Smoking Increases Risk for Cognitive Decline Among Community-Dwelling Older Mexican Americans

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Objectives: Few studies have investigated smoking and cognitive decline (CD) among older Mexican Americans. In this study, the authors explore the relationship between smoking status and cognitive changes over time in a large sample of community-dwelling older adults of Mexican descent. Design: Latent growth curve analyses were used to examine the decreasing growth in the number of correct responses on a test of cognitive functioning with increasing age (7 years with four data collection points). Setting: In-home interviews were obtained from participants residing in the Southwest United States. Participants: Participants were community-dwelling older Mexican Americans. Measurements: Cognitive functioning was assessed at each of the four data collection points with the Mini-Mental State Examination. Participants’ self-reports of health functioning and smoking status were obtained at baseline. Results: With the inclusion of health variables and other control variables, the effect of smoking status on cognitive functioning was significant such that the decrease in the number of correct responses over time was greater for smokers than for nonsmokers. Conclusions: Smoking increases risk for CD among community-dwelling older Mexican Americans. There are numerous health benefits in quitting smoking, even for older adults who have been smoking for many years. Further efforts to ensure that smoking cessation and prevention programs are targeted toward Hispanics are necessary. (Am J Geriatr Psychiatry 2009; 17:934–942)

Key Words: Smoking, cognitive decline, Hispanics, older adults

Smoking affects nearly every organ of the body and is the number one cause of premature death among elderly in the United States. Moreover, smoking has been shown in some studies to accelerate the rate of cognitive decline (CD). Although the mechanisms by which smoking affects CD are not known at this time, several hypotheses have been suggested and include: 1) smoking causes oxidative...
stress damage, 2) effects of smoking are mediated by
cerebrovascular events, and 3) smoking interacts
with other health conditions and variables known to
influence CD.

The characteristics of older Hispanic smokers and
the effects of smoking on CD have not been well
documented empirically and, thus, will be the focus
of this study. Examining the extent to which smoking
influences CD may increase our understanding of the
underlying processes related to CD. In addition, as-
sessing this relationship among elderly Hispanics
can help us to evaluate the health needs of Hispanic
elders who smoke. Specifically, we will examine
smoking as a predictor of CD over time in a sample
of community-dwelling older Hispanic adults of
Mexican American descent, a population that has
been understudied in regard to its association with
known risk factors for CD.

Hispanics, including Mexican Americans, have
higher rates of several risk factors related to CD
compared with whites. Mexican Americans have
fewer years of education,7 greater physical function-
ing problems,8 and greater incidence of stroke9 than
whites, all of which have been linked to CD.10,11 This
may be a result of low socioeconomic status (SES),
life style factors, or reduced access to preventive
healthcare compared with other U.S. subgroups. For
example, Hispanics in general are the least likely
racial or ethnic group to have health insurance12 and
are least likely to receive smoking cessation advice.13

Current evidence suggests that smoking status
predicts CD and dementia. A 2007 meta-analysis of
prospective studies shows that when compared with
individuals who have never smoked, current smok-
ers have an increased risk of CD and Alzheimer
disease (AD),3 the most common cause of dementia.
However, not all studies have found a consistent
relationship between smoking and CD. Indeed, some
studies have found no association.11,14–16 Method-
ological differences across studies may account for
the inconsistencies. Studies examining the effect of
smoking on CD have differed in length of follow-up,
size of samples, and choice of outcome measures
assessing cognitive functioning. For example, a short
follow-up period may not allow sufficient time for
cognitive changes to occur in relation to smoking
status. Also, given that mortality rates are signifi-
cantly higher for smokers than for nonsmokers, a
study design without a relatively large sample may
fail to detect differences because of the premature
death of smokers.

