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Depressive Symptoms and Inductive Reasoning Performance: Findings from the ACTIVE Reasoning Training Intervention

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Abstract

Within the context of the Advanced Cognitive Training for Independent and Vital Elderly (ACTIVE) study, we examined the longitudinal association of baseline depressive symptoms on inductive reasoning performance over a ten-year period between the reasoning training and control conditions (N = 1,375). At baseline, 322 participants (23%) reported elevated depressive symptoms, defined by a score ≥ 9 on the Center for Epidemiological Studies Depression scale (12-item). Differences in baseline depressive status were not associated with immediate post-training gains or with subsequent annual change in reasoning performance, suggesting that the presence of elevated baseline depressive symptoms does not impact the ability to benefit from reasoning training.

Keywords

cognitive training; inductive reasoning ability; cognitive ability; depressive symptoms; older adults

Reasoning ability is vulnerable to decline during the course of the normal aging process; however, significant individual differences exist in the onset, rate, and extent of such decline (Hedden & Gabrieli, 2004; Schaie, 2005). Even in the absence of more impaired function, normative age-related declines can become problematic as they can produce significant difficulties in performing instrumental activities of daily living, especially those which require strong reasoning skills (Willis et al., 2006). For instance, deficits in reasoning performance often limit the ability to solve problems that require linear thinking, such as following step-by-step instructions for the dosing of prescription medications (Zartman, Hilsabeck, Guarnaccia, & Houtz, 2013).

Prior studies have demonstrated the effectiveness of cognitive training interventions for strengthening inductive reasoning skills, suggesting that they may also serve to promote functioning and independence later in life (Baltes, Sowarka, & Kliegl, 1989; Willis & Nesselroade, 1990; Willis & Schaie, 1986; Willis et al., 2006). Although these studies offer great promise, it is not entirely clear whether there are individual characteristics that either facilitate or hinder the benefits of these training programs. Elevated levels of depressive symptoms have been negatively associated with cognitive function (e.g., Comijs, Jonker, Beekman, & Deeg, 2001; Ganguli, Du, Dodge, Ratcliff, & Chang, 2006; Ganguli, Snitz, Bilt, & Chang, 2009) and may represent a modifiable target for improving the outcomes of cognitive interventions. Even if depressive symptoms fail to reach levels sufficient for a clinical diagnosis of a mood disorder, elevated symptoms may be associated with poorer cognitive functioning and cognitive decline in multiple domains (Ganguli et al., 2009), thus limiting the effectiveness of training programs intended to help maintain, or perhaps promote, cognition in later adulthood.

Although the cross-sectional associations between depressive symptoms and cognition have been well-established, the directionality and reasons for these associations are less understood. Longitudinal studies have demonstrated associations between higher levels of depressive symptoms and cognitive decline (e.g., Rosenberg, Mielke, Xue, & Carlson, 2010; Royall, Palmer, Chiodo, & Polk, 2011; Wilson et al. 2002); however, findings have not been consistent (e.g., Dufouil, Fuhrer, Dartigues, & Alperovitch, 1996; Ganguli et al., 2006). Using data from four waves of the English Longitudinal Study of Aging (ELSA), Gale and colleagues (2012) examined the bidirectional relationships between depressive symptoms (using the Center for Epidemiological Studies Depression Scale (CES-D; 8-item)) and general cognitive ability over a 6-year period in a sample of 8,611 adults over 50 years of age at baseline. Overall, their findings suggested that elevated baseline depressive symptoms were associated with poorer cognitive ability at baseline (except in the oldest age group) and with a slightly faster rate of cognitive decline over the 6-year period, but only for people aged 60-80 years. However, it did not appear that initial level of cognitive ability was associated with rate of change in depressive symptoms over time, thus providing little evidence for reciprocal associations.

