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The Association of Pipe and Cigar Use With Cotinine Levels, Lung Function, and Airflow Obstruction

A Cross-sectional Study

Josanna Rodriguez, MD; Rui Jiang, MD, DrPH; W. Craig Johnson, MS; Barbara A. MacKenzie; Lewis J. Smith, MD; and R. Graham Barr, MD, DrPH

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

Objective: To determine whether 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 6 U.S. communities.

Participants: Men and women aged 48 to 90 years without clinical cardiovascular disease at enrollment who were part of MESA (Multi-Ethnic Study of Atherosclerosis).

Measurements: The MESA Lung Study measured spirometry according to American Thoracic Society guidelines and urine cotinine levels by immunoassay on a subsample of MESA. Pipe-years and cigar-years were calculated as years from self-reported age of starting to age of quitting (or to current age in current users) multiplied by pipe-bowls or cigars per day.

Results: Of 3528 participants, 9% 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 urine cotinine levels compared with never-smokers. Pipe-years were associated with decrements in FEV_1, and cigar-years were associated with decrements in the FEV_1–FVC ratio. Participants who smoked pipes or cigars had increased odds of airflow obstruction whether they had also smoked cigarettes (odds ratio, 3.43 [95% CI, 1.75 to 6.71]; $P < 0.001$) or not (odds ratio, 2.31 [CI, 1.04 to 5.11]; $P = 0.039$) compared with participants with no smoking history.

Limitation: Cross-sectional design.

Conclusion: Pipe and cigar smoking increased urine cotinine levels and was associated with decreased lung function and increased odds of airflow obstruction, even in participants who had never smoked cigarettes.

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For author affiliations, see end of text.

Cigarette smoking has decreased substantially in the United States since the 1960s, from a prevalence of 33% in 1983 to 19.8% in 2007, and this decrease has occurred in all age groups (1–3). Although less common than cigarette smoking, pipe and cigar smoking have 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 pipe and cigar tobacco smoking increased by 28% and 8%, respectively, from 2002 to 2006 (5). In 2005, 3% of high school students smoked pipes and 13% smoked cigars. In 2006, the prevalence of pipe and cigar smoking in the United States was 1% and 6%, respectively (5).

Cigarette smoking is the main cause of chronic obstructive pulmonary disease (COPD) (6, 7), the fourth leading cause of death in the United States (8). A large questionnaire-based cohort showed an increased risk for COPD hospitalization and death in pipe smokers, as did a second cohort for cigar smokers (9, 10). Both cohorts, however, relied on hospital-discharge International Classification of Diseases, Ninth Edition, codes that included COPD and asthma as the primary or secondary reason for hospitalization. Chronic obstructive pulmonary disease is defined by accelerated, age-related decrease in lung function (7); however, no U.S. studies have reported on the possible effects of cumulative pipe and cigar smoking on lung function.

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

METHODS

MESA

The MESA (Multi-Ethnic Study of Atherosclerosis) (11) is a multicenter prospective cohort study to investigate

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Context

Cigarettes are the major cause of chronic obstructive pulmonary disease, but few studies have examined the association of pipe and cigar smoking with lung function. In the United States, pipe and cigar smoking has increased in recent years because some view these behaviors as healthier than cigarette smoking.

Contribution

Among 3528 participants in a population-based study, those who did not smoke cigarettes but did smoke pipes or cigars were more likely to have airflow obstruction than never-smokers (odds ratio, 2.31 [95% CI, 1.04 to 5.11]). Decrements in lung function increased with pipe- or cigar-years.

Implication

Advise patients who smoke pipes or cigars that risk for chronic obstructive pulmonary disease is one of the reasons they should quit.

—The Editors

the prevalence, correlates, and progression of subclinical cardiovascular disease in persons without clinical cardiovascular disease. In 2000 to 2002, MESA recruited 6814 men and women aged 45 to 84 years from 6 U.S. communities: Forsyth County, North Carolina; northern Manhattan and Bronx, New York; Baltimore and Baltimore County, Maryland; St. Paul, Minnesota; Chicago, Illinois; and Los Angeles, California. Exclusion criteria included clinical cardiovascular disease, body weight greater than 300 lb, pregnancy, or impediment to long-term participation.

The MESA Lung Study enrolled 3965 MESA participants of 4484 selected persons who were sampled randomly among those who consented to genetic analyses, those who underwent baseline measures of endothelial function, and those who attended an examination during the MESA Lung Study recruitment period in 2004 to 2006 (**Figure 1**). Asian participants were oversampled.