How cognitive functioning is assessed is also im-
portant. Several studies have looked at changes from
nondemented to demented status. This can be prob-
lematic because the power to detect the influence of
smoking on changes in cognitive functioning may be
reduced when using dichotomous rather than con-
tinuous measures. That is, the presence or absence
of dementia or the change from nondemented to de-
mented status may be a less sensitive measure of
change over time than changes in measures of cog-
nitive functioning over time (i.e., continuous mea-
sures). In addition, most studies have measured cog-
nitive functioning at only two occasions rather than
examining change in cognitive functioning scores
across several occasions. Furthermore, variability in
the age of participants, which have ranged from
young adults to the elderly, may influence results,
with younger participants less likely to demonstrate
CD regardless of smoking status. Clearly, studies
examining a high-risk population for CD (e.g., an
older sample) may be more likely to identify the
influence of smoking on CD.

Importantly, research on Hispanics has been lim-
ited. The 2007 meta-analysis3 on smoking and cogni-
tive functioning, which included data from 19 stud-
ies, demonstrated evidence for the effects of smoking
on CD. However, most of the studies’ participants
were non-Hispanic. To date, only three articles,
based on the same large-scale study, examined a
sample with a large percentage (e.g., at least 40%) of
Hispanic individuals (predominately of Dominican,
Puerto Rican, and Cuban descent). In two of these
articles, current smoking was found to increase the risk
of AD.17,18 These authors report that when the analyses
were stratified by ethnic group, the results did not
change appreciably.17 Using data from the same study,
Reitz et al.5 examined measures of memory perfor-
mance and found that elderly smokers experienced
a faster decline compared with nonsmokers. How-
ever, the association was not examined separately
by ethnicity.

Despite the importance of understanding the asso-
ciation between smoking status and CD in older
adults of Mexican origin (the fastest growing popu-
lation in the United States),19 to date, the effects of
smoking on CD have not been studied prospectively
in this subgroup. Therefore, in this study, we will
explore whether being a current smoker at baseline predicts subsequent cognitive functioning scores using data from the Hispanic Established Populations for Epidemiologic Studies of the Elderly. We use several methods to increase our ability to detect the influence of smoking on CD including a) using prospective data in which we followed up participants for a 7-year period, (b) using a continuous measure of global cognitive functioning, the Mini-Mental State Examination (MMSE) that was sensitive to general changes in cognitive functioning over time, c) obtaining a large sample of Mexican Americans aged 65 years and older, and d) controlling for key variables that may also influence the rate of CD among smokers (e.g., indices of SES and health functioning variables). In addition, this study includes four waves of cognitive functioning scores, and the data are analyzed using latent growth curve modeling (LGM). The LGM has several advantages. First, it allows for an examination of growth in cognitive scores (or growth in errors) during several occasions. Moreover, LGM allows for an examination of group differences between smokers and nonsmokers in the rate of change in cognitive functioning with each year of aging (rather than the arbitrary date of data collection).

**METHODS**

**Participants**

The Hispanic Established Populations for Epidemiologic Studies of the Elderly data are from a representative sample of community-dwelling Mexican American adults, aged 65 years and older, residing in five southwestern states. Data were collected from the same individuals over 7 years at four separate waves: baseline interview (1993–1994), 2-year follow-up (1995–1996), 5-year follow-up (1998–1999), and 7-year follow-up (2000–2001). The sampling strategy and methods of the study have been described elsewhere (for human participant protection, study protocols were approved by the institutional review board of the University of Texas Medical Branch at Galveston and the University of Texas at Austin).

The baseline survey consisted of a sample of 3,050 individuals. There was attrition over time; a proportion of the sample had entered a nursing home, was lost to follow-up, or had died, reducing the sample size to 1,557 individuals at Wave 4. Importantly, there were substantial age differences at baseline between the smokers and the nonsmokers who were not retained to Wave 4. Not surprisingly, among this group, smokers were younger than the nonsmokers ($M = 72.6$ years, $SD = 5.7$ versus $M = 74.8$, $SD = 7.7$ years), $F_{[1,1,549]} = 14.9$, $p < 0.001$), likely because of the premature death of smokers, making direct comparisons on cognitive functioning between smokers and nonsmokers difficult. That is, the selective attrition of smokers (because of smoking-related illnesses) in this sample may obscure the effect of smoking on CD over time. Specifically, smokers may die of smoking-related illnesses before there are any observable indicators of CD. To address this problem, we included only those participants (smokers and nonsmokers) for whom we observed cognitive functioning over a 7-year period.

