Depressive Symptoms and Cognitive Change in Older Mexican Americans

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ABSTRACT

To examine the association between presence of clinically relevant depressive symptoms (Center for Epidemiologic Studies Depression Scale [CES-D] score $\geq 16$) and subsequent cognitive function (Mini-Mental State Examination [MMSE]) over a 7-year period in older Mexican Americans, a prospective cohort study was performed. Five southwestern states contributed data to the Hispanic Established Populations for Epidemiologic Studies of the Elderly. Participants included 2812 noninstitutionalized Mexican Americans aged 65 and older followed from 1993-1994 until 2000-2001. Cognitive change was assessed using the MMSE at baseline and at 2, 5, and 7 years of follow-up. Independent variables were sociodemographics, CES-D $\geq 16$, medical conditions (hypertension, diabetes, coronary artery disease, and stroke), and activities of daily living (ADL) status. A general linear mixed model was used to estimate cognitive change. There was a cross-sectional association between CES-D $\geq 16$ and lower MMSE score (estimate = $-0.48$; standard error [SE] = 0.15; $P < .01$), independent of age, gender, education, marital status, time of interview, ADL limitations, vision impairment, and medical conditions. In the fully adjusted longitudinal model, subjects with clinically relevant depressive symptoms had a greater decline in MMSE score over 7 years than those without clinically relevant depressive symptoms (estimate = $-0.17$; SE = 0.05; $P < .001$), adjusting for sociodemographics, ADL and medical conditions. Each point increase in the CES-D score was associated with a decline of 0.010 point in MMSE score per year (SE = 0.002; $P < .0001$), adjusting for relevant confounders. Presence of clinically relevant depressive symptoms was associated with subsequent decline in cognitive function over 7 years in older Mexican Americans, independent of demographic and health factors. (J Geriatr Psychiatry Neurol 2007;20:145-152)

Keywords: depression; cognition; elderly; Mexican Americans

Decline in cognitive ability contributes to loss of independent living and premature deaths in older adults.1-3 An important step toward delaying or stopping cognitive decline is early recognition (and treatment) of conditions associated with high risk of cognitive impairment and subsequent dementia. Among those potentially modifiable conditions are vision impairment, anticholinergic medication use, stroke, diabetes, and depression.4-7

The data on the relationship between depression and cognitive impairment are mixed.6-12 Some studies show association of depressive symptoms with subsequent cognitive impairment. Wilson et al,5 using data from initially nondemented mostly white men and women aged 65 and older, reported a 24% increase in annual cognitive decline over a 7-year period for each baseline depressive symptom on the Center for Epidemiologic Studies Depression Scale (CES-D), adjusting for demographic and
health factors. Other studies did not find an association between depression and cognition. For example, a prospective cohort study of 1265 community-dwelling nondemented older adults reported no association between depressive symptoms and subsequent cognitive decline, despite a cross-sectional association between baseline high depressive symptoms and low cognitive scores.

The reasons for these mixed findings are unclear. One possible reason is the use of different measures to assess depressive symptoms and cognitive function in different studies. It is also possible that the impact of psychological stressors on cognition varies by population characteristics such as level of education, social network, income, and ethnic composition. A recent study showed that proneness to psychological distress is associated with increased odds of developing Alzheimer’s disease in older whites but not in older African Americans. The ethnic differences in cognitive effect of psychological stressors point to possible cultural, educational, and other experiential factors that may influence the cognitive impact of depressive symptoms and other psychological stressors. One way to test this explanation is by examining the association between depression and cognition in a population with low educational attainment and high rates of cognitive impairment. High rates of cognitive impairment in populations with low literacy should be interpreted with caution because of the cultural, linguistic, and educational bias of the most widely used cognitive measures (eg, Mini-Mental State Examination [MMSE]) in aging research.

Past research has demonstrated a high prevalence of depressive symptoms in older Mexican American adults, one of the fastest growing segments of the US population. Because of the reported lower educational attainment in older Mexican Americans compared to older whites, it is not clear if high depressive symptoms will hasten cognitive decline over time in this population. In an effort to examine predictors of cognitive decline in this population, Nguyen et al, in a longitudinal analysis of older Mexican Americans, found an association between stroke, diabetes, and vision impairment, and subsequent decline in the MMSE score over a 5-year period, independent of relevant demographic and health factors. However, information on the impact of depressive symptoms on subsequent cognitive function was not described.