For the current cross-sectional study related to obstructive lung disease, we excluded 322 participants with a restrictive pattern of spirometry, defined as FVC less than the lower limit of normal (12) and an FEV_1–FVC ratio greater than 0.70.

Assessment of Exposure to Pipe and Cigar Smoke

We used self-administered items from the American Thoracic Society questionnaire (13) to identify participants' smoking histories for pipes, cigars, and cigarettes. For pipes, we first asked participants, "Have you smoked at least 20 pipe-bowls in your lifetime?" If they answered "yes," 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?" Similar items were asked about cigar smoking and, separately, cigarette smoking. We used 20 cigars and 100 cigarettes as the thresholds for ever smoking cigars and cigarettes, respectively.

We calculated pipe-years at the time of spirometry as the self-reported age of starting to the age of quitting (or current age if participants still smoked) multiplied by the number of pipe-bowls per day. We calculated cigar-years as the self-reported age of starting to the age of quitting (or current age if participants still smoked) multiplied by 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, Los Angeles, California) at the National Institute for Occupational Safety and Health Core Laboratory. Intra-assay coefficient of variation was 2.02%, and the minimum detectable concentration was 10 ng/mL. On the basis of the standard approach for statistical calculation of undetectable levels of biomarkers, we assigned undetectable values of cotinine to 7.07 ng/mL (14). We did not correct urine cotinine for creatinine clearance because of the generally healthy (mean serum creatinine level, 83.7 μmol/L [SD, 19.0] or 0.9 mg/dL [0.2]) and multiethnic composition of the cohort.

Measurement of Confounders

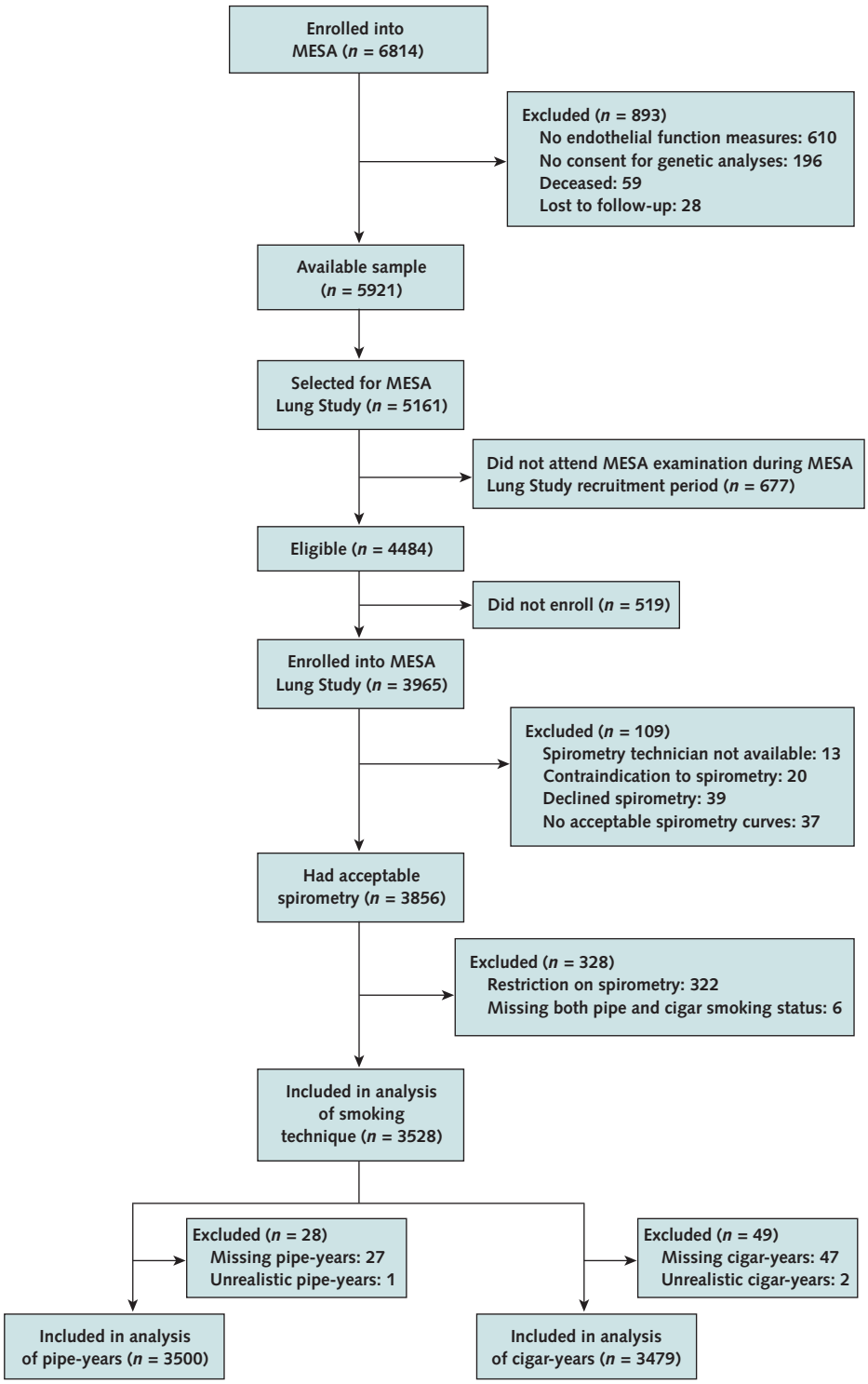
We collected information on age; sex; race or ethnicity; educational attainment; medical history; occupational exposure to dust, fumes, or smoke; depth of inhalation of cigarettes; environmental tobacco smoke exposure; and family history of emphysema by using standardized questionnaire items (13). We defined asthma as self-report of physician-diagnosed asthma before age 45 years. We chose that age to avoid overcorrection for COPD misdiagnosed as asthma in persons older than 45 years, which is common (especially in women) (15). We measured height and weight at the time of spirometry by using calibrated scales and measures and calculated body mass index.

