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Smoking increases risk for cognitive decline among community-dwelling older Mexican Americans

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Abstract

Objectives—Few studies have investigated smoking and cognitive decline among older Mexican Americans. In the current study we 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 4 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 4 data collection points with the Mini-Mental Status 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 non-smokers.

Conclusions—Smoking increases risk for cognitive decline 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.

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Keywords

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 US (1,2). Moreover, smoking has been shown in some studies to accelerate the rate of cognitive decline (CD; (3–6). While 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, 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 the current 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 to Caucasians. Mexican Americans have fewer years of education (7), greater physical functioning problems (8), and greater incidence of stroke (9) than Caucasians, all of which have been linked to CD (10,11). This may be a result of low socioeconomic status (SES), lifestyle factors, or reduced access to preventive health care compared to other US 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 to individuals who have never smoked, current smokers have an increased risk of CD, as well as Alzheimer's 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 non-smokers, 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 non-demented 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 non-demented 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 papers, 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 papers, 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 and colleagues (5) examined measures of memory performance and found that elderly smokers experienced a faster decline compared to non-smokers. 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 US) (19), to date, the effects of smoking on CD have not been studied prospectively in this subgroup. Therefore, in the current 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 (H-EPESE) (20). We employ several methods to increase our ability to detect the influence of smoking on CD including (a) using prospective data in which we followed participants for a seven-year period, (b) using a continuous measure of global cognitive functioning, the Mini-Mental Status Examination (MMSE; (21), that would be sensitive to general changes in cognitive functioning over time, (c) obtaining a large sample of Mexican Americans age 65 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, the current study includes four waves of cognitive functioning scores, and the data are analyzed using latent growth curve modeling (LGM; (22). LGM has several advantages. First, it allows for an examination of growth in cognitive scores (or growth in errors) over several occasions. Moreover, LGM allows for an examination of group differences between smokers and non-smokers in the rate of change in cognitive functioning with each year of aging (rather than the arbitrary date of data collection).

Methods

Participants

The H-EPESE 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 seven years at four separate waves: baseline interview (1993–1994), two-year follow-up (1995–1996), five-year follow-up (1998–1999), and seven-year follow-up (2000–2001). The sampling strategy and methods of the study have been described elsewhere (20).

The baseline survey consisted of a sample of 3050 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 1557 individuals at wave 4. Importantly, there were substantial age differences at baseline between the smokers and non-smokers who were not retained to wave 4. Not surprisingly, among this group, smokers were younger than the non-smokers ($M=72.6$ years, $SD=5.7$ versus $M=74.8$, $SD=7.7$ years), $F(1,1549) = 14.9$, $p<.001$), likely due to the premature death of smokers, making direct comparisons on cognitive

functioning between smokers and non-smokers difficult. That is, the selective attrition of smokers (due to 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 non-smokers) for whom we observed cognitive functioning over a seven-year period.

Control Variables

Several factors associated with CD among older adults were statistically controlled. These include age, gender, education, annual household income, nativity (US 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 if 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 to 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 range 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 $\alpha = .78$, Time 2 $\alpha = .80$, Time 3 $\alpha = .84$, Time 4 $\alpha = .81$.

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 upon 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, income). We expected smokers and non-smokers 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 non-smokers.

Results

Planned Analyses

We first provide descriptive statistics on key variables for smokers and non-smokers who survived to wave 4 (see 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 (see Table 2).