**Control Variables**

Several factors associated with CD among older adults were statistically controlled. These include age, gender, education, annual household income, nativity (U.S. born or not), and health functioning. We were unable to control for alcohol consumption due to a significant amount of missing data.

**Measures**

**Smoking.** At baseline, participants were asked whether they currently were a regular cigarette smoker (no/yes). Self-reports of smoking behavior have generally been found to have high sensitivity and specificity when compared with biochemical measures.

**Cognitive Functioning.** The MMSE was administered at each of the four waves. The MMSE provides a brief and objective measure of global cognitive functioning and assesses five areas of cognitive functioning including orientation, registration, attention and calculation, recall, and language. Scores ranged from 0 to 30, with higher scores indicative of higher cognitive functioning. The MMSE has been used extensively in epidemiologic research of older adults. The internal reliability was as follows: Time
Health Problems. At baseline, respondents were asked whether they had been told by a doctor that they had a heart attack, stroke, hypertension, diabetes, or cancer. Responses were coded 1 (yes), 2 (maybe), or 3 (no). Self-reported health problems have been found to have good agreement with medical records and physician reports.28,29

Procedures for Analyses

LGM was conducted using the software package Mplus.30 LGM involves specifying a factor model for repeated measures in which the factors represent individual-specific aspects of change (intercepts and linear slopes), and factor loadings are fixed to values representing linear growth (here, 0, 2, 5, and 7 to correspond to wave of measurement). The intercept and slope factors, in turn, may be regressed on predictors and covariates. We were interested in examining predictors of growth (i.e., individual differences in the slope factor).

First, the average number of correct responses on the MMSE (i.e., the mean slope for the number correct) was examined, controlling for age at the first wave. Second, smoking status was added as a predictor of intercepts and slopes to examine the impact of smoking on the growth of the number correct with increasing age (e.g., the decrease in number of correct responses over time). Finally, the effect of smoking on growth of the number of correct responses was examined, controlling for demographics, health variables, and SES (education and income). We expected smokers and nonsmokers alike to show a decrease in the number of correct responses over time; however, we expected the decrease to be greater for smokers than for nonsmokers.

RESULTS

Planned Analyses

We first provide descriptive statistics on key variables for smokers and nonsmokers who survived to Wave 4 (Table 1). Then, we conducted LGM analyses to examine the effect of smoking on growth of the number of correct responses on the MMSE (specifically change over time in the number of correct responses) with increasing age over the four waves, including only those participants who survived to Wave 4 (Table 2).

Descriptive Statistics

Of participants who were retained in the study to Wave 4 (N = 1,557), 38% were men, and 62% were women. At Wave 1, 11.9% of participants were smokers. Consistent with previous research, smokers were more likely to be men than women (59% versus 41%). At baseline, the average age of participants was 71.5 (SD = 5.5), but overall, smokers were significantly younger than nonsmokers (M = 69.9, SD = 4.4, and M = 71.7, SD = 5.6, respectively), likely reflecting the earlier death of participants who died from smoking-related illnesses.

As we have found in other studies of older smokers,31,32 because of the younger age of smokers, health problems were actually greater among nonsmokers than smokers. In uncontrolled analyses at baseline, nonsmokers were more likely than smokers to have experienced several health problems including stroke (4% versus 2%), hypertension (42% versus 24%), and diabetes (20% versus 12%). Descriptive data are summarized in Table 1 by smoking status.

Latent Growth Curve Analyses

Latent growth curve analyses were used to examine the change in the number of correct responses on the MMSE with increasing age (7 years with four data collection points). We treated wave as the within-person metric of time and person as the unit of analysis.22 This analysis permitted us to estimate individual differences in cognitive functioning over time and to assess whether variability in change in the number of correct responses could be predicted by key variables while controlling for demographic variables.