Clinical investigations have also demonstrated the co-occurrence of elevated depressive symptoms and cognitive dysfunction within specific patient populations, including those with Parkinson's disease, Huntington's disease, and traumatic brain injury (see Carlson, Eldreth, Chuang, & Eaton, 2012 for review). Moreover, a number of studies have suggested that depressive symptoms may be associated with an increased risk of dementia and Alzheimer's disease (Butters et al., 2008; da Silva, Gonçalves-Pereira, Xavier, & Mukaetova-Ladinska, 2013; Diniz, Butters, Albert, Dew, & Reynolds, 2013; Ownby, Crocco, Acevedo, John, & Loewenstein, 2006). In two recent systematic reviews, da Silva and colleagues (2013) found an increased risk for developing dementia in individuals with depression; however, the likelihood of occurrence was associated with greater frequency and severity of depressive episodes. Likewise, Diniz and colleagues (2013) found that late-life depression was associated with an increased risk for all-cause dementia, vascular dementia, and Alzheimer's disease.

Although work in this area is far from conclusive, both depressive symptomatology and cognitive impairments may be a result of common risk factors or underlying neurodegenerative processes, including frontal lobe pathology, neurotransmitter alterations, vascular changes, and/or genetic factors (Butters et al., 2008; Crocco, Castro, & Loewenstein, 2010). In addition, depressive symptoms can interfere with the ability to concentrate, lower energy levels, and diminish self-confidence. Therefore, depressive symptoms may impact the ability to acquire, practice, and apply the skills taught in cognitive training programs. Unfortunately, relatively few studies have investigated whether the presence of elevated depressive symptoms limits the ability to benefit from cognitive training.

Among the few studies that have investigated the impact of depressive symptoms in the context of cognitive training interventions, Lohman and colleagues (2012) examined the association between depressive symptoms and memory performance across a 5-year period within the memory training arm of the Advanced Cognitive Training for Independent and Vital Elderly (ACTIVE) study (Ball et al., 2002; Jobe et al., 2001; Willis et al., 2006). Although older adults with elevated depressive symptoms scored significantly lower at baseline and declined more rapidly on memory tasks, differences in memory performance between participants with different levels of depressive symptomatology did not vary between the memory-trained and control conditions. Similarly, using data from the Healthy Brain Ageing Clinic in Sydney, Australia; cognitive training was shown to improve memory performance in a small clinical sample ($N = 44$) of cognitively healthy adults aged 45 years or older ($M = 64.8$; $SD = 8.5$; range = 45-82 years) with a lifetime history of major depressive disorder (Naismith, Diamond, Carter, Norrie, Redoblado-Hodge, Lewis, & Hickie, 2011). These studies are promising as findings suggest that the beneficial effects of memory training programs may still be achieved, even among those with elevated depressive symptoms or more severe depressive disorders.

Understanding whether depressive symptoms limit improvements in inductive reasoning skills may be of even greater clinical importance for maintaining independence later in life. To our knowledge, however, no previous study has examined associations of depressive symptomatology on reasoning skills within a training context. Therefore, the present analysis was designed to address this gap and complement the previously described research within the memory-training arm of the ACTIVE study (Lohman et al., 2013) by examining whether elevated depressive symptoms are associated with the ability to benefit from inductive reasoning training within the context of the ACTIVE study (Willis et al., 2006).

Methods

Data were collected as part of the ACTIVE study (see Ball et al., 2002 and Jobe et al., 2001 for details of recruitment procedures and study design). ACTIVE was a randomized, multi-site clinical trial designed to examine the effectiveness and durability of cognitive training on domain-specific cognitive performance, as well as immediate and long-term transfer effects to everyday activities (Jobe et al., 2001). Healthy, community-dwelling individuals aged 65 years and older ($N = 2,802$) were randomized to one of three cognitive training interventions (reasoning ($n = 699$), memory ($n = 703$), and speed of processing ($n = 702$)) or

to a no-contact control group ($n = 698$). Each training program consisted of ten, 60-75 minute sessions in which participants learned and practiced strategies for solving problems, remembering, or responding quickly to stimuli. To evaluate the effects of training, measurements were taken at baseline, immediate posttest, and at 1, 2, 3, 5, and 10 years. Previous research demonstrated that, compared with baseline, each intervention immediately improved the targeted cognitive ability. Effects were maintained through the 5-year (Willis et al., 2006) and 10-year follow-up assessments (Rebok et al., 2014).

Participants

For the present study, we limited our analysis to data from participants in the ACTIVE reasoning-training and control groups. We excluded 22 participants with missing depressive symptom data at baseline, resulting in a final analytic sample of 1,375 participants ($n_{\text{reasoning}} = 684$; $n_{\text{control}} = 691$).

