

Does Cognitive Training Improve Internal Locus of Control Among Older Adults?

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Objectives. We evaluated the effect of cognitive training among 1,534 participants in the Advanced Cognitive Training for Independent and Vital Elderly (ACTIVE) randomized controlled trial (RCT) on 5-year improvements in 3 cognitive-specific measures of locus of control—internal, chance, and powerful others.

Methods. ACTIVE was a multisite RCT (age ≥ 65), with 4 groups (memory, reasoning, speed of processing, and no-contact control). Complete 5-year follow-up data were available for 1,534 (55%) of the 2,802 participants. A propensity score model was used to adjust for potential attrition bias. Clinically important improvements (and decrements) in the cognitive-specific locus of control scale scores were defined as greater than or equal to 0.5 *SD* (medium) and greater than or equal to 1.0 *SD* (large). Multinomial logistic regression was used to simultaneously contrast those who improved and those who declined with those whose locus of control scale score was unchanged.

Results. Statistically significant effects reflecting medium-sized (≥ 0.5 *SD*) improvements in internal locus of control between baseline and the 5-year follow-up were found for the reasoning and speed of processing intervention groups who were 76% ($p < .01$) and 68% ($p < .05$) more likely, respectively, to improve than the no-contact control group. No improvement effects were found on the chance or powerful others locus of control measures or for the memory intervention group.

Conclusion. Cognitive training that targets reasoning and speed of processing can improve the cognitive-specific sense of personal control over one's life in older adults.

Key Words: cognitive status—personal control—randomized controlled trial—speed of processing.

PERSONAL control (Rodin, 1986, 1987, 1990; Rotter, 1966; Skinner, 1996), regardless of whether it is labeled locus of control (Levenson, 1974), sense of control (Mirowsky, 1995, 1997), sense of coherence (Antonovsky, 1979), mastery (Pearlin, Menaghan, Lieberman, & Mullan, 1981), or self-efficacy (Bandura, 1986), is critically important because of its relationship to health and health behavior (Krause & Shaw, 2003; Lachman, 2006; Mirowsky & Ross, 2003; Rodin & Timko, 1991; Schulz & Heckhausen, 1999). Indeed, Rowe and Kahn (1998) consider personal control to be one of the most crucial markers of successful aging. Mirowsky and Ross (p. 66) have simply and succinctly outlined the perceived underlying etiologic process of how and why personal control makes a difference for health outcomes with the statement:

... compared to people who feel powerless to control their lives, people with a sense of control know more about

health, they are more likely to initiate preventive behaviors like quitting smoking, exercising, or maintaining normal weight, and in consequence, they have better self-rated health, fewer illnesses, and lower rates of mortality.

Caplan and Schooler (2003) have recently confirmed in a nationally representative study that personal control plays a crucial deterministic role in the disablement process, especially among older adults, and that this effect reaches forward for two decades.

Beliefs about personal control in life are known to vary by age (Fung, Abeles, & Carstensen, 1999; Pitcher, Spykerman, & Gazi-Tabatabaie, 1987; Schulz & Heckhausen, 1999). For example, cross-sectional analyses of the National Survey of Midlife in the United States have shown (Lachman & Firth, 2004) that although 80% of those 25–39 years old disagreed with the statement that “What happens in my life is often beyond my control” (indicating that they had a sense

of personal control), only 62% of those 60–75 years old disagreed with that statement. Moreover, recent longitudinal evidence has shown age-related declines in personal control beginning after age 50 years (Mirowsky, 1995, 1997; Wolinsky, Wyrwich, Babu, Kroenke, & Tierney, 2003). It is generally assumed that such age-related declines are associated with concomitant age-related degradation in cognition, health, and well-being (Lachman, 2006; Rodin, 1986, 1987, 1990).

More recent work has provocatively begun to explore short-term intra-individual variations in personal control and other personality traits and suggests that the greater the week-to-week variation (vs. more stable patterns), the greater the risk for adverse health outcomes (Eizenman, Nesselroade, Featherman, & Rowe, 1997; Nesselroade, 1988). As the proponents of this approach (Eizenman et al., p. 499) note, however, “This is not to downgrade the role of consistent differences in the level of perceived control. Rather, it is an argument favoring the inclusion of lability in the modeling of apparently key personality processes.”