Descriptive Statistics

At baseline participants who were retained in the study to wave 4 ($N=1557$) were 38% male and 62% female. 11.9% of participants were smokers at wave 1. Consistent with previous research, smokers were more likely to be male than female (59% vs. 41%). At baseline, the average age of participants was 71.5 ($SD=5.5$), but overall, smokers were significantly younger than non-smokers ($M=69.9$, $SD=4.4$, 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), due to the younger age of smokers, health problems were actually greater among non-smokers than smokers. In uncontrolled analyses at baseline, non-smokers were more likely than smokers to have experienced several health problems including stroke (4% vs. 2%), hypertension (42% vs. 24%), and diabetes (20% vs. 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 4 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 variance in cognitive change into between- and within-person components. The factor covariance matrix, therefore, consists only of the intercept variance (ψ_{11}); the level-1 residual variance is denoted 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 = .46, indicating that 46% of the variability was between subjects). The mean intercept was 23.15 ($SE = .11$, Wald $z = 210.5$, $p < .001$) 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 (RMSEA) = .26, 90% Confidence Interval (CI) = (.25, .27); Standardized root mean-square residual (SRMR) = .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 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 $\hat{\alpha}_2 = .47$ ($SE = .02$, $z = 23.5$, $p < .001$) per year of age. We assessed the degree to which the rate of correct responses varied across individuals by freely estimating the slope variance ($\hat{\psi}_{22} = .29$) and the intercept-slope covariance ($\hat{\psi}_{21} = .43$), $\Delta\chi^2(df = 2) = 441.67$, $p < .001$; i.e., the slope variance and intercept-slope covariance were together significantly different from zero). 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 ($\hat{\gamma}_{smoking} = .13$, $SE = .06$, $z = 2.17$, $p = .037$), such that the rate of decrease in the number of correct responses was greater for smokers than for non-smokers. 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 1=smoker, 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 the current study we examined the relationship of smoking status to cognitive decline (CD) in a sample of older Mexican American adults. The prevalence of smoking in our sample (18.4% of males and 8% of females were current smokers) was slightly higher than that reported from other national data. Others have found that among Hispanic Medicare recipients, 12.7% of males and 6.6% of females 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 smoking predicted CD such that current smokers, compared to non-smokers, experienced a greater decline on a measure of cognitive functioning, the MMSE, over seven 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 US.

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). Finally, smoking may interact with other risk factors such as alcohol consumption and genetics (e.g., APOE 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 neurological and cognitive status at baseline, smoking has been shown to accelerate worsening white matter grade (40), leuko-araiosis, 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).

While some factors known to influence CD (e.g., genetics) cannot be changed, 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 to 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 the present 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 to 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 over the seven 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, that is, 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 towards smokers showing more CD than non-smokers ($p = .08$). 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 smoking participants, who died prematurely, long enough to document the changes in cognitive functioning related to smoking. Nonetheless, the current study's results may not generalize to the population as a whole.

Third, there are other variables associated with smoking and CD which were not measured in the current study and which may have enhanced the apparent association between smoking and CD. Specifically, health and lifestyle 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, the current study cannot identify the specific mechanisms by which smoking accelerates CD. Future investigations should employ more specific measures of smoking exposure that can quantify inhaled doses including smoking topography (e.g., puff volume, 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 sum, we found smoking to predict CD in older Mexican American adults. This finding is important because of the consequences for health care in Mexican Americans. Future research should focus on the specific needs of Hispanic elders in addressing smoking cessation.

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Table 1

Comparison of Smokers and Non-Smokers Retained to Wave 4 on Key Variables

	All Participants N=1557	Smokers n=186 (11.9%)	Non-Smokers n=1371 (88.1%)	Test Statistic	p- value
MMSE wave 1	M=24.7, SD=4.2	M=24.7, SD=4.0	M=24.7, SD=4.2	$F(1,1497)=0.00$	.975
MMSE wave 2	M=24.2, SD=4.5	M=24.5, SD=4.1	M=24.2, SD=4.6	$F(1,1378)=0.44$	.507
MMSE wave 3	M=22.8, SD=5.1	M=22.7, SD=4.9	M=22.8, SD=5.1	$F(1,1324)=0.01$	.905
MMSE wave 4	M=21.4, SD=6.4	M=21.2, SD=6.3	M=21.4, SD=6.4	$F(1,1555)=0.14$	.707
Age	M=71.5, SD=5.5	M=69.9, SD=4.4	M=71.7, SD=5.6	$F(1,1555)=17.08$	<.001
Sex				2-sided Fisher's Exact Test	<.001
Male	f=594 (38%)	f=109 (59%)	f=485 (35%)		
Female	f=963 (62%)	f=77 (41%)	f=886 (65%)		
Education	M=5.0, SD=3.9	M=4.8, SD=3.9	M=5.0, SD=3.9	$F(1,1532)=0.30$	.587
Nativity				2-sided Fisher's Exact Test	.581
US Born	899 (58%)	111 (60%)	788 (58%)		
Not US Born	658 (42%)	75 (40%)	583 (43%)		
Household Yearly Income				$\chi^2(7, N=1557)=7.75$	.355
\$0-\$4,999	f=215 (15%)	f=26 (15%)	f=189 (15%)		
\$5,000-\$9,999	f=603 (42%)	f=86 (51%)	f=517 (41%)		
\$10,000-\$14,999	f=344 (24%)	f=33 (19%)	f=311 (25%)		
\$15,000-\$19,999	f=165 (12%)	f=18 (11%)	f=147 (12%)		
\$20,000-\$29,999	f=59 (4%)	f=4 (2%)	f=55 (4%)		
\$30,000-\$39,999	f=27 (2%)	f=2 (1%)	f=25 (2%)		
\$40,000-\$49,999	f=4 (0.3%)	f=0 (0%)	f=4 (0.3%)		
\$50,000+	f=6 (0.4%)	f=1 (0.6%)	f=5 (0.4%)		
Heart Attack wave 1				$\chi^2(2, N=1554)=0.52$	.77

