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## Depressive Symptoms and Cognitive Decline in Older African Americans: Two scales and their factors

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### Abstract

**Objectives**—Depressive symptoms are common in older adults and researchers have explored the possibility of a link between depressive symptoms and cognitive decline, with mixed results. Most studies utilize total score of the Center for Epidemiological Studies Depression Scale (CES-D) with predominately non-Hispanic White participants. We sought to examine the relationship between the four factors of the CES-D and cognitive decline in older African Americans. Generalizability was determined using the Geriatric Depression Scale (GDS) and its factors.

**Methods**—Participants without dementia from the Minority Aging Research Study, ( $n=298$ , mean age= $74\pm 5.68$ ) underwent annual clinical evaluations (mean years= $5\pm 1.9$ ), including depression assessment and cognitive testing, from which global and specific measures were derived. Cognitive decline was examined with linear mixed models adjusted for demographic variables and indicators of vascular risk.

**Results**—Total CES-D score was not related to baseline cognition or change over time, while total GDS score was related to decline in semantic and working memory. In examining CES-D factors, lack of positive affect (e.g., anhedonia) was related to decline in global cognition, episodic memory and perceptual speed. Similarly for the GDS, anhedonia was associated with decline in semantic memory, and increased negative affect was associated with decline in global cognition, episodic, semantic, and working memory.

**Conclusions**—Results suggest that depressive symptoms, particularly anhedonia and negative affect, are related to cognitive decline in older African Americans.

### Keywords

Depressive Symptoms; Older Adults; Cognitive Decline; African American

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Conflicts of Interest:

There are no relationships, financial or otherwise, that could be considered conflicts of interests affecting this manuscript.

## INTRODUCTION

Depressive symptoms are common in older adults, with an estimated 8-20% experiencing significant depressive symptomatology<sup>1,2</sup>. Because many older adults with clinical depression also have cognitive impairment, research has explored the possibility of a link between the two, with mixed results. Some studies indicate that depressive symptoms are linked to faster cognitive decline<sup>3,4,5,6</sup>, even in high functioning older adults<sup>4</sup>. Other studies have found no link between depressive symptoms and cognitive decline<sup>7,8,9</sup>. In addition, the majority of studies have been conducted in predominantly non-Hispanic white samples, and of those that do include African Americans, few have explored the impact of race on depressive symptoms explicitly. Therefore, the nature of the link between depressive symptoms and cognitive decline in older African Americans remains unclear. Some studies suggest that African Americans may be at greater risk of cognitive impairment or cognitive decline<sup>10</sup>. We wanted to examine depressive symptoms and its relation to cognitive decline because depressive symptoms are modifiable and previous studies have reported a range of prevalence rates for depressive symptoms in this population<sup>11</sup>. The findings could have important implications for prevention of cognitive impairment in one of the fastest growing minority populations in the U.S.

Another factor that has been infrequently examined is whether or how specific aspects of depressive symptoms relate to decline. Investigators have most often used the total score of the Center for Epidemiological Studies Depression Scale (CES-D;<sup>12</sup>). Though the CES-D was created with a four-factor structure of negative affect, positive affect, somatic complaints, and interpersonal problems, most researchers rely solely on the total score of the scale. The four factor structure of the CES-D has been validated several times, including across the different versions of the scale (8, 10, and 20-item versions) and in different populations<sup>13</sup>, however no studies have examined the relationship between specific clusters of depressive symptoms and cognitive decline.

The purpose of the current manuscript was to examine the relationship between depressive symptoms and cognitive decline in older African Americans using the 10-item CES-D scale. We hypothesized that depressive symptoms are related to cognitive decline. We also used another common depression inventory, the Geriatric Depression Scale (GDS;<sup>14</sup>), to determine the generalizability of the findings. Since the factors of depressive symptoms have not been previously examined with cognitive decline, in exploratory analyses, we examined their relation with cognitive decline to determine if certain aspects of depressive symptomatology influence decline in older African Americans.