The current study extends the earlier work of Nguyen et al by investigating the association between presence of clinically relevant depressive symptoms (CES-D ≥ 16) and subsequent cognitive function (MMSE) over a 7-year period in a community-based sample of older Mexican Americans. We hypothesize that presence of clinically relevant depressive symptoms will be associated with lower cognitive scores and steeper decline in cognition over time, independent of time-dependent changes in potentially confounding demographic and health factors.

**METHODS**

**Sample**

Data are from the Hispanic Established Population for the Epidemiological Study of the Elderly (H-EPESE). The H-EPESE is a population-based cohort study of 3050 Mexican Americans aged 65 and older at baseline. The sample was designed to be generalizable to approximately 85% of older Mexican Americans living in the southwestern United States: Texas, California, Colorado, Arizona, and New Mexico. A full description of the rationale, methods, and subject characteristics has been previously presented.

Data on cognition, depressive symptoms, and other covariates were collected from H-EPESE participants over a 7-year period starting in 1993/1994 and followed by subsequent interviews in 1995/96, 1998/99, and 2000/2001. Of the 3050 subjects, 2873 were interviewed in person and 177 (5.8%) by proxy. The interviews were conducted in Spanish or English, depending on the respondent’s preference. Subjects (n = 238) with no baseline MMSE or CES-D measurements were excluded. Thus, the present study used the baseline data (1993-94, n = 2812) and the data obtained from the 2-year follow-up (1995-96, n = 2269), the 5-year follow-up (1998-99, n = 1874), and the 7-year follow-up assessment (2000-2001, n = 1598). Over the 7-year follow-up, 414 subjects refused or were lost to follow-up and 800 were confirmed dead through the National Death Index file and from reports from subjects’ relatives.

**Measures**

**Outcome.** The primary outcome was change in MMSE cognitive function over time, independent of time-dependent changes in potentially confounding demographic and health factors.

**Main Independent Variable.** The CES-D scale is a widely used survey measure of depressive symptoms in community-based aging studies. It consists of 20 items. Each item is a question to assess subjects’ experience of
certain positive or negative feelings or symptoms in the past week. Responses are scored on a 4-point scale (0 to 3). Scores for the positive items are reversed and the 20 items summed. The CES-D scores range from 0 to 60. Higher scores indicate increased depressive symptoms. In the analysis, CES-D score was used as both a continuous and a dichotomized variable: absence of clinically relevant depressive symptoms (a score of < 16) versus presence of clinically relevant depressive symptoms (a score \( \geq 16 \)).\textsuperscript{26-28} CES-D has been shown in different population studies to be a reliable and valid instrument for identification of older adults with clinically relevant levels of depressive symptoms.\textsuperscript{6,8-11,13,16,17,26-28} It has a very good internal reliability with a Cronbach’s \( \alpha \) between .80 and .90, and has a high correlation (0.96) with other depression scales.\textsuperscript{26-28} Despite being one of the most frequently used depression screening measures in aging research, the CES-D alone cannot be used to diagnose clinical depression.

**Covariates.** Baseline sociodemographic variables included age, gender, years of education, and marital status. The presence of medical conditions was assessed by asking if respondents had ever been told by a doctor that they had diabetes, stroke, heart attack, or hypertension. Corrected bilateral near vision acuity was measured by having subjects hold cards at least 7 inches from their eyes and asking them to read the numbers, as described by Salive et al.\textsuperscript{29} Each card had 7-digit “telephone numbers” of three different type sizes: 7, 10, and 23 points.\textsuperscript{29} Participants who could only read the 10-point, the 23-point, or unable to read the 23-point were considered to have near vision impairment (code = 0), and participants who could read the 7-point were considered to have adequate near vision (code = 1).

Disability was assessed by 7 items from a modified version of the Katz Activities of Daily Living (ADL) scale. ADLs included walking across a small room, bathing, grooming, dressing, eating, transferring from a bed to a chair, and using the toilet.\textsuperscript{30} Respondents were asked to indicate if they could perform these activities without help, if they needed help, or if they were unable to do them. The validity and reliability of self-reported ADL items has been established in previous studies involving community-living older adults.\textsuperscript{30,31} ADL was used as a continuous variable.