We calculated pack-years of cigarette smoking as follows: the age of starting to the age of quitting (or the current age for current cigarette smokers) × (cigarettes per day/20). The number of cigarettes per day was assessed twice over 4 years 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?" We used the greater of the 2 measures in calculations.

Spirometry

We measured lung function on spirometry in 2004 to 2006 in accordance with the American Thoracic Society/European Respiratory Society recommended guidelines (16). We conducted tests by using a dry-rolling sealed spirometer and software that performed automated quality

Figure 1. **Study flow diagram.**



MESA = Multi-Ethnic Study of Atherosclerosis.

checks as maneuvers were performed (Occupational Marketing, Houston, Texas). All spirometry examinations were reviewed by 1 investigator, and each test was graded for quality (17). We defined low-quality spirometry as only 1 acceptable curve and excluded participants with no acceptable curves.

We defined airflow obstruction as an FEV_1–FVC ratio less than the lower limit of normal (12), in accordance with recent recommendations (16).

Statistical Analysis

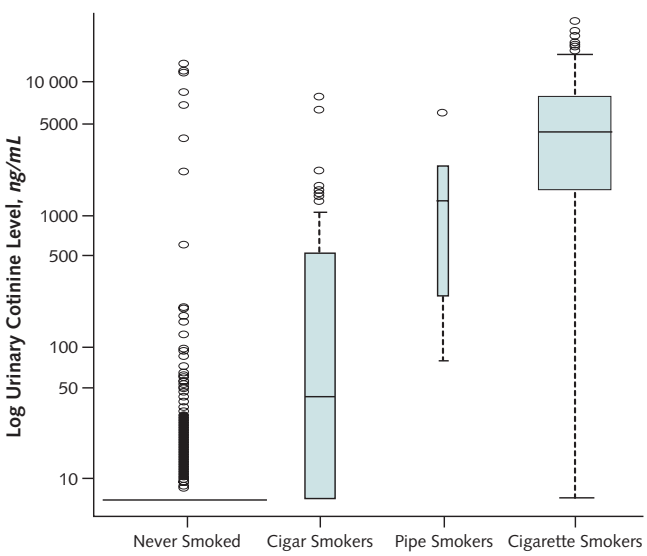
We tested differences in urine cotinine levels between each self-reported current smoking technique and never-smokers with the Wilcoxon rank-sum test. For this analysis, we excluded 4 participants who reported use of nicotine replacement therapy. We hierarchically categorized participants who reported more than 1 smoking technique at the time of the cotinine measurement as described in the footnote to **Figure 2**.

For analyses of smoking technique, the cohort was categorized according to history of pipe or cigar smoking only, cigarette smoking only, and pipe or cigar and cigarette smoking. Decrements in lung function and in the log odds of airflow obstruction were estimated for each category of smoking history compared with never-smokers by using linear regression and logistic regression. We modeled lung function as a function of age, age^2, sex, height, height^2, and race/ethnicity rather than by using the percentage of predicted approach due to known variation in the performance of Third National Health and Nutrition Examination Survey reference equations across racial or ethnic groups in this cohort (17). In addition, we adjusted subsequent models for the following potential causal confounders, all of which are likely to affect lung function in a population-based cohort: educational attainment, cigarette smoking status, pack-years, environmental tobacco smoke exposure, dust exposure, body mass index, asthma before age 45 years, and family history of emphysema. We handled missing data for covariates 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. We performed these analyses in the entire cohort, repeated them after exclusion of participants who smoked cigarettes only, and restricted them to participants who had never smoked cigarettes, thus minimizing residual confounding by cigarette smoking.