Measures

Inductive Reasoning—Inductive reasoning ability was measured using three standardized, timed, paper-and-pencil assessments: *Word Series* (30 items; Time: 6-minutes; Ekstrom, French, Harman, & Derman, 1976), *Letter Series* (30 items; Time: 6-minutes; Thurstone & Thurstone, 1949), and *Letter Sets* (15 items; Time: 6-minutes; Ekstrom et al., 1976). *Word Series* evaluates how well an individual can follow a series of words in logical sequence by providing the appropriate word that follows the last word in a list (ex: January, April, January, May, January, ... Answer: June). The *Letter Series* test is similar to *Word Series*; however, single letters replace the use of words (ex: c, a, d, a, e, a, f, a... Answer: g). The *Letter Sets* test provides five sets of letters with four letters in each set; four of the five sets of letters are similar, and the goal is to identify the one set of letters that is unlike the other sets. For each of the three reasoning tests, scores represent the number of correct responses provided in the time allowed, with higher scores reflecting better performance on the measure. A maximum score of 30 was possible for the *Word Series* and *Letter Series* tasks and a maximum score of 15 for *Letter Sets*. For each task, scores were standardized to their baseline mean and standard deviation. For analysis, individual standardized scores were averaged to create a composite inductive reasoning measure.

Depressive Symptoms—An abbreviated 12-item version of the Center for Epidemiological Studies-Depression (CESD-12) scale was used to measure self-reported depressive symptoms (Mirowsky & Ross, 2003; Radloff, 1977). Respondents were asked to rate the frequency of occurrence of each symptom in the past week on a four-level ordinal scale: *rarely or none of the time*, *some of the time*, *much of the time*, and *most or all of the time*. Summed scores for the CESD-12 range from 0 (no depressive symptoms) to 36 (for all items, most or all of the time). A threshold of greater or equal to 9 points was used to indicate depressive symptomatology, which is comparable to defining depressive status using a cut-off score of 16 points on the well-validated, 20-item version of the CES-D (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993; Wolinsky et al., 2009). Further, we adopted this criterion based on prior research using the ACTIVE sample (e.g., Lohman et al., 2013), as well as for comparability to other population and community-based studies (e.g., National Survey of American Life (Neighbors, Njai, & Jackson, 2007); Iowa Healthy

and Active Minds Study (Wolinsky et al., 2011)). In the present study, participants with elevated depressive symptoms comprised the “depressed” group, although this is not meant to reflect a clinical diagnosis of depression.

Data Analysis

Data were analyzed using *Mplus version 7.11* statistical software (Muthén & Muthén, 1998-2011). Prior to main analyses, we compared demographic characteristics, depressive symptoms, and cognitive variables between the ACTIVE reasoning-training and control groups. Next, we estimated multiple-group latent growth curve models of reasoning performance with groups for intervention assignment (reasoning-training or control group) (Muthén & Muthén, 1998-2011). Specifically, baseline levels and annual changes of reasoning performance (using the reasoning composite measure) were regressed on a binary indicator of depressive symptoms (≥ 9 vs. <9 endorsed symptoms on the CESD-12). We also included a quadratic fixed effect term in our model, which allows us to examine non-linear trends in our data. In a planned sensitivity analysis, we repeated the analysis for each subcomponent of the reasoning composite (*Letter Sets*, *Letter Series*, *Word Series*). All final models were adjusted for baseline age (in years), sex, ethnicity, education (in years), attrition, and baseline global cognitive status as defined by the Mini-Mental State Examination score (MMSE; Folstein, Folstein, & McHugh, 1975). Model-to-data fit was evaluated using the Comparative Fit Index (CFI) and root mean square error of approximation (RMSEA). RMSEA (< 0.05) and CFI (> 0.95) have been shown to perform better than χ^2 given large sample sizes (Muthén & Muthén; 1998-2011). Results are presented as standardized (z-scores) values.

