, the pooled effect sizes changed from g = 0.29 [0.06, 0.52] ($p = .017$) to g = 0.23 [0.04, 0.43] ($p = .024$) at post and from g = 0.44 [0.04, 0.83] ($p = .033$) to g = 0.33 [0, 0.65] ($p = .051$) at follow-up. Figure 3 displays the forest plot for depressive symptomatology at post and Fig. 4 presents the forest plot for follow-up.

A sensitivity analysis was conducted on the post measure of depressive symptomatology and revealed that excluding the within subjects design studies (N = 4; i.e., Brunoni et al., 2014, Moshier & Otto, 2017, Sommer & Plewnia, 2021 and Vervaeke et al., 2021) from the overall analysis altered the pooled effect side from g = 0.29 (95% CI [0.06, 0.52], $p = .017$) to g = 0.17 (95% CI [-0.02, 0.36], $p = .071$). The second sensitivity analysis, excluding the studies using less than five training sessions, changed the pooled effect side from g = 0.29 (95% CI [0.06, 0.52], $p = .017$) to g = 0.35 (95% CI [0.08, 0.62], $p = .016$).

Heterogeneity was visually inspected for the primary outcome at post measure, using a Baujat plot (see Figure A3 in supplementary material), which revealed that several studies (Brunoni et al., 2014; Hoorelbeke et al., 2016; Sommer & Plewnia, 2021) contributed significantly to the heterogeneity. The former and latter studies present high heterogeneity in this meta-analysis due to the absence of a comparable control group, which resulted in within-subjects effect sizes of small samples. For Hoorelbeke et al. (2016), the heterogeneity might be explained by the type of measure used. This study used an ESM design to map depressive affect over time in a healthy, convenience sample. Indeed, people can experience a variety of emotions at different times, which can result in significant variability when calculating effect sizes on aggregated ESM means.

Table 1 Characteristics of the included studies

Studies	Country	Sample	N (included N)	Percentage female	Number of sessions	Outcomes	Follow-up (months)	Control group	Mean age (SD)	RoB assessment
Brunoni et al. (2014)	Brazil	MDD	40 (17)	30.8%	10	BDI-II, HDRS	0.5	Within-group	41.5 (10.6)	Excellent
Calkins et al. (2015)	USA	At-risk	56 (48)	54.2%	3	BDI-II	/	Non-active control	35.7 (14.7)	Good
Ferrari et al. (2021)	Germany	MDD	115 (115)	62.6%	10	BDI-II, RSQ	12	Active control	51.3 (7.4)	Good
Hoorelbeke et al. (2015)	Belgium	At-risk	53 (47)	92.5%	10	BDI-II, RRS	1	Active control	20.6 (2.1)	Good
Hoorelbeke et al. (2016)	Belgium	Healthy	61 (61)	85.3%	10	VAS	/	Active control	21.4 (2.5)	Good
Hoorelbeke and Koster (2017)	Belgium	RMD	68 (68)	66.2%	10	BDI-II, RRS	3	Active control	47.0 (11.5)	Excellent
Hoorelbeke et al. (2021)	Belgium	RMD	92 (82)	64.1%	10	BDI-II, RRS	12	Active control	45.8 (12.8)	Good
Hoorelbeke et al. (2022a)	Belgium	RMD	92 (84)	64.1%	10	VAS	/	Active control	45.8 (12.8)	Good
Hoorelbeke et al. (2022b)	Belgium	At-risk	213 (213)	73.0%	10	DASS, PTQ	1	Within-group	43.1 (13.3)	Good
Lass et al. (2021)	USA	At-risk	72 (47)	75.0%	6	BDI, RRS	/	Active control	21.8 (5.7)	Good
Moshier and Otto (2017)	USA	MDD	34 (34)	52.0%	4	BDI-II, MADRS, RRS	1	Non-active control	35.6 (14.6)	Good
Segrave et al. (2014)	Australia	MDD	27 (18)	37.0%	5	BDI-II	0.75	Non-active control	40.4 (14.5)	Good
Siegle et al. (2014)	USA	MDD	51 (43)	69.8%	6	BDI-II, RSQ	/	Non-active control	39.5 (10.4)	Fair
Sommer and Plewnia (2021)	Germany	MDD	51 (17)	58.8%	12	BDI, MADRS	3	Within-group	34.6 (13.7)	Fair
Van den Bergh et al. (2020)	Belgium	At-risk	102 (89)	79.7%	10	DASS, PTQ	2	Active control	20.78 (2.3)	Good
Vanderhasselt et al. (2015)	Brazil	MDD	37 (14)	79.0%	10	BDI-II, RRS	/	Within-group	41.0 (11.5)	Good
Vanderhasselt et al. (2020)	Belgium	Healthy	41 (41)	53.9%	10	RRS	1.5	Active control	70.3 (3.8)	Fair
Vervaeke et al. (2020)	Belgium	Healthy	147 (147)	74.1%	10	DASS, RRS	/	Active control	21.4 (4.5)	Good
Vervaeke et al. (2021)	Belgium	RMD	68 (68)	64.7%	10	BDI-II, DASS, RRS	3, 6	Active control	39.9 (17.6)	Good