We performed analyses in SAS, version 9.2 (SAS Institute, Cary, North Carolina). We used R, version 2.3.1 (R Foundation, Vienna, Austria), to generate plots.

Figure 2. **Urine cotinine levels, by self-report of current smoking technique.**



Lines represent the median cotinine values (log scale). Upper and lower horizontal bars represent maximum and minimum values, excluding outliers. Box height represents the interquartile range. Box width is proportional to sample size. Circles represent outlier values. $P < 0.001$ for all smoking techniques versus never-smokers. Never-smokers ($n = 1620$) reported smoking fewer than 100 cigarettes, fewer than 20 pipe-bowls, and fewer than 20 cigars in life. Cigar smokers ($n = 47$) reported current cigar use but no current pipe or cigarette smoking. Pipe smokers ($n = 6$) reported current pipe use but no current cigarette smoking. Cigarette smokers ($n = 330$) reported current cigarette smoking.

Role of the Funding Source

The MESA and MESA Lung Study were funded by the National Heart, Lung, and Blood Institute (NHLBI). The MESA Lung Study was designed by the MESA Lung Study investigators. The protocol was approved by the institutional review boards of all collaborating institutions and by the NHLBI. The authors, together with other MESA investigators, collected and analyzed the data, vouched for the data and analysis, and wrote and submitted the paper for publication. The NHLBI staff routinely monitored study performance and participated in the internal review of the manuscript before submission.

RESULTS

Figure 1 shows the recruitment of the 3965 participants in the MESA Lung Study and subsequent exclusions. The 3528 participants in the analysis of smoking technique had a mean age of 66 years (SD, 10) and were 49% male, 35% non-Hispanic white, 26% African American, 22% Hispanic, and 17% Chinese American. Nine percent reported ever smoking pipes (median, 15 pipe-years [interquartile range {IQR}, 4 to 46 pipe-years]), mostly in the past. Eleven percent reported ever smoking cigars (median, 6 cigar-years [IQR, 0 to 26 cigar-years]), of whom approximately one fifth currently smoked cigars. Fifty-two percent reported ever smoking cigarettes, and 9% 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. **Characteristics of Participants, by Smoking History**

Characteristic	History of Smoking			
	None (n = 1620)*	Pipes or Cigars Only (n = 56)†	Cigarettes Only (n = 1424)	Pipes or Cigars and Cigarettes (n = 428)‡
Mean age (SD), *y*	65 (10)	65 (8)	66 (10)	67 (10)
Men, %	34	95	51	93
Race/ethnicity, %				
Non-Hispanic white	28	64	35	57
African American	22	27	30	26
Hispanic	24	9	24	13
Chinese American	25	0	11	4
Educational attainment, %				
Less than 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
Postgraduate degree	19	52	16	28
Mean height (SD), *cm*	163 (10)	174 (9)	167 (10)	174 (8)
Mean body mass index (SD), kg/m^2	27.5 (5.2)	28.6 (3.7)	28.3 (5.7)	28.6 (4.6)
Cigarette smoking status, %				
Never	100	100	–	–
Former	–	–	82	83
Current	–	–	18	17
Mean pack-years of smoking (IQR), %§	–	–	17 (6–35)	20 (7–39)
0–10	–	–	34	32
10–50	–	–	52	52
>50	–	–	14	16
Pipe-smoking status, %				
Never	100	38	100	38
Former	–	59	–	57
Current	–	4	–	4
Median age started pipe smoking (IQR), *y*§	–	24 (20–30)	–	23 (20–30)
Median age at last pipe in quitters (IQR), *y*§	–	30 (25–40)	–	35 (27–45)
Median pipe-bowls smoked per day (IQR), *n*§	–	1 (0–2)	–	2 (1–4)
Median pipe-years (IQR), %§	–	0 (0–12)	–	19 (4–50)
Missing	–	26	–	7
0–10	–	51	–	36
10–50	–	17	–	34
>50	–	6	–	23
Cigar smoking status, %				
Never	100	25	100	20
Former	–	46	–	65
Current	–	29	–	14
Median age started cigar smoking (IQR), *y*§	–	24 (20–40)	–	25 (20–30)
Median age at last cigar in quitters (IQR), *y*§	–	35 (28–55)	–	42 (33–50)
Median cigars smoked per day (IQR), *n*§	–	0 (0–1)	–	1 (0–2)
Median cigar-years (IQR), %§	–	0 (0–10)	–	7 (0–30)
Missing	–	10	–	12
0–10	–	64	–	46
10–50	–	16	–	29
>50	–	10	–	12
Environmental tobacco smoke exposure, %	35	20	51	50
Family history of emphysema, %	4	0	4	6
Asthma before age 45 y, %	8	11	7	10
Exposure to dust, %	30	43	40	47
Mean FEV_1 (SD), % *predicted*‖	99 (16)	95 (13)	94 (19)	92 (18)
Mean FVC (SD), % *predicted*‖	98 (14)	98 (11)	97 (16)	97 (14)
Mean FEV_1–FVC ratio (SD), %	77 (7)	74 (7)	73 (9)	71 (10)

