Original research

Pipe and cigar use, lung function decline and clinical outcomes: an analysis of the NHLBI Pooled Cohorts Study

William M Gardner ,[1] Pallavi P Balte,[1] Christina M Eckhardt ,[1] Jack E Morris,[2] Surya P Bhatt ,[3] David J Couper,[4] Neal D Freedman,[5] David R Jacobs,[6] Ravi Kalhan,[7] Laura R Loehr,[8] Stephanie J London ,[9] Pamela L Lutsey ,[10] Joseph E Schwartz,[1,11] Wendy White,[12] Sachin Yende ,[13] Tiffany R Sanchez,[14] Elizabeth C Oelsner[1]

► Additional supplemental material is published online only. To view, please visit the journal online (https://doi.org/10.1136/thorax-2025-224461).

For numbered affiliations see end of article.

Correspondence to
Dr Elizabeth C Oelsner; eco7@cumc.columbia.edu

Received 5 November 2025
Accepted 16 February 2026

ABSTRACT

Introduction Smoked tobacco is a leading risk factor for cardiopulmonary disease. Pipe and cigar use remains common among US adults, yet its risks remain insufficiently understood.

Methods We analysed data from five pooled cohorts with adults enrolled from 1971 to 2011 with follow-up through 2018. Pipe/cigar use was defined by baseline self-report. Forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and FEV_1/FVC ratio were measured by spirometry. All-cause mortality, coronary heart disease mortality and hospitalisation, respiratory-related mortality, and chronic obstructive pulmonary disease (COPD) mortality and hospitalisation were classified via adjudication or validated algorithms. Associations were estimated with linear mixed models and Fine-Gray subdistribution hazards models adjusted for sociodemographic and clinical factors.

Results Among 22 823 participants (mean (SD) age 48.0 (15.7) years; 44.9% male sex; 70.5% white, 25.4% black, 2.4% Hispanic/Latino), 2621 (11.5%) reported ever pipe/cigar use, including 518 (2.3%) exclusive users without cigarette history. Compared with never tobacco users (n=9931), exclusive pipe/cigar use was associated with faster decline in FEV_1 (3.36 mL/year; 95% CI 1.99 to 4.72), FVC (3.73 mL/year; 95% CI 2.05 to 5.42) and FEV_1/FVC (0.031 per year; 95% CI 0.008 to 0.054). Exclusive users had higher all-cause mortality (adjusted HR (aHR) 1.24; 95% CI 1.08 to 1.41), COPD hospitalisation/mortality (aHR 2.02; 95% CI 1.41 to 2.90) and preserved ratio impaired spirometry (aHR 1.83; 95% CI 1.24 to 2.71).

Conclusion Pipe and cigar use was associated with accelerated lung function decline and increased mortality and cardiopulmonary events, including among never cigarette users. These findings underscore the need for prevention and cessation efforts targeting non-cigarette tobacco.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Cigarette smoking is an established cause of cardiopulmonary disease, but evidence on pipe and cigar use is limited. Prior studies were often small, lacked diversity and did not separate exclusive pipe/cigar users from those who also smoked cigarettes. Despite ongoing use of non-cigarette tobacco products in the USA, their long-term health risks remain poorly defined.

WHAT THIS STUDY ADDS

⇒ In a large, multiethnic US population with prospective follow-up, exclusive pipe or cigar use was associated with accelerated lung function decline, higher all-cause mortality and increased risk of chronic obstructive pulmonary disease-related hospitalisation or death, even among adults who never smoked cigarettes. Exclusive pipe or cigar users also had increased risk of incident preserved ratio impaired spirometry, a pattern linked to cardiopulmonary morbidity and mortality.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ These findings challenge the perception of pipe and cigar smoking as safer alternatives to cigarettes and highlight their independent health risks. Public health messaging, prevention and cessation interventions should address non-cigarette tobacco use explicitly. Clinicians may need to incorporate pipe and cigar use into risk assessment and counselling, while policymakers could consider strengthening regulation and surveillance of these products.

To cite: Gardner WM, Balte PP, Eckhardt CM, *et al*. *Thorax* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/thorax-2025-224461

INTRODUCTION

Smoked tobacco is estimated to account for one in six deaths due to non-communicable disease,[1 2] due in large part to its causal role in the development and progression of cardiovascular disease and chronic obstructive pulmonary disease (COPD). While rates of cigarette use have declined in recent decades, tobacco use remains common, and non-cigarette tobacco products such as pipes and cigars continue to be used by millions of US adults.[3] Cigar use has been linked to cancer and other cardiopulmonary diseases and represents a meaningful source of health loss due to smoked tobacco.[4–6]

Of an estimated 46 million current tobacco users in the USA in 2021, 8.6 million (18.7%) were smoking cigars and 2.3 million (5.0%) were

smoking pipes.[3] Analyses of excise tax data estimated that cigar and pipe consumption increased by 180% and 556%, respectively, from 2000 to 2015.[7] This may be because non-cigarette tobacco use (including pipes and cigars) is frequently perceived as lower risk compared with cigarettes.[8] A recent report from the US Government Accountability Office notes that pipe tobacco and some large cigars are taxed at lower rates compared with cigarettes, and that these tax rate differences may have led to market shifts among these products.[9]

While it was previously thought that cigar and pipe users do not inhale smoke during use, studies have shown that cigar and pipe smoke is likely inhaled, though potentially to a lesser extent than cigarette smoke.[10 11] Other characteristics of pipe and cigar tobacco products may increase the risk of respiratory damage, including a higher pH of pipe and cigar smoke that makes it more readily absorbed across the respiratory lining,[4] higher average weights of tobacco compared with cigarettes[12] and comparable or higher levels of toxic chemicals compared with cigarettes.[12] While major public health agencies note the risks associated with pipe and cigar use, these products did not come under US Food and Drug Administration (FDA) regulatory authority until 2016. Since then, FDA rules requiring warning labels on pipe and cigar products have been struck down by federal courts, making compliance with warning labels largely voluntary for these products.[13]

Research on the health effects of pipe and cigar use remains limited. Many prior studies have been characterised by relatively small and non-diverse samples, limited control for confounding and lack of distinction between pipe and cigar users with or without concurrent cigarette use.[14] We therefore aimed to test associations of pipe and cigar use with rate of lung function decline and risk of adverse health outcomes, including cardiopulmonary events, in a large, multiethnic, US general population-based meta-cohort of adults, with separate analyses including versus excluding those with a history of cigarette use.