## METHODS

### Participants

Participants were enrolled in the Minority Aging Research Study (MARS), a longitudinal epidemiologic study of risk factors for cognitive decline and Alzheimer's disease in older African Americans. The study was approved by the Rush University Medical Center Institutional Review Board. Participants were older community-dwelling persons, without dementia at baseline, recruited from churches, community-based organizations and senior-

subsidized housing facilities in the Chicago metropolitan area. Participants signed an informed consent form in which they agreed to annual clinical evaluations that included an assessment of risk factors (e.g., depressive symptoms) and cognitive testing, as previously described<sup>15</sup> In addition, clinical classification of dementia was based on a uniform, structured clinical evaluation that included a medical history, neurological examination, and assessment of cognitive function (see below). After review of all clinical data, a clinician with experience in evaluating older persons, classified the participants with respect to dementia based on criteria of the joint working group of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association<sup>16</sup>.

At the time of analysis, 402 older adults were enrolled in MARS with complete baseline information. Based on the clinical evaluation at baseline, we excluded 8 persons who met criteria for dementia, and 96 persons who did not have at least two clinical evaluations to measure change in cognition. Two hundred ninety eight participants, with an average length of follow-up of 5.0 years (SD=1.89) were included in the analysis. Participants who were included were slightly younger (73.9 (SD=5.68) v.75.7 (SD=6.79);  $t(393)=2.36$ ,  $p<0.02$ ) and a slightly higher MMSE score (28.3 (SD=1.80) v.27.4 (SD=2.59);  $F(1,391)=12.91$ ,  $p<0.001$ ) than those who were excluded, but did not differ on educational attainment, depressive symptoms or health factors.

### Neuropsychological Testing

The participants' annual neuropsychological testing, reviewed by a board-certified neuropsychologist blinded to previously collected data, consisted of twenty detailed tests selected to assess a broad range of cognitive abilities commonly affected in older adults, including the Mini-Mental State Examination (MMSE) which was used for descriptive purposes only. From the remaining nineteen tests, summary measures of five cognitive domains and a measure of global cognition were derived, as previously described<sup>17</sup>. In brief, there were seven measures of episodic memory (Word List Memory, Recall and Recognition, immediate and delayed recall of the East Boston Story, and Story A from Logical Memory), three measures of semantic memory (Boston Naming, Verbal Fluency, and 15 items from the Wide Range Achievement Test), three measures of working memory (Digit Span forward and backward, and digit ordering), four measures of perceptual speed (Symbol Digit Modalities, Number Comparison, and two indices from a modified version of the Stroop Neuropsychological Screening Test: the number of color names correctly read aloud in 30 seconds minus the number of errors, and the number of colors correctly named in 30 seconds minus the number of errors), and two measures of visuospatial ability (Line Orientation and Progressive Matrices). As described previously<sup>18</sup>, composite scores for the five cognitive domains were created by converting raw scores of the measures comprising each cognitive domain into z scores. The z scores were then averaged to obtain the cognitive domain scores. A global cognitive score was derived by averaging the Z scores of all 19 tests.

## Depression Measures

Number of depressive symptoms was assessed at baseline with the 10-item version of the Center for Epidemiologic Studies Depression Scale (CES-D;<sup>19</sup>), and the 15-item version of the Geriatric Depression Scale (GDS;<sup>20</sup>). The abbreviated version of the CES-D is derived from the original 20-item version<sup>21</sup> and has shown acceptable reliability. Participants were asked whether they experienced each of 10 symptoms in the past week. Item responses are coded in a yes/no format yielding a summary measure with a range of 0-10. A factor structure has been identified for the CES-D<sup>12</sup>, and includes negative affect (3 items), positive affect (2 items), interpersonal problems (2 items), and somatic complaints (3 items). We also used the short form of the GDS, which was derived from the original 30-item version<sup>22</sup>. The 15-item GDS, developed especially for use with older populations, has shown acceptable reliability and validity compared to the original version<sup>20</sup>. Participants were asked about their feelings in the past week. Item responses are coded in a yes/no format yielding a summary measure with a range of 0-15. A factor structure has also been identified for the GDS<sup>2</sup>, and includes negative affect (9 items), positive affect (4 items), and both negative and positive affect (2 items).