The influence of smoking on the growth of the number of correct responses on the MMSE over time, controlling for age, was examined. In the first set of analyses, we fit a model that estimated linear change in cognitive functioning for every person. We first estimated a random intercept model, which contains no predictors and is intended only to partition vari-
ance in cognitive change into between- and within-
person components. Therefore, the factor covariance
matrix consists only of the intercept variance ($\psi_0$); the
Level 1 residual variance is denoted as $\theta$. Factor
loadings for this intercept factor were constrained
equal to 1.0. We found that variability in the number
of correct responses on the MMSE was split almost
evenly between levels, with an estimated within-
person variability of $\hat{\theta} = 15.68$ and a between-person
variability of $\hat{\psi}_{11} = 13.43$ (intraclass correlation = 0.46
indicating that 46% of the variability was between
subjects). The mean intercept was 23.15 (SE = 0.11,
Wald $z = 210.5$, $p < 0.001$) and corresponds to the
average number of correct responses across all sub-
jects at all four waves. Model fit was poor, as ex-
pected for a model that does not accommodate
change in the number of correct responses (e.g., de-
creasing number of correct responses over time)
(root mean square error of approximation = 0.26;
90% confidence interval = 0.25–0.27; and standard-
ized root mean square residual = 0.59).
Wave of measurement was introduced by includ-
ing a linear slope factor with loadings fixed to 0, 2, 5,
and 7. Of key interest in this model were the mean