METHODS

Study population

The National Heart, Lung, and Blood Institute (NHLBI) Pooled Cohorts Study[15] harmonised data from nine US general population-based cohorts that enrolled participants from 1971 to 2011. This report includes data from five cohorts that had comprehensive smoking assessments and two or more longitudinal spirometry measurements: the Atherosclerosis Risk in Communities Study (ARIC),[16] the Coronary Artery Risk Development in Young Adults Study (CARDIA),[17] the Framingham Offspring Study,[18] the Health Aging and Body Composition (HABC) study[19] and the Multi-Ethnic Study of Atherosclerosis (MESA)[20] (online supplemental eTable 1). All studies were approved by the institutional review boards of the collaborating institutions and all participants provided written informed consent at the time of enrolment. This meta-cohort analysis was approved by all cohorts and the institutional review board at Columbia University Irving Medical Center (approval IRB-AAAB1971).

Smoking assessments

Smoking history was self-reported at baseline and follow-up exams. Questionnaires included items on current and previous cigarette, pipe and cigar use (online supplemental eTable 2, eFigure 1). Responses were harmonised systematically and reviewed cross-sectionally and longitudinally for consistency; incongruent responses were reconciled or, if necessary, censored.[21] Baseline pipe or cigar use was classified as never versus ever use (hereafter, 'ever pipe/cigar') and current versus non-current or never use ('current pipe/cigar'). Participants were classified into one of four mutually exclusive categories of pipe/cigar use at baseline: no history of cigarette, pipe or cigar use ('never users'), history of cigarette use only ('exclusive cigarette users'), history of pipe or cigar use only ('exclusive pipe/cigar users') or history of cigarette and pipe or cigar use ('cigarette and pipe/cigar users').

The primary exposures and comparisons of interest were (1) ever pipe/cigar use versus never pipe/cigar use, analysed among the entire study sample, and (2) exclusive pipe/cigar use versus never cigarette or pipe/cigar use, analysed in the subgroup of never cigarette users.

Lung function

Prebronchodilator forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) were measured using water-seal, dry-rolling seal or flow-sensing spirometers following American Thoracic Society (ATS)/European Respiratory Society (ERS) guidelines current at the time of the exams.[15] Lung function measurements were subsequently harmonised and quality-controlled by a scoring rubric based on 2005 ATS/ERS standards.[22] Reference equations derived from the US population using the National Health and Nutrition Examination Survey III data were used to determine per cent predicted values based on age, sex, height, race and ethnicity.[23] Participants with fewer than two valid spirometry measurements meeting quality standards were excluded from the analysis. The first spirometry exam coincided with the baseline assessment of pipe or cigar use in all cohorts (online supplemental eFigure 1).

To account for the physiological decline in lung function associated with ageing and to minimise potential bias from fixed thresholds, incident airflow limitation (AL) and preserved ratio impaired spirometry (PRISm) were defined using the lower limit of normal (LLN) for FEV_1 and FEV_1/FVC. These thresholds were derived from Global Lung Function Initiative (GLI) reference equations, treating the race of all participants as 'other', consistent with prior studies using GLI reference equations in diverse populations.[24 25] Incident AL (FEV_1/FVC below the LLN) and PRISm (FEV_1 less than the LLN and FEV_1/FVC greater than or equal to the LLN) were classified by longitudinal spirometry among participants with normal baseline lung function.

Clinical outcomes

All-cause mortality was ascertained by proxy interview, with validation by death certificate, hospital records or the National Death Index (online supplemental eTable 3). Cause-specific outcomes were respiratory-related mortality, coronary heart disease (CHD)-related mortality and composite outcomes of COPD-related hospitalisation or death ('COPD-related events'), chronic lower respiratory disease (CLRD)-related hospitalisation or death ('CLRD-related events') and CHD-related hospitalisation or death ('CHD-related events'). These outcomes were classified by adjudication via medical record review by clinical events committees or using administrative criteria following previously validated protocols.[15] Data were available from all cohorts for all-cause mortality, CHD-related mortality and events and incident PRISm and AL. Data for respiratory-related mortality were available from ARIC, CARDIA, HABC and MESA, and data on COPD-related events and CLRD-related events were available from ARIC, HABC and MESA.

Covariates

Birth year, age, sex, race/ethnicity and educational attainment were self-reported at the cohort baseline exam. Race/ethnicity was categorised using the 2000 US Census approach.[15] Height and weight were measured using standard methods and used to calculate body mass index (BMI). Hypertension was defined by self-reported physician diagnosis, systolic blood pressure≥140 mm Hg, diastolic blood pressure≥90 mm Hg or use of antihypertensive medications. Diabetes was defined by self-reported physician diagnosis, fasting plasma glucose greater than 125 mg/dL or use of oral hypoglycaemic agents or insulin. Estimated glomerular filtration rate (eGFR) was based on baseline laboratory testing, calculated by the Modification of Diet in Renal Disease equation and categorised as less than 30 mL/min, 30–59 mL/min and 60 or greater mL/min. Histories of clinical CHD, heart failure (HF) and stroke were classified based on self-reported physician diagnosis.

Statistical analyses

Linear mixed models were used to test associations of pipe/cigar use and repeated lung function measures. Separate models were conducted for the three lung function outcomes (FEV_1, FVC and FEV_1/FVC). Lung function was regressed on ever and current pipe/cigar use (vs never pipe/cigar use) at baseline and, in a subgroup analysis limited to never cigarette smokers, exclusive ever and current pipe/cigar use (vs never cigarette or pipe/cigar use). Models were adjusted by age (time-variant), birth year, sex, race/ethnicity, educational attainment, height (time-variant), weight (time-variant), source cohort and site. Birth year was modelled as a potential confounder due to secular changes in the tobacco marketplace that could contribute to differences by birth cohort. Multiplicative interaction terms with age were included for all time-invariant covariates (birth year, sex, race/ethnicity, educational attainment, study cohort and site). For analyses including current or former cigarette users, models were adjusted for cigarette smoking status (time-varying) and pack-years of cigarette smoking at baseline. The models allowed for study-specific unstructured variance/covariance matrices of the visit-specific residuals, which allowed for between-study and between-visit heterogeneity of variances and serial autocorrelation of within-person residuals. Linearity, homoscedasticity and normality assumptions were confirmed using residual plot analyses.

Associations between pipe/cigar use and clinical outcomes were analysed using Kaplan-Meier curves and hazard models with separate analyses for pipe or cigar use among all participants and exclusive pipe or cigar use compared with never users of cigarettes, pipes or cigars. For all-cause mortality, we used Cox proportional hazards models.[26] For cause-specific outcomes, we used Fine-Gray subdistribution hazard models[27] to account for the competing risk of death from other causes. For incident PRISm and AL, we used Fine-Gray models with death as the competing event. Data were available through 2018 with administrative censoring of follow-up after this point. Models were adjusted for all covariates included in the mixed models (with the substitution of BMI for separate height and weight variables) as well as hypertension, diabetes, CHD, HF and stroke at baseline. The proportional hazards assumption was confirmed by examining scaled Schoenfeld residual plots and using the Schoenfeld residual test.