## Covariates

Covariates included sex, age, education (years of formal schooling), and vascular risk factors. As previously described<sup>23</sup>, vascular risk factors (i.e., the sum of hypertension, diabetes mellitus, and smoking, resulting in a score from 0 to 3 for each individual) were computed on the basis of self-report questions, clinical evaluation, and inspection of medications.

## Data Analysis

Global cognition and five cognitive domains were modeled as outcome variables using separate linear mixed models with random intercept and random slope for time (in years). A term for CES-D (predictor) and a term for the interaction between time and CES-D were entered into the model. Subsequent exploratory models included terms for CES-D factors (predictors such as positive affect, negative affect, somatic complaints, and interpersonal problems). All models included terms for age (years), sex, education (years), vascular risk, time, and interaction terms between time and each covariate. The analysis was repeated with GDS and GDS factors. Models were estimated using restricted maximum likelihood and unstructured covariance structure. Overall, these models use individual trajectories of cognitive scores over time to simultaneously estimate effects of variables on initial level of cognition and change in cognition while also taking into account the correlation among scores. Estimates represent the effects of depressive symptoms on the baseline cognitive score. Estimates for time indicate the mean change in cognition per year from the baseline interview, and estimates of the interaction of time and depressive symptoms indicate the effect of depressive symptoms on change in cognitive function over time.

## RESULTS

Among the 298 participants, 70% were female. They had a mean age of 73.9 (SD=5.7), a mean education of 15.0 (SD=3.6) years, and a mean MMSE score of 28.3 (SD=1.8).

Participants had a mean vascular risk factor score of 1.5 (SD=0.90, range=0-3) (See Table 1). The distribution of CES-D scores at baseline was skewed, with 52% of the cohort reporting no symptoms and 48% reporting one or more symptoms. Median CES-D score at baseline was 0.00 (Q3=2, range=0-8). The distribution of GDS scores at baseline was also skewed, with 47% of the cohort reporting no symptoms and 53% reporting one or more symptoms. Median GDS score at baseline was 1.00 (Q3=2, range=0-10). Participants overwhelmingly endorsed more positive items on both the CES-D and GDS (Table 2).

Participants were followed for an average of 5.0 years (SD=1.90). In a linear mixed model, we examined the relation of depressive symptoms to mean cognitive decline, while adjusting for the effects of age, sex, education, and vascular risk factors. In the model with CES-D, there was an average decline of 0.051 unit per year on the global cognitive measure (as indicated by the term for time, but CES-D was not related to cognitive performance at baseline or to change over time (Table 3). To see whether depressive symptoms were associated with decline in some cognitive domains but not others, we repeated the analysis replacing global cognition with each of the five cognitive abilities. Similar to the model for global cognition, there was no relationship between CES-D and decline in any of the cognitive domains.

In subsequent linear mixed models we replaced the global CES-D measure with each of the four specific symptom factors, while adjusting for the effects of age, sex, education, and vascular risk factors. In these models, with global cognition as the outcome, no CES-D factor was related to baseline cognitive performance. Reduced positive affect (also referred to as anhedonia; see<sup>25,26</sup>), however, was associated with a faster rate of decline in global cognition (Table 4). Next, we examined whether the CES-D factors were related to decline in the five cognitive abilities. Anhedonia was associated with faster decline in episodic memory, perceptual speed and a trend for semantic memory (Table 4). Neither negative affect, somatic complaints, nor interpersonal problems were related to cognitive performance at baseline or change in cognitive function.

Next, we repeated the same series of analyses using GDS in place of the CES-D. Analyses with the global score for the GDS showed a similar pattern to the CES-D for global cognition as the outcome. There were no significant associations of the global GDS with baseline global cognition or cognitive decline (Table 5). In contrast, when we examined the effect of global GDS score on the separate cognitive abilities, higher GDS scores were associated with faster declines in semantic and working memory (Table 5).

