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Associations Between Vascular Risk and Mood in Euthymic Older Adults: Preliminary Findings

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Abstract

Objectives—Vascular risk has been associated with late-life depression, but it is less certain whether it is also associated with the endorsement of depressive symptoms among euthymic older adults. We explore whether vascular risk is associated with endorsement of depressive symptoms among euthymic older adults and examine associations with cognitive function.

Methods—Fifty-seven adults (50–89 years), were assessed for: 1) vascular risk (Framingham Stroke Risk Profile, FSRP); 2) depressive mood (Center for Epidemiologic Studies Depression, CESD self-rating questionnaire; Hamilton Depression Rating Scale, HDRS clinical interview); and 3) cognitive domains, (Learning and Memory, L-M; Attention and Information Processing, AIP; Executive Function, EF; Semantic Language, SL).

Results—Significant correlations were observed between FSRP and both depression scales, independent of age. No significant correlations were observed between HDRS and any cognitive domain; in contrast, CESD correlated significantly with L-M, AIP and EF but not SL. FSRP correlated significantly with L-M and EF measures only. Regression analyses revealed that 11.5% of the variance in HDRS scores was explained by FSRP, whereas CESD scores were explained by EF (20.8% of variance).

Conclusions—Vascular risk was associated with endorsement of depressive symptoms in euthymic older adults. However, the patterns of associations with the two depression scales are distinct and may reflect both differences in administration and item characteristics. A limitation of this study was the exclusion of individuals with subclinical depression, leading to a restricted range on depression scales; future studies should include a full population sample to more fully explore low mood in late-life.

Keywords

Aging; cardiovascular risk; depression

Most studies agree that subclinical depressive symptoms are common among otherwise euthymic adults aged 60 years and older who often underreport low mood.^{1,2} In fact, although the prevalence of depression in older adults is low (approximately 7%), the

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presence of undiagnosed and subclinical depressive symptoms is high, estimated to be 15% of the adult population older than 60 years. Vascular risk factors are strongly associated with an increased probability of depression among older adults;^{3–5} however, it is not yet clear whether vascular risk is associated with the endorsement of depressive symptoms in euthymic older adults or whether it represents a risk factor for developing depression. Understanding vascular risk across a continuum of depressive symptoms in aging may clarify mechanisms for development and progression of depression in late life.

It has long been hypothesized that depression in a subset of individuals with late-life depression (LLD) is due, in part, to vascular risk factors and associated cerebrovascular damage that accumulates with increased age, a syndrome commonly described as vascular depression (for more details see previous research^{6–8}). Vascular risk factors both in isolation and in a composite index such as the Framingham Stroke Risk Profile (FSRP)⁹ score have not only been shown to relate to overall white matter integrity but also to the integrity of specific white matter tracts in the brains of individuals with LLD,³ tracts known to regulate emotional expression and mood. The FSRP score has also been associated with white matter damage in healthy aging.¹⁰ Furthermore, individual components of this index (i.e., the presence of cardiovascular disease, hypertension, left ventricular hypertrophy) all contribute to the variance in white matter damage.^{9,11} The extent to which the FSRP and/or its component parts contribute to depressive symptoms in the absence of clinically diagnosed depression in the elderly is less well understood.

The vascular depression hypothesis not only discusses the role of vascular risk factors and associated cerebrovascular damage in LLD, but it also provides insight as to the associated cognitive difficulties, most notably executive function.^{12,13} Evidence of a similar cognitive profile in individuals without a diagnosis of depression but endorsing depressive symptoms is accumulating.¹⁴ For example, in a community-based study, euthymic individuals who endorsed depressive symptoms at baseline were at increased risk of subsequent cognitive decline.¹⁵ In a separate aging study, we found that endorsement of depressive symptoms did not increase with age, nor was it influenced by executive abilities; it was, however, associated with the presence of white matter damage.¹⁶ Although this study demonstrated that the association between depressive symptoms and white matter damage was independent of executive abilities, a more directed analysis of associations with cognition was not performed. A similar pattern of associations between vascular risk factors and cognition has also been shown in healthy middle-aged and older adults,^{11,17} suggesting that these factors may also be measured on a continuum.

