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The Association of Pipe and Cigar Use with Cotinine Levels, Lung Function and Airflow Obstruction: a Cross-sectional Study

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Abstract

Background—Cigarette smoking is the major cause of chronic obstructive pulmonary disease but studies on the contribution of other smoking techniques are sparse.

Objective—We hypothesized that pipe and cigar smoking was associated with elevated cotinine levels, decrements in lung function and increased odds of airflow obstruction.

Design—Cross-sectional study.

Setting—Population-based sample from six US communities.

Participants—The Multi-Ethnic Study of Atherosclerosis (MESA) recruited men and women ages 45–84 years without clinical cardiovascular disease.

Measurements—The MESA Lung Study measured spirometry following American Thoracic Society guidelines and urinary cotinine levels by immunoassay. Pipe-years and cigar-years were calculated as years from self-reported age of starting to quitting (or to current age among current users) × pipefuls or cigars per day.

Results—Of 3,528 participants, 8% reported pipe smoking (median 15 pipe-years), 11% reported cigar smoking (median 6 cigar-years), and 52% reported cigarette smoking (median 18 pack-years). Self-reported current pipe and cigar smokers had elevated urinary cotinine levels compared to never

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RESEARCH STATEMENTS

Protocol for the MESA Lung Study: available to interested readers by contacting Dr. Barr at rgb9@columbia.edu

Statistical Code: available to interested readers by contacting Dr. Barr at rgb9@columbia.edu Data: available as a limited access data set from the National Heart Lung and Blood Institute (www.mesa-nhlbi.org)

smokers. Pipe-years were associated with decrements in the forced expiratory volume in one second (FEV₁) and cigar-years were associated with decrements in the FEV₁ and the ratio of the FEV₁ to the forced vital capacity. Participants who smoked pipes or cigars had an increased odds of airflow obstruction whether they had also smoked cigarettes (Odds ratio 3.43; 95% CI: 1.75, 6.71; P<0.001) or had never smoked cigarettes (Odds ratio 2.31; 95% CI: 1.04, 5.11; P=0.039) compared to participants with no smoking history.

Limitations—Cross-sectional design.

Conclusions—Pipe and cigar smoking increased urinary cotinine levels and was associated with decrements in lung function and increased odds of airflow obstruction, even among participants who never smoked cigarettes.

INTRODUCTION

Cigarette smoking has decreased significantly in the United States since the 1960s. A sustained decline in cigarette smoking occurred in all age groups from a prevalence of 33% in 1983 to a prevalence of 19.8% in 2007 (1-3). Although less common than cigarette smoking, the prevalence of pipe and cigar smoking has increased substantially in the United States in recent years. Smoking of all types of cigars increased by 46.4% from 1993 to 1997 (4), and consumption of pipe and cigar tobacco increased by 28% and 8%, respectively, from 2002 to 2006 (5). In 2005, 3% of high school students smoked pipes and 13% smoked cigars. The prevalence of current pipe and cigar smoking in the US was 1% and 6%, respectively, in 2006 (5).

Cigarette smoking is the main cause of chronic obstructive pulmonary disease (COPD) (6,7), the fourth leading cause of death in the US (8). A large questionnaire-based cohort found an increased risk of COPD hospitalization and death among pipe smokers, as did a second cohort for cigar smokers (9,10). Both cohorts, however, relied upon hospital discharge ICD-9 codes that included COPD *and* asthma as the primary *or* secondary reason for hospitalization. COPD is defined by accelerated, age-related decline in lung function (7); however, no US studies have reported on the possible effects of cumulative pipe and cigar smoking on lung function, in part since few contemporary large US epidemiologic studies cohorts collected information on duration of pipe and cigar smoking.

We therefore examined if current pipe and cigar smoking resulted in biological absorption of tobacco smoke, assessed by urinary cotinine levels, in a large cohort study and tested the hypothesis that pipe and, separately, cigar smoking was associated with decrements in lung function and increased odds of airflow obstruction.