Finally, in models with the GDS factor scores, we again found a similar pattern to the results for the CES-D. When global cognition was used as the outcome, none of the GDS factors were related to cognitive performance at baseline, but increased negative affect was associated with faster decline in global cognition (Table 6). In models with the separate cognitive abilities as the outcome measure, anhedonia was associated with faster decline in semantic memory, and increased negative affect was associated with faster decline in episodic, semantic, and working memory (Table 6). As a sensitivity analysis, we excluded persons with mild cognitive impairment at baseline and repeated all models. The results

were similar for both scales with one exception; the relation between the global measure of GDS and working memory was no longer significant.

## DISCUSSION

In this study of 298 community-dwelling African Americans without dementia at baseline, we found that depressive symptoms are related to cognitive decline, but the pattern was mixed depending on which depression scale was used and whether the global score or the individual factors were used. Using the full scale, the CES-D was not associated with decline in global cognition or with any of the cognitive domains. The full scale GDS, although not related to decline in global cognition, was related to a faster rate of decline in semantic and working memory. Upon examination of the individual factors for each depression scale, we found that for the CES-D, reduced positive affect/anhedonia was related to faster declines in global cognition, episodic memory and perceptual speed. Similarly, for the GDS, anhedonia and increased negative affect were both associated with faster declines in semantic, episodic, and working memory. The results suggest that depressive symptoms are related to a faster rate of cognitive decline in older African Americans and the effect appears to be primarily driven by negative affect and anhedonia.

Previous research is mixed with regard to whether depressive symptoms are linked to cognitive decline, with some reporting that depressive symptoms predict decline in executive function, attention, memory, and global cognitive status<sup>3,5,6</sup>, but others reporting either conflicting results or negative associations<sup>4,7,8,9</sup>. For example, one study reported that for each depressive symptom on the CES-D, decline in global cognition increased by approximately 24%,<sup>18</sup> and another indicated that for each depressive symptom reported on the CES-D, the rate of global cognitive decline increased by about 5% over the annual decline measured in those without depressive symptoms<sup>6</sup>. Some studies have demonstrated no relation between depressive symptoms and cognitive decline<sup>7,9</sup>, while others have shown that depressive symptoms influence cognition at baseline only<sup>4,9</sup>, and one indicated that only certain aspects of cognition are affected<sup>7</sup>. Perhaps the literature is mixed because studies have focused only on global depressive symptoms and their relationship to cognition, an idea that is consistent with the current results. In addition, the majority of these studies have been conducted in predominantly non-Hispanic whites, so whether depressive symptoms are related to cognitive decline in African Americans is not firmly established. While there are a few studies that include large numbers of African Americans<sup>3,27,28</sup>, results have been inconclusive here as well. Though some studies have reported a positive relationship<sup>3,6</sup>, in some cases, it has been dependent on other factors, like medical conditions including stroke<sup>27,28</sup>. Importantly, even in studies with African Americans, few have focused explicitly on race. The current study fills important gaps in the literature by focusing specifically on an older African American sample, by examining a comprehensive battery of cognitive function, including separate cognitive abilities, and by examining the established factors of the depressive symptoms scales to determine if any particular factor may drive the relationship. Our results demonstrate that depressive symptoms are related to a faster rate of cognitive decline, which is consistent with most literature in predominately non-Hispanic white populations.