### TABLE 1. Comparison of Smokers and Nonsmokers Retained to Wave 4 on Key Variables

<table>
<thead>
<tr>
<th>All Participants, $N = 1,557$</th>
<th>Smokers, $n = 186$ (11.9%)</th>
<th>Nonsmokers, $n = 1,371$ (88.1%)</th>
<th>Test Statistic</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MMSE, $M$ (SD)</strong></td>
<td>24.7 (4.2)</td>
<td>24.7 (4.0)</td>
<td>24.7 (4.2)</td>
<td>$F_{[1, 1,497]} = 0.00$</td>
</tr>
<tr>
<td><strong>Wave 1</strong></td>
<td>24.2 (4.5)</td>
<td>24.5 (4.1)</td>
<td>24.2 (4.6)</td>
<td>$F_{[1, 1,576]} = 0.44$</td>
</tr>
<tr>
<td><strong>Wave 3</strong></td>
<td>22.8 (5.1)</td>
<td>22.7 (4.9)</td>
<td>22.8 (5.1)</td>
<td>$F_{[1, 1,524]} = 0.01$</td>
</tr>
<tr>
<td><strong>Wave 4</strong></td>
<td>21.4 (6.4)</td>
<td>21.2 (6.3)</td>
<td>21.4 (6.4)</td>
<td>$F_{[1, 1,555]} = 0.14$</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>71.5 (5.5)</td>
<td>69.9 (4.4)</td>
<td>71.7 (5.6)</td>
<td>$F_{[1, 1,555]} = 17.08$</td>
</tr>
<tr>
<td><strong>Sex, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>594 (38)</td>
<td>109 (59)</td>
<td>485 (35)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>963 (62)</td>
<td>77 (41)</td>
<td>886 (65)</td>
<td>two-sided Fisher’s exact test $&lt;0.001$</td>
</tr>
<tr>
<td><strong>Education, $M$ (SD)</strong></td>
<td>5.0 (3.9)</td>
<td>4.8 (3.9)</td>
<td>5.0 (3.9)</td>
<td>$F_{[1, 1,555]} = 0.30$</td>
</tr>
<tr>
<td><strong>Nativity, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>U.S. born</td>
<td>899 (58)</td>
<td>111 (60)</td>
<td>788 (58)</td>
<td></td>
</tr>
<tr>
<td>Not U.S. born</td>
<td>658 (42)</td>
<td>75 (40)</td>
<td>583 (45)</td>
<td>two-sided Fisher’s exact test 0.581</td>
</tr>
<tr>
<td><strong>Household yearly income, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$0–$4,999</td>
<td>215 (15)</td>
<td>26 (15)</td>
<td>189 (15)</td>
<td></td>
</tr>
<tr>
<td>$5,000–$9,999</td>
<td>603 (42)</td>
<td>86 (51)</td>
<td>517 (41)</td>
<td></td>
</tr>
<tr>
<td>$10,000–$14,999</td>
<td>344 (24)</td>
<td>33 (19)</td>
<td>311 (25)</td>
<td></td>
</tr>
<tr>
<td>$15,000–$19,999</td>
<td>165 (12)</td>
<td>18 (11)</td>
<td>147 (12)</td>
<td></td>
</tr>
<tr>
<td>$20,000–$29,999</td>
<td>59 (4)</td>
<td>4 (2)</td>
<td>55 (4)</td>
<td></td>
</tr>
<tr>
<td>$30,000–$39,999</td>
<td>27 (2)</td>
<td>2 (1)</td>
<td>25 (2)</td>
<td></td>
</tr>
<tr>
<td>$40,000–$49,999</td>
<td>4 (0.3)</td>
<td>0 (0)</td>
<td>4 (0.3)</td>
<td></td>
</tr>
<tr>
<td>$50,000 +</td>
<td>6 (0.4)</td>
<td>1 (0.6)</td>
<td>5 (0.4)</td>
<td>$\chi^2 (7, 7 = 1,557) = 7.75$</td>
</tr>
<tr>
<td><strong>Heart attack Wave 1, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>119 (8)</td>
<td>12 (7)</td>
<td>107 (8)</td>
<td></td>
</tr>
<tr>
<td>Maybe</td>
<td>20 (1)</td>
<td>2 (1)</td>
<td>18 (1)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1,415 (91)</td>
<td>172 (93)</td>
<td>1,243 (91)</td>
<td>$\chi^2 (2, N = 1,554) = 0.52$</td>
</tr>
<tr>
<td><strong>Stroke Wave 1, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>62 (4)</td>
<td>4 (2)</td>
<td>58 (4)</td>
<td></td>
</tr>
<tr>
<td>Maybe</td>
<td>4 (0.3)</td>
<td>2 (1)</td>
<td>2 (0.1)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1,488 (96)</td>
<td>180 (97)</td>
<td>1,308 (96)</td>
<td>$\chi^2 (2, N = 1,554) = 7.31$</td>
</tr>
<tr>
<td><strong>Hypertension Wave 1, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>615 (40)</td>
<td>44 (24)</td>
<td>571 (42)</td>
<td></td>
</tr>
<tr>
<td>Maybe</td>
<td>23 (2)</td>
<td>3 (2)</td>
<td>20 (2)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>914 (59)</td>
<td>138 (75)</td>
<td>776 (57)</td>
<td>$\chi^2 (2, N = 1,552) = 22.13$</td>
</tr>
<tr>
<td><strong>Cancer Wave 1, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>56 (4)</td>
<td>2 (1)</td>
<td>54 (4)</td>
<td></td>
</tr>
<tr>
<td>Maybe</td>
<td>4 (0.3)</td>
<td>1 (0.5)</td>
<td>3 (0.2)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1,497 (96)</td>
<td>183 (98)</td>
<td>1,314 (96)</td>
<td>$\chi^2 (2, N = 1,557) = 4.49$</td>
</tr>
<tr>
<td><strong>Diabetes Wave 1, $f$ (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>294 (19)</td>
<td>23 (12)</td>
<td>271 (20)</td>
<td></td>
</tr>
<tr>
<td>Maybe</td>
<td>63 (4)</td>
<td>10 (5)</td>
<td>53 (4)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1,195 (77)</td>
<td>153 (82)</td>
<td>1,042 (76)</td>
<td>$\chi^2 (2, N = 1,552) = 6.49$</td>
</tr>
</tbody>
</table>