METHODS

Multi-Ethnic Study of Atherosclerosis

The Multi-Ethnic Study of Atherosclerosis (MESA) is a multicenter prospective cohort study to investigate the prevalence, correlates and progression of subclinical cardiovascular disease in individuals without clinical cardiovascular disease (11). In 2000-2002, MESA recruited 6,814 men and women ages 45-84 years old from six U.S. communities: Forsyth County, NC; Northern Manhattan and the Bronx, NY; Baltimore City and Baltimore County, MD; St. Paul, MN; Chicago, IL; and Los Angeles, CA. Exclusion criteria included clinical cardiovascular disease, weight > 300 lbs, pregnancy or impediment to long-term participation.

The MESA Lung Study enrolled 3,965 MESA participants of 4,484 selected who were sampled randomly among those who consented to genetic analyses, underwent baseline measures of endothelial function, and attended an examination during the MESA-Lung recruitment period

in 2004–2006 (99%, 89%, and 91% of the MESA cohort, respectively). Asians were over-sampled.

For the current cross-sectional study related to obstructive lung disease, we excluded 322 participants with a restrictive pattern of spirometry, defined as a forced vital capacity (FVC) less than the lower limit of normal (12) with a forced expiratory volume in one second (FEV_1)/FVC ratio above 0.70.

Assessment of Exposure to Pipe and Cigar Smoke

Self-administered items from the American Thoracic Society (ATS) questionnaire (13) were used to identify participants' smoking histories for pipes, cigars, and cigarettes. For pipes, participants were asked first, "Have you smoked at least 20 pipe-bowls in your lifetime?" If they answered in the affirmative, additional questions included, "How old were you when you first started smoking pipes?"; "On average, about how many pipe-bowls a day do/did you smoke?"; "Have you smoked a pipe within the last 30 days?" and, if relevant, "How old were you when you quit smoking pipes?" The same items inquired about cigar smoking and, separately, cigarette smoking. Twenty cigars and 100 cigarettes used as the threshold for ever smoking cigars and cigarettes, respectively.

Pipe-years were calculated as age of starting to quitting (or current age if current pipe smoker) $\times$ pipe-bowls per day. Cigar-years were calculated as self-reported age of starting to quitting (or current age if current cigar smoker) $\times$ cigars per day.

Cotinine Assessment

Cotinine measurements from urine collected on the same day as questionnaire information were performed by immunoassay (Immulite 2000 Nicotine Metabolite Assay; Diagnostic Products Corp., Los Angeles, CA) at the National Institute for Occupational Safety and Health Core Laboratory. Intra-assay coefficient of variation was 2.02% and the minimal detectable concentration was 10 ng/mL. Based on standard approach used for statistical calculation of undetectable levels of biomarkers, we assigned undetectable values of cotinine to 7.07 (14). Urinary cotinine was not corrected for creatinine clearance due to the generally healthy (mean serum creatinine 79.6 ± 17.7 μ mol/L) and multiethnic composition of the cohort.

Measurement of Confounders

Information on age, gender, race/ethnicity, educational attainment, medical history, occupational exposure to dust, fumes, or smoke, depth of inhalation of cigarettes, environmental tobacco smoke exposure and family history of emphysema was collected using standardized questionnaire items (13). Asthma was defined as self-report of physician-diagnosed asthma before age 45 years in order to avoid over-correction for COPD misdiagnosed as asthma above that age, which is common and differential by gender (15). Height and weight were measured at the time of the spirometry exam using calibrated scales and measures, and body mass index was calculated as weight (kg)/height (m)².

Pack-years of cigarette smoking was calculated as age of starting to quitting (or current age if current cigarette smoker) $\times$ (cigarettes per day/20). Cigarettes per day was assessed twice over a 4-year interval by the ATS questionnaire item, "On average, about how many cigarettes per day do you smoke?" (13) and by a second time item, "On the average of the entire time you smoked, how many cigarettes did you smoke per day?" The greater of the two measures were used in calculations.

Spirometry

Spirometry was conducted in accordance with the ATS/European Respiratory Society (ERS) recommended guidelines (16) with all participants performing at least three acceptable maneuvers. Tests were conducted using a dry-rolling-sealed spirometer and software that performed automated quality checks as maneuvers were performed (Occupational Marketing, Inc.). All spirometry exams were reviewed by one investigator and each test was graded for quality (17). Low-quality spirometry was defined as only one acceptable curve; participants with no acceptable curves were excluded.