The primary aim of the current study was to examine the associations between vascular risk, depressive symptoms, and cognition in a sample of euthymic adults older than 50 years. We hypothesized that: 1) greater vascular risk as measured by using the FSRP will be associated with greater endorsement of depressive symptoms; and 2) depressive symptoms will be associated with poorer cognitive function across a number of composite indices, including executive function. Long term, we see understanding the interplay between these mechanisms in the continuum between healthy (euthymic) aging and LLD as having the potential to inform disease-monitoring techniques and treatment. The first step, however, is

to determine the direction of association between vascular risk, depressive symptoms, and cognition in euthymic older adults.

METHODS

Participants

Data were collected as part of a larger program of research investigating depression and diabetes across the lifespan within the Department of Psychiatry at the University of Illinois at Chicago. Specifically, participants were recruited from two studies of depression: one that recruited individuals with and without depression who were older than 60 years and another study of diabetes and depression across the lifespan in which individuals were aged 30 years and older. Further details of the proportions of those individuals included from each study are presented in the following discussion. Only nondepressed individuals were included in the current study regardless of the presence or absence of type 2 diabetes. Both studies were approved by the University of Illinois at Chicago institutional review board and were conducted in accordance with the Declaration of Helsinki. All participants gave written informed consent.

Data presented here include only healthy (i.e., nondemented, nondepressed) adults aged 50 years or older, recruited via community outreach (newspaper, radio, and television advertisements). As previously stated, individuals with diabetes were not excluded. Participants underwent a preliminary telephone screen. Exclusion criteria were current or history of brain disorders (i.e., dementia, stroke, seizure), history of head injury or loss of consciousness, current or history of substance abuse or dependence, current or history of an Axis I psychiatric diagnosis, and psychotropic medication use including antidepressant medication.

After passing the telephone screen, all participants completed a more detailed evaluation including cognitive (i.e., Mini-Mental State Examination¹⁸) and affective (i.e., Structured Clinical Interview for DSM-IV¹⁹) screens for final inclusion determination. Measures were administered by a trained research assistant and were followed by an evaluation by a board-certified (AK) or board-eligible (OA) psychiatrist who completed the Hamilton Depression Rating Scale (HDRS).²⁰ Raters were blind to telephone screen information. Final inclusion criteria for healthy older participants included absence of symptoms of depression based on the Structured Clinical Interview for DSM-IV and score <8 on the HDRS. All participants had a Mini-Mental State Examination score >24, indicating that cognitive impairment was unlikely, and all were native English speakers.

Participants completed the Center for Epidemiologic Studies Depression scale (CESD)²¹ as a subjective measure of depressive symptoms. A CESD score ≥ 23 is considered probable depression and ≥ 16 possible depression. In the current sample, CESD scores ranged from 0–21; no individuals were classified as probable depression, and 4 individuals had scores ≥ 16, indicating that they may have had subclinical or possible depression. Participants received an assessment of vascular risk according to the FSRP.⁹ History of stable (e.g., hypertension) or remitted (e.g., cancer) medical illness was not an exclusionary factor. The mean delay

between assessment of vascular risk factors and neuropsychological assessment was 17.25 days.

Ninety-four individuals aged 50 years and older attended the initial screening. Twenty-one individuals were excluded based on screen results: five had past substance abuse/dependence; six had English as a second language; one was on contraindicative medication; two had psychiatric diagnoses; two had recent neuropsychological testing; one individual suffered a previous sustained loss of consciousness; and four had sleep apnea. Of the 73 individuals who passed screening, 29 (40%) had diabetes. Although we did not want to exclude individuals with diabetes (it represents a significant vascular risk factor), this rate is proportionally higher than the state of Illinois average (23.8%)²² and was likely due to active recruitment of individuals with diabetes for one of our two studies. To represent the population as a whole and avoid overrepresenting persons with diabetes, the proportion of individuals with diabetes was reduced to 23% by random sampling and using SPSS (IBM SPSS 20 Statistics, IBM Corporation, Armonk, NY) to better represent the larger population from which this sample was taken. The final sample (n = 57) included 44 healthy individuals recruited as controls in the aforementioned LLD study (1 of whom had type 2 diabetes) and 13 individuals with diabetes recruited from the previously described diabetes and depression study (Table 1).