IQR = interquartile range.
* Lifetime smoking history of fewer than 100 cigarettes, 20 pipe-bowls, or 20 cigars.
† Lifetime smoking history of at least 20 pipe-bowls or at least 20 cigars but fewer than 100 cigarettes. Among these 56 participants, 21 reported a history of smoking both cigars and pipes.
‡ Lifetime smoking history of at least 20 pipe-bowls or at least 20 cigars in life and at least 100 cigarettes in life.
§ In participants with a history of smoking the given tobacco product.
‖ Predicted values calculated by using Third National Health and Nutrition Examination Survey reference equations, with 0.88 correction factor for Chinese Americans (12, 17).

Table 1 shows the study sample stratified by smoking technique into 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, to be white or African American, and to have higher educational attainment.

Cotinine Levels, by Current Smoking Technique

To evaluate the biological plausibility of the 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 in never-smokers ($n = 1620$), 43 ng/mL in current cigar smokers ($n = 47$), 1324 ng/mL in current pipe smokers ($n = 6$), and 4304 ng/mL in current cigarette smokers ($n = 330$) (all $P < 0.001$ vs. never-smokers). **Figure 2** shows box plots 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 [IQR, 7 to 206 ng/mL]; $P < 0.001$) and 2 current pipe smokers (164 ng/mL [IQR, 81 to 248 ng/mL]; $P < 0.001$) who reported never smoking cigarettes.

Lung Function, Airflow Obstruction, and Smoking Technique

Table 2 shows decrements in lung function and in odds ratios for airflow obstruction according to categories of smoking technique. Decrements in the FEV_1 were consistent among participants with a history of pipe or cigar smoking only, cigarette smoking only, and pipe or cigar and cigarette smoking compared with never-smokers. The decrement was modest and not statistically significant in the 56 participants who smoked pipes or cigars only, was greater and statistically significant in the much-larger group who smoked cigarettes only, and was greatest in 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 to 5.11]; $P = 0.039$) compared with never-smokers and were greatest among participants who smoked pipes or cigars in addition to cigarettes.

Cumulative Pipe Smoking

In the entire sample, pipe-years were inversely associated with FEV_1 in the analyses adjusted for age, race/ethnicity, sex, and height (**Table 3**). This association persisted in the fully adjusted model and after exclusion of participants who smoked cigarettes only. On restriction to participants who never smoked cigarettes, effect estimates were of larger magnitude but were not statistically significant. Consistent, although generally nonsignificant, patterns were evident for the FEV_1–FVC ratio. The odds ratio for airflow obstruction was increased in all analyses, and the increase was statistically significant in fully adjusted analyses in participants who never smoked cigarettes (odds ratio, 2.13 [CI, 1.13 to 4.0]; $P = 0.019$).

Table 2. **Decrement in Lung Function and Odds Ratio for Airflow Obstruction, by Smoking History***