Included N refers to the sample that was used for effect size calculation in this meta-analysis. BDI, Beck Depression Inventory; BDI-II, Beck Depression Inventory – 2nd edition; DASS, Depression Anxiety Stress Scales - depression subscale; HDRS, Hamilton Depression Rating Scale; MADRS, Montgomory Asberg Depression Rating Scale; MDD, Major Depressive Disorder; PTQ, Perseverative Thinking Questionnaire; RMD, Remitted depressed; RoB, Risk of Bias; RRS, Ruminative Response Scale; RSQ, Response Style Questionnaire; VAS, Visual Analogue Scale

Table 2 Extracted data and calculated effect sizes

Study	Outcome	Measure	N CCT	Mean CCT	SD CCT	N control	Mean control	SD control	Hedges' g (SE)	Timing
Brunoni et al. (2014)	Depressive sympt.	BDI-II	15	23.00	12.00	15	34.00	8.00	0.98 (0.29)	post
Brunoni et al. (2014)	Depressive sympt.	HDRS	17	20.00	9.00	17	27.00	5.70	1.11 (0.35)	post
Brunoni et al. (2014)	Depressive sympt.	BDI-II	15	24.00	11.40	15	34.00	8.00	1.00 (0.31)	follow-up
Brunoni et al. (2014)	Depressive sympt.	HDRS	17	20.00	8.70	17	27.00	5.70	1.14 (0.35)	follow-up
Calkins et al. (2015)	Depressive sympt.	BDI-II	24			24			0.72 (0.30)	post
Ferrari et al. (2021)	Depressive sympt.	BDI-II	56	17.28	9.50	59	19.40	11.58	0.20 (0.19)	post
Ferrari et al. (2021)	Rumination	RSQ	56	50.23	10.24	59	51.25	13.48	0.08 (0.19)	post
Hoorelbeke et al. (2015)	Depressive sympt.	BDI-II	25	12.12	11.33	22	9.41	7.84	-0.27 (0.29)	follow-up
Hoorelbeke et al. (2015)	Rumination	RRS	25	48.88	11.27	22	50.95	9.88	0.19 (0.29)	post
Hoorelbeke et al. (2015)	Rumination	RRS	20	50.05	10.16	17	51.24	8.74	0.12 (0.33)	follow-up
Hoorelbeke et al. (2016)	Depressive sympt.	VAS	29	10.44	10.09	32	8.12	6.65	-0.27 (0.26)	post
Hoorelbeke et al. (2016)	Rumination	VAS	29	28.05	14.50	32	27.07	12.76	-0.07 (0.26)	post
Hoorelbeke and Koster (2017)	Depressive sympt.	BDI-II	34	5.38	7.10	34	9.29	9.37	0.47 (0.25)	post
Hoorelbeke and Koster (2017)	Depressive sympt.	BDI-II	34	4.50	5.10	34	9.29	7.28	0.75 (0.25)	follow-up
Hoorelbeke and Koster (2017)	Rumination	RRS	34	34.71	10.01	34	44.29	12.58	0.83 (0.25)	post
Hoorelbeke and Koster (2017)	Rumination	RRS	34	30.06	6.72	34	40.35	14.06	0.92 (0.26)	follow-up
Hoorelbeke et al. (2021)	Depressive sympt.	BDI-II	43	9.51	8.75	39	11.38	10.16	0.20 (0.22)	follow-up
Hoorelbeke et al. (2021)	Rumination	RRS	43	45.95	14.44	39	48.90	13.30	0.21 (0.22)	follow-up
Hoorelbeke et al. (2022a)	Depressive sympt.	VAS	46	10.36	10.51	38	15.06	16.82	0.34 (0.22)	post
Hoorelbeke et al. (2022a)	Rumination	VAS	46	22.15	17.34	38	29.47	21.47	0.38 (0.22)	post
Hoorelbeke et al. (2022b)	Depressive sympt.	DASS	213	9.29	8.80	213	12.60	9.32	0.29 (0.06)	post
Hoorelbeke et al. (2022b)	Rumination	PTQ	213	34.98	9.08	213	39.43	8.22	0.42 (0.06)	post
Hoorelbeke et al. (2022b)	Depressive sympt.	DASS	213	9.27	9.10	213	12.60	9.32	0.33 (0.06)	follow-up
Hoorelbeke et al. (2022b)	Rumination	PTQ	213	31.77	9.15	213	39.43	8.22	0.79 (0.07)	follow-up
Moshier and Otto (2017)	Depressive sympt.	BDI-II	21	23.10	10.80	13	18.60	13.30	-0.37 (0.36)	post
Moshier and Otto (2017)	Depressive sympt.	BDI-II	21	22.00	11.80	13	18.20	14.60	-0.29 (0.35)	follow-up
Moshier and Otto (2017)	Depressive sympt.	MADRS	21	19.76	11.10	13	17.00	9.90	-0.25 (0.35)	post
Moshier and Otto (2017)	Depressive sympt.	MADRS	21	20.00	9.80	13	17.50	11.70	-0.23 (0.35)	follow-up
Moshier and Otto (2017)	Rumination	RRS	21	54.60	12.20	13	48.90	15.20	-0.42 (0.36)	post
Moshier and Otto (2017)	Rumination	RRS	21	52.00	11.20	13	44.70	14.30	-0.57 (0.21)	follow-up
Segrave et al. (2014)	Depressive sympt.	BDI-II	9	23.44	12.37	9	25.33	10.05	0.16 (0.47)	post
Segrave et al. (2014)	Depressive sympt.	BDI-II	9	15.33	10.37	9	27.78	8.61	1.24 (0.52)	follow-up
Siegle et al. (2014)	Depressive sympt.	BDI-II	23	24.13	12.14	20	30.55	11.31	0.54 (0.31)	post
Siegle et al. (2014)	Rumination	RSQ	23	49.70	9.52	20	59.80	9.87	1.02 (0.33)	post
Sommer and Plewnia (2021)	Depressive sympt.	BDI	17	17.35	7.54	17	25.59	6.68	1.08 (0.29)	post
Sommer and Plewnia (2021)	Depressive sympt.	MADRS	17	18.35	8.85	17	29.0	7.45	1.41 (0.36)	post
Sommer and Plewnia (2021)	Depressive sympt.	BDI	17	14.73	8.41	17	25.59	6.68	1.26 (0.30)	follow-up

