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

Nicole Collins, B.A., Natalie Sachs-Ericsson, Ph.D., Kristopher J. Preacher, Ph.D., Kristin M. Sheffield, B.S., Kyriakos Markides, Ph.D.

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.[1,2] Moreover, smoking has been shown in some studies to accelerate the rate of cognitive decline (CD).[3–6] 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

Received September 10, 2008; revised February 12, 2009; accepted March 23, 2009. From the Department of Psychology, Florida State University, FL (NC, NS-E); Department of Psychology, University of Kansas, KS (KJP); and Preventative Medicine and Community Health, University of Texas Medical Branch, TX (KMS, KM). Send correspondence and reprint requests to Natalie Sachs-Ericsson, Ph.D., Department of Psychology, 1107 West Call Street, Tallahassee, FL 32306-4301. e-mail: sachs@psy.fsu.edu

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, assessing 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 functioning problems,[8] and greater incidence of stroke[9] 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 insurance[12] 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 smokers 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] Methodological 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 significantly 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 important. Several studies have looked at changes from nondemented to demented status. This can be problematic because the power to detect the influence of smoking on changes in cognitive functioning may be reduced when using dichotomous rather than continuous measures. That is, the presence or absence of dementia or the change from nondemented to demented status may be a less sensitive measure of change over time than changes in measures of cognitive functioning over time (i.e., continuous measures). In addition, most studies have measured cognitive 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 limited. The 2007 meta-analysis[3] on smoking and cognitive functioning, which included data from 19 studies, 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 performance and found that elderly smokers experienced a faster decline compared with nonsmokers. However, the association was not examined separately by ethnicity.

Despite the importance of understanding the association between smoking status and CD in older adults of Mexican origin (the fastest growing population 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.[20] 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[21]) that would be 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).[22] 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).[20]

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.[23–25] 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.[26]

Cognitive Functioning. The MMSE was administered at each of the four waves. The MMSE provides a brief and objective measure of global cognitive functioning[21] 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.[27] The internal reliability was as follows: Time

1, $\alpha = 0.78$; Time 2, $\alpha = 0.80$; Time 3, $\alpha = 0.84$; and Time 4, $\alpha = 0.81$.