Linear mixed models of lung function decline were performed using SAS software V.9.4 (Cary, North Carolina). Cox proportional hazards and Fine-Gray subdistribution hazard models were fit in R (V.4.0.2). We used complete case analysis, restricting each analysis to participants with non-missing values for the outcome, exposure and all covariates. To assess potential selection bias, we compared baseline characteristics of included versus excluded participants (online supplemental eTable 4). A two-tailed alpha of 0.05 was considered statistically significant for all models. Due to the potential for type 1 error, subgroup analyses should be interpreted with caution.

RESULTS

Participant characteristics

There were 22 823 participants across five cohorts included in lung function analyses (table 1, online supplemental eFigure 2). At baseline, the mean age of participants was 48.0±15.7 years, 10 253 (44.9%) were male, 5806 (25.4%) were non-Hispanic black, 16 080 (70.5%) were non-Hispanic white and 537 (2.4%) were Hispanic/Latino. With respect to tobacco exposures, 9931 (43.5%) were never-users, 10 271 (45.0%) were exclusive cigarette users, 518 (2.3%) were exclusive pipe/cigar users and 2103 (9.2%) were cigarette and pipe/cigar users. Pipe or cigar users were predominantly male (96.7%). Among ever pipe/cigar users, the mean age of initiation was 29.0±10.5 years, the mean total years of pipe/cigar use was 11.3±11.3 years, and 24.8% were current users. Among exclusive pipe/cigar users, mean age of initiation was 25.9±8.6 years, mean total years of pipe/cigar use was 15.0±12.4 years and 42.7% were current users. Mean pack-years of cigarette use among exclusive cigarette users was 21.7±21.4 years compared with 28.5±15.9 years among users of both cigarettes and pipes or cigars. Median duration of follow-up was 12.6±9.9 years, with median number of spirometry assessments of 2 (Q1–Q3 2–3). Comparing study participants to those excluded due to insufficient spirometry or covariate values, included participants were more likely to be younger but otherwise had similar rates of ever and current pipe/cigar use (online supplemental eTable 4). The characteristics of participants in the clinical outcomes analyses were similar (online supplemental eTables 5 and 6).

Lung function

Compared with never pipe/cigar users, ever pipe/cigar use was associated with a greater rate of decline in FEV_1 (2.38 mL/year, 95% CI 1.71 to 3.04), FVC (2.60 mL/year, 95% CI 1.78 to 3.41) and FEV_1/FVC (0.017 per year, 95% CI 0.005 to 0.029, figure 1). Associations with current pipe/cigar use (vs never pipe/cigar use) were similar. Ever/pipe cigar use among all participants was not associated with incident AL or PRISm (online supplemental eTable 7).

Among never cigarette users (N=10 449), exclusive ever pipe/cigar use was also associated with a greater rate of decline in FEV_1 (3.36 mL/year, 95% CI 1.99 to 4.72), FVC (3.73 mL/year, 95% CI 2.05 to 5.24) and FEV_1/FVC (0.031 per year, 95% CI 0.008 to 0.054; Figure 1) compared with never pipe/cigar users. Results for current exclusive pipe/cigar use versus never pipe/cigar use among never cigarette users were similar, although the association with FEV_1/FVC decline was not statistically significant. Exclusive pipe/cigar use was also associated with incident PRISm among never cigarette users (adjusted subdistribution HR (asHR) 1.83, 95% CI 1.24 to 2.71; online supplemental eTable 7), but not incident AL. Results were qualitatively similar in sensitivity models including males only, though no models of FEV_1/FVC were statistically significant (online supplemental eFigure 3). In those cohorts that measured pipe and cigar use separately, sensitivity analyses with separate models for pipe and

Table 1 Baseline characteristics, stratified by cigarette, pipe and cigar use

	Never users of cigarette or pipe/cigar (N=9931)	Exclusive cigarette users (N=10 271)	Exclusive pipe/cigar users (N=518)	Cigarette and pipe/cigar users (N=2103)	Total (N=22 823)
Age, mean±SD, years	46.7±16.4	47.2±15.0	54.2±13.2	56.7±12.7	48.0±15.7
Male, number (%)	3230 (32.5)	4488 (43.7)	508 (98.1)	2027 (96.4)	10 253 (44.9)
Race/ethnicity, number (%)*					
White	6479 (65.2)	7441 (72.4)	425 (82.0)	1735 (82.5)	16 080 (70.5)
Black	2886 (29.1)	2500 (24.3)	89 (17.2)	331 (15.7)	5806 (25.4)
Hispanic	276 (2.8)	231 (2.2)	4 (0.8)	26 (1.2)	537 (2.4)
Other race	290 (2.9)	99 (1.0)	0 (0.0)	11 (0.5)	400 (1.8)
Education, number (%)*					
Less than high school	755 (7.6)	1226 (11.9)	37 (7.1)	204 (9.7)	2222 (9.7)
High school	2874 (28.9)	3227 (31.4)	111 (21.4)	475 (22.6)	6687 (29.3)
Some college	2040 (20.5)	1964 (19.1)	52 (10.0)	348 (16.5)	4404 (19.3)
College or more	4262 (42.9)	3854 (37.5)	318 (61.4)	1076 (51.2)	9510 (41.7)
BMI, mean±SD	26.9 (5.5)	26.8 (5.2)	27.8 (4.1)	27.6 (4.1)	26.9 (5.2)
Cigarette pack-years, mean±SD	NA	21.7 (21.4)	NA	28.5 (15.9)	22.9 (22.4)
Cigarettes per day, mean±SD	NA	17.3 (12.3)	NA	21.3 (13.6)	18.0 (12.6)
Pipe/cigar use					
Current pipe/cigar use, number (%)	NA	NA	221 (43.4)	429 (20.9)	650 (2.9)
Pipe/cigar age of initiation, mean±SD	NA	NA	25.9 (8.6)	29.7 (10.7)	29.0 (10.5)
Pipe/cigar years, mean±SD	NA	NA	15.0 (12.4)	10.7 (11.0)	11.3 (11.3)
Lung function					
% predicted FEV_1 baseline, mean±SD,	98.3 (14.2)	93.0 (16.5)	97.0 (14.4)	92.3 (17.1)	95.3 (15.8)
% predicted FVC baseline, mean±SD	99.9 (13.4)	97.8 (14.1)	98.2 (13.1)	96.6 (14.2)	98.6 (13.8)
FEV_1/FVC baseline, mean±SD	0.78 (0.07)	0.75 (0.08)	0.75 (0.06)	0.72 (0.09)	0.76 (0.08)
Number of spirometry measures, median (Q1–Q3)	3 (2-4)	2 (2-3)	2 (2-3)	2 (2-3)	2 (2-3)
Years of follow-up, mean±SD	13.7 (10.3)	12.2 (9.7)	10.5 (8.9)	9.3 (8.3)	12.6 (9.9)
Cohort*					
ARIC	4699 (47.3)	5211 (50.7)	297 (57.3)	1240 (59.0)	11 447 (50.2)
CARDIA	2534 (25.5)	1906 (18.6)	32 (6.2)	120 (5.7)	4592 (20.1)
FHS-O	877 (8.8)	1642 (16.0)	75 (14.5)	111 (5.3)	2705 (11.9)
HABC	654 (6.6)	506 (4.9)	83 (16.0)	368 (17.5)	1611 (7.1)
MESA	1167 (11.8)	1006 (9.8)	31 (6.0)	264 (12.6)	2468 (10.8)