Table 2 (continued)

Study	Outcome	Measure	N CCT	Mean CCT	SD CCT	N control	Mean control	SD control	Hedges' g (SE)	Timing
Sommer and Plewnia (2021)	Depressive sympt.	MADRS	17	15.87	8.94	17	29.0	7.45	2.07 (0.47)	follow-up
Van den Bergh et al. (2020)	Depressive sympt.	DASS	47	6.10	5.50	42	5.80	5.10	-0.06 (0.21)	post
Van den Bergh et al. (2020)	Depressive sympt.	DASS	47	5.90	5.00	42	5.80	4.90	-0.02 (0.21)	follow-up
Van den Bergh et al. (2020)	Rumination	PTQ	47	35.90	10.10	42	37.10	9.80	0.12 (0.21)	post
Van den Bergh et al. (2020)	Rumination	PTQ	47	33.90	9.90	42	35.30	9.80	0.14 (0.21)	follow-up
Vanderhasselt et al. (2015)	Rumination	RRS	14	58.57	13.25	14	66.00	8.44	0.67 (0.30)	post
Vanderhasselt et al. (2020)	Rumination	RRS	21	35.29	9.54	20	34.89	11.17	-0.04 (0.31)	post
Vanderhasselt et al. (2020)	Rumination	RRS	21	33.52	10.29	20	35.82	14.75	0.18 (0.31)	follow-up
Vervaeke et al. (2020)	Depressive sympt.	DASS	74	18.74	5.78	73	19.89	6.56	0.19 (0.17)	post
Vervaeke et al. (2020)	Rumination	RRS	74	41.58	11.93	73	42.11	10.76	0.05 (0.17)	post
Vervaeke et al. (2021)	Depressive sympt.	BDI-II	34	9.00	5.70	34	9.00	6.50	0.00 (0.24)	follow-up
Vervaeke et al. (2021)	Depressive sympt.	DASS	34	6.50	7.90	34	5.90	7.90	-0.08 (0.24)	post
Vervaeke et al. (2021)	Depressive sympt.	DASS	34	4.50	4.80	34	7.70	8.50	0.46 (0.25)	follow-up
Vervaeke et al. (2021)	Depressive sympt.	DASS	34	4.80	5.40	34	7.60	7.60	0.42 (0.25)	follow-up
Vervaeke et al. (2021)	Rumination	RRS	34	50.50	10.30	34	51.90	12.22	0.12 (0.24)	post
Vervaeke et al. (2021)	Rumination	RRS	34	46.10	12.00	34	45.90	12.50	-0.02 (0.24)	follow-up
Vervaeke et al. (2021)	Rumination	RRS	34	48.10	12.80	34	47.50	11.40	-0.05 (0.24)	follow-up
Lass et al. (2021)	Depressive sympt.	BDI	24	17.08	10.22	23	18.26	9.54	0.12 (0.29)	post
Lass et al. (2021)	Rumination	RRS	24	58.08	12.69	23	56.35	10.70	0.14 (0.29)	post

For Calkins et al., 2015, no means and standard deviations were reported. The effect size was calculated from the reported t test. BDI, Beck Depression Inventory; BDI-II, Beck Depression Inventory – 2nd edition; CCT, Cognitive Control Training; DASS, Depression Anxiety Stress Scales - depression subscale; HDRS, Hamilton Depression Rating Scale; MADRS, Montgomory Asberg Depression Rating Scale; PTQ, Perseverative Thinking Questionnaire; RRS, Ruminative Response Scale; RSQ, Response Style Questionnaire; VAS, Visual Analogue Scale