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-

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

	All Participants, N = 1,557	Smokers, n = 186 (11.9%)	Nonsmokers, n = 1,371 (88.1%)	Test Statistic	p
MMSE, *M* (SD)					
Wave 1	24.7 (4.2)	24.7 (4.0)	24.7 (4.2)	$F_{[1, 1,497]} = 0.00$	0.975
Wave 2	24.2 (4.5)	24.5 (4.1)	24.2 (4.6)	$F_{[1, 1,378]} = 0.44$	0.507
Wave 3	22.8 (5.1)	22.7 (4.9)	22.8 (5.1)	$F_{[1, 1,324]} = 0.01$	0.905
Wave 4	21.4 (6.4)	21.2 (6.3)	21.4 (6.4)	$F_{[1, 1,555]} = 0.14$	0.707
Age	71.5 (5.5)	69.9 (4.4)	71.7 (5.6)	$F_{[1, 1,555]} = 17.08$	<0.001
Sex, *f* (%)					
Male	594 (38)	109 (59)	485 (35)		
Female	963 (62)	77 (41)	886 (65)	two-sided Fisher's exact test	<0.001
Education, *M* (SD)	5.0 (3.9)	4.8 (3.9)	5.0 (3.9)	$F_{[1, 1,532]} = 0.30$	0.587
Nativity, *f* (%)					
U.S. born	899 (58)	111 (60)	788 (58)		
Not U.S. born	658 (42)	75 (40)	583 (43)	two-sided Fisher's exact test	0.581
Household yearly income, *f* (%)					
$0–$4,999	215 (15)	26 (15)	189 (15)		
$5,000–$9,999	603 (42)	86 (51)	517 (41)		
$10,000–$14,999	344 (24)	33 (19)	311 (25)		
$15,000–$19,999	165 (12)	18 (11)	147 (12)		
$20,000–$29,999	59 (4)	4 (2)	55 (4)		
$30,000–$39,999	27 (2)	2 (1)	25 (2)		
$40,000–$49,999	4 (0.3)	0 (0)	4 (0.3)		
$50,000+	6 (0.4)	1 (0.6)	5 (0.4)	χ^2 (7, 7 = 1,557) = 7.75	0.355
Heart attack Wave 1, *f* (%)					
Yes	119 (8)	12 (7)	107 (8)		
Maybe	20 (1)	2 (1)	18 (1)		
No	1,415 (91)	172 (93)	1,243 (91)	χ^2 (2, N = 1,554) = 0.52	0.77
Stroke Wave 1, *f* (%)					
Yes	62 (4)	4 (2)	58 (4)		
Maybe	4 (0.3)	2 (1)	2 (0.1)		
No	1,488 (96)	180 (97)	1,308 (96)	χ^2 (2, N = 1,554) = 7.31	0.026
Hypertension Wave 1, *f* (%)					
Yes	615 (40)	44 (24)	571 (42)		
Maybe	23 (2)	3 (2)	20 (2)		
No	914 (59)	138 (75)	776 (57)	χ^2 (2, N = 1,552) = 22.13	<0.001
Cancer Wave 1, *f* (%)					
Yes	56 (4)	2 (1)	54 (4)		
Maybe	4 (0.3)	1 (0.5)	3 (0.2)		
No	1,497 (96)	183 (98)	1,314 (96)	χ^2 (2, N = 1,557) = 4.49	0.106
Diabetes Wave 1, *f* (%)					
Yes	294 (19)	23 (12)	271 (20)		
Maybe	63 (4)	10 (5)	53 (4)		
No	1,195 (77)	153 (82)	1,042 (76)	χ^2 (2, N = 1,552) = 6.49	0.039

ance in cognitive change into between- and within-person components. Therefore, the factor covariance matrix consists only of the intercept variance (ψ_{11}); the Level 1 residual variance is denoted as θ. 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 subjects at all four waves. Model fit was poor, as expected for a model that does not accommodate change in the number of correct responses (e.g., decreasing number of correct responses over time) (root mean square error of approximation = 0.26; 90% confidence interval = 0.25–0.27; and standardized root mean square residual = 0.59).

Wave of measurement was introduced by including a linear slope factor with loadings fixed to 0, 2, 5, and 7. Of key interest in this model were the mean

TABLE 2. Primary Results From Fitting Latent Growth Curve Models

	Model Values (SE)	χ^2	p	Model Fit: RMSEA, 90% CI	Model Fit: SRMR
Random intercept model					
Mean intercept	23.15 (0.11)				
Intercept variance	13.43 (0.67)				
Residual variance	15.68 (0.35)	1,167.26 (11, N = 1,557)	<0.001	0.260 (0.247–0.273)	0.586
Random intercept/random slope model					
Mean intercept	24.82 (0.10)				
Mean slope	−0.47 (0.02)				
Intercept variance	8.01 (0.62)				
Slope variance	0.29 (0.03)				
Intercept-slope covariance	0.43 (0.09)				
Residual variance	10.61 (0.29)	81.72 (8, N = 1,557)	<0.001	0.077 (0.062–0.092)	0.071
Smoking as a predictor of slopes, age as only covariate					
Age → slope	−0.03 (0.004)				
Smoking → slope	0.11 (0.06)	89.17 (12, N = 1,557)	<0.001	0.064 (0.052–0.077)	0.052
Smoking as a predictor of slopes, all covariates					
Age → slope	−0.04 (0.004)				
Education → slope	−0.004 (0.01)				
Sex → slope	−0.04 (0.04)				
Nativity → slope	0.02 (0.04)				
Heart attack → slope	0.03 (0.04)				
High blood pressure → slope	0.01 (0.02)				
Cancer → slope	0.01 (0.06)				
Diabetes → slope	0.02 (0.03)				
income → slope	−0.02 (0.02)				
Smoking → slope	0.13 (0.06)	110.58 (30, N = 1,557)	<0.001	0.042 (0.033–0.050)	0.024