The CES-D and GDS are the two most commonly used screening tools for depressive symptoms in older adults but there are important differences between the two. First, the CES-D screens for somatic symptoms (e.g., “My Sleep was restless.”) while the GDS does not. Somatic questions were not included in the GDS so as not to confuse the physical disturbances common in older age, such as changes in sleep or appetite, with similar somatic symptoms of depression<sup>14</sup>. It is possible that the inclusion of somatic complaints on the CES-D may obscure or weaken the effect of depressive symptoms on cognitive decline in older adults, and this may, at least in part, account for why we did not see an effect with the global score and could explain the inconsistent results reported in the literature. Another difference between the two scales is that the CES-D includes interpersonal items (e.g, people were unfriendly and people disliked me) that may not be particularly reflective of depression in African Americans. In fact, some have argued that this factor may assess perceptions of discrimination rather than depression<sup>29,30</sup>. Unfortunately, we did not have sufficient power to assess the relationship between interpersonal items and cognitive decline due to a relatively low proportion of the sample endorsing this factor<sup>31</sup>.

Our results showed that only anhedonia and negative affect items were related to cognitive decline. The reasons for this are not clear. However, previous studies have shown that negative mood in this population is related to adverse health outcomes<sup>32, 33</sup>. This is interesting because studies have also demonstrated that negative mood is lower in African Americans compared with other groups<sup>32, 34</sup>. In an examination of day-to-day emotions and emotional responses to conflict across races, one study found that African Americans were less likely to use emotional tactics when they cope with conflict<sup>34</sup>. They propose that the predilection to emotionally self control and the reluctance to emotionally self disclose within African Americans may have created a blunted response or desensitization to negative emotion and thereby the reporting of it<sup>34</sup>. This desensitization theory is consistent with our finding of an abundance of affirmative responses on positive items on both the CES-D and the GDS. In addition, given that the symptoms predicting cognitive decline in this sample are affect-related, it must be noted that these symptoms could possibly be more reflective of an apathy syndrome, which previous research suggests is associated with cognitive decline, and often the first indicator of dementia<sup>35,36</sup>.

Though the basis of the association of depressive symptoms and cognitive decline is not completely understood, several theories have been advanced. For example, it has been suggested that depressive symptoms are a reaction to cognitive decline. However, studies like this one, which utilize a longitudinal design, and only include individuals without frank cognitive impairment or dementia at baseline, make it unlikely that reactions to existing cognitive decline are the basis of the depressive symptoms in the current sample. It has also been suggested that depressive symptoms are an early indicator/prodrome of Alzheimer's disease, but most studies have not found a link between depressive symptoms and plaques and tangles, the hallmark of AD<sup>37</sup>. Another possibility is vascular disease, which has been shown to be related to both depression and cognitive decline<sup>38, 39</sup>, and African Americans tend to have a high prevalence of vascular risk factors including hypertension and diabetes<sup>40, 41</sup>. However, we controlled for vascular risk factors in our analyses, and still demonstrated a relationship between depressive symptoms and cognitive decline. It is most widely believed that depression is related to volumetric brain changes. This belief is based

on stress research and the dysregulation of the hypothalamic – pituitary - adrenal (HPA) axis in both animals and humans, which shows that prolonged stress can lead to atrophy of the hippocampus. Animal models show that stressful experiences are associated with structural changes in the hippocampus, and within humans hippocampal atrophy has been reported in individuals who suffer from major depressive disorder. Therefore, the theory is that depressive symptoms are associated with dysfunction of neural systems that regulate the HPA axis<sup>42</sup>. Though brain atrophy was not examined, the current study supports the idea behind this theory in that depressive symptoms appeared to be primarily related to memory, which is governed by the hippocampus. However, neuroimaging studies in African Americans are needed to further explore this possibility.

This study has some limitations. First, the results are based on a volunteer cohort that may not be representative of the US population in education or lifestyle. For instance, our results may be affected by the relatively high educational status of our sample, which might reflect a healthier population with low depressive symptoms. The homogeneity of the sample and the low prevalence of depressive symptoms may have resulted in a more modest association with cognition than would normally be revealed. The generalizability of the findings will need to be demonstrated with representative samples. Second, individuals who were excluded from this study because they had not yet reached their follow-up evaluation had a slightly lower MMSE score than the study participants, which could have potentially introduced a slight bias to the results. In addition, although we adjusted analyses for important covariates such as education and health, this is a relatively conservative approach to address type 1 error. Third, the study focused on depressive symptoms and not the clinical syndrome of depression. Research indicates that the cognitive consequences associated with depression increase with severity, therefore utilizing the clinical syndrome of depression rather than depressive symptoms could lead to findings that are more robust. Finally, we assessed depressive symptoms at a single time point. The influence of any past depression on the rate of cognitive decline is unclear.