Value	History of Smoking						
	None ($n = 1620$)†	Pipes or Cigars Only ($n = 56$)‡	*P* Value	Cigarettes Only ($n = 1424$)	*P* Value	Pipes or Cigars and Cigarettes ($n = 428$)§	*P* Value
FEV_1, *mL*							
Mean	2314	2299		2217		2187	
Mean difference for model 1‖	0 (reference)	−50 (−146 to 46)	0.31	−98 (−128 to −68)	<0.001	−150 (−199 to −100)	<0.001
Mean difference for model 2¶	0 (reference)	−39 (−134 to 55)	0.41	−82 (−142 to −22)	0.007	−160 (−266 to −55)	0.003
FVC, *mL*							
Mean	3032	3075		3001		3017	
Mean difference for model 1‖	0 (reference)	22 (−93 to 137)	0.71	−32 (−66 to 2.4)	0.068	−28 (−85 to 29)	0.33
Mean difference for model 2¶	0 (reference)	33 (−81 to 146)	0.57	−20 (−89 to 48)	0.56	−7 (−129 to 115)	0.91
FEV_1–FVC ratio, %							
Mean	76.5	73.5		73.3		71.1	
Mean difference for model 1‖	0 (reference)	−1.4 (−3.3 to 0.5)	0.150	−2.6 (−3.2 to −2.0)	<0.001	−3.5 (−4.4 to −2.5)	<0.001
Mean difference for model 2¶	0 (reference)	−1.4 (−3.2 to 0.5)	0.150	−2.2 (−3.4 to −1.1)	<0.001	−4.3 (−6.3 to −2.3)	<0.001
Airflow obstruction, %							
Prevalence	7.6	17.9		16.1		21.04	
Odds ratio for model 1‖	1 (reference)	2.24 (1.04 to 4.80)	0.040	2.15 (1.68 to 2.74)	<0.001	2.42 (1.67 to 3.52)	<0.001
Odds ratio for model 2¶	1 (reference)	2.31 (1.04 to 5.11)	0.039	2.01 (1.31 to 3.08)	0.001	3.43 (1.75 to 6.71)	<0.001

* 95% CIs are shown in parentheses.
† Lifetime smoking history of fewer than 100 cigarettes, 20 pipe-bowls, or 20 cigars.
‡ Lifetime smoking history of at least 20 pipe-bowls or at least 20 cigars but fewer than 100 cigarettes. In these 56 participants, 21 reported a history of smoking both cigars and pipes.
§ Lifetime smoking history of at least 20 pipe-bowls or at least 20 cigars and at least 100 cigarettes.
‖ Adjusted for age, race/ethnicity, sex, and height.
¶ Adjusted for age, race/ethnicity, sex, height, cigarette smoking status and pack-years, environmental tobacco smoke exposure, body mass index, education, family history of emphysema, asthma before age 45 years, and dust exposure.

Table 3. **Decrement in Lung Function and Odds Ratio for Airflow Obstruction per 10 Pipe-Years***

Value	Entire Sample†	*P* Value	Excluding Participants Who Smoked Cigarettes Only‡	*P* Value	Restricted to Participants Who Never Smoked Cigarettes§	*P* Value
Mean difference for FEV_1, *mL*						
Model 1‖	−10 (−16 to −4)	0.002	−13 (−19 to −7)	<0.001	−28 (−69 to 12)	0.168
Model 2¶	−6 (−12 to −0.1)	0.045	−8 (−13 to −2)	0.010	−34 (−76 to 8)	0.108
Mean difference for FVC, *mL*						
Model 1‖	−7 (−13 to −0.2)	0.043	−9 (−16 to −3)	0.005	6 (−42 to 54)	0.80
Model 2¶	−6 (−12 to 1)	0.103	−9 (−15 to −2)	0.011	−2 (−52 to 48)	0.94
Mean difference for FEV_1–FVC ratio, %						
Model 1‖	−0.10 (−0.2 to 0.02)	0.093	−0.16 (−0.3 to −0.05)	0.003	−0.7 (−1.5 to 0.1)	0.089
Model 2¶	−0.02 (−0.1 to 0.1)	0.74	−0.02 (−0.1 to 0.1)	0.70	−0.8 (−1.6 to 0.06)	0.069
Odds ratio for airflow obstruction						
Model 1‖	1.03 (1.0 to 1.06)	0.075	1.04 (1.01 to 1.07)	0.018	1.57 (0.91 to 2.72)	0.103
Model 2¶	1.02 (0.98 to 1.05)	0.28	1.02 (0.98 to 1.06)	0.27	2.13 (1.13 to 4.0)	0.019

* 95% CIs are shown in parentheses.
† 3500 participants, 272 pipe smokers.
‡ 2076 participants, 272 pipe smokers.
§ 1667 participants, 26 pipe smokers.
‖ Adjusted for age, race/ethnicity, sex, and height.
¶ Adjusted for age, race/ethnicity, sex, height, cigarette smoking status and pack-years, cigar-years, environmental tobacco smoke exposure, body mass index, education, family history of emphysema, asthma before age 45 years, and dust exposure.

The decrease in lung function from pipe smoking was much larger in pipe smokers who smoked heavily. For example, in fully adjusted analyses, the mean FEV_1 in the 64 participants in the full sample with 50 or more pipe-years was 154 mL lower (CI, −262 to −47 mL; $P = 0.005$) than that of participants who had never smoked pipes, and the mean FEV_1–FVC ratio was 2.1 percentage points lower (CI, −4.1 to −0.1 percentage points; $P = 0.039$).

