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Lung function decline over 25 years of follow-up among black and white adults in the ARIC study cohort

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Abstract

Background—Interpretation of longitudinal information about lung function decline from middle to older age has been limited by loss to follow-up that may be correlated with baseline lung function or the rate of decline. We conducted these analyses to estimate age-related decline in lung

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function across groups of race, sex, and smoking status while accounting for dropout from the Atherosclerosis Risk in Communities Study.

Methods—We analyzed data from 13,896 black and white participants, aged 45–64 years at the 1987–1989 baseline clinical examination. Using spirometry data collected at baseline and two follow-up visits, we estimated annual population-averaged mean changes in forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) by race, sex, and smoking status using inverse-probability-weighted independence estimating equations conditioning-on-being-alive.

Results—Estimated rates of FEV_1 decline estimated using inverse-probability-weighted independence estimating equations conditioning on being alive were higher among white than black participants at age 45 years (e.g., male never smokers: black: −29.5 ml/year; white: −51.9 ml/year), but higher among black than white participants by age 75 (black: −51.2 ml/year; white: −26). Observed differences by race were more pronounced among men than among women. By smoking status, FEV_1 declines were larger among current than former or never smokers at age 45 across all categories of race and sex. By age 60, FEV_1 decline was larger among former and never than current smokers. Estimated annual declines generated using unweighted generalized estimating equations were smaller for current smokers at younger ages in all four groups of race and sex compared with results from weighted analyses that accounted for attrition.

Keywords

Aging; Epidemiology; Lung function tests; Respiratory; Spirometry

1. Introduction

Low measures of pulmonary function are diagnostic for chronic obstructive pulmonary disease (COPD) and characteristic of other lung conditions. Longitudinal research provides evidence that spirometric measures of pulmonary function also predict the development of arrhythmias, risk of coronary heart disease, heart failure, cognitive decline, and mortality in the general population, even among individuals without known lung disease and among non-smokers [1–8]. Because of these associations, however, pulmonary function may affect continued participation in the very prospective studies used to evaluate trajectories of lung function. If baseline lung function affects continued participation, then this potential source of bias may interfere with the interpretation of results on rates of lung function decline over time [9].

A recent study of the association between smoking and cognitive decline attempted to account for loss to follow-up that may depend on baseline levels of both smoking and cognitive function [10]. Similar methods have not been applied to the study of decline of lung function with age. To date, epidemiologic studies of rates of lung function decline have focused largely on describing discrepancies between estimates generated using cross-sectional versus longitudinal data [11–13], assessing modification of the effects of smoking on lung function decline [14–17], and evaluating associations between genetic variation and lung function decline [18,19]. While one study examined lung function declines leading to COPD [20] and several have examined differences in lung function decline by race, sex, and

smoking status [12,13,17], longitudinal studies have not generally accounted for the potential influences of dropout during study follow-up. Results from such longitudinal studies, based on participants healthy enough to continue participating, may underestimate rates of lung function decline in the target population.

In longitudinal studies of lung function, individuals who do not continue to participate may do so due to death during the study follow-up or withdrawal (i.e., dropout) from the study for reasons other than death. While existing literature cautions against making inferences as if death did not occur by extrapolating observations beyond death [21,22], new statistical methods are now available to address loss to follow-up–that is, study attrition–among individuals who were alive at the time of the follow-up observation, but did not continue to participate [22,23]. Considering the documented associations between lung function and both morbidity and mortality [1–8], distinguishing between attrition due to death and dropout for reasons other death could plausibly influence estimates of lung function decline. By taking into account non-death loss to follow-up, new statistical methods may improve estimation of lung function decline generated from longitudinal studies [22,23].

The Atherosclerosis Risk in Communities (ARIC) study provides an opportunity to extend our understanding about lung function decline by evaluating variation in patterns of age-related changes in lung function in a large, population-based cohort of black and white adults in the United States. We used data from three clinical examinations spanning approximately 25 years to estimate rates of decline in forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) across groups of race, sex, and cigarette smoking status. Our analyses examined quantitative changes in pulmonary function, rather than diagnoses of chronic obstructive pulmonary disease or other conditions. Because low measures of FVC and FEV_1 are risk factors for morbidity and mortality in the general population, even among individuals with measures in the normal range [24,25], quantitative changes in FVC and FEV_1 are valuable metrics of pulmonary health regardless of whether such changes reach a threshold for impairment. We accounted for non-participation in follow-up visits using inverse-probability-weighted independence estimating equations with population-averaged linear models for regression conditioning-on-being-alive [22,23].