Results

Demographic characteristics

Table 1 summarizes the baseline demographic characteristics of the sample by training group (reasoning vs. control) and presence of depressive symptoms. Overall, there were no differences between the reasoning-training and control groups with respect to age, sex, ethnicity, education, or cognitive status. Further, because ACTIVE selected for cognitively-healthy older adults, participants did not show impaired functioning on any of the reasoning tasks at baseline (see Ball et al., 2002). A few differences were noted, however, between individuals with and without elevated depressive symptoms in both reasoning-training and control groups. In general, individuals with elevated depressive symptoms were slightly older, had fewer years of education, were more likely to be lost to follow-up, and had lower scores on a measure of global cognition (as measured by the MMSE; Folstein et al., 1975), as well as lower scores on reasoning measures.

Associations between depressive symptoms and reasoning performance

We fit a latent growth curve model to the data to describe the association of depressive symptoms on trajectories of reasoning performance. Findings by intervention group and depressive status are shown in Table 2 and Figure 1. Conventional fit indices indicated that the model fit well to the data (RMSEA: 0.05; CFI: 0.97).

Among participants without elevated depressive symptoms, mean levels of baseline reasoning performance were similar between the reasoning-training and control groups (estimate = 0.04 vs. 0.07, respectively; $p > 0.05$); however, the reasoning-training group showed greater improvements in performance than the control group immediately after receiving the intervention (estimate = 0.71 vs. 0.25, respectively; $p < 0.05$). In addition, among individuals without elevated depressive symptoms, the subsequent annual rate of decline in reasoning performance was steeper for the reasoning-training than for the control group (estimate = -0.03 vs. 0.00, respectively; $p < 0.05$) (Table 2). This may be, in part, because the reasoning group showed immediate improvements as a result of training but were not able to sustain these immediate training gains over time. The control group did not show initial improvements of this magnitude, and therefore did not have the same opportunity to decline.

Among individuals with elevated depressive symptoms, mean levels of baseline reasoning performance in both the reasoning-training and control groups (estimate = -0.08 vs. -0.25, respectively) were somewhat lower than those reported above for the non-elevated depressive group, although the magnitude of this difference was only significant among the control group (estimate = -0.32, $p < 0.05$). Importantly, the association between baseline depressive symptoms and reasoning performance did not differ by intervention status. Similar to findings for the non-elevated depressive group, the reasoning-training group (compared to the control group) showed greater immediate gains in reasoning performance (estimate = 0.70 vs. 0.24, respectively; $p < 0.05$) and steeper annual rate of decline (estimate = -0.05 vs. 0.00, respectively; $p < 0.05$) (Table 2).

Of greatest interest, when examining intervention-related differences between the non-elevated and elevated depressive groups, depressive status at baseline was not associated with differences in mean level reasoning performance immediately following training (estimate = 0.01) or with subsequent annual change in the composite measure of inductive reasoning performance (estimate = -0.01) ($ps > 0.05$). Together, these findings suggest that depressive symptoms do not appear to affect the ability to benefit from training programs (Table 2; Figure 1).

In subsequent analyses, we repeated the models for individual components of the reasoning composite score. For the *Letter Series* and *Letter Sets* tasks, among individuals without elevated depressive symptoms, the annual rate of cognitive decline did not differ by intervention group. The inferences for *Word Series* within the non-elevated depressive group were similar to those previously described for the reasoning composite. When examining the differences between the non-elevated and elevated depressive groups, findings for the individual reasoning measures were largely consistent with those reported for the reasoning composite (see Tables 3-5 in Appendix).

Discussion

We examined the longitudinal association between depressive symptoms and inductive reasoning ability within the context of the ACTIVE reasoning-training intervention. Although elevated depressive symptoms were associated with lower reasoning performance

at baseline, our findings failed to detect differences between depressive status groups at baseline and immediate post-training gains or with subsequent training-related annual change on the composite measure of inductive reasoning performance.

Several studies have demonstrated the effectiveness of cognitive training programs; however, only a few investigations have been conducted to determine who benefits from such interventions (see Ball, Ross, Roth, & Edwards, 2013; Rebok et al., 2013; Willis & Caskie, 2013). The present findings are generally consistent with those previously found within the memory training arm of the ACTIVE study (Lohman et al., 2013), demonstrating that elevated depressive symptoms were associated with memory performance at baseline, but performance did not differ between training conditions. Together, these findings are particularly noteworthy as they suggest that the presence of elevated depressive symptoms at baseline does not appear to impact the ability to benefit from the memory or reasoning-training arms of the ACTIVE study.