Neuropsychological Assessment

Qualifying participants were scheduled for a second visit, at which a comprehensive neuropsychological assessment was administered by a trained research assistant. The cognitive domains of Learning and Memory (L-M), Attention and Information Processing (AIP), Executive Function (EF), and Semantic Language (SL) were assessed by using variables from the following measures. L-M was assessed by using California Verbal Learning Test–II²³ immediate and long delay free recall of a 16-item word list and the Wechsler Memory Scale–III²⁴ Logical Memory I and II, as well as Visual Reproduction I and II, both immediate and delay free recall of prose passages and simple geometric shapes, respectively. AIP was assessed by using the Stroop Color and Word reading trials raw scores,²⁵ Trail Making Test A time to completion for connecting dots in sequential numerical order,²⁶ and the Digit-Symbol Coding raw score from the Wechsler Adult Intelligence Scale–III²⁷ requiring completion of symbols matched to digits. EF was assessed by using the Category Switching total correct responses when alternating between the generation of fruit and furniture words from the Delis-Kaplan Executive Function System,²⁸ Trail Making Test B time to completion for connecting dots in alternating number and letter sequential order,²⁶ Stroop Interference Score²⁵ when reading the color ink of a particular color word, Backwards Digit Span raw score from the Wechsler Adult Intelligence Scale–III,²⁷ and self-ordered pointing task total errors when attempting to monitor responses across an increasing string of visually presented geometric shapes.²⁹ SL was assessed by using the total correct responses from animal fluency³⁰ as well as a (semantic) association index and a percent (of total output) in (semantic) cluster.³¹ Raw scores were transformed into z-scores and coded so that high scores reflected good performance across all variables; z-scores were then collated to produce a mean score for each of the aforementioned domains. For each cognitive domain, Cronbach's alpha was computed to assess how well the variables

measured a latent construct. All values were considered good, indicating that each variable measured a unidimensional latent construct (L-M, $\alpha = 0.876$; AIP, $\alpha = 0.807$; EF, $\alpha = 0.753$; SL, $\alpha = 0.751$). Appendix 1 presents detailed descriptions of these neuropsychological measures.

Statistical Analyses

Correlational analyses were used to explore the associations between age and depression scales, vascular risk, and cognitive function. Given the expected associations with age, further analyses between variables used partial correlations to control for age effects. Stepwise regression analyses examined the contribution of variables to explaining the variance in depression scales. Two participants had missing individual data points (Table 1); thus, degrees of freedom for individual analyses vary accordingly. Given the number of analyses performed in this study of vascular risk, depressive symptoms, and cognition associations, results should be viewed as preliminary.

RESULTS

Correlations With Age

A significant correlation was noted between the FSRP score and age ($r[53] = 0.389$; $p = 0.003$). As expected, the HDRS and CESD correlated significantly with each other ($r[54] = -0.368$; $p = 0.008$). Age did not correlate significantly with either HDRS or CESD scales.

Correlations Between Vascular Risk and Mood

Correlations between the FSRP and depression scales are reported, controlling for age. The FSRP correlated significantly with both the HDRS and CESD. It is worth noting that the association between the FSRP and CESD did not reach significance ($r[52] = 0.262$; $p = 0.056$) until we controlled for age.