Smoking Increases Risk for Cognitive Decline
intercept ($\alpha_1$), mean slope ($\alpha_2$), and intercept and slope variances ($\psi_{11}$ and $\psi_{22}$), and covariance ($\psi_{21}$) (conditional on age as a covariate). The number of correct responses decreased by an average of $\hat{\alpha}_2 = 0.47$ (SE = 0.02, z = 23.5, p < 0.001) per year of age. We assessed the degree to which the rate of correct responses varied across individuals by freely estimating the slope variance ($\hat{\psi}_{22} = 0.29$) and the intercept-slope covariance ($\hat{\psi}_{21} = 0.43$), $\Delta \chi^2 (df = 2) = 441.67$, p < 0.001; that is, the slope variance and intercept-slope covariance were together significantly different from 0). Modeling results are reported in Table 2.

We controlled for age by entering it as a person-level predictor of both intercepts and slopes. Smoking was added as a person-level predictor; this yielded a nonsignificant effect of smoking status, controlling for age. With the inclusion of health variables and other control variables in an additional model, the effect of smoking status on slope became significant ($\hat{\gamma}_{\text{smoking}} = 0.13$, SE = 0.06, z = 2.17, p = 0.037), such that the rate of decrease in the number of correct responses was greater for smokers than for nonsmokers. It is important to note that we found that the effect of smoking on slope to be positive because the dependent measure is scored in terms of number correct, and smoking is scored as 1 = smoker and 2 = nonsmoker. Controlling for covariates, nonsmokers decreased by an average of 0.45 correct responses per year and smokers by an average of 0.58 correct responses per year.

### CONCLUSIONS

In this study, we examined the relationship of smoking status to CD in a sample of older Mexican American adults. The prevalence of smoking in our sample (18.4% of men and 8% of women were current smokers) was slightly higher than that reported from other national data. Others have found that among Hispanic Medicare recipients, 12.7% of men and 6.6% of women were current smokers.\(^{31}\) Data also show that smoking prevalence differs by race and ethnicity. The prevalence of smoking among older Hispanic men...
is generally greater than that of older white men (11.9%) but less than that of older black men (20.5%).

For older Hispanic women, smoking prevalence is less than that of both white (10.4%) and black women (11.3%).

Consistent with studies of non-Hispanics, we found that smoking predicted CD such that current smokers, compared with nonsmokers, experienced a greater decline on a measure of cognitive functioning, the MMSE, over 7 years. Importantly, this study involved several features that have been absent from other such studies, including a prospective design, a continuous measure of global cognitive functioning assessed at four occasions, a large sample size, a relatively long follow-up period, and the use of latent growth curve analysis. Moreover, this study focused on older adults of Mexican origin, one of the fastest growing populations in the United States.

There are several hypotheses regarding the mechanisms by which smoking may affect CD. One hypothesis is that smoking causes oxidative stress, or cumulative damage caused by free radicals, to cells and organs including the brain. Oxidative stress is evident in the pathogenesis of AD and may cause neuron degeneration. Cigarette smoke contains free radicals and is involved in the generation of oxidative stress. Furthermore, smokers tend to have both a lower dietary intake and circulation of antioxidants that neutralize free radicals.

A second hypothesis is that long-term exposure to cigarettes may lead to atherosclerosis, resulting in stroke and subsequent vascular dementia. Tobacco smoke has been shown to increase risk of atherosclerosis, which is caused by the formation of plaques within the arteries. Several ingredients in cigarettes and cigarette smoke, including nicotine monoxide, damage the endothelium and lead to the narrowing of blood vessels, increasing the likelihood of a blockage and, thus, of a heart attack or stroke.

Smoking may also affect cognition and the brain due to indirect effects on other conditions such as lung functioning. For example, smoking has been shown to cause lung injury that leads to chronic obstructive pulmonary disease. Poor lung functioning is associated with both poorer cognitive functioning and brain atrophy. Smoking may interact with other risk factors such as alcohol consumption and genetics (e.g., apolipoprotein E gene) that are associated with increased CD.