Of particular interest in this regard was the noteworthy drop in personal control immediately following the attacks of September 11, 2001 that were serendipitously captured in an ongoing longitudinal study of health-related quality of life (Wolinsky, Wyrwich, Kroenke, Babu, & Tierney, 2003). Those data indicated that a negative intervention (i.e., the 9–11 terrorist acts) had an immediate and lasting (at least for 6 months thereafter) detrimental effect on personal control levels and age-related trajectory slopes among older adults.

That finding raises the question of whether with appropriate positive intervention, personal control levels could be enhanced among older adults, potentially resulting in subsequent lasting improvements in health and health behavior. Emerging evidence partially supports this view, at least from the perspective that control beliefs in specific domains (such as fear of falling, a common concern among older adults) can be modified (Lachman, 2006). In particular, Tennstedt and colleagues (1998) used cognitive restructuring to significantly improve falling-specific control beliefs in older adults. Those altered falling-specific control beliefs did not, however, result in changes in fall rates (in part because the targeted behavior was avoidance of activity related to concerns about falling), and there was no evidence of a more generalized (i.e., nontask-specific) improvement in personal control.

In this research note, we explore the intriguing possibility of improving cognitive-specific measures of personal control in older adults using data from the National Institutes of Health funded multisite Advanced Cognitive Training for Independent and Vital Elderly (ACTIVE) randomized controlled trial (RCT; Jobe et al., 2001). Specifically, we examine, from an intent-to-treat perspective, whether the three distinct cognitive interventions fielded in ACTIVE—memory, reasoning, and speed of processing—resulted in improvements in cognitive-specific locus of control that lasted as

long as 5 years among older adults. That is, we look for enduring changes in cognitive-specific personal control among the study participants that can be causally attributed to the three cognitive interventions. Because each of the three cognitive interventions focused on strengthening individual skills and abilities rather than external factors, we hypothesize that if these interventions had an effect, then that effect should have been manifested only on internal locus of control and not on chance or powerful others locus of control measures.

METHODS

ACTIVE was designed to examine the long-term effects of cognitive interventions on daily functioning among independently living older adults. Detailed descriptions of the conceptual model on which the ACTIVE study was based, as well as the overall study design, are available elsewhere (Ball et al., 2002; Jobe et al., 2001; Willis et al., 2006). Briefly, ACTIVE hypothesized that each of the three cognitive interventions would have a direct effect on their targeted cognitive outcomes and that the effects on both the primary (daily function) and the secondary (health) study outcomes would be mediated through these targeted cognitive outcomes. Furthermore, ACTIVE expected the reasoning and memory interventions to affect everyday problem-solving aspects of instrumental activities of daily living (IADLs), whereas the speed of processing intervention was expected to affect everyday speed aspects of IADLs. In this research note, we focus on the effect of each of the three cognitive interventions on locus of control at 5-year postbaseline.

All ACTIVE participants were living independently in the community and were aged 65 years or older at baseline. Recruitment strategies were unique to each site. From March 1998 through October 1999, 5,000 potential participants were identified (Jobe et al., 2001), of whom 935 (18%) were subsequently excluded for prespecified reasons. Another 1,263 potential participants (25%) were unwilling to participate. The 2,802 remaining potential participants were screened, signed written Institutional Review Board-approved informed consent, and were enrolled in ACTIVE.

Baseline data were collected after enrollment, and each site randomly assigned participants to four study groups using a computerized program. At all follow-ups, data collectors were blinded to treatment assignment. Participants were reassessed immediately after training and at 1-, 2-, 3-, and 5-year postbaseline. We focus on the baseline to 5-year follow-up because only long-term changes in personal control levels would be expected to result in the improvements in health and health behavior that we have already observed in ACTIVE (Wolinsky, Unverzagt, Smith, Jones, Stoddard, et al., 2006; Wolinsky, Unverzagt, Smith, Jones, Wright, et al., 2006; Wolinsky et al., 2009).

The memory, reasoning, and speed of processing interventions each involved ten 1-hr intervention sessions over

6 weeks, with an optimum group size of 3–4 participants per group. These sessions were standardized such that they shared nine key elements (e.g., practice, individual and group components, fostering self-efficacy, and social interaction). The first five sessions focused on strategy instruction and practice exercises, whereas the last five provided additional practice. Both laboratory-type and everyday activities were well specified in trainer protocol manuals (Jobe et al., 2001). About 1 month prior to the first and third annual follow-ups, booster training was offered to a 60% random sample of cognitive intervention participants who had completed at least 80% of the initial training sessions. These participants received up to four additional standardized sessions at each of those two follow-ups under equivalent circumstances.