Other research has suggested that observed differences in cognitive performance between depressed and non-depressed individuals may be reduced, or perhaps eliminated, in structured situations but remain when individuals have to rely on their own initiative (Hertel, 2004). The structured nature of both the ACTIVE memory and reasoning-training programs (e.g., small group sessions, structured practice activities led by a group facilitator; see Jobe et al., 2001), may have increased the likelihood that individuals with elevated depressive symptoms would benefit from these training programs.

Although findings converged regarding the ability to benefit from memory-training and reasoning-training programs, the impact of baseline depressive symptoms on trajectories of cognitive change differed between the present study and the work by Lohman et al. (2013). Specifically, it seems that (at least within the ACTIVE sample) depressive symptoms have a greater impact on memory performance, than on tasks of inductive reasoning over time. Specifically, Lohman and colleagues (2013) found greater declines in memory performance among individuals with elevated depressive symptoms across a 5-year period, whereas we did not find that depressive symptoms were associated with cognitive change over the 10-year period. This may be due to differences in the analytic sample selected (memory vs. reasoning trained groups) or the length of time (5 vs. 10 years) investigated. With this said, the literature in this area remains inconsistent and several community-based observational studies have also failed to show a relationship between depressive symptoms and cognitive functioning over time (Dufouil et al., 1996; Gale et al., 2012; Ganguli et al., 2006). To better address such questions, studies should include a comprehensive assessment of brain-behavior functioning to capture discrete areas of cognitive decline. Further, future studies should use a multivariate, multi-domain approach to examine associations between depressive status and cognition. This would explicate how specific cognitive domains are affected by depressive status and test how specific abilities impact the delivery of and ability to benefit from interventions to improve cognitive functioning.

Nevertheless, our findings suggest the utility of non-pharmacological interventions for improving cognitive function, even among those with elevated depressive symptoms. As the population is rapidly aging, determining ways to maintain (or potentially enhance) cognition

is critical for reducing the risk of dementia and, subsequently, the associated health, social, and economic burdens. Further, given the prevalence of sub-threshold depressive symptoms (as opposed to major depression) within older community-based populations (Blazer, 2003), our findings suggest that all individuals, regardless of depressive symptomatology, are responsive to cognitive interventions. However, it is important to note that within this community-based sample, older adults with more severe depression or exhibiting significant cognitive impairments were excluded from participation at baseline. Further, individuals were not asked whether they were currently taking any medications or seeking other treatments for depression. Thus, it is possible that the beneficial effects of training would be limited for individuals displaying a greater severity of depressive symptoms.

To date, however, only a few other studies have demonstrated the benefits of memory training programs for clinically depressed older adults (e.g., Naismith et al., 2011), and to our knowledge, reasoning training programs have not been examined within a clinical population. Some clinical studies have shown the effectiveness of problem-solving therapy for treatment of patients with late-life depression and co-morbid executive dysfunction (Alexopoulos, Raue, Kanellopoulos, Mackin, & Arean, 2008). As a clinical approach, problem-solving therapy trains individuals to become more effective problem solvers, which helps develop and exercise similar cognitive capacities and skills to those taught and practiced in the ACTIVE reasoning training program. The limited work in clinical settings offers important future directions for the implementation and evaluation of cognitive interventions targeting individuals with clinical depressive symptoms, and determining whether this group at elevated risk for cognitive impairment may benefit from a structured, cognitive intervention.

The ACTIVE trial included a large, representative sample of healthy, cognitively normal, community-dwelling older adults randomized to one of three cognitive interventions or a no-contact control. Further, the prospective study design allows for longitudinal measurements of outcomes as functions of baseline and time-varying measures with the use of continuous assessments of cognition and depressive symptoms allowing for a more sensitive evaluation of associations across time and with a specific training intervention group. However, this research must also be presented alongside several limitations.