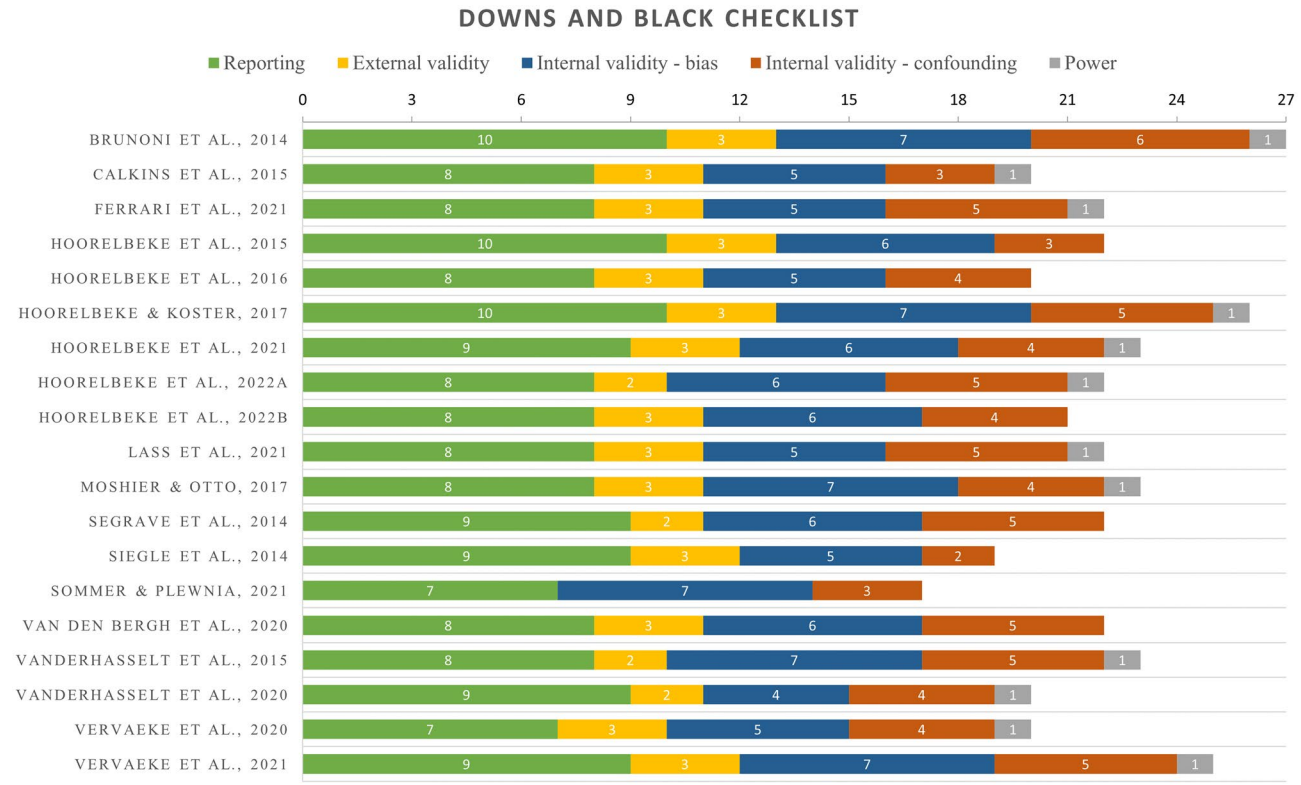

Fig. 2 Downs and Black checklist – summary

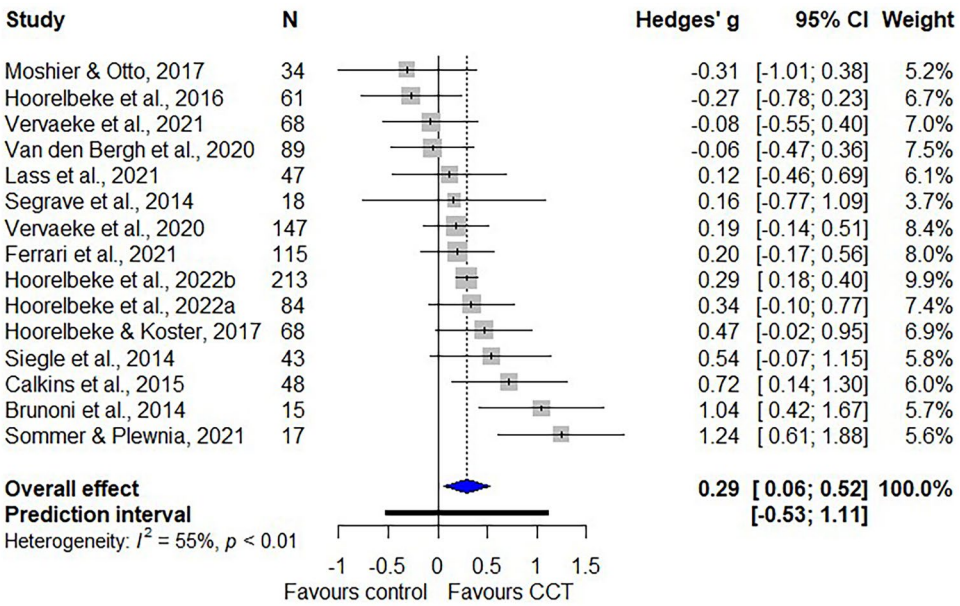

Fig. 3 Forest plot for depressive symptomatology at post

Effect of aPASAT Training on Rumination

The secondary outcome, rumination, showed similar results as the primary outcome, depressive symptomatology. The pooled effect size for the 14 analyzed standardized mean differences at post measure was significant (g = 0.25, 95% CI = [0.05, 0.45], p = .018 and heterogeneity was moderate (τ^2 = 0.09, I^2 = 52.4%, 95% CI [12.5%, 74.1%]). At follow-up, Hedges' g was no longer significant (g = 0.27, 95% CI = [-0.12, 0.65],