The focus of “reasoning training” was on inductive reasoning, specifically the ability to solve problems involving linear thinking or following a serial pattern. Examples included understanding daily medication dosing patterns or using a bus schedule to plan a trip. “Memory training” focused on verbal episodic memory and used multiple mnemonic strategies for remembering lists, item sequences, text material, and story details. Examples included shopping and “to-do” lists. “Speed of processing training” focused on visual search and the ability to identify and locate visual information quickly in a divided attention computerized format. Stimulus target duration was systematically reduced, and the divided visual attention difficulty level and field size were progressively increased over time in response to improved performance.

Locus of control was measured using a shortened version (half of the original items) of reliable and validated 12-item indices of Lachman, Baltes, Nesselroade, and Willis (1982) that specifically target the intellectual (i.e., cognitive-specific) aspects of the internal, chance, and powerful others dimensions of locus of control that were originally identified by Levenson (1974). Each of our cognitive-specific locus of control scales contains six items with a standard 6-point response set—*strongly agree* to *strongly disagree*. Illustrative questions include the following: (a) “If I studied a map carefully, I could figure out how to get around in a strange place” (internal); (b) “I have little control over my mental state” (chance); and (c) “I can’t figure out sale prices of items unless someone helps me” (powerful others).

The internal consistency reliability of the original 12-item cognitive-specific scales of Lachman and colleagues (1982) was clearly acceptable (alpha coefficients were .84, .76, and .76 for the internal, chance, and powerful others scales, respectively), and the multitrait, multimethod, and confirmatory factor analytic evidence for their validity was substantial (Lachman et al.). We found comparable results in ACTIVE for our six-item versions of these cognitive-specific locus of control scales, with exploratory factor analyses indicating simple factor structures for each scale; minimum factor loadings greater than or equal to 0.50; and alpha coefficients of .62, .79, and .73 for the internal, chance, and powerful

others scales, respectively. Scores on our six-item versions of these cognitive-specific locus of control scales ranged from 6 to 36, with high scores indicating greater endorsement of that particular locus of control dimension. Thus, improvements reflecting a greater sense of personal control or individual responsibility over time would involve gain scores (positive 5-year minus baseline values) on internal locus of control and loss scores (negative 5-year minus baseline values) on chance and powerful others locus of control.

Two thresholds were used to define meaningful improvements in each of the cognitive-specific locus of control scales. This involved greater than or equal to 0.5 *SD* and greater than or equal to 1.0 *SD* increases in the scores between baseline and the 5-year follow-up for the internal locus of control scale (because higher numerical scores on this scale are desirable) and greater than or equal to 0.5 *SD* and greater than or equal to 1.0 *SD* decreases in the scores between baseline and the 5-year follow-up for the chance and powerful others locus of control scales (because lower numerical scores on these scales are considered preferable). We used the same thresholds to define meaningful decrements in each of the locus of control scales. Those involved greater than or equal to 0.5 *SD* and greater than or equal to 1.0 *SD* decreases in the scores between baseline and the 5-year follow-up for the internal locus of control scale and greater than or equal to 0.5 *SD* and greater than or equal to 1.0 *SD* increases in the scores between baseline and the 5-year follow-up for the chance and powerful others locus of control scales. Those with stable values (i.e., neither meaningful increments nor decrements in locus of control scale scores) were used as the reference (i.e., comparison) group. This approach is consistent with traditional criteria for medium and large effect sizes, respectively (Cohen, 1969), has been well validated in a large meta-analysis of anchor-based quality of life studies (Norman, Sloan, & Wyrwich, 2003), and is somewhat more stringent than meta-analysis-based draft recommendations from the Section on Clinical Geropsychology of the American Psychological Association’s review of memory training programs in older adults that suggested a minimum threshold for clinical relevance greater than 0.20 *SD* (Rebok, Carlson, & Langbaum, 2007).