First, depressive symptoms were assessed using a questionnaire, rather than clinical evaluation and diagnosis; therefore, no conclusions can be made on the basis of our results regarding the association between clinical depression and cognitive decline in older adults. Further, we used a CESD-12 threshold score of 9 to classify individuals as “depressed” versus “non-depressed.” It is important to note that although this cut-off score is not equivalent to a clinical diagnosis, it has been used in previous studies to identify subjects with and without elevated depressive symptoms (see Mirowsky & Ross, 2003; Wolinsky et al., 2009).

Moreover, the overall percentage of participants in the present analysis with baseline depressive symptoms was 23.4% (322/1,375), which is consistent with reported prevalence estimates of depressive symptoms (between 3% and 26%) from a prior review of epidemiological studies (e.g., Duke Established Populations for Epidemiologic Studies of

the Elderly (EPESE), Hispanic EPESE, Longitudinal Aging Study Amsterdam (LASA)), using the 20-item version of the CES-D in community-dwelling, older adult populations (see Hybels & Blazer, 2003 for review). In comparing across studies, however, it is important to note that variations in sample characteristics, measurement, and definitions of depressive symptomatology have the potential to affect prevalence rates.

In addition, only baseline levels of depressive symptoms were included in the present analysis. This does not account for the fact that depressive symptoms may change over time. However, Wolinsky and colleagues (2009) evaluated the effects of the three ACTIVE cognitive intervention groups (reasoning, memory, processing speed) on future depressive status among individuals with and without baseline depressive symptoms (CESD-12 = 9). Among individuals with elevated depressive symptoms at baseline, there was no improvement in depressive symptoms from baseline to year 1 of follow-up as a result of participation in any of the intervention conditions. Future analyses should incorporate longitudinal measures of depressive symptoms to account for the significant changes in symptoms, which are likely to alter cognitive performance.

Lastly, there were a few initial differences between individuals with and without elevated depressive symptoms within both the reasoning-training and control groups. Although our final models adjusted for sample attrition, age, sex, race, MMSE, and years of education, it is possible that initial differences were not fully eliminated, and therefore, limiting our ability to draw any causal claims. However, one particular advantage of examining these relations within the context of the ACTIVE randomized trial is that demographic characteristics at baseline did not differ between the reasoning-training and control groups, minimizing the likelihood that the estimate of the effect is biased. An additional concern is the possibility of attrition bias over the 10-year period. We conducted a sensitivity analysis in which we tested our models using only those individuals who were retained in the trial over the entire 10-year period (N = 460). Findings from our sensitivity analysis were consistent with those from the larger sample (N = 1,375) and there was no indication that these individuals started worse or benefited less from reasoning training. Moreover, there was no evidence of differential attrition with respect to the intervention arm at the 10-year assessment (Rebok et al., 2014).

As both depressive symptoms and cognitive impairment may contribute to additional health impairments and disability later in life, future research should focus on the dynamic interplay between depressive symptoms and cognition, as well as on the important clinical, functional, social, and neurobiological implications of mild to moderate depressive symptoms on cognitive function in late-life.

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Appendix

Models for individual components (*Letter Sets*, *Letter Series*, *Word Series*) of the reasoning composite score

Table 3

Growth model for baseline depressive symptoms and reasoning performance: Findings for Letter Sets

Parameter	Reasoning (n = 699) Estimate (SD)	Control (n = 698) Estimate (SD)	Intervention Group difference Estimate (SD)
Non-elevated depressive symptoms			
Baseline reasoning performance	0.02 (0.05)	0.06 (0.04)	-0.05 (0.06)
Gain at immediate post-training	0.38 (0.04) *	0.14 (0.04) *	0.25 (0.06) *
Annual rate of change in reasoning	-0.03 (0.02) *	0.00 (0.02)	-0.03 (0.02)
Elevated depressive symptoms			
Baseline reasoning performance	-0.12 (0.12)	-0.13 (0.08)	0.01 (0.15)
Gain at immediate post-training	0.54 (0.09) *	0.03 (0.07)	0.51 (0.12) *
Annual rate of change in reasoning	-0.05 (0.02) *	0.00 (0.02)	-0.05 (0.03)
Differences by depressive status			
Baseline reasoning performance	-0.14 (0.13)	-0.20 (0.09) *	0.06 (0.16)
Gain at immediate post-training	0.16 (0.09)	-0.11 (0.08)	0.27 (0.12) *
Annual rate of change in reasoning	-0.01 (0.01)	0.00 (0.02)	0.02 (0.02)

Note.