2. Methods

2.1. Study population

The ARIC study is a prospective cohort study designed to evaluate the etiology of atherosclerosis and its clinical sequelae in a general population based sample of adults [26]. Men and women, aged 45–64 years, were recruited and enrolled from four U.S. communities: Forsyth County, North Carolina; Jackson, Mississippi; suburbs of Minneapolis, Minnesota; and Washington County, Maryland. Initial examination of the cohort took place in 1987–1989 (‘visit 1’, n = 15,792), when participants responded to health-related questionnaires and completed a clinical examination. Follow-up examinations occurred in 1990–1992 (‘visit 2’, n = 14,348; 93% of those still alive) when participants were 48–67 years of age, 1993–1995 (‘visit 3’), 1996–1998 (‘visit 4’), and 2011–2013 (‘visit 5’, n = 6538; 65% of those still alive) when participants were 65–90 years of age. Of the 15,792 adults who completed visit 1, 13,896 were included in our final analysis after

sequentially excluding participants based on the following criteria: data use restricted by participant consent (n = 41), race other than black or white (n = 48), black participants recruited from suburbs of Minneapolis, Minnesota or Washington County, Maryland (n = 54), incomplete spirometry at visit 1 (n = 138), errors in recording spirometry data (n = 182), and inadequate participant effort (n = 1433). The study protocol and instruments were approved by institutional review boards at each of the four participating exam sites and the data coordinating center, and all participants provided written informed consent. The analyses presented here were exempted from institutional review board review at the Centers for Disease Control and Prevention.

2.2. Spirometry

FEV_1 and FVC were measured at visits 1 and 2 using Collins Survey II water-seal spirometers (Warren E. Collins Inc., Braintree, MA) and at visit 5 using SensorMedics model 1022 dry rolling seal spirometers (OMI, Houston, TX). At each visit, spirometry testing protocols were standardized across the four ARIC field centers, calibration checks were performed daily, and the standardization of data collection and management was coordinated across field centers by a single pulmonary function reading center. For the present analysis, we used two spirometry measurements: FEV_1 and FVC, both in milliliters (ml). At each visit, we selected each participant's best FEV_1 and FVC of three acceptable maneuvers, based on the centralized expert review.

2.3. Covariates

Age, race, and sex were self-reported and height was measured at the visit 1 exam. Body mass index, cigarette smoking status, history of asthma, and history of chronic lung diseases other than asthma were defined and updated at visits 1, 2, and 5, respectively, using information collected at each of the visits and via annual follow-up telephone calls.

2.4. Statistical analysis

To estimate FEV_1 and FVC at age 45 years, the FEV_1/FVC ratio at age 45 years, and annual changes in FEV_1 and FVC across categories of race, sex, and smoking status, we used inverse-probability-weighted independence estimating equations with population-averaged linear models for regression conditioning-on-being-alive [22,23,27]. The statistical models are described in detail in the online supplement.

Because the statistical models included a non-linear specification of time, our results generated annual changes in FEV_1 and FVC that are not constant over the follow-up period. That is, the estimated annual changes are not constant across the range of ages included in our analysis and therefore cannot be summarized with a single set of race-, sex-, and smoking status-specific coefficients. To most clearly present these results, we calculated population-averaged mean estimates of FEV_1 and FVC, with 95% confidence intervals (CIs) at age 45 years to represent baseline measures of lung function for each of the 12 categories of race, sex, and smoking status and present estimated annual declines in FEV_1 and FVC at the a priori selected ages of 45, 60, and 75 years. Estimated annual declines at age 45 years were calculated as the change from ages 45 to 46; estimated annual declines at age 60 years

were calculated as the change from ages 60 to 61, and estimated annual declines at age 75 years were calculated as the change from ages 75–76 years.

For comparison, we also estimated annual declines in FEV_1 and FVC in models fitted using unweighted generalized estimating equations (GEE) with independence working correlation matrices. These models were specified identically to the regression conditioning-on-being-alive models. Empirical sandwich variance estimators were used for both weighted and unweighted modeling approaches.

3. Results

3.1. Characteristics of the study population

Characteristics at visit 1 of the 13,896 participants in the analysis are shown in Table 1. Participant dropouts and deaths that occurred in the follow-up periods between visits 1, 2, and 5, the visits at which spirometry was conducted, are shown in Table S1 in the online supplement. At visit 1, the prevalence of asthma was 6.4% and the prevalence of other chronic obstructive lung diseases was 5.2%. Participants identified as former smokers or never smokers at visit 1 largely maintained that same smoking status throughout follow-up, with 95.2% of former smokers and 88.9% of never smokers maintaining their smoking status through visits 2 and 5. For traits that changed over time, characteristics at visit 5 are shown in Table S2.