No evidence existed that these associations were modified by cigarette smoking in the entire sample (for example, the *P* value for the interaction of pack-years with pipe-years for the FEV_1 was 0.22). Because most pipe 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, more cigar-years were associated with decreased FEV_1, decreased FEV_1–FVC ratio, and increased odds ratio for airflow obstruction in the analyses adjusted for age, race/ethnicity, sex, and height (**Table 4**). Fully adjusted models yielded consistent associations, which remained statistically significant for the FEV_1–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 with FEV_1 in the entire sample was modified by pack-years of cigarette smoking (*P* for interaction < 0.001).

The associations were qualitatively consistent in white persons and in men (data not shown).

Cumulative Cigarette Smoking

By comparison, cigarette pack-years were inversely associated with FEV_1 (−46 mL per 10 pack-years [CI, −54 to −38 mL per 10 pack-years]; $P < 0.001$) and the FEV_1–FVC ratio (−0.8% per 10 pack-years [CI, −1.0% to −0.7% per 10 pack-years]; $P < 0.001$) and were associated with increased airflow obstruction (odds ratio, 1.20 [CI, 1.14 to 1.26]; $P < 0.001$) 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 sex. Results were similar in sensitivity analyses in which pack-years of cigarettes were increased by 25% selectively among those who reported no current smoking but had cotinine levels greater than 100 ng/mL and after exclusion of participants with low-quality spirometry (data not shown).

DISCUSSION

Pipe and cigar smoking were associated with an obstructive pattern of spirometry characterized by decreased FEV_1 and FEV_1–FVC ratio and by an increased risk for 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 measurable increase in the risk for COPD.

Tobacco smoke is the major cause of COPD (6, 19). However, it is not entirely clear whether pipe and cigar smoking damages the lungs via the same mechanism as cigarette smoking. Some pipe and cigar smokers say they do not inhale, or inhale less, than cigarette smokers. The elevated cotinine levels in the current study, however, belie

Table 4. **Decrement in Lung Function and Odds Ratio for Airflow Obstruction per 10 Cigar-Years***

Value	Entire Sample†	*P* Value	Excluding Participants Who Smoked Cigarettes Only‡	*P* Value	Restricted to Participants Who Never Smoked Cigarettes§	*P* Value
Mean difference for FEV_1, *mL*						
Model 1‖	−11 (−19 to −3)	0.005	−14 (−22 to −7)	<0.001	−3 (−18 to 11)	0.65
Model 2¶	−5 (−12 to 3)	0.23	−7 (−14 to 1)	0.075	7 (−10 to 24)	0.43
Mean difference for FVC, *mL*						
Model 1‖	−1 (−9 to 8)	0.91	−2 (−10 to 6)	0.60	4 (−14 to 22)	0.65
Model 2¶	4 (−4 to 13)	0.32	1 (−7 to 10)	0.80	15 (−6 to 36)	0.166
Mean difference for FEV_1–FVC ratio, %						
Model 1‖	−0.3 (−0.4 to −0.1)	0.001	−0.3 (−0.5 to −0.2)	<0.001	−0.1 (−0.4 to 0.2)	0.40
Model 2¶	−0.2 (−0.3 to −0.03)	0.019	−0.2 (−0.3 to −0.05)	0.009	−0.1 (−0.4 to 0.3)	0.74
Odds ratio for airflow obstruction						
Model 1‖	1.05 (1.01 to 1.09)	0.015	1.06 (1.02 to 1.11)	0.003	1.01 (0.90 to 1.14)	0.80
Model 2¶	1.03 (0.99 to 1.08)	0.135	1.04 (0.99 to 1.08)	0.130	0.63 (0.26 to 1.54)	0.31

* 95% CIs are shown in parentheses.
† 3479 participants, 334 cigar smokers.
‡ 2055 participants, 334 cigar smokers.
§ 1672 participants, 38 cigar smokers.
‖ Adjusted for age, race/ethnicity, sex, and height.
¶ Adjusted for age, race/ethnicity, sex, height, cigarette smoking status and pack-years, pipe-years, environmental tobacco smoke exposure, body mass index, education, family history of emphysema, asthma before age 45 years, and dust exposure.

this notion and provide a biological measure of nicotine exposure. Our results are also consistent with previous observations (20, 21) of elevated carboxyhemoglobin saturations in pipe and cigar smokers, particularly among former cigarette smokers who may be more likely to inhale than persons who have never smoked cigarettes. 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 were lower among current pipe and cigar smokers than among current cigarette smokers; however, relative differences in cotinine levels reflect differences in nicotine absorption but not necessarily exposure to harmful products of tobacco smoke. Most cigarettes are filtered, 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 that activates carcinogens from tobacco smoke, and DNA adduct levels have not been found, possibly because of differences in inhalation (25).