Airflow obstruction was defined as the FEV₁/FVC ratio < the lower limit of normal (12), in accordance with recent recommendations (16).

Statistical Analysis

Differences in urinary cotinine levels between each self-reported current smoking technique and never smokers were tested with the Wilcoxon Rank Sum test. For this analysis, four participants who reported use of nicotine replacement therapy were excluded and participants who reported current use of more than one smoking technique were categorized hierarchically as described in the footnote to the figure.

For analyses of smoking technique, the cohort was categorized according to history of: 1) pipe or cigar smoking only, 2) cigarette smoking only, and 3) pipe or cigar and cigarette smoking. Decrements in lung function and the log odds of airflow obstruction were estimated for each category of smoking history compared to never smokers using linear regression and logistic regression. We modeled lung function as a function of age, age², sex, height, height² and race/ethnicity rather than using the percent of predicted due to known variation in the performance of NHANES III reference equations across race/ethnic groups in this cohort (17). Subsequent models were additionally adjusted for the following potential causal confounders, all of which are likely to affect lung function in a population-based cohort such as MESA: educational attainment, cigarette smoking status, pack-years, environmental tobacco smoke exposure, dust exposure, body mass index, asthma prior to age 45 years, and family history of emphysema. Missing data for covariates was handled with multiple imputation (18).

Analyses of the relationships of cumulative pipe smoking and cumulative cigar smoking to lung function and airflow limitation used a similar modeling approach. These analyses were performed in the entire cohort, repeated after the exclusion of participants who smoked cigarettes only, and restricted to participants who never smoked cigarettes in order to minimize residual confounding by cigarette smoking.

Analyses were performed in SAS version 9.2 (SAS Institute, Cary, NC); R (version 2.3.1; R Foundation, Vienna, Austria) was used to generate plots.

Role of Funding Source

The study was funded by the National Heart, Lung and Blood Institute (NHLBI) and designed by the MESA Lung investigators in collaboration with NHLBI staff. The protocol was approved by the Institutional Review Boards of all collaborating institutions and the NHLBI. The authors, together with other MESA investigators, collected and analyzed the data, vouch for the data and analysis, and wrote and submitted the paper for publication. NHLBI staff routinely monitored study performance and participated in the internal review of this manuscript prior to submission.

RESULTS

Figure 1 shows the recruitment of the 3,965 participants in the MESA Lung Study and subsequent exclusions. The 3,528 participants in the analysis of smoking technique had a mean age of 66 ± 10 years and were 49% male, 35% non-Hispanic White, 26% African-American, 22% Hispanic, and 17% Chinese-American. Eight percent reported ever pipe smoking (median 18 pipe-years [interquartile range 6, 36]), mostly in the past. Eleven percent reported ever cigar smoking (median 6 cigar-years [interquartile range 0, 26]), of whom approximately one fifth smoked cigars currently. Fifty-two percent reported ever smoking cigarettes and nine percent were current cigarette smokers. Of 484 participants with a history of pipe or cigar smoking, 88% also reported a history of cigarette smoking.

Table 1 shows the study sample stratified by history of pipe or cigar smoking only, cigarette smoking only, and pipe or cigar and cigarette smoking. Participants with a history of pipe or cigar smoking were more likely to be male, white or African-American, and of higher education attainment.

Cotinine Levels by Current Smoking Technique

To evaluate the biological plausibility of effects of pipe and cigar smoking in the lung, we assessed urine cotinine levels according to self-reported smoking technique. Median cotinine levels were less than 10 ng/mL among never smokers ($n=1620$), 43 ng/mL among current cigar smokers ($n=47$), 1324 ng/mL among current pipe smokers ($n=6$), and 4304 ng/mL among current cigarette smokers ($n=330$) (all $P<0.0001$ vs. never smokers). Figure 2 shows boxplots of urine cotinine levels by type of current smoking technique. Median cotinine levels were also elevated among the 16 current cigar smokers (11 ng/mL; interquartile range, 7, 206; $P<0.001$) and 2 current pipe smokers (164 ng/mL; interquartile range, 81, 248; $P=0.002$) who denied ever smoking cigarettes.