3.2. Lung function at age 45 years

Adjusted population-averaged estimates of FEV_1, FVC, and the FEV_1/FVC ratio at age 45 were each lower for current smokers than for former and never smokers across all categories of race and sex (Table 2). Estimates of both FEV_1 and FVC at age 45 were lower among black than white participants within categories of sex and smoking status and among women than men within categories of race and smoking status. Within categories of smoking status, FEV_1/FVC ratios at age 45 years were largely similar across categories of race and sex.

3.3. Lung function decline by age, race, and sex

As expected, predicted values of FEV_1 and FVC declined with advancing age (Fig. 1). Broadly speaking, estimated rates of FEV_1 decline varied by race and were higher among white than black participants at age 45 years (e.g., among male never smokers: black: −29.5 ml/year [95% CI: −43.9, −15.0]; white: −51.9 ml/year [95% CI: −58.9, −44.8]), but higher among black than white participants by age 75 years (black: −51.2 ml/year [95% CI: −73.3, −29.2]; white: -26.6 ml/year [95% CI: −37.4, −15.7]), though 95% confidence intervals of the estimates largely overlap (Table 3). FVC declined similarly to FEV_1 at age 45 years (e.g., among male never smokers: black: −45.0 [95% CI: −62.9, −27.0]; white: −66.8 [95% CI: −74.7, -59.0)] and at age 75 years (black: −33.3 [95% CI: −59.5, −7.1]; white: −18.3 [95% CI: −30.8, −5.8]). These observed differences in estimated rates of decline by race were more pronounced among men than among women.

3.4. Lung function decline by smoking status

By smoking status, estimated rates of FEV_1 decline were larger among current smokers than former or never smokers at age 45 years across all categories of race and sex (Table 3). By age 60, FEV_1 decline was larger among former and never smokers than among current smokers across all categories of race and sex. At age 75 years, the small number of current smokers in all groups resulted in FEV_1 declines that were imprecise. In fact, for current smokers, changes in both FEV_1 and FVC at ages older than 75 years were imprecise and we opted not to present them in Fig. 1. With increasing age and within categories of race and sex, FEV_1 decline among former and never smokers attenuated among white men and women, whereas it remained relatively constant among black women and increased among black men.

3.5. Comparison of results generated using inverse-probability-weighted regression conditioning-on-being-alive and unweighted GEE models

Regression coefficients for the inverse-probability-weighted regression conditioning-on-being-alive models used to generate estimates shown in Fig. 1 and Tables 2–4 are shown in Tables S3 and S4, in the online supplement. Estimates of lung function at age 45 years generated using unweighted GEE (Tables S5, in the online supplement) were similar in magnitude and precision to those generated in our main analysis. In contrast, the annual declines in FEV_1 and FVC generated using unweighted GEE were smaller for current smokers at younger ages in all four groups of race and sex compared with results from weighted analyses that accounted for attrition. For example, for FEV_1, the rate of change in 45 year-old, white, male, current smokers was −59.6 ml/year in unweighted analyses (Table 4) versus −67.0 ml/year in analyses conducted using inverse-probability-weighted regression conditioning-on-being-alive (Table 3). A similar pattern was observed for FVC. Differences between the unweighted and weighted methods attenuated by age 60 years. At age 75 years, by which time substantial attrition of current smokers had occurred, estimates generated using both methods were correspondingly unstable.

4. Discussion

Using approximately 25 years of follow-up information, we estimated annual population-averaged changes in FEV_1 and FVC within each category of race, sex, and smoking status. Our analyses generated patterns of declines in FEV_1 and FVC that varied notably by smoking status and age, with largest declines at younger ages among current smokers and at older ages among former and never smokers. Despite the methods we applied to account for the decline in study participation over time this finding may be a function of the lower baseline FEV_1 values among current smokers than among former or never smokers or differences between smokers who survived to old age and other smokers. Our analyses also generated differences in estimated rates of decline by race; declines were higher among white than black participants at age 45 years, but higher among black than white participants by age 75. Differences observed by race were more pronounced among men than among women.