To be included in our analytic sample, participants had to have locus of control scores available at baseline and at the 5-year follow-up. Fifty-five percent (1,534) of the 2,802 original ACTIVE participants met these criteria. Because this created the potential for selection and attrition bias, we used a propensity score adjustment model (D’Agostino, 1998; Robins, Rotnitzky, & Zhao, 1994; Rosenbaum & Rubin, 1983; Rubin, 1979). We estimated a logistic regression model of whether participants were included in the analytic sample and computed their predicted probability of inclusion (Hosmer & Lemeshow, 1989). This model included treatment group assignment; demographic; socioeconomic; and cognitive, health, and functional status measures at baseline (complete list available on request). The propensity

score model fits the data well (C -statistic = .67; Hosmer–Lemeshow statistic p value = .98; Hanley & McNeil, 1982; Hosmer & Lemeshow). Within each propensity score (predicted probability) quintile, we determined the average participation rate (i.e., inclusion in the analytic sample or P) and used the inverse ($1/P$) to weight the data. This gave greater influence to participants in the analytic sample most like those not included. We then adjusted the propensity score weights so that the final weighted N was equal to the actual number of participants (i.e., 1,534).

We used multinomial logistic regression (Hosmer & Lemeshow, 1989) to model the effects of the three treatment groups on both meaningful improvements and decrements in our three cognitive-specific locus of control scales. Although somewhat more complicated than traditional binomial logistic regression, multinomial logistic regression allowed us to simultaneously contrast meaningful improvements and decrements in locus of control levels against participants whose personal control did not change (i.e., the reference or comparison group). This is especially important because the hypothesized effects of the cognitive interventions could (a) facilitate improvements over time, (b) protect against decrements over time, or (c) do both. Our analyses included three dummy variables contrasting each treatment group with the no-contact control group as well as the baseline locus of control score (in order to adjust for initial levels of personal control and to address the potential for floor and ceiling effects). We did this for both the medium (≥ 0.5 SD) and the large (≥ 1.0 SD) improvement thresholds. Because the method of selecting participants to receive booster training was conditioned (i.e., dependent) on participant adherence, we did not consider the booster effect in our main analyses in order to maintain fidelity to the intent-to-treat approach; however, we report additional (exploratory) analyses to address this dose–response issue.

RESULTS

Of the 1,534 participants in the analytic sample, there were 389, 393, 392, and 360 in the memory, reasoning, speed of processing, and no-contact control groups, respectively. After weighting the data to adjust for potential selection and attrition bias (weighted $N = 1,534$), the mean age at baseline was 73 years, 22% were men, 27% were Black, and the average educational attainment was 13.5 years. The mean number of chronic health conditions was 2.2, and 15% reported being in fair or poor (vs. excellent, very good, or good) health.

Table 1 contains the locus of control scale means by intervention group at baseline. As expected in a RCT, there were no statistically significant differences between intervention groups on these baseline means. Table 2 contains the percent who met medium (≥ 0.5 SD) or large (≥ 1.0 SD) thresholds for improvement in the locus of control scales between baseline and the 5-year follow-up by treatment

Table 1. Unadjusted Baseline Means on Internal, Chance, and Powerful Others Locus of Control Scales by Treatment Group ($N = 1,534$)

Treatment group	Internal locus of control	Chance locus of control	Powerful others locus of control
Baseline			
Memory	31.0	17.3	14.7
Reasoning	30.9	17.3	15.3
Speed of processing	31.0	17.5	15.0
No-contact control	31.0	17.2	14.7

group, unadjusted for baseline values. Although there were no statistically significant crude (i.e., unadjusted for baseline values) overall differences between the treatment groups in this table, there was trend evidence for medium improvements on internal locus of control ($p = .06$). Moreover, pair-wise contrasts indicated that at the medium improvement threshold, both the reasoning ($p = .01$) and the speed of processing ($p = .04$) training interventions resulted in significantly more medium-sized improvements relative to the no-contact control group. There was also pair-wise trend evidence that the memory ($p = .06$), reasoning ($p = .07$), and speed of processing ($p = .07$) interventions resulted in more large improvements relative to the no-contact controls on internal locus of control. Both the medium and the large improvements in internal locus of control resulted from there being fewer ACTIVE participants with unchanged internal locus of control scales in the reasoning and speed of processing intervention groups (49% and 50%, respectively) than in the no-contact control group (55%). Indeed, there were no meaningful differences across the treatment groups in terms of the percentage of participants whose internal locus of control scales reflected medium-sized changes (range = 26.8%–28.6%). Thus, although declines in internal locus of control were more common than improvements, being assigned to either the reasoning or the speed of processing groups increased the likelihood of improved locus of control scores.