Lung Function, Airflow Obstruction and Smoking Technique

Table 2 shows decrements in lung function and odds ratios for airflow obstruction according to categories of smoking technique. There were consistent decrements in the FEV_1 among participants with a history of pipe or cigar smoking only, cigarette smoking only, and pipe or cigar and cigarette smoking compared to never smokers. The decrement was modest and not statistically significant among the 55 participants who smoked pipes or cigars only, greater and statistically significant among the much larger group who smoked cigarettes only, and greatest among those who smoked pipes or cigars in addition to cigarettes. A similar pattern was evident for the FEV_1/FVC ratio.

The odds of airflow obstruction were increased among participants who smoked pipes or cigars only (Odds ratio 2.31; 95% CI: 1.04, 5.11; $P=0.039$) compared to never smokers, and were greatest among participants who smoked pipes or cigars in addition to cigarettes.

Cumulative Pipe Smoking

Pipe-years were inversely associated with the FEV_1 in age-race-sex-height-adjusted analyses in the entire sample (Table 3). This association persisted in the fully adjusted model and after exclusion of participants who smoked cigarettes only. Upon restriction to participants who never smoked cigarettes, effect estimates were of larger magnitude but did not attain statistical significance. Consistent although generally non-significant patterns were evident for the FEV_1/FVC ratio. The odds ratio for airflow obstruction was increased in all analyses, statistically significantly so in fully adjusted analyses among never cigarette smokers (Odds ratio 2.13; 95% CI 1.13, 4.0; $P=0.02$).

The magnitude of the decrement in lung function related to pipe smoking was considerable larger among heavy pipe smokers. For example, the mean FEV₁ among the 64 participants in the full sample with 50 or more pipe-years was 150 ml lower (95% CI -257, -43; P=0.006) than participants who had never smoked pipes in fully adjusted analyses and the mean FEV₁/FVC ratio was 2.1% lower (95% CI: -4.1, -0.1; P=0.042).

There was no evidence that these associations were modified by cigarette smoking in the entire sample (e.g., the P-value for the interaction of pack-years with pipe-years for the FEV₁ was P=0.22). Since most cigar smokers in this cohort were white and male, we repeated analyses restricted to these groups and found consistent associations (data not shown).

Cumulative Cigar Smoking

In the entire sample, greater cigar-years were associated with a decrement in the FEV₁, a decrement in the FEV₁/FVC ratio and an increased odds ratio for airflow obstruction in age-race-sex-height-adjusted analyses (Table 4). Fully adjusted models yielded consistent associations, which remained statistically significant for the FEV₁/FVC ratio. Results were similar after the exclusion of participants who smoked only cigarettes but were attenuated after restriction to participants who never smoked cigarettes, and there was evidence that the association of cigar-years to the FEV₁ in the entire sample was modified by pack-years of cigarette smoking (P-interaction <0.001).

The associations were qualitatively consistent among whites and men (data not shown).

Cumulative Cigarette Smoking

By comparison, cigarette pack-years were inversely associated with the FEV₁ (-46 ml per 10 pack-years; 95% CI -54, -38; P<0.001) and the FEV₁/FVC ratio (-0.8% per 10 pack-years; 95% CI -1.0, -0.7; P<0.001) and were associated with increased airflow obstruction (Odds ratio 1.20; 95% CI 1.17, 1.23; P<0.001) in the entire sample in fully adjusted models.

Sensitivity Analyses

There was no evidence for effect modification of associations of pipe-years and cigar-years with lung function by race/ethnicity or gender. Results were similar in sensitivity analyses in which pack-years of cigarettes were increased by 25% selectively among those who reported no current smoking status but had cotinine levels > 100 ng/mL and after the exclusion of participants with low quality spirometry (data not shown).

DISCUSSION

Pipe and cigar smoking was associated with an obstructive pattern of spirometry characterized by decrements in the FEV₁ and FEV₁/FVC ratio and by an increased risk of airflow obstruction in this large, multiethnic study. These results, together with the extensive literature on the effects of tobacco smoke on the development of COPD and the increase in cotinine levels among current pipe and cigar smokers in this cohort, suggest that pipe and cigar smoking produce a measureable increase in the risk of COPD.