Correlations With Cognitive Variables

No significant correlations were observed between HDRS and any of the four cognitive variables (Table 2). For CESD, significant correlations were observed with L-M ($r[54] = -0.460$; $p < 0.001$), AIP ($r[54] = -0.371$; $p = 0.005$), and EF ($r[54] = -0.497$, $p < 0.001$) but not with SL ($r[54] = -0.057$; $p = 0.678$). Age did not correlate with any of the cognitive variables. Correlations are reported controlling for age, but results were not substantially altered without this covariate. The associations between FSRP and cognitive abilities also controlled for age. FSRP correlated significantly with both L-M and EF but not with AIP or SL. Table 2 presents full details of correlations and partial correlations.

Given the association between FSRP and cognition, the association between CESD and cognitive variables was explored while controlling for FSRP. The associations between CESD and L-M ($r[53] = -0.383$; $p = 0.005$), AIP ($r[53] = -0.325$; $p = 0.019$), and EF ($r[53] = -0.414$; $p = 0.002$) were not substantially altered by controlling for FSRP as well as age.

Regression Analyses

Separate stepwise regressions were performed with HDRS or CESD as dependent variables; independent variables included were age, FSRP, and cognitive variables (L-M, AIP, EF, and SL). Missing data for two subjects reduced the degrees of freedom in the analysis.

The model explained 11.5% of the variance in HDRS scores, with only FSRP contributing significantly ($F_{[1, 53]} = 6.86$; $p = 0.011$ [R^2 95% confidence interval: 0.006–0.309]).

For the CESD, 20.8% of the variance in CESD scores was explained, with only EF significantly contributing to the variance ($F_{[1, 52]} = 13.63$; $p = 0.001$ [R^2 95% confidence interval: 0.045–0.417]).

CONCLUSIONS

In this study of euthymic older adults, we demonstrated a significant association between vascular risk and depressive symptoms as rated by an experienced psychiatrist as well as by self-report. For HDRS, this association was independent of age; for CESD, the association did not reach significance until age was included as a covariate. Thus, presence of vascular risk was associated with an increase in both objectively (psychiatrist) and subjectively (self) rated depressive symptoms even when these symptoms were below standard cutoffs for depression. Results are complementary to those from a previous study, which used a different self-report measure (the Geriatric Depression Scale); depressive symptoms were significantly correlated with white matter damage (measured with diffusion tensor imaging) associated with vascular risk in euthymic middle-aged and older adults.¹⁶ Furthermore, our results revealed a pattern of associations between depression severity and vascular risk factors similar to that observed in LLD.^{3,5,6}

Different patterns of correlations were observed between the depression scales and the cognitive domain measures. HDRS did not correlate with any of the cognitive variables, whereas CESD negatively correlated with three of the four cognitive domains (L-M, AIP, and EF). This dissociation between the mood scales was also apparent in stepwise regression results, in which different factors contributed to explaining the variance in HDRS and CESD. Stroke risk (FSRP) explained 11.5% of the variance in HDRS, with no significant contribution from age or cognitive variables. In contrast, 20.8% of the variance in CESD was explained by EF; no other variable contributed significantly to the model. Associations between CESD and cognition have precedence in the literature, particularly the association with EF, as a core feature of depression–executive dysfunction among older adults with LLD.^{7,13} Furthermore, patients with comorbid depression and executive dysfunction show poorer treatment response and more frequent recurrence of disease.⁷ If the same pattern holds in euthymic older adults, greater vigilance monitoring of both affect and cognition is warranted.

The different associations with the depression scales may reflect differences in the items included on the scales, the way in which they are administered, and the range of possible scores on each depression scale. Although the HDRS and CESD both include items such as low mood, anxiety, and insomnia, more emphasis is placed on somatic items in the HDRS;

interpersonal items feature more strongly in the CESD. In addition, the HDRS does not assess cognitive difficulties, whereas the CESD asks about concentration difficulties. The CESD also requires more cognitive capacity for completion, including aspects of self-monitoring and working memory, to maintain mental set for the Likert scale and task at hand compared with the HDRS, externally driven by a board-certified or board-eligible psychiatrist. Furthermore, vascular damage is associated with cognitive deficits, and, although rare, anosognosia for depression has been reported after stroke.³² The vascular damage occurring in aging may lead to a lack of insight and impede the ability of individuals to evaluate their own depressive symptoms. Thus, as well as requiring self-awareness, CESD may be more cognitively demanding than a clinical interview, leading to the dissociation between the two depression scales. Future studies may consider whether the discrepancy between self-report and psychiatrist-report represents a mild form of anosognosia that could be associated with cardiovascular damage. All of these factors may have influenced the lack of cognitive contributions to the HDRS, as well as the opposite pattern for the CESD.