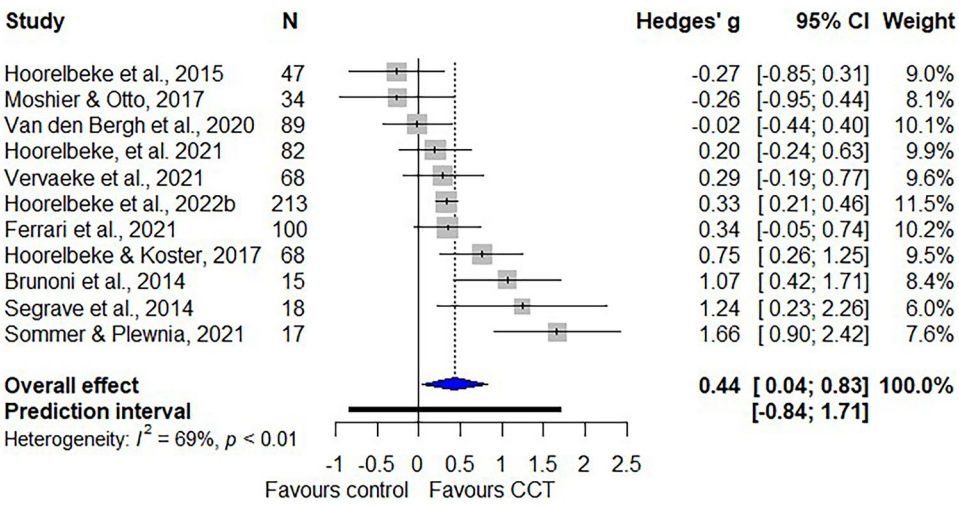

Fig. 4 Forest plot for depressive symptomatology at follow-up

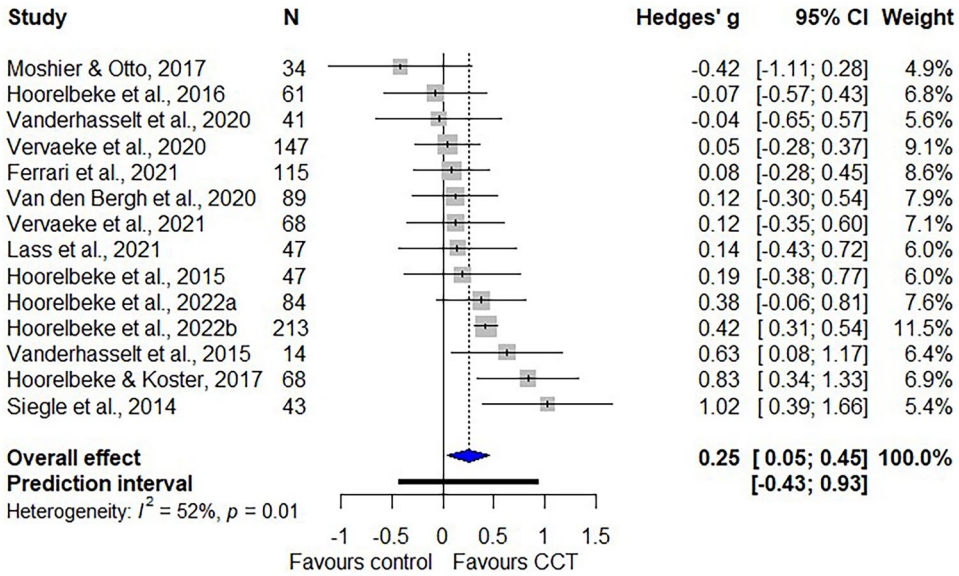

Fig. 5 Forest plot for rumination at post

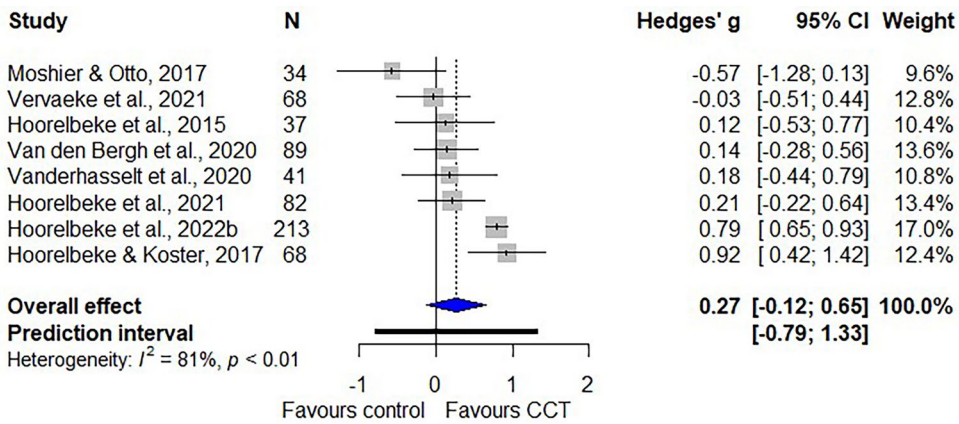

Fig. 6 Forest plot for rumination at follow-up

$p = .142$) for the 8 included studies. Heterogeneity was considerable ($\tau^2 = 0.16$, $I^2 = 81.4\%$, 95% CI [64.4%, 90.3%]). Figures 5 and 6 display the forest plots for rumination at post and follow-up, respectively.