Tobacco smoke is the major cause of COPD (6,19). However, it is not entirely clear whether pipe and cigar smoke may damage the lung via the same mechanism as cigarettes. Some people who smoke pipes and cigars claim to not inhale, or at least inhale less, than cigarette smokers. The elevated cotinine levels in the current study, however, belie this notion and provide a biological measure of nicotine exposure. Our results are also consistent with prior observations of elevated carboxyhemoglobin saturations in pipe and cigar smokers, particularly among former cigarette smokers who may be more likely to inhale than never cigarette smokers (20,

21). Cigar smoke particles have been shown to be deposited in the lung, regardless of report of inhalation (22). These findings strongly suggest that tobacco smoke from cigars and particularly from pipes is absorbed systemically.

Cotinine levels among pipe and cigar smokers were lower than among people who smoke cigarettes; however, relative differences in cotinine levels reflect differences in nicotine absorption but not necessarily exposure to harmful products of tobacco smoke. Most cigarettes contain filters whereas pipes and cigars are unfiltered and may therefore yield a higher dose of tobacco smoke for the same dose of nicotine. Furthermore, pipe and cigar smoke exposes the smoker to more side-streamed smoke, which may be particularly harmful (23,24). However, similar effects of pipe and cigar to cigarette smoking on CYP1A2 activity, the major pathway activating carcinogens from tobacco smoke, and DNA adduct levels have not been found, possibly due to differences in inhalation (25).

Despite recent increases in pipe and cigar smoking, prior studies on pipe and cigar smoking and lung function are few. We conducted an English-language MEDLINE search through July, 2009 to identify studies that examined the association between pipe and/or cigar smoking and lung function. Consistent with our results, pipe and cigar smokers in the Copenhagen City Heart Study had an increased rate of decline in lung function compared to non-smokers (26-28), as well as an increased risk of mucous hypersecretion (28). The Copenhagen City Heart Study demonstrated this association in a relatively homogeneous European sample, whereas the current study extends these findings to a multiethnic sample in the US.

Other studies of pipe and cigar smoking and COPD have relied upon ICD-based measures. In addition to the findings in the two US cohorts (9,10), pipe and cigar smoking were associated with an increased mortality rate from emphysema and chronic bronchitis in Sweden (29), and British men who switched from cigarettes to pipes and cigars had an increased risk of dying from COPD, ischemic heart disease or lung cancer compared to those who quit smoking all together (20). A prospective cohort study in the Netherlands recently showed that pipe and cigar smoking was associated with reduce life expectancy, although to a lesser extent than cigarette smoking (30). Other studies investigating the effects of pipe and cigar smoking are limited to overall mortality, coronary artery disease, and risk of lung cancer (31,32).

Major strengths of this study include the large, multi-ethnic, population-based sample with standardized measures of spirometry, cotinine and pipe and cigar smoking. The major limitations are the cross-sectional design, the retrospective ascertainment of cumulative pipe and cigar smoking, and the relatively small proportion of participants who smoked pipes or cigars but not cigarettes. Cross-sectional studies of lung function can yield different results from longitudinal studies and are potentially subject to selection bias. However, our cross-sectional findings are consistent with the longitudinal results from Copenhagen (26-28). Confounding, particularly by cigarette smoking, may have contributed to the observed associations. However, we controlled for precise measures of the major potential confounders, in addition to performing analyses stratified by smoking history, restricting to participants who had never smoked cigarettes, and using up-weighting estimates of cigarette pack-years, all of which yielded consistent results.

Misclassification of smoking technique is unlikely to have accounted for the differences in urinary cotinine levels between cigar and pipe smokers who never smoked cigarettes. A small number of the 1,620 participants who reported never smoking had detectable levels of cotinine; almost all of these were likely due to environmental tobacco smoke exposure (i.e., levels <100 mg/dl) but only 7 were unequivocally consistent with active smoking. Given the generally healthy cohort and generally subclinical decrements in lung function, recall bias of smoking history is unlikely to have been substantial or differential with respect to the outcomes.

The proportion of participants who smoked pipes or cigars but not cigarettes was small. Effect estimates in this group were therefore relatively imprecise; however, results for pipe or cigar smokers as a group and pipe-years as a cumulative measure were highly consistent with those from the entire sample and statistically significant for airflow obstruction. The interaction term for cigar-years suggested a possible greater impact of cigar smoking among participants who had ever smoked cigarettes compared to those who never smoked cigarettes, which further research will have to refute or confirm.