We cannot deny that study recruitment criteria restricted the range of scores possible on the HDRS more severely (0–6) than the CESD (0–21). This limited range may also contribute to the differences in association as well as the modest correlations seen between the depression scales and cognition. As this was one of only a few attempts to determine associations between vascular risk, cognition, and depressive symptoms in otherwise euthymic older adults, this restriction of range could not be avoided; however, future studies should examine a wider continuum incorporating not only subclinical depression but also milder forms of the disorder for a larger range of scores across multiple assessment tools. Regardless of inherent restrictions of range or overall global differences between the depression measures, the different pattern of associations across the scales may suggest that these measures are also differentially sensitive to items associated with vascular risk and/or low mood without depression. One recent item analysis of the CESD and HDRS suggests that depression severity may best predict group membership in LLD.³³ A detailed analysis of items within these scales may elucidate facets of depressive symptoms specifically associated with vascular risk, as well as clarifying the pattern of depressive symptoms among euthymic older adults.

FSRP correlated significantly with L-M and EF ability independent of age effects. In fact, age did not correlate significantly with these or any other cognitive ability in our healthy aging cohort. Given that the FSRP encompasses multiple risk factors known to negatively affect white matter integrity, this resulting vascular damage might have a greater impact on these higher cognitive functions because they rely on a distributed cognitive network in the brain³⁴ and, therefore, a small amount of damage may disproportionately disrupt these functions. This association between vascular damage and higher order cognitive dysfunction has been observed in healthy aging populations.^{34,35} Follow-up studies assessing the link between vascular risk and vascular damage will confirm this assumption.

The lack of correlations between age and cognition alone is not in keeping with the cognitive aging literature,^{34,36} in which cognitive decline has been observed in most abilities with relative sparing of language function. One reason for these findings may be the small number of older adults in our sample (only three individuals were older than 75 years).

Although the results were not substantially altered by excluding these three individuals, the restricted age range may reduce our ability to observe the expected cognitive associations in older adults, and we acknowledge this limitation. An alternative explanation for the lack of age associations may be the high mean education level for this sample. This education level might contribute to cognitive reserve or resiliency, which has been hypothesized to protect against age-related cognitive decline.³⁷ It is also possible that the modest sample size may have contributed to these nonsignificant findings, although significant associations were observed between FSRP and depressive symptoms.

Despite the restrictions to both the age and depression range of our sample, we did show that depressive symptoms are associated with vascular risk and select cognitive functions in a nondepressed aging cohort. Although we omitted individuals scoring 8–14 on the HDRS, CESD scores suggest we retained a few individuals with subclinical depression. Subclinical depression and mild forms of depression not meeting full diagnostic criteria for major depression (not included in this study) may be particularly common in an elderly cohort.^{38,39} Omitting all such individuals from analyses could result in biased samples that do not represent the full continuum of depression symptoms present in the wider population. Likewise, close investigation of a true “control” sample presumes these individuals to be “risk-free,” but we would argue consideration of cardiovascular risk factors in this cohort is also critical. We included some subjects with subclinical depression ($n = 4$) in our study of the associations between vascular risk, depressive symptoms, and cognition in otherwise euthymic older adults; however, future studies should include euthymic, subclinical, and mildly depressed individuals to integrate information about cardiovascular disease and depression across the full continuum of the aging population, to better identify those at risk for adverse outcomes.