Table 2. Unadjusted Percent With Medium (≥ 0.5 SD) and Large (≥ 1.0 SD) Improvements in Internal, Chance, and Powerful Others Locus of Control Scales at 5 Years by Treatment Group ($N = 1,534$)

Improvement level	Internal locus of control	Chance locus of control	Powerful others locus of control
Medium (≥ 0.5 SD)			
Memory	20.6	27.0	26.7
Reasoning	23.3	26.7	29.8
Speed of processing	22.3	26.6	25.7
No-contact control	16.1	23.3	22.9
Large (≥ 1.0 SD)			
Memory	12.6	11.9	11.8
Reasoning	12.5	10.6	10.4
Speed of processing	12.6	13.0	11.2
No-contact control	8.4	10.6	8.9

Table 3. Adjusted Odds Ratios From Multiple Multinomial Logistic Regression of Medium ($\geq 0.5 SD$) and Large ($\geq 1.0 SD$) 5-Year Improvements and Decrements on the Internal Locus of Control Scale ($N = 1,534$)

Risk factor	Medium decrement	No change	Medium improvement
Treatment group			
Memory	0.98	1.00	1.37
Reasoning	1.10	1.00	1.76**
Speed of processing	1.07	1.00	1.68*
Baseline internal locus of control score	1.01	1.00	0.76***
Risk factor	Large decrement	No change	Large improvement
Treatment group			
Memory	0.99	1.00	1.49
Reasoning	1.13	1.00	1.56†
Speed of processing	1.17	1.00	1.62†
Baseline internal locus of control score	1.08***	1.00	0.75***

Notes: Improvements in personal control reflect higher 5-year minus baseline numerical scores on the internal locus of control scale.

Chi-square for the medium-sized changes = 320.6 at 8 *df*, $p < .001$.

Chi-square for the large-sized changes = 270.0 at 8 *df*, $p < .001$.

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

Table 3 contains the results for the internal locus of control scale obtained from the multinomial logistic regression analyses using the medium improvement (upper panel) and large improvement (lower panel) thresholds, adjusting for the baseline level of the internal locus of control measure. Relative to the no-contact control group, the reasoning and the speed of processing interventions had statistically significant effects on medium improvements in the internal locus of control measure. The adjusted odds ratios (AORs) of 1.76 ($p = .01$) for the reasoning and 1.68 ($p = .02$) for the speed of processing interventions indicate that ACTIVE participants assigned to these treatment groups had 76% and 68%, respectively, greater odds than those in the no-contact control group to have achieved a medium level ($\geq 0.5 SD$) improvement in internal locus of control. This pattern of effects for the internal locus of control scale was also observed at the large improvement threshold, albeit at a marginally nonsignificant level (AORs = 1.56 and 1.62, $ps < .10$). This was to be expected, given the reduced power available (as shown in Table 2, fewer participants met this more stringent threshold). At both the medium and the large improvement levels, ceiling effects were evident such that those with higher baseline internal locus of control scores were less likely to improve, and some evidence of a floor effect was found in that those with higher baseline scores were more likely to decline at the large decrement level.

To explore the dose–response relationship associated with the booster training, we conducted additional analyses. Specifically, we replaced the set of three dummy variables contrasting the intervention groups with the no-contact control group in the multinomial logistic regression analyses with a set of six dummy variables that separate each inter-

Table 4. Adjusted Odds Ratios From Multiple Multinomial Logistic Regression of Medium ($\geq 0.5 SD$) and Large ($\geq 1.0 SD$) 5-Year Improvements and Decrements on the Chance Locus of Control Scale ($N = 1,534$)

Risk factor	Medium decrement	No change	Medium improvement
Treatment group			
Memory	0.84	1.00	1.17
Reasoning	1.06	1.00	1.20
Speed of processing	0.87	1.00	1.15
Baseline chance locus of control score	0.94***	1.00	1.11***
Risk factor	Large decrement	No change	Large improvement
Treatment group			
Memory	0.60*	1.00	0.99
Reasoning	0.83	1.00	0.97
Speed of processing	0.70†	1.00	1.18
Baseline chance locus of control score	0.90***	1.00	1.15***

Notes: Improvements in personal control reflect lower 5-year minus baseline numerical scores on the chance locus of control scale.

Chi-square for the medium-sized changes = 221.1 at 8 *df*, $p < .001$.

Chi-square for the large-sized changes = 179.6 at 8 *df*, $p < .001$.