In conclusion, pipe and cigar smoking was associated with decrements in lung function consistent with obstructive lung disease. These findings, together with increased cotinine levels in current pipe and cigar smokers, suggest that long-term pipe and cigar smoking may damage the lungs and contribute to the development of COPD. Practitioners should consider pipe and cigar smoking a risk factor for COPD and counsel cessation of pipe and cigar smoking regardless of smoking history.

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Figure 1.
Recruitment of the MESA Lung Study and Exclusions for the Current Study Sample
MESA = Multi-Ethnic Study of Atherosclerosis

Figure 2.

Urinary cotinine levels by self-report of current smoking technique

Thick lines = median values of cotinine (log scale); box height = interquartile range; box width is proportional to sample size; upper and lower horizontal thin bars = maximum and minimum values excluding outliers; small circles = outlier values. $P < 0.001$ for all smoking techniques vs. never smokers.

Never smoked (n=1,620): reported smoking less than 100 cigarettes, less than 20 pipe-bowls and less than 20 cigars in life. **Cigar smokers** (n=47): reported current cigar use but denied current pipe or cigarette smoking. **Pipe Smokers** (n=6): reported current pipe use but denied current cigarette smoking. **Cigarette smokers** (n=330): reported current cigarette smoking.

Table 1

Characteristics of participants stratified by smoking technique.

	History of Smoking			
	None*	Pipes or Cigars only [†]	Cigarettes Only	Pipes or Cigars and Cigarettes [‡]
	n=1620	n=56	N=1424	n=428
Age, mean (SD), years	65 (10)	65 (8)	66 (10)	67 (10)
Male gender, %	34	95	51	93
Race/ Ethnicity %				
White	28	64	35	57
African-American	22	27	30	26
Hispanic	24	9	24	13
Chinese-American	25	0	11	4
Educational Attainment, %				
< High school	18	0	18	8
High school graduate	19	5	19	15
Some college	25	18	31	29
College graduate	19	25	16	20
> Bachelor's degree	19	52	16	28
Height, mean (SD), cm	163 (10)	174 (9)	167 (10)	174 (8)
Body mass index, mean (SD), Kg/m ²	27.5 (5.2)	28.6 (3.7)	28.3 (5.7)	28.6 (4.6)
Cigarette Smoking Characteristics				
Cigarette smoking status %				
Never	100	100	--	--
Former	--	--	82	84
Current	--	--	18	16
Pack-years of smoking, median (IQR) ^{//}	--	--	17 (6, 35)	20 (7, 39)
0-10 pack-years, %	--	--	34	32
10-50 pack-years, %	--	--	52	52
>50 pack-years, %	--	--	14	16
Pipe Smoking Characteristics				
Pipe smoking status %				
Never	100	38	100	38
Former	--	59	--	57
Current	--	4	--	4
Age started pipe smoking, median (IQR) ^{//}	--	24 (20, 30)	--	23 (20, 30)
Age at last pipe among quitters, median (IQR) ^{//}	--	30 (25, 40)	--	35 (27, 45)
Number of pipe bowls smoked per day, median (IQR) ^{//}	--	1 (0, 2)	--	2 (1, 4)
Pipe-years, median (IQR) ^{//}	--	0 (0, 12)	--	19 (4, 50)
Missing pipe-years, %	--	26	--	7
0-10 pipe-years, %	--	51	--	36

	History of Smoking			
	None [*]	Pipes or Cigars only [†]	Cigarettes Only	Pipes or Cigars and Cigarettes [‡]
10-50 pipe-years, %	--	17	--	34
>50 pipe-years, %	--	6	--	23
Cigar Smoking Characteristics				
Cigar smoking status %				
Never	100	25	100	20
Former	--	46	--	65
Current	--	29	--	14
Age started cigar smoking, median (IQR) ^{//}	--	24 (20, 40)	--	25 (20, 30)
Age at last cigar among quitters, median (IQR) ^{//}	--	35 (28, 55)	--	42 (33, 50)
Number of cigars smoked per day, median (IQR) ^{//}	--	0 (0, 1)	--	1 (0, 2)
Cigar-years, median (IQR) ^{//}	--	0 (0, 10)	--	7 (0, 30)
Missing cigar-years, %	--	10	--	12
0-10 cigar-years, %	--	64	--	46
10-50 cigar-years, %	--	16	--	29
>50 cigar-years, %	--	10	--	12
Environmental tobacco smoke exposure, %	35	20	51	50
Family history of emphysema, %	4	0	4	6
Asthma before age 45 years, %	8	11	7	10
Exposure to dust, %	30	43	40	47
FEV ₁ percent of predicted, ** mean (SD)	98 (16)	95 (13)	94 (19)	92 (18)
FVC percent of predicted, ** mean (SD)	98 (14)	98 (11)	97 (16)	97 (14)
FEV ₁ /FVC ratio, mean (SD)	76 (7)	73 (7)	73 (9)	71 (10)