Table 3 Subgroup analyses on population type

Population	k	Hedges' g	95% CI	τ^2	I^2	*p*
Depressive symptomatology – post						
MDD	6	0.49	[-0.12, 1.10]	0.24	69.3%	0.524
RMD	3	0.25	[-0.45, 0.94]	0.04	26.4%	
At-risk	4	0.25	[-0.22, 0.72]	0.06	40.1%	
Healthy	2	0.00	[-2.84, 2.84]	0.05	54.9%	
Rumination – post						
MDD	4	0.33	[-0.64, 1.30]	0.29	74.3%	< 0.001
RMD	3	0.44	[-0.44, 1.32]	0.08	52.4%	
At-risk	4	0.33	[0.09, 0.57]	< 0.01	2.1%	
Healthy	3	0.00	[-0.16, 0.16]	< 0.01	0.0%	

Meta-Regression on Number of aPASAT Sessions

A meta-regression was conducted to examine if the number of sessions a study used had an effect on the outcome measures. For both outcomes at both post and follow-up measures, the number of sessions did not significantly affect treatment effect (depressive symptomatology at post: $\gamma = 0.02$, $SE = 0.04$, $p = .680$, $R^2 = 0\%$ and at follow-up: $\gamma = 0.07$, $SE = 0.08$, $p = .471$, $R^2 = 0\%$); rumination at post: $\gamma = 0.03$, $SE = 0.05$, $p = .621$, $R^2 = 0\%$ and at follow-up: $\gamma = 0.16$, $SE = 0.08$, $p = .097$, $R^2 = 32\%$). Figure A4 displays a bubble plot to visualize this meta-regression on the number of aPASAT sessions (see Supplementary Material).

Subgroup Analysis on Population Type

As preregistered, a subgroup analysis was performed examining the potential moderating role of type of population for effects of aPASAT training. For depressive symptomatology immediately after training, no significant effect of population was observed. In contrast, a random-effects model showed a significant difference between population types for rumination, indicating a higher mean effect of aPASAT training on MDD, RMD and at-risk populations immediately following training, compared to a healthy population (Table 3). For both depressive symptomatology and rumination no significant effect of population was observed at follow-up ($ps > 0.11$; for a more detailed account, we refer to supplemental Table A5).

Publication Bias

As preregistered, indications for publication bias were examined using a visual inspection of the funnel plot, Egger's test and Duval and Tweedie's trim and fill method, performed on the primary outcome at post measure. The funnel plot showed no asymmetry or indication of publication bias (Fig. 7). Egger's test was not significant ($p = .850$). Duval and Tweedie's trim and fill procedure did not remove, nor add any studies, thus not changing the pooled effect size. No evidence for publication bias was found, suggesting a successful and complete search strategy.

To examine the quality of the evidence in this meta-analysis, the GRADE approach was used. For both outcomes, the quality of evidence scored moderate. One level was deducted due to some concerns in the inconsistency of results (see Table 4), because a few of the included studies did not observe significant effects and the subgroup analysis did not find any moderating effect of population for the primary outcome, which did not point to additional insight into the observed heterogeneity. No large effects were found and no dose-response gradient was observed for either depressive symptomatology or rumination, so neither outcome was upgraded.

To ease interpretation of the pooled effect size for clinical relevance, the standardized mean difference of depressive symptomatology after the training was converted into a Number Needed to Treat (NNT) using the Kraemer & Kupfer method. The pooled effect size $g = 0.29$ converted into a NNT of 6.15.

Discussion

In this systematic review and meta-analysis, we set out to examine the efficacy of an aPASAT training on depression vulnerability. First, overall effectiveness was examined for this specific type of CCT on depressive symptomatology as primary outcome and rumination as secondary outcome, both immediately after the training and at follow-up. The pooled effect sizes directly after training revealed small significant effects on both depressive symptomatology and rumination. For depressive symptomatology, the pooled effect size remained significant at follow-up. Although a similar effect size was obtained for rumination at follow-up, this did not reach significance. One possible explanation for this might be that fewer studies included a