In a population sample such as presented here, it would be particularly informative to monitor longitudinal change in mood and associations with vascular risk over time. Although not possible in the current cross-sectional design, it should be explored in future longitudinal studies, allowing for the development of adverse neurologic events as the study progressed. It is the conundrum of all cross-sectional studies on healthy aging that vascular risk often excludes (via self-selection) individuals likely to have the highest FSRP scores, but we feel that our range of scores (from 1–18), representing the lower two tertiles of the FSRP scale (maximum score: 30), is reflective of other cross-sectional analyses in aging adults.⁹

This study has demonstrated significant associations between vascular risk and the endorsement of depressive symptoms in euthymic older adults, independent of age. Endorsement of depressive symptoms is also associated with cognitive decline, although these associations are present for subjective self-report measures only. Within the general aging population, further research is required to investigate profiles of depressive symptoms beyond nondepressed and subclinical depression, as well as their interactions with vascular risk and cognitive function. In addition, investigations examining how severity of depressive symptoms, including mild to severe clinical depression symptoms, mediates associations between these factors will clarify whether the pattern observed in LLD and in euthymic older adults represent a true continuum. LLD is a heterogeneous disorder that often goes

untreated in the elderly,⁴⁰ affected not only by age of onset, duration of illness, and number of episodes of depression but also by vascular risk and subjective report. As a result, population-based studies are necessary to fully describe the range of symptoms and associations within this population so that targeted interventions associated with specific patterns of disease comorbidity and onset may be developed.

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APPENDIX 1. Description of Neuropsychological Measures

Learning and Memory

California Verbal Learning Test–II¹

A list of 16 words is read at a pace of slightly longer than one word per second. After the list is completed, participants are asked to recall as many words as they can remember in any order. The same list of words is read again with the same procedure for recall; this is repeated for a total of five recall trials. The total number of correctly recalled items across the five trials is the immediate free recall score. After approximately 30 minutes, participants are asked to recall the original list. The number of correctly recalled items is the long delay free recall score.

Wechsler Memory Scale–III² Logical Memory

One short story is read to participants who are then asked to recall as much of the story as possible in as similar words as possible; a second story is read twice with the same recall procedure for each hearing of the story. The total score across story A and story B (twice) are summed to produce Logical Memory I score. After approximately 30 minutes, individuals are asked to recall as much of the two stories as possible. Recall on both stories are summed for the Logical Memory II score.

Wechsler Memory Scale–III² Visual Reproduction

A total of five line drawings are presented for 10 seconds in turn, and individuals are requested to remember as much of the drawing as possible. Immediately after 10 seconds, the individual is asked to reproduce each drawing. Drawings are scored for accuracy according to strict scoring criteria; total score across the five drawings is the Visual Reproduction I score. After approximately 30 minutes, individuals are asked to redraw as much of the line drawings as possible. Recall across all drawings are summed for the Visual Reproduction II score.

Attention and Information Processing

Stroop test³

A list of 100 color words (e.g. red, blue, etc.) or blocks of color are presented to the participant who is required to say aloud either the color (Color trial) or the word (Word trial). The total number of items correctly read aloud in 45 seconds provides the raw score.

Trail Making Test A⁴

A random array of numbers is presented on a page. Participants must start with the number 1 and connect the number in sequential order. Time to completion is the outcome measure.

Wechsler Adult Intelligence Scale–III⁵ Digit-Symbol Coding

Participants are presented with a page containing 1×2 boxes in which the top of the box contains a number (1–9) and the bottom of the box contains a different symbol for each digit. Farther down the page, a series of 1×2 boxes are repeated with the top box containing

a digit, but the symbol section is blank. Participants must draw in the appropriate symbol that goes with each number. Number completed correctly in 2 minutes provides the score.

Executive Function

Delis-Kaplan Executive Function System⁶ Category Switching

Participants must alternate between saying as many different types of fruits and furniture as they can in 60 seconds. Score is the total number of correct items produced.

Trail Making Test B⁴

A random array of numbers and letters is presented on a page. Participants must start with the number 1 and must draw a line between the items alternating between the numbers and the letters, sequentially. Time to completion is the outcome measure.