Notes: RMSEA: root mean square error of approximation; SRMR: standardized root mean square residual.

intercept (α_1), mean slope (α_2), and intercept and slope variances ($\hat{\psi}_{11}$ and $\hat{\psi}_{22}$), and covariance ($\hat{\psi}_{21}$) (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%).[31] 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,[3] 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.[33] Oxidative stress is evident in the pathogenesis of AD and may cause neuron degeneration.[34] Cigarette smoke contains free radicals[35] and is involved in the generation of oxidative stress.[36] Furthermore, smokers tend to have both a lower dietary intake and circulation of antioxidants that neutralize free radicals.[37]

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,[38] 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.[38]

Smoking may also affect cognition and the brain due to indirect effects on other conditions such as lung functioning.[33] For example, smoking has been shown to cause lung injury that leads to chronic obstructive pulmonary disease.[38] Poor lung functioning is associated with both poorer cognitive functioning and brain atrophy.[39] Smoking may interact with other risk factors such as alcohol consumption and genetics (e.g., apolipoprotein E gene) that are associated with increased CD.[33]

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.[33] In individuals with normal neurologic and cognitive status at baseline, smoking has been shown to accelerate worsening white matter grade,[40] 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.[41,42] On the other hand, it is important to note that others have not detected an effect of smoking on total brain atrophy.[43] 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.[44]

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.[3,6] 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.[32]

Despite the many potential benefits of smoking cessation, there has been more focus on offering smoking cessation programs to young and middle-aged adults[45] and to non-Hispanics.[46] 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.[47] Additionally, older smokers may be unaware that there are significant health benefits of smoking cessation late in life.[48] Studies of community samples have found the cessation rate among older adults to be 10%.[49] Importantly, when offered the tools they need, older smokers quit smoking at rates comparable with those of younger smokers.[48] In particular, tailoring cessation programs in ways that are appropriate to age and ethnicity/culture has been effective in some studies for older adults[50] and Hispanics.[51]

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 nonsmokers), 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[52] 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.

This work was supported in part by grant RO1 AG10939 from the National Institute on Aging; the Center for Population Health and Health Disparities at the University of Texas Medical Branch; and the Robert Wood Johnson Network for Multicultural Research on Health and Health Care at UCLA.

References

1. Centers for Disease Control and Prevention (CDC). Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. MMWR Morb Mortal Wkly Rep 2002; 51:300–303
2. LaCroix AZ, Lang J, Scherr P, et al: Smoking and mortality among older men and women in three communities. N Engl J Med 1991; 324:1619–1625
3. Anstey KJ, von Sanden C, Salim A, et al: Smoking as a risk factor for dementia and cognitive decline: a meta-analysis of prospective studies. Am J Epidemiol 2007; 166:367–378
4. Deary IJ, Pattie A, Taylor MD, et al: Smoking and cognitive change from age 11 to age 80. J Neurol Neurosurg Psychiatry 2003; 74:1006–1007
5. Reitz C, Luchsinger J, Tang MX, et al: Effect of smoking and time on cognitive function in the elderly without dementia. Neurology 2005; 65:870–875
6. Richards M, Jarvis MJ, Thompson N, et al: Cigarette smoking and cognitive decline in midlife: evidence from a prospective birth cohort study. Am J Public Health 2003; 93:994–998
7. Public Policy Institute of California: The Economic Progress of Mexican Americans. Research Brief. CA, Public Policy Institute of California, 2002
8. Ostchega Y, Harris TB, Hirsch R, et al: The prevalence of functional limitations and disability in older persons in the US: data from the National Health and Nutrition Examination Survey III. J Am Geriatr Soc 2000; 48:1132–1135
9. Morgenstern LB, Smith MA, Lisabeth LD, et al: Excess stroke in Mexican Americans compared with non-Hispanic Whites: the Brain Attack Surveillance in Corpus Christi Project. Am J Epidemiol 2004; 160:376–383
10. Sachs-Ericsson N, Blazer DG: Racial differences in cognitive decline in a sample of community-dwelling older adults: the mediating role of education and literacy. Am J Geriatr Psychiatry 2005; 13:968–975