Two notable strengths of our study are the approximately 25 years of follow-up information available and the statistical approach we applied to account for attrition during the lengthy follow-up period. Broadly speaking, previous studies of lung function decline have not implemented formal statistical methods to account for attrition. Previous studies of other outcomes have handled attrition using different methods [28,29]. For example, under attrition missing-at-random assumptions, longitudinal random effects [30] and traditional inverse-probability-weighted regression [31] approaches have addressed the potential bias resulting from the diminished representativeness of the study population at later visits through specification of the intra-subject correlation structure or the use of weights derived from a model that does not distinguish between sources of attrition. In a refinement of the latter approach, Weuve et al. [10] derived weights from inverse-probability-weighted regression by joining results from separate models for death and dropout, allowing for different mechanisms of death and dropout, and thus weighting the data such that the study population at later visits more closely represents the original study population. Making inferences as if death did not occur and, as a result, extrapolating health metrics beyond death have been noted as important limitations of these approaches [21,22].

In contrast, the approach we applied assumes that the study population is continuously re-defined by death and attempts to estimate lung function decline over time, conditioning on being alive (and therefore eligible for continued participation). Using this method, we generated weights based on predicted probabilities of each participant continuing to participate in the ARIC study conditioning on his/her being alive. We then applied the predicted probabilities to up-weight the contribution of participants with demographic and health-related characteristics of those not observed due to non-death dropout. If dropout from the ARIC study were differentially associated with lung function, then we expect that applying these methods would reduce the underestimation of lung function decline. Indeed, comparing results generated using this modeling strategy to those generated using unweighted GEE provides evidence that unweighted GEE models resulted in estimates of lung function decline that were smaller in magnitude, though 95% CIs for estimates generated using the two methods largely overlapped. Due to the methods we applied, our results are presented as population-averaged estimates and cannot be used to draw conclusions about individual-level lung function declines. Overall, the results presented here suggest that these novel methods are a valuable analytic improvement in estimating lung function decline in long follow-up studies.

Comparing our results to previously published age-related rates of lung function decline is not straight-forward. Because we regressed FEV_1 and FVC on an array of covariates that included quadratic terms for follow-up time, the final estimated annual rates of decline varied non-linearly with age. To simplify the presentation of our results, we selected *a priori* three ages for which to present point estimates of the estimated annual rates of decline: ages 45, 60, and 75 years. In light of previous research reporting higher rates of lung function decline among smokers than among non-smokers [13,15,32], our finding that within categories of race and sex, annual declines in both FEV_1 and FVC were greater among current smokers than former smokers and never smokers at younger ages, but greater among former smokers and never smokers than current smokers at older ages, is novel. Better measures of health among current smokers than among former and never smokers, such as

the estimated annual declines we report at older ages, is characteristic of the effect of selection bias described as the ‘healthy smoker effect’ [33]. For comparison with a study conducted in a population ranging in age from 19 to 70 years that did not estimate declines at different ages, overall rates of annual decline over 13 years of follow-up were greater among smokers (men: −59.8 ml/year; women:−42.2 ml/year) than among non-smokers (men: −45.8 ml/year; women: −38.6 ml/year) [13]. From a study conducted among lifetime non-smokers, Ware et al. (1990) reported an age-related acceleration of the annual decline in FEV_1 among both men and women [12]. Together, these findings provide evidence of variation in longitudinal FEV_1 decline by sex and smoking status, but did not account for the extent to which loss to follow-up due to death, illness, and dropout affected the magnitude of or variation in the observed declines. In a recent meta-analysis of 16 longitudinal studies, the combined estimate for FEV_1 decline was −26.9, −29.2 and −35.7 ml/year in never, former, and persistent smokers, respectively [32]. Most recently, Lange et al. reported on trajectories in lung function in three cohorts and reported declines in FEV_1 ranging from −19 ml/year in the Lovelace Smokers Cohort to −25 ml/year in the Copenhagen City Heart Study [20]. In our study, results from models that accounted for attrition for reasons other than death and results from GEE models that did not differed most notably among current smokers at the youngest ages. Given the differential loss to follow-up among smokers, this finding may not be unexpected, since rapid decline in lung function rendered smokers underrepresented at our last follow-up visit, as shown by the comparison of population characteristics at baseline (Table 1) versus at visit 5 (Table S2). Indeed, weights used in our analyses did not take into account the increased mortality among smokers (Table S1) and, because pulmonary function testing was not included in clinic exams 3 and 4, we are unable to draw conclusions about changes in lung function, particularly among younger smokers, that may have been detected at those visits.