	All Participants N=1557	Smokers n=186 (11.9%)	Non-Smokers n=1371 (88.1%)	Test Statistic	p- value
Yes	f=119 (8%)	f=12 (7%)	f=107 (8%)		
Maybe	f=20 (1%)	F=2 (1%)	f=18 (1%)		
No	f=1415 (91%)	F=172 (93%)	f=1243 (91%)		
Stroke wave 1				$\chi^2(2, N=1554)=7.31$	.026
Yes	f=62 (4%)	F=4 (2%)	f=58 (4%)		
Maybe	f=4 (0.3%)	F=2 (1%)	F=2 (0.1%)		
No	f=1488 (96%)	F=180 (97%)	f=1308 (96%)		
Hypertension wave 1				$\chi^2(2, N=1552)=22.13$	<.001
Yes	f=615 (40%)	f=44 (24%)	f=571 (42%)		
Maybe	f=23 (2%)	f=3 (2%)	f=20 (2%)		
No	f=914 (59%)	f=138 (75%)	f=776 (57%)		
Cancer wave 1				$\chi^2(2, N=1557)=4.49$	.106
Yes	f=56 (4%)	f=2 (1%)	f=54 (4%)		
Maybe	f=4 (0.3%)	f=1 (0.5%)	f=3 (0.2%)		
No	f=1497 (96%)	f=183 (98%)	f=1314 (96%)		
Diabetes wave 1				$\chi^2(2, N=1552)=6.49$	.039
Yes	f=294 (19%)	f=23 (12%)	f=271 (20%)		
Maybe	f=63 (4%)	f=10 (5%)	f=53 (4%)		
No	f=1195 (77%)	f=153 (82%)	f=1042 (76%)		

Table 2

Primary Results from Fitting Latent Growth Curve Models

	Model Values (SE)	χ^2	p-value	Model Fit		
				RMSEA (90% CI)	SRMR	
Random Intercept Model		$\chi^2(11, N=1557) = 1167.26$	<.001	.260 (.247, .273)	.586	
Mean intercept	23.15 (.11)					
Intercept variance	13.43 (.67)					
Residual variance	15.68 (.35)					
Random Intercept / Random Slope Model		$\chi^2(8, N=1557) = 81.72$	<.001	.077 (.062, .092)	.071	
Mean intercept	24.82 (.10)					
Mean slope	-.47 (.02)					
Intercept variance	8.01 (.62)					
Slope variance	.29 (.03)					
Intercept-slope covariance	.43 (.09)					
Residual variance	10.61 (.29)					
Smoking as a Predictor of Slopes, Age as Only Covariate		$\chi^2(12, N=1557) = 89.17$	<.001	.064 (.052, .077)	.052	
age $\rightarrow$ slope	-.03 (.004)					
smoking $\rightarrow$ slope	.11 (.06)					
Smoking as a Predictor of Slopes, All Covariates		$\chi^2(30, N=1557) = 110.58$	<.001	.042 (.033, .050)	.024	
age $\rightarrow$ slope	-.04 (.004)					
education $\rightarrow$ slope	-.004 (.01)					
sex $\rightarrow$ slope	-.04 (.04)					
nativity $\rightarrow$ slope	.02 (.04)					
heart attack $\rightarrow$ slope	.03 (.04)					
high blood pressure $\rightarrow$ slope	.01 (.02)					
cancer $\rightarrow$ slope	.01 (.06)					
diabetes $\rightarrow$ slope	.02 (.03)					

Model Fit				
	Model Values (SE)	χ^2	p-value	RMSEA (90% CI) SRMR
income $\rightarrow$ slope	-.02 (.02)			
smoking $\rightarrow$ slope	.13 (.06)			