11. Knopman D, Boland LL, Mosley T, et al: Cardiovascular risk factors and cognitive decline in middle-aged adults. Neurology 2001; 56:42–48
12. DeNavas-Walt C, Proctor BD, Smith J: Current Population Reports, P60–233, Income, Poverty, and Health Insurance Coverage in the United States: 2006. Washington, DC, US Government Printing Office, 2007
13. Houston TK, Scarinci IC, Person SD, et al: Patient smoking cessation advice by health care providers: the role of ethnicity, socioeconomic status, and health. Am J Public Health 2005; 95:1056–1061
14. Broe GA, Creasey H, Jorm AF, et al: Health habits and risk of cognitive impairment and dementia in old age: a prospective study on the effects of exercise, smoking and alcohol consumption. Aust N Z J Public Health 1998; 22:621–623
15. Chen WT, Wang PN, Wang SJ, et al: Smoking and cognitive performance in the community elderly: a longitudinal study. J Geriatr Psychiatry Neurol 2003; 16:18–22
16. Whittington JE, Huppert FA: Smoking and cognitive decline. Hum Psychopharmacol 1997; 12:467–480
17. Luchsinger JA, Reitz C, Honig LS, et al: Aggregation of vascular risk factors and risk of incident Alzheimer disease. Neurology 2005; 65:545–551
18. Merchant C, Tang MX, Albert S, et al: The influence of smoking on the risk of Alzheimer's disease. Neurology 1999; 52:1408–1412
19. Guzmán B: The Hispanic Population. Census 2000 Brief. Washington, DC, U.S. Census Bureau, 2001
20. Markides KS, Rudkin L, Angel RJ, et al: Health status of Hispanic elderly in the United States, in Racial and Ethnic Differences in Late Life Health in the United States. Edited by Martin LG, Soldo BJ, Foote K. Washington, DC, National Academy Press, 1997, pp 285–300
21. Folstein MF, Folstein SE, McHugh PR: "Mini-mental state." A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975; 12:189–198
22. Preacher KJ, Wichman AL, MacCallum RC, et al: Latent Growth Curve Modeling. Thousand Oaks, CA, Sage Publications, 2008
23. Anstey K, Christensen H: Education, activity, health, blood pressure and apolipoprotein E as predictors of cognitive change in old age: a review. Gerontology 2000; 46:163–177
24. Heller PL, Briones DF, Schiffer RB, et al: Mexican-American ethnicity and cognitive function: findings from an elderly southwestern sample. J Neuropsychiatry Clin Neurosci 2006; 18:350–355
25. Sachs-Ericsson N, Joiner T, Plant EA, et al: The influence of depression on cognitive decline in community-dwelling elderly persons. Am J Geriatr Psychiatry 2005; 13:402–408
26. Patrick DL, Cheadle A, Thompson DC, et al: The validity of self-reported smoking: a review and meta-analysis. Am J Public Health 1994; 84:1086–1093
27. Dufouil C, Clayton D, Brayne C, et al: Population norms for the MMSE in the very old: estimates based on longitudinal data. Mini-Mental State Examination. Neurology 2000; 55:1609–1613
28. Kehoe R, Wu SY, Leske MC, et al: Comparing self-reported and physician-reported medical history. Am J Epidemiol 1994; 139: 813–818
29. Okura Y, Urban LH, Mahoney DW, et al: Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure. J Clin Epidemiol 2004; 57:1096–1103
30. Muthén LK, Muthén BO: Mplus User's Guide. Los Angeles, CA, Muthén & Muthén, 1998–2007
31. Moser JW, Langwell K: Differences By Race/Ethnicity in Smoking Behavior and Smoking Cessation of the Medicare Population. Baltimore, Centers for Medicare & Medicaid Services, 2002