**Statistical Analyses**

We examined sociodemographic and health characteristics at baseline for our sample (\( n = 2812 \)) stratified by CES-D scores (< 16 vs \( \geq 16 \)) using descriptive and univariate statistics for continuous variables and contingency tables (\( \chi^2 \)) for categorical variables.

To test whether CES-D was related to decline of MMSE score over 7 years of follow-up, we fitted a general linear mixed model using the MIXED procedure in SAS, while adjusting for age, gender, education, marital status, ADL limitations, medical conditions (diabetes, stroke, heart attack, and arthritis), and near vision impairment. All the variables were analyzed as time-dependent covariate (potential to change over time) except variables of education and gender. We chose mixed models for analysis of the H-EPESE data for several reasons. First, the models best accounted for missing or incomplete observations, thus enabling us to use all available information. Second, the mixed model approach allowed us to model time-dependent change in our variables (MMSE and CES-D scores, among others). Third, the approach allowed modeling of time-dependent change in the magnitude of association between these variables. Finally, using mixed models to analyze the 7-year repeated measures in the H-EPESE study allowed us flexibility in modeling the effects of time on change in MMSE score.\textsuperscript{32} Three mixed models were constructed to test the relationship between depressive symptoms and MMSE change over 7 years. Model 1 included age, gender, education, marital status, CES-D \( \geq 16 \), and time. In model 2, an interaction term—CES-D * time—was added to assess the association of depressive symptoms (presence of clinically relevant depressive symptoms, CES-D \( \geq 16 \) vs absence of clinically relevant depressive symptoms, CES-D < 16) and slope of MMSE score over time. Model 3 included additional variables of ADL limitation, selected medical conditions, and near vision impairment.

All analyses were performed using the SAS System for Windows, Version 8 (SAS Institute, Cary, NC).

**RESULTS**

Table 1 presents characteristics of our sample at baseline as a function of CES-D < 16 versus \( \geq 16 \). At baseline, 24% (\( n = 674 \)) of the subjects had clinically relevant depressive symptoms (CES-D \( \geq 16 \)). Mean MMSE was significantly lower in subjects with CES-D score \( \geq 16 \) compared to those with low depressive symptoms (MMSE = 23.8 ± 4.9 vs MMSE = 25.1 ± 4.3; \( P < .0001 \)). Subjects with CES-D score \( \geq 16 \) were significantly more likely to be female, less educated, unmarried, and to have ADL limitations and near vision impairment compared to subjects with CES-D < 16. Clinically relevant depressive symptoms were also significantly associated with a history of diabetes, stroke, and hypertension.

Figure 1 presents a scattered plot of correlation between change in MMSE score and change in CES-D score over a 7-year period. There is a significant longitudinal negative
correlation \( r = -0.12; P < .0001 \) between change in MMSE scores and change in CES-D scores, indicating a decrease in cognitive function with increasing depressive symptoms.

Figure 2 presents mean (with 95% confidence intervals) of MMSE scores at baseline and at 2, 5, and 7 years of follow-up by category of nondepressed (CES-D < 16) and depressed (CES-D ≥ 16). Subjects with CES-D ≥ 16 had significantly lower scores of MMSE than subjects with CES-D < 16 at baseline and at 2, 5, and 7 years of follow-up; the slopes of MMSE decline were steadily deeper for subjects with clinically relevant depressive symptoms after 2 and 5 years of follow-up.

Table 2 presents a mixed model estimate of the relationship between CES-D scores and MMSE scores over a 7-year period. In model 1, there was a significant cross-sectional association between having clinically relevant depressive symptoms (CES-D ≥ 16) and lower MMSE scores, independent of age, gender, education, marital status, and time (estimate = −1.14, standard error [SE] = 0.12; \( P < .0001 \)). In model 2, there was a significant longitudinal association between CES-D—by—time interaction on change in MMSE score (estimate = −0.18; SE = 0.05; \( P < .0001 \)), indicating that subjects with clinically relevant depressive symptoms had a significantly greater decline in MMSE scores over time compared to those without clinically relevant depressive symptoms. As shown in model 3, this longitudinal association of clinically relevant depressive symptoms with cognitive decline (estimate = −0.17; SE = 0.05; \( P < .001 \)) still remained significant after controlling for additional time-dependent variables of ADL limitations, self-reports of diabetes, stroke, heart attack, and hypertension, and near vision impairment.