The study also had several strengths. First, this study utilized a comprehensive battery of cognitive tests that sampled 5 domains which have been shown to be valid and reliable in this sample<sup>19</sup>. Second, longitudinal data was available on close to 300 people, providing adequate statistical power, and the use of composite measures of cognition enhanced our ability to identify subtle cognitive changes over time. Third, we used two validated scales in order to measure depressive symptoms and found the results to be similar across the scales. Fourth, we examined the impact of specific dimensions of depression on cognition, allowing us to better target the domains most related to cognitive decline in older African Americans. Finally, all analyses controlled for important clinical and demographic variables that might influence depressive symptoms, the risk of cognitive decline, or their association.

In summary, our findings suggest that affective symptoms, in particular anhedonia and negative affect, are related to cognitive decline in older African Americans. Further, it is possible that the use of the total score of the CES-D in this population may obscure the link between depressive symptoms and cognitive decline, possibly due to the inclusion of somatic complaints and interpersonal problems. Our results suggest that scales that focus on negative affect may be more useful in this population.



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Table 1

## Clinical Characteristics

	Mean	SD
Age, y	73.93	5.68
Education, y	15.01	3.56
Women, N %	210	70
MMSE score	28.29	1.80
CES-D, median (Q3)	0.00	2.00
GDS, median (Q3)	1.00	2.00
Vascular Risk Score	1.52	0.90

Notes: Participant N= 298. SD=Standard Deviation; MMSE=Mini-Mental State Examination; CES-D=Center for Epidemiological Studies Depression Scale; GDS=Geriatric Depression Scale.

**Table 2**

Factor Structure of Depressive Scales

Items	Factor*	%Endorsing Yes
<b>CES-D</b>		
4. I was happy	Positive Items	91.11
7. I enjoyed life	Positive Items	97.78
3. I felt depressed	Negative Items	11.85
5. I felt lonely	Negative Items	12.10
8. I felt sad	Negative Items	12.10
1. I felt that everything I did was an effort	Somatic Complaints	19.01
2. My sleep was restless	Somatic Complaints	22.22
10. I could not get going	Somatic Complaints	11.85
6. People were unfriendly	Interpersonal Items	2.22
9. I felt that people disliked me	Interpersonal Items	1.98
<b>GDS</b>		
1. Are you basically satisfied with your life?	Positive Affect	95.85
5. Are you in good spirits most of the time?	Positive Affect	96.79
7. Do you feel happy most of the time?	Positive Affect	92.59
11. Do you think it is wonderful to be alive?	Positive Affect	97.28
2. Have you dropped many of your activities and interests?	Negative Affect	15.10
4. Do you often get bored?	Negative Affect	10.37
8. Do you often feel helpless?	Negative Affect	4.44
9. Do you prefer to stay at home, rather than going out and doing new things?	Negative Affect	19.01
10. Do you feel that you have more problems with memory than most?	Negative Affect	11.11
12. Do you feel pretty worthless the way you are now?	Negative Affect	2.22
13. Do you feel full of energy?	Negative Affect	72.10
14. Do you feel that your situation is hopeless?	Negative Affect	1.98
15. Do you think that most people are better off than you are?	Negative Affect	5.19
3. Do you feel that your life is empty?	Positive & Negative Affect	5.19
6. Are you afraid that something bad is going to happen to you?	Positive & Negative Affect	3.46

Note.