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

vention group into those who were or were not invited to participate in the booster sessions about a month before the first and third annual follow-ups. Even though statistical power was substantially reduced (due to the smaller-sized groups), those results (complete data not shown but available on request) indicated that among participants invited to receive boosters, the effects for the speed of processing and reasoning interventions on medium-sized ($\geq 0.5 SD$) improvements in the cognitive-specific internal locus of control scale were statistically significant (AOR = 2.02, $p < .01$ and AOR = 1.86, $p < .01$, respectively) and were noticeably larger than those for participants who were not invited to participate in the boosters (AOR = 1.36, $p = .27$ and AOR = 1.60, $p = .10$, respectively). Furthermore, we observed remarkably similar results when using the large-sized ($\geq 1.00 SD$) improvement criterion in the cognitive-specific internal locus of control scale for the speed of processing and memory interventions (AOR = 2.00, $p < .05$ and AOR = 1.85, $p < .05$, respectively, for these boosted groups vs. AOR = 1.39, $p = .35$ and AOR = 1.35, $p = .37$, respectively, for these nonboosted groups).

As noted, we did not expect to find that any of the cognitive interventions would have an effect on the chance or powerful others locus of control scales because these interventions focused on strengthening individual abilities rather than addressing external influences. Results for the chance and powerful others locus of control scales are shown in Tables 4 and 5. Contrary to our expectations, the results in Table 4 revealed that being assigned to the memory (AOR = 0.60, $p < .05$) or speed of processing (AOR = 0.70, $p < .10$) interventions marginally protected against large decrements

Table 5. Adjusted Odds Ratios From Multiple Multinomial Logistic Regression of Medium (≥ 0.5 SD) and Large (≥ 1.0 SD) 5-Year Improvements and Decrements on the Powerful Others Locus of Control Scale ($N = 1,534$)

Risk factor	Medium decrement	No change	Medium improvement
Treatment group			
Memory	0.83	1.00	1.04
Reasoning	0.80	1.00	1.04
Speed of processing	0.84	1.00	0.93
Baseline powerful others locus of control score	0.98*	1.00	1.13***

Risk factor	Large decrement	No change	Large improvement
Treatment group			
Memory	0.80	1.00	1.43
Reasoning	0.89	1.00	0.88
Speed of processing	1.06	1.00	1.05
Baseline powerful others locus of control score	0.98	1.00	1.16***

Notes: Improvements in personal control reflect lower 5-year minus baseline numerical scores on the internal locus of control scale.

Chi-square for the medium-sized changes = 188.8 at 8 *df*, $p < .001$.

Chi-square for the medium-sized changes = 122.6 at 8 *df*, $p < .001$.

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

in the cognitive-specific chance locus of control scale. They did not, however, have any effect on improving scores on the chance locus of control scale. Table 5 shows that, as expected, none of the three cognitive interventions had significant effects on any changes in the cognitive-specific powerful others locus of control scale.

DISCUSSION

In a large multisite RCT, we have shown that two cognitive training interventions—reasoning and speed of processing—resulted in long-term medium-sized (≥ 0.5 SD) statistically significant relative improvements (AORs = 1.76 and 1.68, respectively) in a short version of cognitive-specific internal locus of control scale of Lachman and colleagues (1982). We also observed similar improvements in the cognitive-specific internal locus of control scale when the large-sized (≥ 1.0 SD) effect threshold was used, although given the reduction in statistical power associated with that more stringent criterion those results were marginally nonsignificant ($p < .10$). This pattern of effects on the cognitive-specific locus of control scale was expected because the reasoning and speed of processing training interventions focused on maintaining or improving the cognitive processing abilities in older adults to preserve their independence during a period of their lives when cognitive abilities and performance are commonly on the decline.

In contrast, we did not expect that any of the three cognitive interventions would generalize to short versions of cognitive-specific chance and powerful others locus of control scales of Lachman and colleagues (1982). Our reason-

ing here was that these independent dimensions of locus of control are believed to be influenced more by factors external to the individual rather than by their own abilities. That was the case for the powerful others locus of control scale. We did, however, find that being assigned to the memory or speed of processing interventions protected against large decrements in the chance locus of control scale.