*Table shows column percentage.
ARIC, Atherosclerosis Risk in Communities; BMI, body mass index; CARDIA, Coronary Artery Risk Development in Young Adults; FEV_1, forced expiratory volume in 1 s; FHS-O, Framingham Offspring Study; FVC, forced vital capacity; HABC, Health Aging and Body Composition; MESA, Multi-Ethnic Study of Atherosclerosis.

cigar use showed similar magnitude of effects on lung function as the results with the pooled exposure definition (online supplemental eFigure 4). Sensitivity analyses among former smokers showed qualitatively similar, though statistically insignificant, results (online supplemental eFigure 5).

Clinical outcomes

Of 27 129 participants with follow-up for clinical events, there was a median follow-up of 22.7 years (Q1–Q3 12.2–28.4) for all-cause mortality and CHD-related mortality, 22.2 years (Q1–Q3 11.8–28.5) for respiratory-related mortality, 21.6 years (Q1–Q3 11.7–28.2) for CHD-related events and 14.9 years (Q1–Q3 10.0–24.0) for COPD-related events (online supplemental eTable 6).

Among never users of cigarettes, exclusive pipe/cigar use was associated with increased hazards of all-cause mortality (aHR 1.24, 95% CI 1.08 to 1.41) and COPD-related hospitalisation or death (asHR 2.02, 95% CI 1.41 to 2.90) (table 2, figure 2). Significant associations with CHD-related and respiratory-related mortality were not observed, although event rates were relatively low. In sensitivity analyses including males only (online supplemental eTable 8) and additional control for eGFR (online supplemental eTable 9), results were qualitatively similar. Results were consistent when stratified by cohort (online supplemental eFigure 6). Survival curves for all clinical outcomes for pipe/cigar use among all participants (online supplemental eFigure 7) and among never cigarette users (online supplemental eFigure 8) are available in the online supplemental materials.

DISCUSSION

Pipe or cigar use was associated with greater decline in lung function and adverse clinical outcomes in a large, multiethnic,

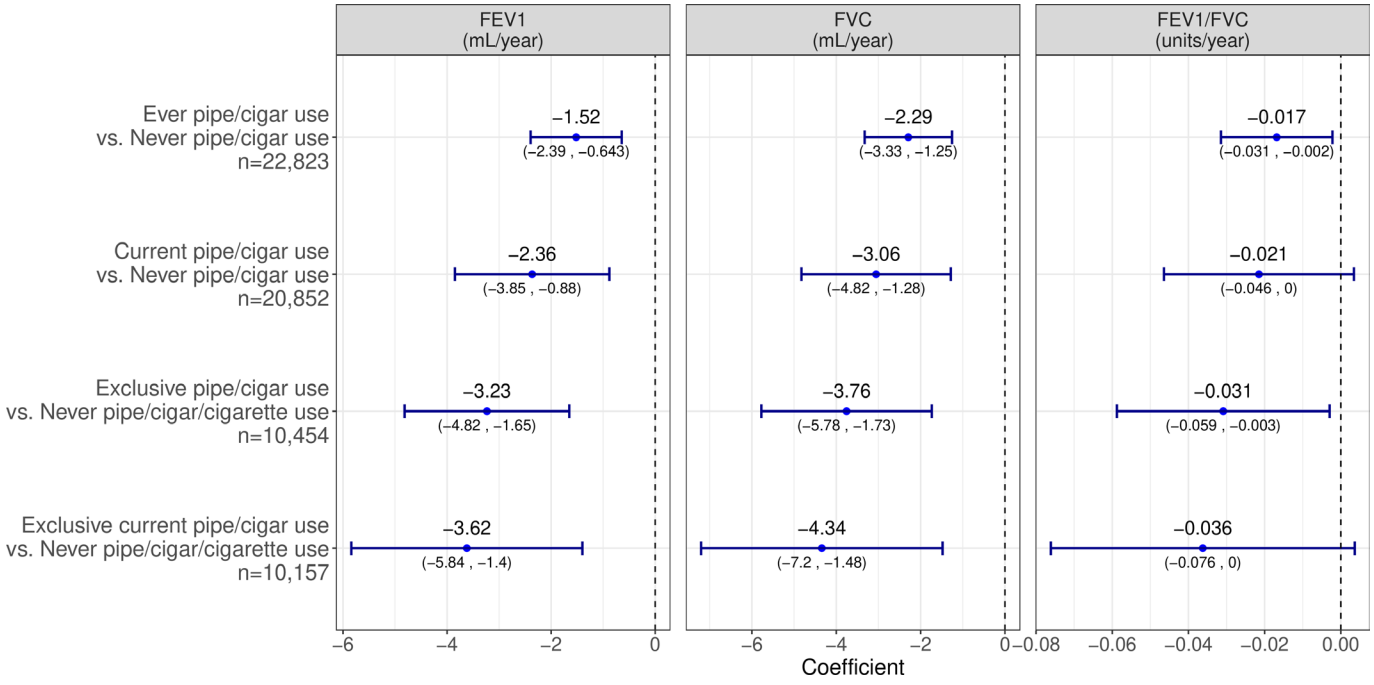

Figure 1 Associations between pipe or cigar use and lung function decline. Lung function decline was estimated using linear mixed models with study-specific unstructured variance/covariance matrices of the visit-specific residuals. The coefficient for the interaction term of exposure and age was interpreted as the longitudinal association with rate of change in lung function. All models were adjusted by age, sex, race/ethnicity, educational attainment, height, weight, study cohort and site. For analyses including current or former cigarette users, models were adjusted for cigarette smoking status (time-varying) and pack-years of cigarette smoking at baseline. Multiplicative interaction terms with age were included for all time-invariant covariates. Error bars represent 95% CIs. FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity.