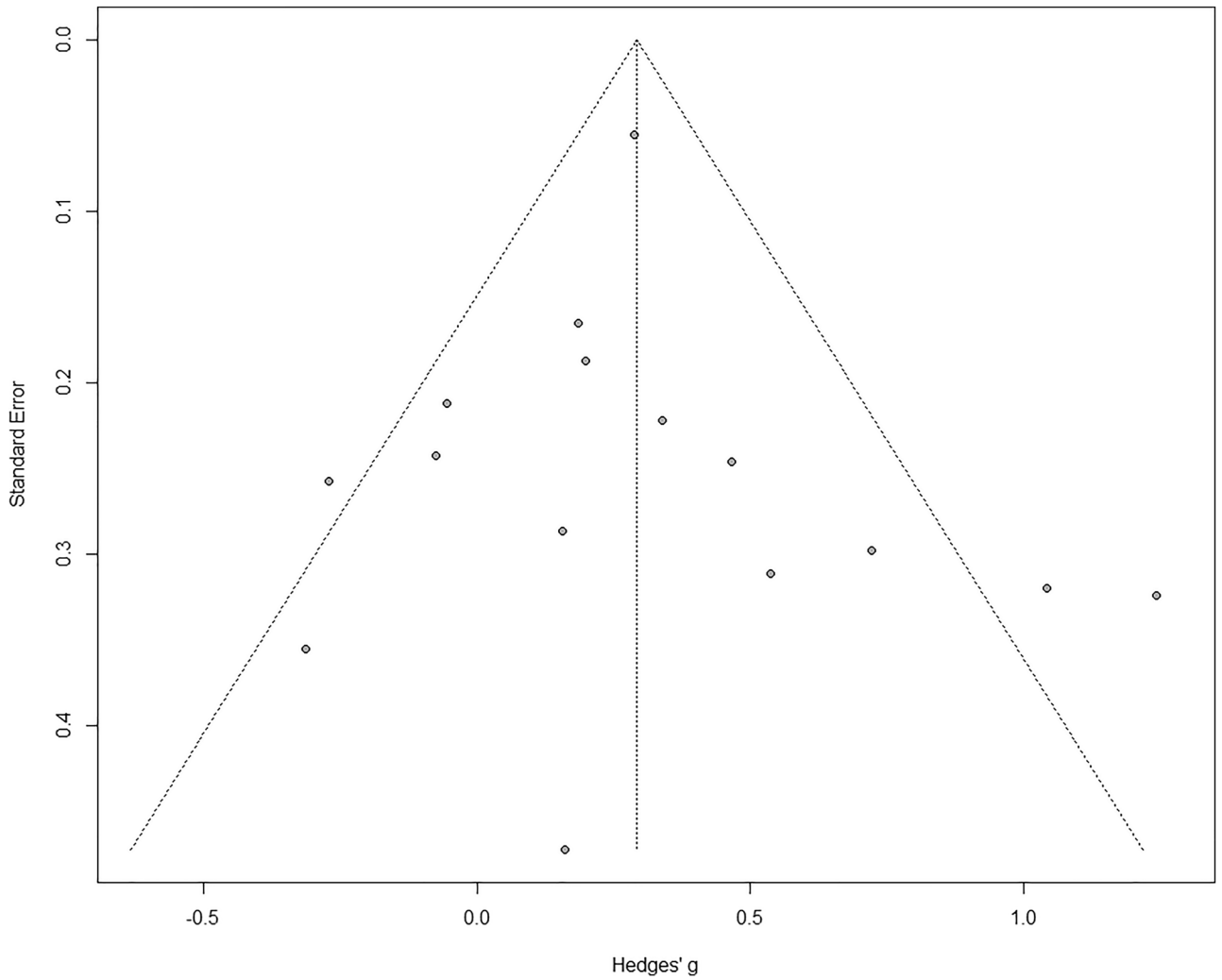

Fig. 7 Funnel plot

Table 4 GRADE certainty of evidence table

Outcome	Number of participants (studies)	Study limitations	Inconsistency of results	Indirectness of evidence	Imprecision	Publication bias	Quality of evidence	Comments
Depressive symptomatology	1208 (17 studies)	No serious concerns	Some serious concerns	No serious concerns	No serious concerns	No serious concerns	⊕⊕⊕◯ Moderate	Inconsistency of results was downgraded due to substantial levels of unexplained heterogeneity.
Rumination	1163 (15 studies)	No serious concerns	Some serious concerns	No serious concerns	No serious concerns	No serious concerns	⊕⊕⊕◯ Moderate	

follow-up measure, resulting in lower statistical power. A second possibility is that not all included studies examined their statistical power and some studies assumed a medium or high effect size in their power analysis and likely did not recruit enough participants to find a significant small effect. Lastly, the timing of follow-up measurement ranged widely, from just a few weeks up to one year, which might also contribute to the heterogeneity.

The effects from this meta-analysis are similar in size compared with previous meta-analyses examining the effects of cognitive training (e.g., Launder et al., 2021; Motter et al., 2016), resulting in small effect sizes for the effects of CCT. This leads to several interesting conclusions. First, this seems to confirm that the aPASAT is a promising CCT operationalization with a robust effectiveness in the context of depression vulnerability. Second, the effect sizes are small, both immediately after training, as well as long-term. This is noteworthy given the relative low intensity of training procedures used, and strong heterogeneity in follow-up periods. Although the effects are small, they appear to be sustainable long-term. Previous CCT research has hinted at slightly larger long-term effect sizes than immediately after training for some outcomes (Hoorelbeke & Koster, 2017). However, given the limited aPASAT studies reporting follow-up measures and the heterogeneity in follow-up examinations, we conclude that both immediate and long-term effects are modest. Lastly, both depressive symptomatology and rumination as risk factor for depression appear susceptible for improvements after training of cognitive control.

In contrast to our expectations, the results suggested no effect of number of training sessions. Our sensitivity analysis, which excluded the studies using only five or less CCT sessions, did not meaningfully change these results. Whilst just three studies used five or less sessions and most recent studies have used ten or twelve sessions, an optimal dose of an aPASAT training has currently not been investigated and the required number of sessions for a sustained effect is presently unknown. In addition, some of the early training studies have relied on a multifactorial training approach, in which the aPASAT was combined with Wells' Attention Training (e.g. Calkins et al., 2015; Siegle et al., 2007, 2014), which may explain the absence of effects of training dosage for the primary and secondary outcomes. Future research should investigate both the required number of sessions for sustainable results, as well as the optimal time frame during which the CCT sessions are administered. The potential role of booster sessions (i.e., administering additional sessions) at critical moments could also be examined to see if an individually tailored approach, using a more flexible and personalized administration method, would increase CCT effects on depression vulnerability.