Between clinical examinations at study visits 2 and 5, there were changes in spirometry equipment. Variability in spirometry measurements collected using different devices has been described [34], however, side-by-side comparisons that would allow us to assess the validity or precision of the spirometry measurements across visits were not available for this analysis. To improve the internal validity of the spirometry data, Collins Survey II spirometers were used at all four field centers at visits 1 and 2, calibration checks were performed daily, and the standardization of data collection and management was overseen by a single pulmonary function reading center. At visit 5, all spirometry was conducted using SensorMedics spirometers and quality control was again coordinated across field centers. For all four field centers, quality assurance methods included training and practice for all technicians performing spirometry testing, review of spirometry tests performed, and feedback about the quality of the testing performed. The value of such practical training for spirometry technicians is well-described [35]. Changes in the spirometry equipment over time are not expected to have affected estimates of lung function decline differentially across groups of race, sex, or smoking status. Nonetheless, because our group-specific estimates of lung function decline pertain to the cohort of ARIC participants who were aged 45–64 years at the baseline visit, our models were adjusted for period effects that may have accounted for changes in spirometry equipment over time. The role of period effects in our statistical models is further described in the online supplement.

Our analyses did not take into account secondhand tobacco smoke and occupational exposures or other exposures that may affect lung function over time. The analyses also did not take into account pack-years of cigarette smoking or the duration of smoking prior to visit 1, nor did they take into account precise quit dates among participants who began the study as current smokers and transitioned to former smokers during follow-up. If these or other factors that may affect lung function over time are distributed unequally across categories of race, sex, or smoking status, then our results may have been affected by residual confounding. By including time-varying information about body mass index, our analyses did account for changes in participant body weight over time. Our models did not evaluate whether the observed declines in lung function with increasing weight were equivalent in men and women.

In summary, we applied inverse-probability-weighted regression conditioning-on-being-alive methods to generate estimates of age-related declines in FEV_1 and FVC across categories of race, sex, and smoking status, while accounting for non-death dropout during approximately 25-years of follow-up of the ARIC study population. Our findings demonstrate a consistent pattern of decline in FEV_1 and FVC over time in all categories of race, sex, and smoking status. Declines varied most notably by smoking status and age, with largest declines occurring among current smokers at the youngest ages. Compared with the weighted methods we used, unweighted methods not accounting for attrition underestimated declines in lung function among current smokers at younger ages. Thus, our findings suggest that using statistical methods to account for differential dropout may be worthwhile when evaluating decline in lung function over a lengthy follow-up period. Since spirometric measures of pulmonary function are diagnostic for COPD and predictive of cardiovascular morbidity and mortality in the general population, our estimates of lung function decline provide valuable information about the extent to which race, sex, and smoking status may influence trajectories of lung function decline during middle age and later in life. Additional improvements in our understanding of these and other risk factors for lung function decline may continue to improve our insight into the mechanisms by which lung function influences illness and predicts mortality.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

ARIC	Atherosclerosis Risk in Communities
CI	confidence interval
COPD	chronic obstructive pulmonary disease
FEV_1	forced expiratory volume in one second
FVC	forced vital capacity
GEE	generalized estimating equations
ml	milliliters