US general population-based meta-cohort of adults. Among adults with no history of cigarette use, exclusive pipe or cigar use was associated with greater all-cause mortality as well as CHD-related and COPD-related hospitalisation or death. These findings support the need for prevention and cessation efforts targeting non-cigarette tobacco use and suggest that adults with a history of pipe or cigar use, with or without cigarette smoking, warrant enhanced cardiopulmonary risk mitigation.

This is the first large US general population-based study, to our knowledge, to establish associations of pipe or cigar use with accelerated loss of lung function, including in adults who never smoked cigarettes. The observed lung function decline among pipe and cigar users is likely mediated through similar pathophysiological mechanisms as cigarette smoking. Combusted tobacco smoke contains chemical constituents including reactive oxygen species, carcinogens and inflammatory mediators. These compounds trigger oxidative stress through generation of free radicals, causing DNA damage, lipid peroxidation and protein oxidation in airway epithelium.[28] Chronic inflammation in turn promotes protease-antiprotease imbalance and airway remodelling characteristic of obstructive lung disease.[29]

While the estimated annualised decrease of 3–4 mL per year in FEV_1 and FVC is modest in absolute terms, it was equivalent to approximately 13% of the unadjusted mean decline in non-users of pipes or cigars. A prior systematic review of 26 studies found that continuing cigarette smokers had 10.8 mL per year greater decline in FEV_1 compared with never smokers,[30] and more recent results from the NHLBI Pooled Cohorts Study demonstrated an approximately 9 mL/year greater FEV_1 decline among current cigarette smokers compared with never smokers.[21] Thus, the effect magnitude we observed for pipe/cigar use represents approximately 30%–40% of that seen with cigarette smoking. This attenuated effect could be due to differences in inhalation patterns, with pipe/cigar users practising more, but not exclusively, oropharyngeal inhalation.[10] Differences in use frequency may also result in differences in cumulative exposure,[14] though the unfiltered nature of pipe and cigar smoke may partially offset this through increased toxicant delivery per smoking episode.[4]

Our results are consistent with prior cross-sectional results from the MESA study, showing that greater years of pipe smoking were associated with lower FEV_1 and greater years of cigar smoking were associated with lower FEV_1/FVC.[31] However, whereas that cross-sectional analysis associated ever and exclusive pipe/cigar use with prevalent AL, we did not identify associations with incident AL over longitudinal follow-up; rather, we identified an association of exclusive pipe/cigar use with incident PRISm. This finding nonetheless has clinical implications, since PRISm has been linked to increased risks of cardiovascular and respiratory morbidity and mortality.[25] Our results regarding accelerated lung function decline are broadly consistent with results from the Copenhagen City Heart Study, where decline in FEV_1 was greatest for exclusive cigar or cheroot users compared with any other tobacco use group or non-users of tobacco.[32] Our study examined both former and current pipe or cigar users, of whom current users demonstrated slightly greater acceleration in lung function decline, whereas the Copenhagen City study was limited to current users.

Pipe or cigar use was also associated with increased risk of all-cause mortality and cardiopulmonary events among never-users of cigarettes. Our results showing increased risks of all-cause mortality among exclusive pipe or cigar users are consistent with recent results from the National Longitudinal Mortality Study, showing elevated risks of mortality associated with exclusive cigar use.[5] Although prior research generated mixed results, our findings confirm and extend several prior studies that had notable limitations, including reliance on data collected

Table 2 Associations between pipe or cigar use and clinical outcomes

	All-cause mortality	CHD-related mortality	CHD-related events	Respiratory-related mortality	COPD-related events
Entire sample (N=27 129)					
Ever pipe/cigar					
Events (at risk)	1753 (3454)	260 (3454)	608 (3325)	107 (3092)	534 (2951)
Incidence density rate per 10K person-years	291.00	43.16	109.89	20.47	123.09
Never pipe/cigar					
Events (at risk)	7804 (23 675)	799 (23 675)	2593 (23 327)	440 (19 516)	2134 (15 882)
Incidence density rate per 10K person-years	159.93	16.37	55.46	11.14	82.81
HR for ever pipe/cigar vs never pipe/cigar (95% CI, p value)	1.05 (0.99 to 1.11, 0.12)	0.94 (0.80 to 1.10, 0.44)	1.00 (0.90 to 1.10, 0.94)	0.98 (0.77 to 1.25, 0.90)	1.09 (0.98 to 1.21, 0.12)
Never cigarette users (N=11 781)					
Exclusive pipe/cigar					
Events (at risk)	305 (640)	49 (640)	117 (614)	9 (520)	46 (493)
Incidence density rate per 10K person-years	250.80	40.29	105.59	9.32	57.13
Never cigarette or pipe/cigar					
Events (at risk)	2887 (11 141)	291 (11 141)	915 (11 029)	81 (9490)	324 (7423)
Incidence density rate per 10K person-years	120.75	12.17	39.36	4.05	25.67
HR for exclusive pipe/cigar vs never cigarette or pipe/cigar (95% CI, p value)	1.24 (1.08 to 1.41, 0.002)	1.10 (0.78 to 1.54, 0.60)	1.16 (0.93 to 1.44, 0.19)	1.48 (0.68 to 3.23, 0.32)	2.02 (1.41 to 2.90, <0.001)

HRs for all-cause mortality were calculated using Cox proportional hazards regression.
All other HRs are subdistribution HRs from Fine-Grey subdistribution hazard models. Models were adjusted for baseline age, sex, race/ethnicity, education, study site, BMI, and diagnosis of hypertension, coronary heart disease, heart failure and stroke. Regressions were stratified by study, allowing for cohort-specific differences in the underlying baseline risk.
BMI, body mass index; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease.

over 50 years ago, limited racial and ethnic diversity and/or minimal adjustment for potential confounders, such as prevalent cardiometabolic disease.[33–35]

Of note, concurrent or alternating use of cigarettes, pipes and cigars is common,[36] as evidenced by the relatively small sample size of exclusive pipe/cigar users in our population. We found no statistically significant increased risk of all-cause mortality, COPD-related events and CHD-related events among ever pipe/cigar users when including concurrent or former cigarette users. We suspect that this was due to the strong association of these outcomes with cigarette use—which was adjusted for in these analyses—and thus should not detract from the overall findings that pipe or cigar use was associated with a range of adverse health outcomes.