CES-D= Center for Epidemiological Studies Depression Scale, GDS=Geriatric Depression Scale

\* Factor is based on published factor analyses<sup>2, 13</sup>

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**Table 3**

Relation of CES-D to baseline cognition and cognitive decline

Model Term	Model 1			Model 2			Model 3			Model 4			Model 5			Model 6		
	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df
Time	<b>-0.051 (.012)</b>	<b>.001</b>	<b>1254</b>	<b>-0.058 (.007)</b>	<b>.001</b>	<b>1280</b>	<b>-0.071 (.017)</b>	<b>.001</b>	<b>1280</b>	<b>-0.029 (.015)</b>	<b>.046</b>	<b>1277</b>	<b>-0.047 (.012)</b>	<b>.001</b>	<b>1255</b>	<b>-0.029 (.015)</b>	<b>.054</b>	<b>1250</b>
CES-D	-.008 (.019)	.683	291	-.011 (.024)	.667	291	-.022 (.025)	.365	291	.034 (.031)	.270	291	-.012 (.027)	.662	290	-.027 (.027)	.317	290
CES-D $\times$ Time	-.004 (.004)	.315	1254	-.001 (.006)	.865	1280	-.009 (.006)	.118	1280	-.010 (.005)	.072	1277	-.005 (.004)	.248	1255	-.005 (.006)	.415	1250

Note. Six separate linear mixed models, each controlling for age, sex, education, and vascular risk. Participant N=298; Observation N=1585. p values are based on t values of the coefficients. SE: standard error; CES-D=Center for Epidemiologic Studies Depression scale

**Table 4**

Relation of CES-D factors to baseline cognition and cognitive decline

Model Term	Model 1			Model 2			Model 3			Model 4			Model 5			Model 6		
	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df
Time	<b>-0.52 (.012)</b>	<b>.001</b>	<b>1254</b>	<b>-0.61 (.019)</b>	<b>.001</b>	<b>1280</b>	<b>-0.66 (.016)</b>	<b>.001</b>	<b>1280</b>	<b>-0.22 (.015)</b>	.145	1277	<b>-0.50 (.012)</b>	<b>.001</b>	<b>1255</b>	<b>-0.28 (.016)</b>	.072	1250
Positive Affect	-.055 (.084)	.510	291	-.045 (.113)	.694	291	-.058 (.111)	.602	291	-.076 (.135)	.574	291	-.025 (.119)	.831	290	.036(.121)	.767	290
Positive Affect $\times$ time	<b>-0.38 (.019)</b>	<b>.048</b>	<b>1254</b>	<b>-0.58 (.029)</b>	<b>.045</b>	<b>1280</b>	<b>-0.49 (.025)</b>	.053	<b>1280</b>	<b>-0.20 (.024)</b>	.395	1277	<b>-0.50 (.019)</b>	<b>.009</b>	<b>1255</b>	<b>-0.16 (.025)</b>	.537	1250
Negative Affect	-.006 (.034)	.859	291	-.030 (.045)	.509	291	.011(.045)	.805	291	.048 (.055)	.383	291	.019 (.048)	.693	290	-.050 (.049)	.307	290
Negative Affect $\times$ time	-.004 (.008)	.600	1254	-.001 (.012)	.923	1280	-.012 (.011)	.280	1280	-.007 (.010)	.454	1277	-.007 (.008)	.390	1255	-.004 (.011)	.718	1250
Somatic Complaints	-.027 (.037)	.469	291	-.008 (.050)	.876	291	-.067 (.049)	.170	291	.045 (.060)	.454	291	-.068 (.053)	.194	290	-.036 (.054)	.501	290
Somatic Complaints $\times$ time	-.003 (.009)	.697	1254	.007 (.013)	.610	1280	-.018 (.012)	.124	1280	-.016 (.011)	.137	1277	.000 (.009)	.986	1255	-.010 (.011)	.385	1250
Interpersonal Problems	.168 (.129)	.194	291	.223 (.177)	.207	291	.034 (.173)	.843	<b>1280</b>	.456 (.211)	.300	291	.097 (.184)	.598	290	-.048 (.191)	.802	290
Interpersonal Problems $\times$ time	-.008 (.029)	.771	1254	-.005 (.043)	.906	1280	.006 (.038)	.873	291	-.029 (.036)	.407	1277	-.010 (.029)	.724	1255	-.012 (.037)	.746	1250