SD = standard deviation; IQR = interquartile range; FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity.

* Smoked less than 100 cigarettes, less than 20 pipe-bowls and less than 20 cigars in life.

[†] Smoked at least 20 pipe-bowls or at least 20 cigars but less than 100 cigarettes in life. Among these 56 participants, 21 reported a history of smoking both cigars and pipes.

[‡] Smoked at least 20 pipe-bowls or at least 20 cigars in life and smoked at least 100 cigarettes in life.

^{//} Among participants with a history of smoking the given tobacco product.

** Predicted values calculated using NHANES III reference equations with 0.88 correction factor for Chinese-Americans (12, 17).

Table 2

Decrement in lung function and odds ratio for airflow obstruction among pipe or cigar smokers, cigarette smokers, and pipe or cigar and cigarette smokers.

	History of Smoking				
	None* n=1,620	Pipes or Cigars only [†] n=56	Cigarettes Only N=1,424	Pipes or Cigars and Cigarettes [‡] n=428	P-value
FEV ₁ , mL					
Mean	2314	2299	2217	2187	
Mean Difference – Model 1	0 (ref)	-50 (-146, 46)	-98 (-128, -68)	-150 (-199, -100)	<0.001
Mean Difference – Model 2	0 (ref)	-39 (-134, 55)	-82 (-142, -22)	-160 (-266, -55)	0.003
FVC, mL					
Mean	3032	3075	3001	3017	
Mean Difference – Model 1	0 (ref)	22 (-93, 137)	-32 (-66, 2.4)	-28 (-85, 29)	0.33
Mean Difference – Model 2	0 (ref)	33 (-81, 146)	-20 (-89, 48)	-7 (-129, 115)	0.91
FEV ₁ /FVC, %					
Mean	76.5	73.3	73.3	71.1	
Mean Difference – Model 1	0 (ref)	-1.4 (-3.3, 0.5)	-2.6 (-3.2, -2.0)	-3.5 (-4.4, -2.5)	<0.001
Mean Difference – Model 2	0 (ref)	-1.4 (-3.2, 0.5)	-2.2 (-3.4, -1.1)	-4.3 (-6.3, -2.3)	<0.001
Airflow obstruction					
Prevalence, %	7.6	18.2	16.1	20.6	
Odds ratio – Model 1	1 (ref)	2.24 (1.04, 4.80)	2.15 (1.68, 2.74)	2.42 (1.67, 3.52)	<0.001
Odds ratio – Model 2	1 (ref)	2.31 (1.04, 5.11)	2.01 (1.31, 3.08)	3.43 (1.75, 6.71)	<0.001

FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity; 95% Confidence intervals are shown in the parenthesis; results are bolded when p-value is less than 0.05.

Model 1: adjusted for age, race, gender and height.

Model 2: adjusted for variables in Model 1 plus cigarette smoking status and pack-years, environmental tobacco smoke exposure, body mass index, education, family history of emphysema, asthma before 45 years of age, and dust exposure.

* Smoked less than 100 cigarettes, less than 20 pipe-bowls and less than 20 cigars in life.

[†] Smoked at least 20 pipe-bowls or at least 20 cigars but less than 100 cigarettes in life. Among these 56 participants, 21 reported a history of smoking both cigars and pipes.

[‡] Smoked at least 20 pipe-bowls or at least 20 cigars in life and smoked at least 100 cigarettes in life.