Our findings have several implications for public health messaging and clinical practice. First, they strengthen the evidence base supporting health warnings on pipe and cigar products. Second, our results showing adverse health effects of pipe and cigar use support recent government reports advocating for the elimination of excise tax disparities between tobacco products to reduce economic incentives for substitution.[9] Finally, our results highlight the need for greater clinical attention to pipe and cigar use in smoking cessation guidelines and clinical pathways, which often focus predominantly on cigarette smoking without explicit guidance or evidence for pipe/cigar use.[37]

Strengths of our study include a large, multiethnic, US general population-based sample; prospective longitudinal lung function measurement and event ascertainment; and analysis of both ever and exclusive pipe/cigar use, with robust confounder adjustment. Nevertheless, there are several relevant limitations. First, use of cigarettes, pipes and cigars was self-reported and thus subjected to potential reporting biases and misclassification. Previous research based on these data has suggested that misclassification of smoking status, if present, is likely small.[21] Second, we were unable to capture variation in frequency, duration and volume of pipe or cigar use, or inhalation patterns, which could modify the health risks of these products. Relatedly, we were unable to capture variation in the design and types of pipes and cigars, which may carry different cardiopulmonary risks.[14] Included cohorts also did not assess for cannabis use, and some cigar use may be more related to cannabis rather than tobacco use. Recent evidence has shown that consumption of cannabis in a tobacco-derived cigar shell may increase the risk of starting cigarettes or tobacco-based cigars.[38] Additionally, pipe or cigar users in our sample were predominantly male, which may limit generalisability of the results to female pipe or cigar users. This limitation is common to most pipe and cigar research, as males are significantly more likely to use non-cigarette tobacco products.[39] Nonetheless, our results in analyses limited to male participants were consistent with the main results. Our estimates of lung function were based on prebronchodilator spirometry, as postbronchodilator spirometry was unavailable. However, prebronchodilator spirometry has been shown to predict health outcomes with similar accuracy to postbronchodilator spirometry in population studies.[40] Finally, pooling data across cohorts can introduce heterogeneity, yet data were systematically

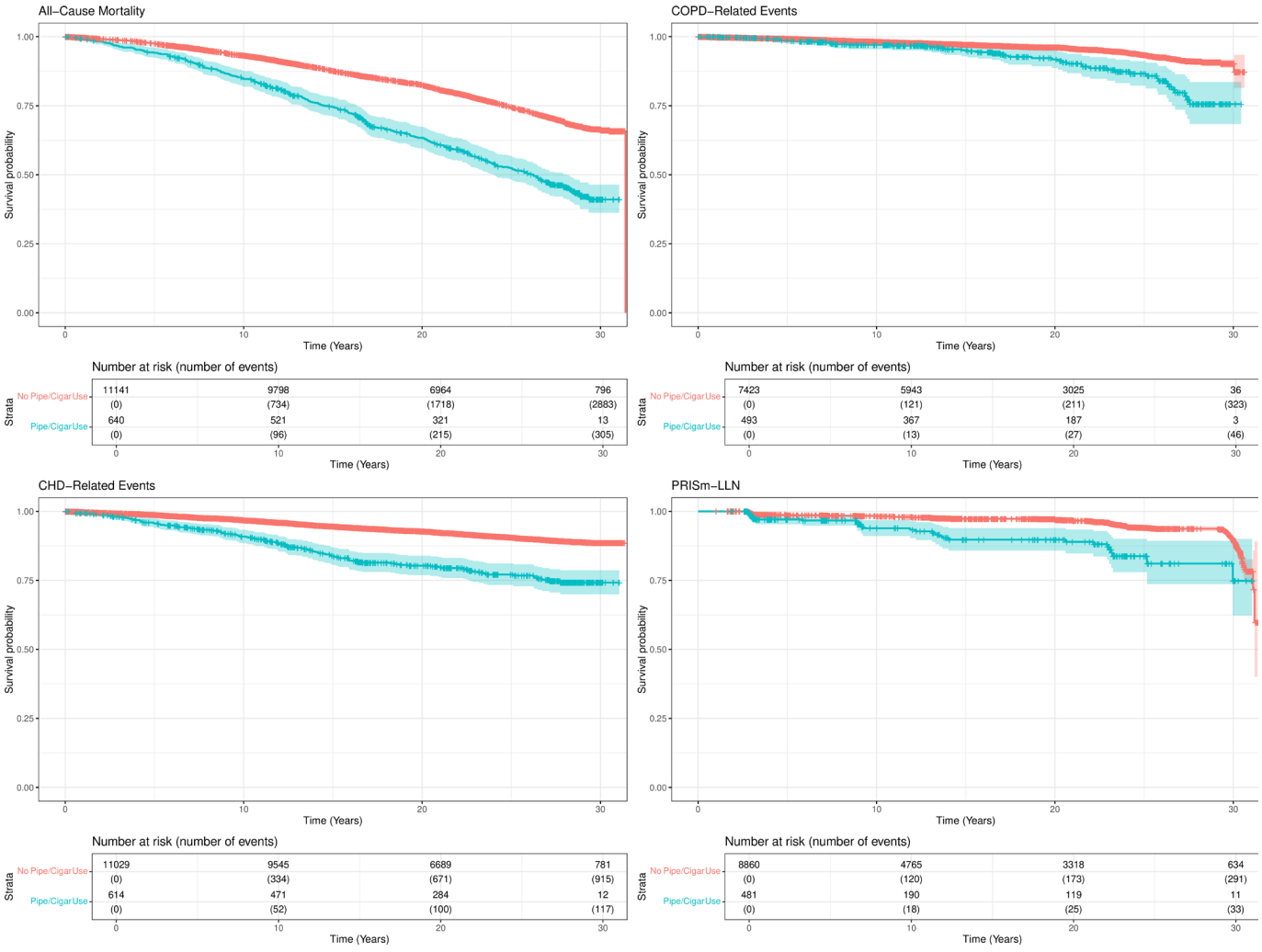

Figure 2 Survival curves for exclusive pipe/cigar use among never cigarette users. CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; LLN, limit of normal; PRISm, preserved ratio impaired spirometry.

harmonised, analyses accounted for both cohort and site effects, and analyses fully stratified by cohort yielded similar results.

These findings show that pipe or cigar use was associated with accelerated lung function decline and adverse cardiovascular and respiratory health outcomes, independent of cigarette use, in a multiethnic population of US adults. Our results highlight the need for comprehensive interventions for education, prevention, cessation and mitigation of the risks of non-cigarette tobacco use.