A similar trend was found when CES-D was analyzed as a continuous variable; each point increase in the CES-D score was associated with a decline of 0.010 point in
MMSE score per year (SE = 0.002; P < .0001), adjusting for relevant confounders. Other significant predictors of lower MMSE scores at follow-up were increasing age, longer follow-up time, self-report of stroke and hypertension, ADL limitations, and near vision impairment.

We also assessed whether antidepressant medication use moderated the relationship between CES-D scores and change in MMSE. We conducted a subset analysis using available data for self-report of use of antidepressive medications at baseline (3.3%, n = 94). There was a nonsignificant trend for subjects using antidepressive medications to have higher CES-D scores (estimate = −0.68, SE = 0.36; P = .0616) than nonantidepressant users. There was also a nonsignificant trend for antidepressant users to have steeper MMSE decline (estimate = −0.12; SE = 0.11; P = .2600) than nonusers, adjusting for confounding covariates in Table 2. Finally, we found no significant interaction between depressive symptoms and antidepressive use on the decline of MMSE (slope).

To account for the possibility that subjects with more advanced cognitive impairment (MMSE < 18) might not accurately answer CES-D questions, we repeated the mixed-model analyses by excluding subjects with MMSE score < 18 at baseline. Having CES-D ≥ 16 (compared to CES-D < 16) was still significantly associated with lower MMSE scores and with greater MMSE decline over time, adjusting for covariates listed in Table 2. For example, when subjects with MMSE < 18 were excluded from the analyses, the results in the fully adjusted model 3 in Table 2 still showed a significant longitudinal relationship between CES-D ≥ 16 and greater decline in MMSE scores over time (estimate = −0.22, SE = 0.05; P < .0001). In general, with analyses restricted to those with MMSE ≥ 18, the magnitude of the CES-D and MMSE cross-sectional association effects declines while the magnitude of the longitudinal association effects increases.

Based on a prior study by Haringsma et al.,³³ we also examined the effects of very high levels of clinically relevant depressive symptoms on MMSE change by repeating the mixed-model analyses using higher CES-D cutoff points of 22 or 25. Less than 10% of our subjects had CES-D ≥ 22 or ≥ 25 in each wave. With the cutoff of CES-D ≥ 22, the results in the fully adjusted model 3 in Table 2 showed a nonsignificant trend between CES-D ≥ 22 and greater decline in MMSE scores over time (CES-D ≥ time, estimate = −0.06, SE = 0.07; P > .05). A similar nonsignificant trend of greater MMSE decline with very high levels of clinically relevant depressive symptoms (estimate = −0.10, SE = 0.08; P > .05) was also found using the cutoff score of CES-D ≥ 25.

DISCUSSION

Our findings can be summarized as follows. There was a significant cross-sectional association between presence of clinically relevant depressive symptoms (CES-D ≥ 16) and lower MMSE scores among community-dwelling older Mexican Americans. We also found that presence of clinically relevant levels of depressive symptoms at baseline was significantly associated with steeper decline in MMSE scores over 7 years compared to absence of clinically relevant depressive symptoms. This association was independent of age, gender, education, baseline MMSE, ADL limitations, diabetes, stroke, heart attack, and vision impairment. Other significant predictors of lower MMSE scores at follow-up were increasing age, longer follow-up time, self-report of stroke and hypertension, ADL limitations, and near vision impairment.