SD = standard deviation. Statistical significance in the reasoning and control group columns indicate the parameter is significantly different from 0 in the group. Statistical significance in the group difference column indicates a significant interaction between the parameter and training group membership. Final models adjusted for sample attrition, age, sex, race, MMSE, and years of education. RMSEA = 0.029.

* $p < 0.05$.

Table 4

Growth model for baseline depressive symptoms and reasoning performance: Findings for Letter Series

Parameter	Reasoning (n = 699) Estimate (SD)	Control (n = 698) Estimate (SD)	Intervention Group difference Estimate (SD)
Non-elevated depressive symptoms			
Baseline reasoning performance	0.04 (0.04)	0.05 (0.04)	-0.01 (0.05)
Gain at immediate post-training	0.74 (0.04) *	0.26 (0.03) *	0.48 (0.04) *
Annual rate of change in reasoning	-0.00 (0.02)	0.02 (0.01) *	-0.02 (0.02)
Elevated depressive symptoms			
Baseline reasoning performance	-0.07 (0.07)	-0.29 (0.07) *	0.21 (0.10) *

	Reasoning (n = 699)	Control (n = 698)	Intervention Group difference
Parameter	Estimate (SD)	Estimate (SD)	Estimate (SD)
Gain at immediate post-training	0.71 (0.07) *	0.31 (0.07) *	0.40 (0.10) *
Annual rate of change in reasoning	-0.02 (0.01) *	0.01 (0.02)	-0.03 (0.02)
Differences by depressive status			
Baseline reasoning performance	-0.11 (0.08)	-0.33 (0.08) *	0.22 (0.11)
Gain at immediate post-training	-0.03 (0.08)	0.05 (0.07)	-0.08 (0.10)
Annual rate of change in reasoning	-0.02 (0.01) *	-0.01 (0.01)	-0.01 (0.02)

Note.

SD = standard deviation. Statistical significance in the reasoning and control group columns indicate the parameter is significantly different from 0 in the group. Statistical significance in the group difference column indicates a significant interaction between the parameter and training group membership. Final models adjusted for sample attrition, age, sex, race, MMSE, and years of education. RMSEA = 0.065.

* $p < 0.05$.

Table 5

Growth model for baseline depressive symptoms and reasoning performance: Findings for Word Series

	Reasoning (n = 699)	Control (n = 698)	Intervention Group difference
Parameter	Estimate (SD)	Estimate (SD)	Estimate (SD)
Non-elevated depressive symptoms			
Baseline reasoning performance	0.03 (0.04)	0.06 (0.04)	-0.03 (0.05)
Gain at immediate post-training	0.73 (0.03) *	0.29 (0.03) *	0.45 (0.04) *
Annual rate of change in reasoning	-0.03 (0.01) *	0.01 (0.01)	-0.04 (0.02) *
Elevated depressive symptoms			
Baseline reasoning performance	-0.02 (0.08)	-0.25 (0.06) *	0.23 (0.10) *
Gain at immediate post-training	0.63 (0.05) *	0.29 (0.05) *	0.34 (0.07) *
Annual rate of change in reasoning	-0.04 (0.01) *	0.00 (0.01)	-0.04 (0.02) *
Differences by depressive status			
Baseline reasoning performance	-0.05 (0.09)	-0.31 (0.07) *	0.26 (0.11) *
Gain at immediate post-training	-0.11 (0.05) *	0.00 (0.05)	-0.11 (0.08)
Annual rate of change in reasoning	-0.01 (0.01)	-0.01 (0.01)	-0.00 (0.02)

Note.

SD = standard deviation. Statistical significance in the reasoning and control group columns indicate the parameter is significantly different from 0 in the group. Statistical significance in the group difference column indicates a significant interaction between the parameter and training group membership. Final models adjusted for sample attrition, age, sex, race, MMSE, and years of education. RMSEA = 0.065.

* $p < 0.05$.