Stroop Interference Score³

Participants must read aloud color-words (i.e., Red) written in conflicting colored ink. A total of 100 words are presented; the total number of items correctly read aloud in 45 seconds provides the score.

Wechsler Adult Intelligence Scale–III⁵ Backwards Digit Span

Individuals are read a list of numbers, beginning with a list length of 2 digits, and are required to repeat them back in reverse order. The list length increases until the participant is unable to successfully remember the list. Total number of sequences recalled provides the score.

Self-Ordered Pointing Task⁷

Participants are presented with a page containing a number of abstract designs (6, 9, or 12), that are repeated on subsequent pages (pages equal to the number of designs) but with the order (layout) of the designs rearranged. The participant must aim to point to a different design on each page. Three trials are performed at each item length (i.e., 6, 9, or 12). The total number of errors over the whole task provides the score.

Semantic Language

Animal Fluency⁸

Requires individuals to produce the names of as many animals as possible in 60 seconds. Individuals often cluster similar stimuli together in different ways (e.g., farm animals, ocean-dwelling creatures) that reflect semantic integrity. Several measures of semantic language were measured. Total correct response is the total number of items correctly produced in the allotted time. Also, the association index is the degree of associated-ness between similar items (Carew et al.⁹ provides full details) and percentage of items that are included in any cluster.

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TABLE 1

Demographic Data and Depressive Scales

	Mean (SD) Range	Correlation with Age	Correlation with FSRP	Correlation with FSRP, Controlling for Age
Age (n = 57)	64.53 (7.85) 50–89		<i>r = 0.389, p = 0.003</i>	
Gender (male:female) (n = 57)	21:36			
Highest degree (n = 57)	15.54 (3.06) 10–24			
Stroke risk factor total score (n = 55)	9.49 (4.32) 1–18			
HDRS (n = 57)	1.19 (1.52) 0–6	r(55) = 0.191, p = 0.155	r(53) = 0.339, <i>p = 0.011</i>	r(52) = 0.333, <i>p = 0.015</i>
CESD (n = 56)	5.88 (5.32) 0–21	r(54) = -0.062, p = 0.651	r(52) = 0.262, p = 0.056	r(51) = 0.317, <i>p = 0.021</i>

Notes: Italics indicates significance at $p < 0.05$. CESD: Center for Epidemiologic Studies Depression; FSRP: Framingham Stroke Risk Profile; HDRS: Hamilton Depression Rating Scale; SD: standard deviation.

TABLE 2

Cognitive Variables

	Range for z-Scores (Mean [SD] = 0 [1])	Correlation with Age	Partial Correlation with FSRP, Controlling for Age	Partial Correlation with HDRS Controlling for Age	Partial Correlation with CESD Controlling for Age
L-M	-2.47 to 1.49	$r = -0.098, p = 0.468$	$r = -0.386, p = .004$	$r = -0.093, p = 0.502$	$r = -0.460, p < 0.001$
AIP	-2.32 to 1.76	$r = -0.199, p = 0.137$	$r = -0.168, p = 0.224$	$r = -0.006, p = 0.965$	$r = -0.371, p = 0.005$
EF	-1.76 to 1.47	$r = -0.251, p = 0.059$	$r = -0.423, p = 0.001$	$r = -0.031, p = 0.824$	$r = -0.497, p < 0.001$
SL	-1.91 to 1	$r = 0.007, p = 0.959$	$r = -0.219, p = 0.111$	$r = -0.155, p = 0.258$	$r = -0.057, p = 0.678$
df		55	53	55	54

Notes: Italics indicates significance at $p < 0.05$. AIP: Attention and Information Processing; CESD: Center for Epidemiologic Studies Depression; EF: Executive Function; FSRP: Framingham Stroke Risk Profile; HDRS: Hamilton Depression Rating Scale; L-M: Learning and Memory; SD: standard deviation; SL: Semantic Language.