### Table 2. General Linear Mixed Models Estimates of MMSE as a Function of Depressive Symptoms Status

<table>
<thead>
<tr>
<th>Explanatory Variables</th>
<th>Model 1 Estimate (SE)</th>
<th>Model 2 Estimate (SE)</th>
<th>Model 3 Estimate (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>25.14 (0.09)*</td>
<td>25.06 (0.10)*</td>
<td>25.29 (0.10)*</td>
</tr>
<tr>
<td>Age (years)</td>
<td>−0.16 (0.01)*</td>
<td>−0.16 (0.01)*</td>
<td>−0.12 (0.01)*</td>
</tr>
<tr>
<td>Male</td>
<td>−0.20 (0.14)</td>
<td>−0.19 (0.14)</td>
<td>−0.25 (0.13)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>0.44 (0.02)*</td>
<td>0.44 (0.02)*</td>
<td>0.42 (0.02)*</td>
</tr>
<tr>
<td>Married</td>
<td>0.38 (0.13)*</td>
<td>0.38 (0.13)*</td>
<td>0.40 (0.12)*</td>
</tr>
<tr>
<td>Depression (CES-D ≥ 16)</td>
<td>−1.14 (0.12)*</td>
<td>−0.81 (0.15)*</td>
<td>−0.48 (0.15)*</td>
</tr>
<tr>
<td>Time</td>
<td>−0.54 (0.02)*</td>
<td>−0.51 (0.02)*</td>
<td>−0.46 (0.02)*</td>
</tr>
<tr>
<td>CES-D* time</td>
<td>−0.18 (0.05)*</td>
<td>−0.17 (0.05)*</td>
<td>−0.17 (0.05)*</td>
</tr>
<tr>
<td>Number of ADL limitation</td>
<td></td>
<td>−0.57 (0.04)*</td>
<td>−0.57 (0.04)*</td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td>0.03 (0.13)</td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td>−0.83 (0.21)*</td>
<td></td>
</tr>
<tr>
<td>Heart attack</td>
<td></td>
<td>0.35 (0.18)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td>−0.35 (0.12)*</td>
<td></td>
</tr>
<tr>
<td>Near vision</td>
<td></td>
<td>−1.61 (0.15)*</td>
<td></td>
</tr>
</tbody>
</table>

Note: Estimate, estimate change in MMSE score; SE, standard error; CES-D, Center for Epidemiological Studies Depression Scale, range 0–60; ADL, Activities of Daily Living limitations, range 0–7; Variable time is testing the slope of decline in MMSE scores over time. The term* for the interaction between high depressive symptoms and time represents the longitudinal effect of the baseline measure of depressive symptoms on the annual rate of decline in performance of the MMSE.

a. P < .0001.
b. P < .001.
c. P < .01.
Our findings are consistent with the results of previous studies and different from others.6,14,34 A community-based study of 4392 subjects aged 65 and older (38% white and 62% black) showed that, independent of social, demographic, and health factors, each depressive symptom at baseline CES-D was associated with a 5% increase in rate of global cognitive decline at 5-year follow-up.34 Others described no such association.8,11,14,35,36 For example, a community-based study of older adults (61% with greater than high school education) found no significant relationship between baseline depressive symptoms and cognitive decline over time.8 Unlike past studies in well-educated older cohorts, our study in older Mexican Americans (mean years of education = 4.9 ± 3.9) showed significant association between presence of clinically relevant depressive symptoms and increased risk of cognitive decline over time, independent of time-dependent changes in demographic and health covariates. One possible reason for the association between depression and cognition in our study might be the low educational status of the participants. The relation between low educational levels and psychological stress is complex. It is possible that low educational level creates chronic stress that contributes to depressive symptomatology. In the current study, adjusting for years of education did not affect the depression-cognition association.

The finding of nonsignificant association between antidepressant use and MMSE decline in our study likely reflects the small sample size (n = 94) of subjects on antidepressant medications. Thus, our analysis of the effect of antidepressant use (only 3.3% of our sample) versus nonuse on MMSE change over time is underpowered to detect any significant difference between the 2 groups. A longitudinal study with large sample will be needed to examine any potential effect of depression treatment on cognitive function. Because antidepressant users likely represent patients with clinically diagnosed depression (ie, clinically relevant levels of depression at baseline), the trend for these patients to have steeper cognitive decline, though not statistically significant, is consistent with our overall finding of association of high CES-D scores with steeper MMSE decline. Our finding, however, does not prove cause and effect relationship between depression and cognitive decline. Late-life depressive symptoms could simply be an early marker for incipient cognitive decline or preclinical dementia. In that scenario, treating depression, though laudable and clinically recommended, may not necessarily alter the course of ongoing cognitive decline. Regardless, an important area for future study is assessing the long-term effect of depression treatment on subsequent cognitive function in depressed older adults.

The finding of nonsignificant association between presence of very high levels of clinically relevant depressive symptoms (very high CES-D cutoff score ≥ 22 or ≥ 25) and MMSE decline likely reflects the small number of our subjects with the high CES-D cutoff score. Thus, our analysis of the effect of very high CES-D scores (< 10% of our sample) on MMSE change over time is underpowered to detect any significant difference over time. The wide confidence intervals and loss of statistical power thus reflect the low percentage of subjects (< 10% in each wave had CES-D ≥ 22 or ≥ 25) with very high levels of clinically relevant depressive symptoms in our community-based study. In contrast, a clinic-based or hospital-based study is likely to have a higher proportion of subjects with very high CES-D scores. Such a clinic-based study with a larger sample size of clinically depressed subjects may allow enough power to detect longitudinal effects of very high CES-D scores (≥ 22 or ≥ 25) on cognitive function.