Contrary to our initial hypothesis, a subgroup analysis on the type of sample population (MDD, RMD, at-risk, healthy) found no significant differences for depressive symptomatology, indicating we found no evidence that aPASAT training was more or less effective in reducing depressive symptoms in a specific population. For the secondary outcome rumination however, subgroup differences were found at post measure, suggesting a higher mean effect in MDD, RMD and at-risk populations, as opposed to a healthy population. This is in line with our hypothesis that populations with higher cognitive impairments gain more from cognitive training. It is noteworthy that differential hypotheses can be made with regard to the effects of CCT in function of population type. On the one hand, it is plausible that aPASAT training is more effective for people with greater cognitive deficits (i.e., MDD and RMD populations). On the other hand, it could also be the case that individuals with higher levels of cognitive control profit more from training. Related to this, it is possible that individuals with greater cognitive deficits may benefit from a combined remediation approach (e.g., combining other neuromodulation techniques such as tDCS with CCT; Segrave et al., 2014). Currently, it is unclear which of these hypotheses is correct. Moreover, based on the overall effect size reported in the current meta-analyses one could argue that the current average sample size of previous studies could have been too small to detect meaningful differences in differential efficacy of training between different subgroups. More sufficiently powered research should be performed in several types of population samples. Furthermore, in CCT studies participants show substantial heterogeneity in treatment response. Several factors might play a role, such as the context of how CCT is administered. In particular, in the context of aPASAT training, differences exist in terms of performance feedback provided during and following training sessions. For instance, in early studies performance feedback was typically kept to a minimum (e.g., only providing information on the median ITI and consecutive number of (in)correct responses during sessions; Hoorelbeke et al., 2015; Hoorelbeke & Koster, 2017). In contrast, in some of the recent studies more detailed feedback has been provided regarding online task performance. For instance, Lass et al. (2021) made training task performance explicit by providing auditory feedback. Upon providing an incorrect response, participants were presented with a low-pitched beep. Sommer and Plewnia (2021) on the other hand, added visual feedback, where correct versus incorrect responses were followed by presentation of green or red stimuli respectively. Moreover, gamified versions of the aPASAT have typically also included motivational messages, as well as feedback on training progress over sessions (e.g., Vervaeke et al., 2020; Hoorelbeke et al., 2022b; Van den Bergh et al., 2020). Given the strong heterogeneity in types of performance feedback provided, gamification elements used, and limited number of available aPASAT studies, the current meta-analysis did not explore effects of performance feedback or gamification. However, recent studies have shown that the use of gamification techniques can effectively bolster treatment adherence, without influencing treatment effectiveness (Mohammed et al., 2017; Vervaeke et al., 2020).

To determine the clinical relevance of our meta-analytic results, the pooled effect sizes were converted into a NNT. For depressive symptomatology at post measure, an effect size of $g = 0.29$ was converted to NNT = 6.15, meaning that around six patients would have to be treated with CCT for one patient to improve. In comparison to antidepressant medication ($g = 0.30$, NNT = 5.95; Khan & Brown, 2015) or psychotherapy ($g = 0.25$, NNT = 7.13; Cuijpers et al.,

2013), aPASAT training has a similar NNT, meaning a similar amount of patients need to be treated with the aPASAT to approximate the effects of psychopharmacological and psychotherapeutic interventions, separately. In clinical practice, the combination of antidepressant medication and psychotherapy occurs often. A recent meta-analysis examining this combination therapy found a standardized mean difference g = 0.43, resulting in a NNT of 4.19 (Cuijpers et al., 2014). For future research, the role of the combination of CCT and other types of antidepressant therapies should be examined (see Van den Bergh et al., 2018).

Although the number of aPASAT studies was sufficient for a meta-analysis, more in depth analysis on specific subgroups or specific doses would require more studies. The number of studies examining long-term effects of depressive symptomatology and rumination are limited, resulting in lower power for the long-term measure. Moreover, studies show strong variability in follow-up period, ranging from 2 weeks to 12 months. Due to unexplained heterogeneity and some inconsistency in results, the confidence in certainty of evidence was moderate. These limitations are mitigated by several strengths of this meta-analysis, among which that calculations were performed on the post mean effect sizes, where possible, rather than on pre-post effect sizes (Cuijpers et al., 2017). Additionally, by focusing on the aPASAT, task-related heterogeneity of different CCT procedures was avoided. Lastly, this study design was preregistered on Prospero and the data and analysis code needed for reproducing the results is available on OSF.

Considering the positive effect sizes of this meta-analysis, suggesting beneficial effects on depressive symptomatology and rumination, together with the relative low cost of internet-delivered CCT and its accessibility, clinical implementation of aPASAT training could be considered as an effective strategy to remediate cognitive control impairments in the context of depression.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11065-023-09581-8.

Authors' Contributions Yannick Vander Zwalmen, Kristof Hoorelbeke and Ernst Koster developed the framework of the review. Yannick Vander Zwalmen and Eveline Liebaert performed study selection and risk of bias assessment, for which Kristof Hoorelbeke served as arbitrator. All authors provided critical contributions and revisions.

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Data Availability All data and a script for the analyses is available on OSF: https://osf.io/dhqfk.

Declarations

Ethical Approval Not applicable.

Competing Interests The authors declare that there are no competing interests.

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