On the one hand, we believe that the combination of the long-term and medium-sized effects of the reasoning and speed of processing cognitive interventions on improving levels of the cognitive-specific internal locus of control scale is noteworthy. These effects were observed at 5-year postintervention, despite the well-documented, normative age-related decline in personal control that has been found repeatedly in both cross-sectional (Lachman, 2006; Lachman & Firth, 2004; Rodin, 1986, 1987, 1990) and longitudinal studies (Mirowsky, 1995, 1997; Wolinsky, Wyrwich, Babu, et al., 2003). Moreover, these improvements resulted from a rather low-dose initial exposure to just ten 1-hr training sessions, supplemented among about half of the training participants by four additional sessions about 1 month before the first and third annual follow-ups. As such, our results corroborate findings by Tennstedt and colleagues (1998) that control beliefs in specific domains (such as fear of falling, a common concern among older adults) can be modified by cognitive interventions and extend that work from a focus on personal control for specific situations (such as the fear of falling) to the broader pattern of cognitive-specific personal control.

On the other hand, the absolute percentages of participants in the reasoning and speed of processing groups who achieved these improvements in internal locus of control were modest. Indeed, only an additional 7.2% of participants in the reasoning and 6.2% of participants in the speed of processing groups had medium-sized improvements (relative to the no-contact control group), and only an additional 4.1% of participants in the reasoning and 4.2% of participants in the speed of processing groups had large-sized improvements. When coupled with the absence of any reduction in the percentages of participants in these groups with normative age-related declines, the modest absolute improvements brought about by ACTIVE's cognitive interventions may not be viewed by some as particularly noteworthy. We disagree with that perspective for two reasons. First, based on these attributable (absolute) percentages of medium-sized increases in internal locus of control, the numbers needed to treat (Guyatt, Sackett, & Cook, 1993a, 1993b) to bring about one additional success (i.e., medium-sized increase) were not large—13.9 for the reasoning group and 16.1 for the speed of processing group. Second, although our exploratory analyses of the dose–response relationship associated with the booster training did not adhere strictly to an intent-to-treat approach (because invitation to participate in the boosters was conditioned on participant adherence), those results did reflect a clear dose–response relationship such that larger doses (initial + booster training)

resulted in meaningfully larger effects than smaller doses (initial only). Thus, it is plausible that larger doses of the initial memory, reasoning, and speed of processing interventions or an aggressive recurring pattern of booster treatments would have increased the size of the memory, reasoning, and speed of processing effects on cognitive-specific internal locus of control.

FUNDING

The ACTIVE cognitive training trial was supported by grants from the National Institutes of Health to six field sites and the coordinating center, including the following: Hebrew Senior-Life, Boston (NR04507), the Indiana University School of Medicine (NR04508); the Johns Hopkins University (AG14260); the New England Research Institutes (AG14282); the Pennsylvania State University (AG14263); the University of Alabama at Birmingham (AG14289); and the University of Florida (AG014276). F.D.W. was Associate Director of and M.W.V.W. and R.M. are Investigators at the Center for Research in the Implementation of Innovative Strategies in Practice at the Iowa City Veterans Administration Medical Center, which is funded through the Department of Veterans Affairs, Veterans Health Administration, Health Services Research and Development Service (HFP 04-149).

ACKNOWLEDGMENTS

F.D.W. has a limited consulting arrangement with Posit Science Corporation, which in October 2007 acquired ownership of the speed of processing intervention used in the ACTIVE cognitive training trial; however, F.D.W. has not received any support from Posit Science Corporation for the analysis and writing of this manuscript. The speed of processing intervention used in ACTIVE was originally developed by K.K.B. and colleagues. As a result of the sale of that intervention to Posit Science Corporation, K.K.B. is now a stockholder in Posit Science Corporation and serves on its Advisory Board. G.W.R. is an investigator with Compact Disc Incorporated for the development of an electronic version of the ACTIVE memory intervention. He has received no financial support from them for ACTIVE. The opinions expressed here are those of the authors and do not necessarily reflect those of the funding agencies, academic, research, governmental institutions, or corporations involved. F.D.W. conceived of the study, designed and executed the statistical analyses, and drafted and revised the manuscript. M.W.V.W. and R.M. assisted with the conception of the study; provided counsel on the statistical analyses and their interpretation; and read, commented upon, and approved all versions of the manuscript. F.W.U., S.L.W., M.M., G.W.R., J.N.M., K.K.B., and S.L.T. are ACTIVE Site Principal Investigators and members of the ACTIVE Executive Committee provided counsel on the conception of the study and statistical analyses and read, commented upon, and approved all versions of the manuscript.

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