Note. Six separate linear mixed models, each controlling for age, sex, education, and vascular risk. Participant N=298; Observation N=1581. p values are based on t values of the coefficients. SE: standard error; CES-D=Center for Epidemiologic Studies Depression scale



Table 5

Relation of GDS to baseline cognition and cognitive decline

Model Term	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	$\beta$ (SE)	p	$\beta$ (SE)	p	$\beta$ (SE)	p	$\beta$ (SE)	p	$\beta$ (SE)	p	$\beta$ (SE)	p
Time	<b>-.048 (.012)</b>	<b>.001</b>	<b>-.050 (.017)</b>	<b>.004</b>	<b>-.067 (.015)</b>	<b>.001</b>	<b>-.027 (.014)</b>	<b>.062</b>	<b>-.053 (.012)</b>	<b>.001</b>	<b>-.033 (.015)</b>	<b>.029</b>
GDS	.006 (.016)	.716	.013 (.021)	.541	.000 (.021)	.991	.031 (.026)	.246	-.022 (.023)	.332	.015 (.024)	.533
GDS $\times$ Time	-.007 (.004)	.072	-.011 (.006)	.062	<b>-.013 (.005)</b>	<b>.010</b>	<b>-.012 (.005)</b>	<b>.012</b>	.001 (.004)	.808	-.001 (.008)	.891

Note. Six separate linear mixed models, each controlling for age, sex, education, and vascular risk. Participant N=298; observation N=1585. p values are based on t values of the coefficients. SE: standard error; GDS = Geriatric Depression Scale.

**Table 6**

Relation of GDS factors to baseline cognition and cognitive decline

Model Term	Model 1			Model 2			Model 3			Model 4			Model 5			Model 6		
	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df	$\beta$ (SE)	p	df
Time	<b>-.052(.012)</b>	<b>.001</b>	1254	<b>-.055(.018)</b>	<b>.002</b>	1280	<b>-.071(.015)</b>	<b>.001</b>	1280	<b>-.028(.014)</b>	<b>.050</b>	1277	<b>-.051(.012)</b>	<b>.001</b>	1255	<b>-.033(.015)</b>	<b>.025</b>	1250
Positive Affect	-.065(.049)	.183	291	-.034(.066)	.606	291	-.067(.065)	.303	291	-.018(.079)	.823	291	-.114(.069)	.097	290	-.091(.070)	.197	290
Positive Affect × time	-.015(.011)	.188	1254	-.027(.017)	.109	1280	<b>-.047(.015)</b>	<b>.002</b>	1280	-.007(.014)	.610	1277	-.007(.012)	.578	1255	.007(.015)	.641	1250
Negative Affect	-.018(.022)	.425	291	.029(.030)	.336	291	-.004(.029)	.891	291	.046(.036)	.197	291	-.018(.031)	.569	290	.050(.032)	.108	290
Negative Affect × time	<b>-.011(.005)</b>	<b>.044</b>	1254	<b>-.017(.008)</b>	<b>.031</b>	1280	<b>-.015(.007)</b>	<b>.031</b>	1280	<b>-.017(.006)</b>	<b>.008</b>	1277	-.003(.005)	.598	1255	-.004(.007)	.586	1250
Positive and Negative Affect	.015(.086)	.185	291	.119(.116)	.304	291	.183(.113)	.108	291	.162(.138)	.242	291	.036(.121)	.766	290	.146(.124)	.241	290
Positive and Negative Affect × time	.001(.021)	.954	1254	-.008(.032)	.792	1280	-.022(.028)	.438	1280	-.023(.026)	.375	1277	.042(.022)	.056	1255	.006(.029)	.842	1250

Note. Six separate linear mixed models, each controlling for age, sex, education, and vascular risk. Participant N=298; observation N=1581. p values are based on t values of the coefficients. SE: standard error; GDS = Geriatric Depression Scale.