32. Sachs-Ericsson N, Schmidt NB, Zvolensky MJ, et al: Smoking cessation behavior in older adults by race and gender: the role of health problems and psychological distress. Nicotine Tob Res 2009; 11:433–443
33. Swan GE, Lessov-Schlaggar CN: The effects of tobacco smoke and nicotine on cognition and the brain. Neuropsychol Rev 2007; 17:259–273
34. Smith MA, Rottkamp CA, Nunomura A, et al: Oxidative stress in Alzheimer's disease. Biochim Biophys Acta 2000; 1502:139–144
35. Church DF, Pryor WA: Free-radical chemistry of cigarette smoke and its toxicological implications. Environ Health Perspect 1985; 64:111–126
36. Traber MG, van der Vliet A, Reznick AZ, et al: Tobacco-related diseases. Is there a role for antioxidant micronutrient supplementation? Clin Chest Med 2000; 21:173–187
37. Alberg A: The influence of cigarette smoking on circulating concentrations of antioxidant micronutrients. Toxicology 2002; 180: 121–137
38. The 2004 United States Surgeon General's Report: The health consequences of smoking. N S W Public Health Bull 2004; 15:107
39. Sachdev PS, Anstey KJ, Parslow RA, et al: Pulmonary function, cognitive impairment and brain atrophy in a middle-aged community sample. Dement Geriatr Cogn Disord 2006; 21:300–308
40. Longstreth WT Jr, Arnold AM, Beauchamp NJ Jr, et al: Incidence, manifestations, and predictors of worsening white matter on serial cranial magnetic resonance imaging in the elderly: the Cardiovascular Health Study. Stroke 2005; 36:56–61
41. Akiyama H, Meyer JS, Mortel KF, et al: Normal human aging: factors contributing to cerebral atrophy. J Neurol Sci 1997; 152: 39–49
42. Meyer JS, Rauch GM, Crawford K, et al: Risk factors accelerating cerebral degenerative changes, cognitive decline and dementia. Int J Geriatr Psychiatry 1999; 14:1050–1061
43. Enzinger C, Fazekas F, Matthews PM, et al: Risk factors for progression of brain atrophy in aging: six-year follow-up of normal subjects. Neurology 2005; 64:1704–1711
44. Swan GE, DeCarli C, Miller BL, et al: Biobehavioral characteristics of nondemented older adults with subclinical brain atrophy. Neurology 2000; 54:2108–2114
45. Cox JL: Smoking cessation in the elderly patient. Clin Chest Med 1993; 14:423–428
46. Cokkinides VE, Halpern MT, Barbeau EM, et al: Racial and ethnic disparities in smoking-cessation interventions: analysis of the 2005 National Health Interview Survey. Am J Prev Med 2008; 34:404–412
47. Riegel B, Bennett JA: Cardiovascular disease in elders: is it inevitable? J Adult Dev 2000; 7:101–111
48. Donze J, Ruffieux C, Cornuz J: Determinants of smoking and cessation in older women. Age Ageing 2007; 36:53–57
49. Salive ME, Cornoni-Huntley J, LaCroix AZ, et al: Predictors of smoking cessation and relapse in older adults. Am J Public Health 1992; 82:1268–1271
50. Rimer BK, Orleans CT, Fleisher L, et al: Does tailoring matter? The impact of a tailored guide on ratings and short-term smoking-related outcomes for older smokers. Health Educ Res 1994; 9:69–84
51. Munoz RF, Marin BV, Posner SF, et al: Mood management mail intervention increases abstinence rates for Spanish-speaking Latino smokers. Am J Community Psychol 1997; 25:325–343
52. Dallongeville J, Marecaux N, Fruchart J-C, et al: Cigarette smoking is associated with unhealthy patterns of nutrient intake: a meta-analysis. J Nutr 1998; 128:1450–1457