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Figure 1.
Reasoning performance over 10-year interval by intervention group and depressive status

Table 1

Demographic characteristics of the ACTIVE sample

Characteristic	Reasoning training group			Control group		
	Elevated depressive symptoms (n=165)	Non-elevated depressive symptoms (n=519)	p	Elevated depressive symptoms (n=157)	Non-elevated depressive symptoms (n=534)	p
Age (in years), mean (SD)	74.6 (6.0)	73.2 (5.6)	0.004	74.7 (6.3)	73.8 (5.9)	0.07
Sex (Female), n (%)	134 (81.2)	390 (75.1)	0.11	134 (85.4)	374 (70.0)	<0.001
Education (in years), mean (SD)	12.7 (2.6)	13.7 (2.7)	<0.001	12.3 (2.6)	13.7 (2.7)	<0.001
MMSE total, mean (SD)	26.8 (2.1)	27.4 (2.0)	0.001	26.6 (2.1)	27.5 (1.9)	<0.001
Race (White), n (%)	120 (72.7)	372 (71.7)	0.79	105 (66.9)	390 (73.0)	0.14
CESD-12, mean (SD)	13.2 (4.6)	3.1 (2.5)	<0.001	12.4 (3.8)	2.9 (2.5)	<0.001
Attrition, n (% followed up after 10 years)	323 (62.2)	128 (81.5)	0.02	351 (65.7)	119 (72.1)	<0.001
Reasoning composite, mean (SD)						
Baseline	-0.08 (0.09)	0.04 (0.04)	<0.001	-0.25 (0.06)	0.07 (0.04)	<0.001
Immediate post-test	0.62 (0.08)	0.74 (0.04)	<0.001	-0.01 (0.06)	0.32 (0.03)	<0.001
Year 1	0.57 (0.08)	0.71 (0.04)	<0.001	-0.02 (0.06)	0.31 (0.03)	<0.001
Year 2	0.49 (0.08)	0.65 (0.04)	<0.001	-0.05 (0.06)	0.29 (0.03)	<0.001
Year 3	0.40 (0.07)	0.57 (0.04)	<0.001	-0.11 (0.06)	0.24 (0.03)	<0.001
Year 5	0.16 (0.06)	0.36 (0.04)	<0.001	-0.28 (0.07)	0.07 (0.04)	<0.001
Year 10	-0.80 (0.16)	-0.51 (0.15)	0.08	-1.09 (0.18)	-0.73 (0.14)	0.001

Note. SD: standard deviation; Scores for the CESD-12 range from 0 (no depressive symptoms) to 36 (for all items, most or all of the time). A threshold of 9 points was used to indicate moderate to severe (i.e., elevated) depressive symptoms.

Table 2

Growth model for baseline depressive symptoms and reasoning performance: Findings for Reasoning Composite

Parameter	Reasoning (n = 699)	Control (n = 698)	Intervention Group difference
	Estimate (SD)	Estimate (SD)	Estimate (SD)
Non-elevated depressive symptoms			
Baseline reasoning performance	0.04 (0.04)	0.07 (0.04)	−0.03 (0.05)
Gain at immediate post-training	0.71 (0.03) *	0.25 (0.02) *	0.45 (0.03) *
Annual rate of change in reasoning	−0.03 (0.01) *	0.00 (0.01)	−0.03 (0.01) *
Elevated depressive symptoms			
Baseline reasoning performance	−0.08 (0.09)	−0.25 (0.06) *	0.17 (0.11)
Gain at immediate post-training	0.70 (0.06) *	0.24 (0.04) *	0.47 (0.07) *
Annual rate of change in reasoning	−0.05 (0.01) *	0.00 (0.01)	−0.05 (0.02) *
Differences by depressive status			
Baseline reasoning performance	−0.11 (0.10)	−0.32 (0.07) *	0.21 (0.12)
Gain at immediate post-training	0.00 (0.06)	−0.02 (0.04)	0.01 (0.07)
Annual rate of change in reasoning	−0.02 (0.01) *	0.00 (0.01)	−0.01 (0.02)

Note.

SD = standard deviation. Statistical significance in the reasoning and control group columns indicate the parameter is significantly different from 0 in the group. Statistical significance in the group difference column indicates a significant interaction between the parameter and training group membership. Final models adjusted for sample attrition, age, sex, race, MMSE, and years of education. RMSEA = 0.054.

*
 $p < 0.05$.