Despite increases in pipe and cigar smoking, studies on such smoking and lung function are few. We conducted an English-language MEDLINE search through December 2009 to find studies that examined the association between pipe 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 versus nonsmokers (26–28), as well as an increased risk for mucous hypersecretion (28). The Copenhagen City Heart Study showed this association in a relatively homogeneous European sample, whereas our study extends these findings to a multiethnic sample in the United States.

Other studies of pipe and cigar smoking and COPD have relied on International Classification of Disease–based measures. In addition to the findings in the 2 U.S. cohorts (9, 10), pipe and cigar smoking was associated with increased mortality from emphysema and chronic bronchitis in Sweden (29), and British men who switched from cigarettes to pipes and cigars had an increased risk for COPD death, ischemic heart disease, and lung cancer compared with those who quit smoking all together (20). A prospective cohort study in the Netherlands (30) showed that pipe and cigar smoking was associated with reduced life expectancy, although to a lesser extent than cigarette smoking. Other studies (31, 32) investigating the effects of pipe and cigar smoking are limited to overall mortality, coronary artery disease, and risk for lung cancer.

Major strengths of this study include the large, multiethnic, 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, restricted to participants who 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 urine cotinine levels between cigar and pipe smokers who never smoked cigarettes. Only 13 (0.8%) of the 1620 participants who reported never smoking had urine cotinine levels that suggested active smoking (>100 ng/mL), and only 7 (0.4%) had levels that were unequivocally consistent with active smoking (>500 ng/mL). Because of 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.

Few participants smoked pipes or cigars but not cigarettes. 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 results from the entire sample and were statistically significant for airflow obstruction. The interaction term for cigar-years suggested a possible greater effect of cigar smoking in participants who had ever smoked cigarettes than in 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. Physicians should consider pipe and cigar smoking a risk factor for COPD and counsel cessation of pipe and cigar smoking regardless of cigarette smoking history.

From Columbia University, New York, New York; University of Washington, Seattle, Washington; National Institute for Occupational Safety and Health, Cincinnati, Ohio; and Northwestern University, Chicago, Illinois.

Note: A full list of participating MESA investigators and institutions can be found at www.mesa-nhlbi.org.

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Reproducible Research Statement: *Study protocol (for the MESA Lung Study) and statistical code:* Available from Dr. Barr (e-mail, rgb9@columbia.edu). *Data set:* Available as a limited access data set from the NHLBI (www.mesa-nhlbi.org).

Requests for Single Reprints: R. Graham Barr, MD, DrPH, Columbia University Medical Center, 630 West 168th Street, PH 9 East, Room 105, New York, NY 10032; e-mail, rgb9@columbia.edu.

Current author addresses and author contributions are available at www.annals.org.

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Current Author Addresses: Drs. Rodriguez, Jiang, and Barr: Columbia University Medical Center, PH 9 East, Room 105, 630 West 168th Street, New York, NY 10032.
Mr. Johnson: University of Washington, Collaborative Health Studies Coordinating Center, Building 29, Suite 310, 6200 NE 74th Street, Seattle, WA 98115-8160.
Ms. MacKenzie: Robert A. Taft Labs, Centers for Disease Control and Prevention, The National Institute for Occupational Safety and Health, 4676 Columbia Parkway, Cincinnati, OH 45226.
Dr. Smith: Northwestern University, 750 North Lake Shore Drive, Room 707, Chicago, IL 60611.

Author Contributions: Conception and design: R.G. Barr.
Analysis and interpretation of the data: J. Rodriguez, B.A. MacKenzie, L.J. Smith, R.G. Barr.
Drafting of the article: J. Rodriguez, R.G. Barr.
Critical revision of the article for important intellectual content: R. Jiang, L.J. Smith, R.G. Barr.
Final approval of the article: J. Rodriguez, R. Jiang, W.C. Johnson, L.J. Smith, R.G. Barr.
Provision of study materials or patients: L.J. Smith.
Statistical expertise: W.C. Johnson, R.G. Barr.
Obtaining of funding: R.G. Barr.
Administrative, technical, or logistic support: W.C. Johnson, R.G. Barr.
Collection and assembly of data: W.C. Johnson, R.G. Barr.