Table 3

Decrement in lung function and odds ratios for airflow obstruction per 10 pipe-years in the entire sample, excluding participants who smoked cigarettes only, and restricted to participants who never smoked cigarettes.

	Entire Sample		Excluding Participants who Smoked Cigarettes Only		Restricted to Participants who Never Smoked Cigarettes	
Number of participants	3,500	P-value	2,097	P-value	1,675	P-value
Number of pipe smokers	272		272		26	
FEV₁, mean difference, mL						
Model 1	-10 (-16, -4)	0.002	-13 (-19, -7)	<0.001	-28 (-69, 12)	0.168
Model 2	-6 (-12, -0.1)	0.045	-8 (-13, -2)	0.010	-34 (-76, 8)	0.108
FVC, mean difference, mL						
Model 1	-7 (-13, -0.2)	0.043	-9 (-16, -3)	0.005	6 (-42, 54)	0.80
Model 2	-6 (-12, 1)	0.103	-9 (-15, -2)	0.011	-2 (-52, 48)	0.94
FEV₁/FVC, mean difference, %						
Model 1	-0.10 (-0.2, 0.02)	0.093	-0.16 (-0.3, -0.05)	0.003	-0.7 (-1.5, 0.1)	0.089
Model 2	-0.02 (-0.1, 0.1)	0.74	-0.02 (-0.1, 0.1)	0.70	-0.8 (-1.6, 0.06)	0.069
Airflow obstruction						
Odds ratio – Model 1	1.03 (1.0, 1.06)	0.075	1.04 (1.01, 1.07)	0.018	1.57 (0.91, 2.72)	0.103
Odds ratio – Model 2	1.02 (0.98, 1.05)	0.28	1.02 (0.98, 1.06)	0.27	2.13 (1.13, 4.0)	0.019

FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity; 95% Confidence intervals are shown in the parenthesis; results are bolded when p-value is less than 0.05.

Model 1: adjusted for age, race, gender and height.

Model 2: adjusted for variables in Model 1 plus cigarette smoking status and pack-years, cigar-years, environmental tobacco smoke exposure, body mass index, education, family history of emphysema, asthma before 45 years of age, and dust exposure.

Table 4

Decrement in lung function and odds ratios for airflow obstruction per 10 cigar-years in the entire sample, excluding participants who smoked cigarettes only, and restricted to participants who never smoked cigarettes.

	Entire Sample		Excluding Participants who Smoked Cigarettes only		Restricted to Participants who Never Smoked Cigarettes	
Number of participants	3,521	P-value	2,097	P-value	1,675	P-value
Number of cigar smokers	334		334		38	
FEV₁, mean difference, mL						
Model 1	-11 (-19, -3)	0.005	-14 (-21, -7)	<0.001	-3 (-18, 11)	0.65
Model 2	-5 (-12, 3)	0.23	-7 (-14, 1)	0.075	7 (-10, 24)	0.43
FVC, mean difference, mL						
Model 1	-1 (-9, 8)	0.91	-2 (-10, 6)	0.60	4 (-13, 22)	0.65
Model 2	4 (-4, 13)	0.32	1 (-7, 10)	0.80	15 (-6, 36)	0.166
FEV₁/FVC, mean difference, %						
Model 1	-0.3 (-0.4, -0.1)	0.001	-0.3 (-0.5, -0.2)	<.0001	-0.1 (-0.4, 0.2)	0.40
Model 2	-0.2 (-0.3, -0.03)	0.019	-0.2 (-0.3, -0.05)	0.009	-0.1 (-0.4, 0.3)	0.74
Airflow obstruction						
Odds ratio – Model 1	1.05 (1.01, 1.09)	0.015	1.06 (1.02, 1.11)	0.003	1.01 (0.90, 1.14)	0.80
Odds ratio – Model 2	1.03 (0.99, 1.08)	0.135	1.04 (0.99, 1.08)	0.130	0.64 (0.26, 1.53)	0.31

FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity; 95% Confidence intervals are shown in the parenthesis; results are bolded when p-value is less than 0.05.

Model 1: adjusted for age, race, gender and height.

Model 2: adjusted for variables in Model 1 plus cigarette smoking status and pack-years, pipe-years, environmental tobacco smoke exposure, body mass index, education, family history of emphysema, asthma before 45 years of age, and dust exposure.