Author affiliations
[1]Department of Medicine, Columbia University Vagelos College of Physicians and Surgeons, New York, New York, USA
[2]Columbia University Mailman School of Public Health, New York, New York, USA
[3]Division of Pulmonary, Allergy and Critical Care Medicine, University of Alabama At Birmingham, Birmingham, Alabama, USA
[4]Department of Biostatistics, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA
[5]Tobacco Control Research Branch, Division of Cancer Control and Population Sciences, National Cancer Institute, Rockville, Maryland, USA
[6]Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, Minnesota, USA
[7]Division of Pulmonary/Critical Care, Northwestern University, Chicago, Illinois, USA
[8]Department of Medicine, The University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA
[9]National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, USA
[10]Division of Epidemiology and Community Health, University of Minnesota School of Public Health, Minneapolis, Minnesota, USA
[11]Department of Psychiatry, Stony Brook University Renaissance School of Medicine, Stony Brook, New York, USA
[12]Undergraduate Training and Education Center, Tougaloo College, Jackson, Mississippi, USA
[13]Department of Critical Care Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania, USA
[14]Department of Epidemiology, Colorado School of Public Health, University of Colorado Anschutz Medical Center, Aurora, Colorado, USA

Contributors WMG is the guarantor of the content of the manuscript and takes responsibility for the integrity of the data and the accuracy of the data analysis. WMG, PB, JES, TRS and EO contributed to study design. WMG performed the data analysis and drafted the manuscript. WMG, PB, CME, JEM, SPB, DC, NDF, DRJ, RK, LL, SJL, PLL, JES, WW, SY, TRS and EO revised the manuscript and had final responsibility for the decision to submit for publication.

Funding This work was supported by The National Heart, Lung, and Blood Institute Pooled Cohorts Study that has been funded with federal funds from the National Heart, Lung, and Blood Institute and National Institutes of Health (R21-HL121457, R21-HL-129924, and K23-HL-130627). The Atherosclerosis Risk in Communities study has been funded in whole or in part with Federal funds from the National Heart, Lung, and Blood Institute, National Institutes of Health, Department of Health and Human Services, under contract numbers (75N92022D00001, 75N92022D00002, 75N92022D00003, 75N92022D00004, 75N92022D00005). The authors thank the staff and participants of the ARIC study for their important contributions. The Coronary Artery Risk Development in Young Adults Study (CARDIA) is conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the University of Alabama at Birmingham (75N92023D00002 & 75N92023D00005), Northwestern University (75N92023D00004), University

of Minnesota (75N92023D00006) and Kaiser Foundation Research Institute (75N92023D00003).This manuscript has been reviewed by CARDIA for scientific content. The Framingham Heart Study (FHS) acknowledges the support of contracts NO1-HC-25195, HHSN268201500001I and 75N92019D00031 from the National Heart, Lung, and Blood Institute and grant supplement R01 HL092577-06S1 for this research. We also acknowledge the dedication of the FHS study participants without whom this research would not be possible. From the Health, Aging, and Body Composition Study, this research was supported by National Institute on Aging (NIA) Contracts N01-AG-6-2101; N01-AG-6-2103; N01-AG-6-2106; NIA grant R01-AG028050 and NINR grant R01-NR012459. This research was funded in part by the Intramural Research Program of the NIH, National Institute on Aging. From the Multi-Ethnic Study of Atherosclerosis, this research was supported by contracts 75N92020D00001, HHSN268201500003I, N01-HC-95159, 75N92020D00005, N01-HC-95160, 75N92020D00002, N01-HC-95161, 75N92020D00003, N01-HC-95162, 75N92020D00006, N01-HC-95163, 75N92020D00004, N01-HC-95164, 75N92020D00007, N01-HC-95165, N01-HC-95166, N01-HC-95167, N01-HC-95168 and N01-HC-95169 from the National Heart, Lung, and Blood Institute, and by grants UL1-TR-000040, UL1-TR-001079, and UL1-TR-001420 from the National Center for Advancing Translational Sciences (NCATS). The authors thank the other investigators, the staff, and the participants of the MESA study for their valuable contributions. A full list of participating MESA investigators and institutions can be found at http://www.mesa-nhlbi.org. This paper has been reviewed and approved by the MESA Publications and Presentations Committee. CME was supported by National Institutes of Health grant KL2TR001874.

Disclaimer The views expressed in this manuscript are those of the authors and do not necessarily represent the views of the National Heart, Lung, and Blood Institute; the National Institutes of Health; or the US Department of Health and Human Services.

Competing interests CME is an employee and shareholder in Merck & Co., Inc. SPB has received fees for consulting or advisory boards from Apreo, AstraZeneca, Boehringer Ingelheim, Chiesi, Connect Biopharma, Genentech, GSK, Merck, Polarean, Sanofi, Regeneron and Verona Pharma. His institute has received funds for research from COPD Foundation, Sanofi, Genentech and Nuvaira. He has received honouraria for CME from Horizon CME, Illuminate Health, Integritas Communications, IntegrityCE and Medscape. RK has had consultancy agreements with CVS Caremark, AstraZeneca, GlaxoSmithKline and Boehringer Ingelheim. He has participated in research with AstraZeneca, PneumRx/BTG and Spiration, for which his institution has been remunerated.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by Columbia University Irving Medical Center Institutional Review Board (approval number IRB-AAAB1971). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer-reviewed.

Data availability statement Data are available upon reasonable request. Data are available on reasonable request to the corresponding author.

ORCID iDs
William M Gardner https://orcid.org/0000-0002-7607-3586
Christina M Eckhardt https://orcid.org/0000-0002-3249-926X
Surya P Bhatt https://orcid.org/0000-0002-8418-4497
Stephanie J London https://orcid.org/0000-0003-4911-5290
Pamela L Lutsey https://orcid.org/0000-0002-1572-1340
Sachin Yende https://orcid.org/0000-0002-6596-8034