It is not clear why depressed people might be at a higher risk of cognitive decline. Is depression a marker of incipient dementia? Is the cognitive decline simply reflecting interference with cognitive testing by the presence of depressive symptoms such as poor motivation and inattention? Are depressive symptoms clustering with other factors (eg, social disengagement) known to be associated with poor cognitive function? Are cognitive and mood disorders reflecting common etiologic factors? Preliminary answers to these questions have begun to emerge in population studies of brain aging.10,37-43

Findings from these studies support the possibility of shared etiologic factors (eg, apolipoprotein E4 genotype, diabetes, stroke, and high inflammation markers) that may contribute to both depressive symptoms and cognitive impairment.36-42,44 For example, community-based studies in older adults suggest that high blood levels of interleukin-6 (a proinflammatory cytokine) are significantly associated with increased odds of depression37,39 and high risk of cognitive decline,38 independent of demographic and other health factors. Other studies in older community-dwelling subjects showed that apolipoprotein E4 and radiologic findings of brain infarcts are independently associated with increased risk of subsequent depression and dementia.41,42 These data suggest common pathogenic factors accounting for both depressive symptoms and cognitive impairment. In this scenario, depressive symptoms might manifest earlier, but over time and with persistence of the pathogenic factors, progressive loss of brain function, as captured by MMSE, becomes apparent. Studies involving blood markers and brain imaging are needed to explore the hypothesis of common pathogenic factors contributing to depression and cognitive decline.
It is also possible that coexisting factors (e.g., diabetes and ApoE4 genotype) might interact and lead to increased likelihood of depression and cognitive loss. The risk factors may also vary by different ethnic groups. For example, in older Mexican Americans, a population with low frequency of ApoE4, the high prevalence of type 2 diabetes mellitus may be contributing to both the depressive symptoms and the cognitive decline, with the depression being captured much earlier and the cognitive loss becoming greater and clinically manifest over time among the depressed versus nondepressed. There are no data, however, on whether depressive symptoms occur early in pathogenesis of cognitive decline and whether antidepressant drugs can alter subsequent progression to clinical dementia in diabetes patients. This is an important area for future study. Future study should also address the interaction effect of genetic susceptibility factor (such ApoE4 genotype) and diabetes on the association between depression and subsequent cognitive change.

A limitation of our study is the reliance on self-reports of medical conditions, ADL measures, and other covariates in subjects with potential for impaired cognition. However, past studies have reported good agreement between patients’ self-reported ADLs and medical conditions and proxy assessments of patients’ ADL performance, irrespective of patients’ cognitive functioning. Another limitation included using only the MMSE—as opposed to a more comprehensive test of cognition including the executive function—for assessment of cognitive functioning. For example, the MMSE does not reflect the change in executive cognition, a cognitive domain known to be important for daily living activities in the elderly. The MMSE may also be affected by educational level, cultural and linguistic factors, and visual impairment. Finally, although it is possible that repeated MMSE administration may result in improvement of some items due to learning phenomena, this possibility is less likely in our study because of long intervals of at least 2 years between each interview wave. Despite these potential biases, past studies have shown that MMSE is a reliable, valid, easy-to-use, and fast measure of cognitive function in community-based research.

Our study has several strengths including its large community-based sample, its prospective design, and its exploration of the potential role of emotional health in cognitive disablement in older Mexican Americans, a rapidly growing segment of the older population in the United States. Another important strength of our study is the use of mixed models, an analytic approach that allowed the use of all available data and evaluation of time-dependent effects.

In conclusion, our study showed that older Mexican Americans with clinically relevant levels of depressive symptoms had greater decline in cognitive function over a period of 7 years compared to persons without clinically relevant depressive symptoms, independent of other demographic and health factors. It is not clear whether interventions aimed at treating depression will reduce the onset of cognitive decline and subsequent dementia. More studies are needed to unravel the biologic and genetic factors mediating depressive symptomatology and cognitive decline in the elderly, with the goal of creating strategies to prevent or delay the onset of cognitive and physical disability in the elderly.

References
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