Although the mechanism by which smoking directly affects CD is as yet unknown, there is evidence to suggest that smoking does negatively affect brain structure. In individuals with normal neurologic and cognitive status at baseline, smoking has been shown to accelerate worsening white matter grade, leukoaraiosis, cerebral atrophy, and cerebral perfusional declines, which are markers of depleted neuronal synaptic reserves that predispose individuals to CD and the onset of dementia. On the other hand, it is important to note that others have not detected an effect of smoking on total brain atrophy. However, some research has shown that reduction in total brain volume is independent of other degenerative changes, such as white matter hyperintensities, although this study found that smoking was related to both types of degeneration over time.

Some factors known to influence CD (e.g., genetics) cannot be changed, but smoking is a potentially modifiable behavior. Therefore, the benefits of smoking cessation among older Hispanics, in relation to CD in particular, should be explored. Some studies have suggested that quitting smoking may have benefits on cognition. These findings point to the positive impact of smoking cessation on cognition even among older adults. In addition, there are other significant health benefits to quitting smoking even at an older age.

Despite the many potential benefits of smoking cessation, there has been more focus on offering smoking cessation programs to young and middle-aged adults and to non-Hispanics. Risk factors for smoking-related health conditions may not be addressed by clinicians because many assume that it is too late and too difficult for older adults to attempt to modify smoking behavior. Additionally, older smokers may be unaware that there are significant health benefits of smoking cessation late in life. Studies of community samples have found the cessation rate among older adults to be 10%. Importantly, when offered the tools they need, older smokers quit smoking at rates comparable with those of younger smokers. In particular, tailoring cessation programs in ways that are appropriate to age and ethnicity/culture has been effective in some studies for older adults and Hispanics.

As in every study there are limitations that should be considered. One limitation of this study was that there was an implicit assumption that the covariates...
were time invariant. It was assumed, for example, that the demographic and health status variables remained invariant. Our model did not account for the likely change in health status over time.

Second, there was considerable attrition over time through the death of participants. Given the selective mortality of younger smokers (compared with non-smokers), we may have underestimated the influence of smoking on CD due to the premature death of smokers who could have experienced CD had they survived during the 7-year follow-up period. In contrast, it should be noted that an additional latent growth curve analysis was conducted including all participants with missing data, i.e., the data from participants who died between Wave 1 and Wave 4. With the inclusion of participants with missing data, there was only a trend toward smokers showing more CD than nonsmokers (p = 0.08). This may have been the result of smokers dying prematurely of smoking-related illnesses before smoking affected cognitive functioning. That is, we may not have followed up smoking participants, who died prematurely, long enough to document the changes in cognitive functioning related to smoking. Nonetheless, the results of this study may not generalize to the population as a whole.

Third, there are other variables associated with smoking and CD, which were not measured in this study and may have enhanced the apparent association between smoking and CD. Specifically, health and life style factors associated with both smoking and CD may explain, in part, the observed association between smoking and CD. For example, smokers may have poorer nutrition be more likely to drink harmful levels of alcohol or undertake less physical activity than nonsmokers.3

Future research could expand on the present investigation in several ways. First, there were no comparisons to other racial or ethnic groups to examine the possibility of a differential effect of smoking on CD. Second, this study cannot identify the specific mechanisms by which smoking accelerates CD. Future investigations should use more specific measures of smoking exposure that can quantify inhaled doses including smoking topography (e.g., puff volume and duration) or measures of cotinine,26 rather than rely on self-reports of smoking behavior. In addition, biomarkers of oxidative stress or atherosclerosis could be included. Third, the benefits of smoking cessation on cognitive functioning should be explored perhaps through the inclusion of cognitive measures in large-scale studies of smoking cessation.

In summary, we found smoking as a predictor of CD in older Mexican American adults. This finding is important because of the consequences for healthcare in Mexican Americans. Future research should focus on the specific needs of Hispanic elders in addressing smoking cessation.

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