REFERENCES

1 Beaglehole R, Bonita R, Horton R, *et al*. Priority actions for the non-communicable disease crisis. *The Lancet* 2011;377:1438–47.
2 Brauer M, Roth GA, Aravkin AY, *et al*. Global burden and strength of evidence for 88 risk factors in 204 countries and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. *The Lancet* 2024;403:2162–203.
3 Cornelius ME, Loretan CG, Jamal A, *et al*. Tobacco Product Use Among Adults – United States, 2021. *MMWR Morb Mortal Wkly Rep* 2023;72:475–83.
4 National Cancer Institute. *Cigars: Health Effects and Trends. Tobacco Control Monograph No.9*. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, 1998.
5 Christensen CH, Rostron B, Cosgrove C, *et al*. Association of Cigarette, Cigar, and Pipe Use With Mortality Risk in the US Population. *JAMA Intern Med* 2018;178:469–76.
6 Sharma E, Tang Z, Lauten K, *et al*. Cardiovascular disease outcomes among established cigar users 40 years and older: Findings from the population assessment of tobacco and health (PATH) study, waves 1-5 (2013-2019). *Prev Med Rep* 2024;37:102569.
7 Wang TW, Kenemer B, Tynan MA, *et al*. Consumption of Combustible and Smokeless Tobacco - United States, 2000-2015. *MMWR Morb Mortal Wkly Rep* 2016;65:1357–63.
8 Bernat JK, Ferrer RA, Margolis KA, *et al*. US adult tobacco users' absolute harm perceptions of traditional and alternative tobacco products, information-seeking behaviors, and (mis)beliefs about chemicals in tobacco products. *Addict Behav* 2017;71:38–45.
9 U.S. Government Accountability Office. *Tobacco Taxes: Federal Revenue Implications of Tax Rate Differences and Drawback Refunds*. Washington (DC): U.S. Government Accountability Office, 2025.
10 Pechacek TF, Folsom AR, de Gaudermaris R, *et al*. Smoke exposure in pipe and cigar smokers. Serum thiocyanate measures. *JAMA* 1985;254:3330–2.
11 McDonald LJ, Bhatia RS, Hollett PD. Deposition of cigar smoke particles in the lung: evaluation with ventilation scan using (99m)Tc-labeled sulfur colloid particles. *J Nucl Med Off Publ Soc Nucl Med* 2002;43:1591–5.
12 IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Tobacco smoke and involuntary smoking. *IARC Monogr Eval Carcinog Risks Hum* 2004;83:1–1438.
13 Wackowski OA, Kurti M, Schroth KRJ, *et al*. Examination of Voluntary Compliance with New FDA Cigar Warning Label Requirements. *Tob Regul Sci* 2020;6:379–83.
14 The National Academies of Sciences, Engineering, and Medicine; Health and Medicine Division; Board on Population Health and Public Health Practice; Committee on Patterns of Use and Health Effects of "Premium Cigars" and Priority Research. In: *Premium Cigars: Patterns of Use, Marketing, and Health Effects*. Washington (DC): National Academies Press (US), 2022.
15 Oelsner EC, Balte PP, Cassano PA, *et al*. Harmonization of Respiratory Data From 9 US Population-Based Cohorts: The NHLBI Pooled Cohorts Study. *Am J Epidemiol* 2018;187:2265–78.
16 THE ARIC INVESTIGATORS. THE ATHEROSCLEROSIS RISK IN COMMUNITIES (ARIC) STUDY: DESIGN AND OBJECTIVES. *Am J Epidemiol* 1989;129:687–702.
17 Friedman GD, Cutter GR, Donahue RP, *et al*. CARDIA: study design, recruitment, and some characteristics of the examined subjects. *J Clin Epidemiol* 1988;41:1105–16.
18 Manson JE, Bassuk SS. Invited Commentary: The Framingham Offspring Study-A Pioneering Investigation Into Familial Aggregation of Cardiovascular Risk. *Am J Epidemiol* 2017;185:1103–8.
19 Goodpaster BH, Carlson CL, Visser M, *et al*. Attenuation of skeletal muscle and strength in the elderly: The Health ABC Study. *J Appl Physiol* 2001;90:2157–65.
20 Bild DE, Bluemke DA, Burke GL, *et al*. Multi-Ethnic Study of Atherosclerosis: objectives and design. *Am J Epidemiol* 2002;156:871–81.
21 Oelsner EC, Balte PP, Bhatt SP, *et al*. Lung function decline in former smokers and low-intensity current smokers: a secondary data analysis of the NHLBI Pooled Cohorts Study. *Lancet Respir Med* 2020;8:34–44.
22 Miller MR, Hankinson J, Brusasco V, *et al*. Standardisation of spirometry. *Eur Respir J* 2005;26:319–38.
23 Hankinson JL, Odencrantz JR, Fedan KB. Spirometric Reference Values from a Sample of the General U.S. Population. *Am J Respir Crit Care Med* 1999;159:179–87.
24 Quanjer PH, Brazzale DJ, Boros PW, *et al*. Implications of adopting the Global Lungs Initiative 2012 all-age reference equations for spirometry. *Eur Respir J* 2013;42:1046–54.
25 Wan ES, Balte P, Schwartz JE, *et al*. Association Between Preserved Ratio Impaired Spirometry and Clinical Outcomes in US Adults. *JAMA* 2021;326:2287–98.
26 Cox DR. Regression Models and Life-Tables. *Journal of the Royal Statistical Society Series B* 1972;34:187–202.
27 Fine JP, Gray RJ. A Proportional Hazards Model for the Subdistribution of a Competing Risk. *J Am Stat Assoc* 1999;94:496–509.
28 Aoshiba K, Nagai A. Oxidative stress, cell death, and other damage to alveolar epithelial cells induced by cigarette smoke. *Tob Induc Dis* 2003;1:219–26.
29 Macnee W, Rahman I. Oxidants and antioxidants as therapeutic targets in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1999;160:S58–65.
30 Lee PN, Fry JS. Systematic review of the evidence relating FEV1 decline to giving up smoking. *BMC Med* 2010;8:84.
31 Rodriguez J, Jiang R, Johnson WC, *et al*. The association of pipe and cigar use with cotinine levels, lung function, and airflow obstruction: a cross-sectional study. *Ann Intern Med* 2010;152:201–10.
32 Lange P, Groth S, Nyboe J, *et al*. Decline of the lung function related to the type of tobacco smoked and inhalation. *Thorax* 1990;45:22–6.
33 Chang CM, Corey CG, Rostron BL, *et al*. Systematic review of cigar smoking and all cause and smoking related mortality. *BMC Public Health* 2015;15:390.

34 Henley SJ, Thun MJ, Chao A, *et al*. Association between exclusive pipe smoking and mortality from cancer and other diseases. *J Natl Cancer Inst* 2004;96:853–61.
35 Wald NJ, Watt HC. Prospective study of effect of switching from cigarettes to pipes or cigars on mortality from three smoking related diseases. *BMJ* 1997;314:1860–3.
36 Antognoli E, Koopman Gonzalez S, Trapl E, *et al*. Cigarettes, Little Cigars, and Cigarillos: Initiation, Motivation, and Decision-Making. *Nicotine Tob Res* 2018;20:S5–11.
37 Leone FT, Zhang Y, Evers-Casey S, *et al*. Initiating Pharmacologic Treatment in Tobacco-Dependent Adults. An Official American Thoracic Society Clinical Practice Guideline. *Am J Respir Crit Care Med* 2020;202:e5–31.
38 Fairman BJ, Kimmel HL, Blanco C, *et al*. Blunt and non-blunt cannabis use associated with cigarette, e-cigarette, and cigar initiation: Findings from the population assessment of tobacco and health (PATH) study. *Drug Alcohol Depend* 2023;246:109837.
39 Corey CG, Holder-Hayes E, Nguyen AB, *et al*. US Adult Cigar Smoking Patterns, Purchasing Behaviors, and Reasons for Use According to Cigar Type: Findings From the Population Assessment of Tobacco and Health (PATH) Study, 2013–2014. *Nicotine Tob Res* 2018;20:1457–66.
40 Mannino DM, Diaz-Guzman E, Buist S. Pre- and post-bronchodilator lung function as predictors of mortality in the Lung Health Study. *Respir Res* 2011;12:136.