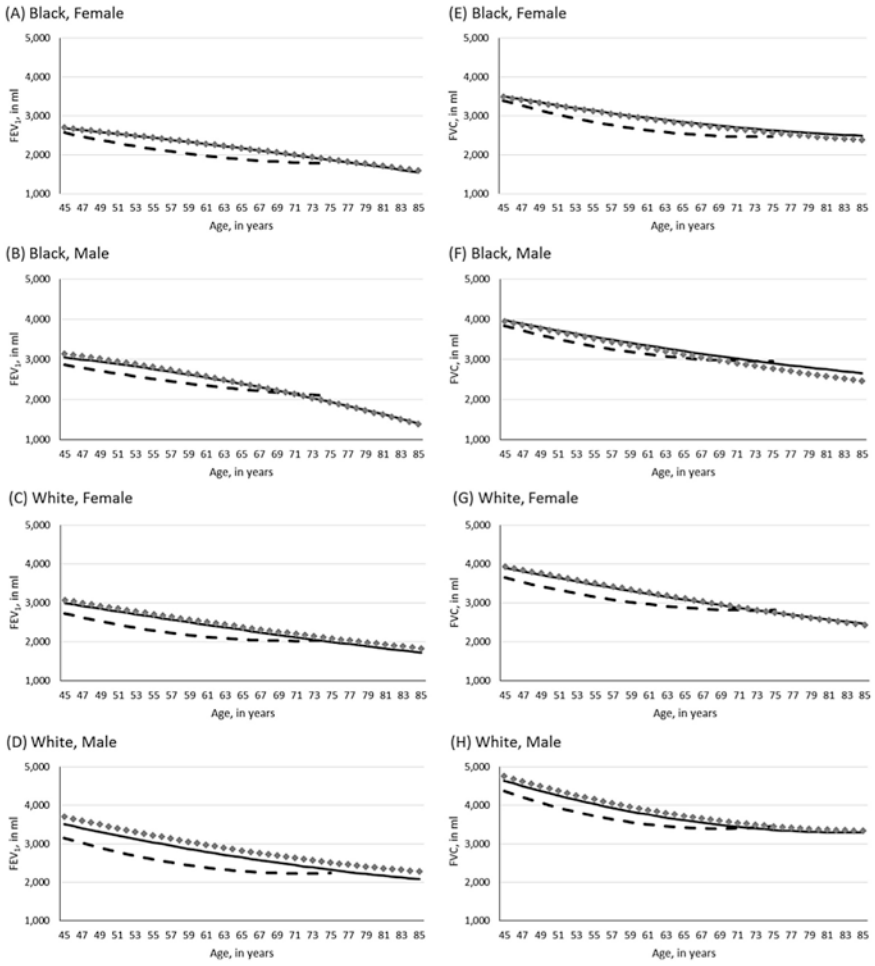

Fig. 1.

Predicted FEV_1 (panels A-D) and FVC (panels E-H) among current smokers (dashed black line), former smokers (solid black line), and never smokers (dotted gray line) for the cohort of ARIC study participants aged 45–64 years at baseline, by race and sex.

Table 1

Demographic and health-related characteristics of the final study sample at visit 1: the ARIC Study.

	All Participants	Race	
		Black	White
	No. (%)[a]	No. (%)[a]	No. (%)[a]
Total	13,896 (100.0)	3428 (24.7)[b]	10,468 (75.3)[b]
Study center			
Forsyth County, NC	3627 (26.1)	383 (11.2)	3244 (31.0)
Jackson, MS	3045 (21.9)	3045 (88.8)	–
Suburbs of Minneapolis, MN	3655 (26.3)	–	3655 (34.9)
Washington County, MD	3569 (25.7)	–	3569 (34.1)
Demographic characteristics			
Age			
45–49	3768 (27.1)	1094 (31.9)	2674 (25.5)
50–54	3639 (26.2)	926 (27.0)	2713 (25.9)
55–59	3362 (24.2)	732 (21.4)	2630 (25.1)
60–64	3127 (22.5)	676 (19.7)	2451 (23.4)
Sex			
Female	7705 (55.4)	2112 (61.6)	5593 (53.4)
Male	6191 (44.6)	1316 (38.4)	4875 (46.6)
Health-related characteristics			
Asthma			
No	13,006 (93.6)	3208 (93.6)	9798 (93.6)
Yes	890 (6.4)	220 (6.4)	670 (6.4)
Body mass index			
≤18.4	122 (0.9)	37 (1.1)	85 (0.8)
18.5–24.9	4529 (32.6)	712 (20.8)	3817 (36.5)
25.0–29.9	5498 (39.6)	1297 (37.8)	4201 (40.1)
≥30.0	3747 (27.0)	1382 (40.3)	2365 (22.6)
Chronic obstructive lung disease[c]			
No	13,173 (94.8)	3270 (95.4)	9903 (94.6)
Yes	723 (5.2)	158 (4.6)	565 (5.4)
Smoking status			
Current smoker	3603 (25.9)	1037 (30.3)	2566 (24.5)
Former smoker	4500 (32.4)	808 (23.6)	3692 (35.3)
Never smoker	5793 (41.7)	1583 (46.2)	4210 (40.2)
Pack-years of smoking			
0	5854 (42.1)	1595 (46.5)	4259 (40.7)
0.01–10.99	1913 (13.8)	555 (16.2)	1358 (13.0)
11.00–24.74	1989 (14.3)	530 (15.5)	1459 (13.9)
24.75–38.99	1947 (14.0)	356 (10.4)	1591 (15.2)

	All Participants	Race	
		Black	White
	No. (%)[a]	No. (%)[a]	No. (%)[a]
≥39.00	1876 (13.5)	268 (7.8)	1608 (15.4)
Unknown	317 (2.3)	124 (3.6)	193 (1.8)

[a]Column percentages, unless otherwise noted.

[b]Row percentages.

[c]Other than asthma.

Table 2

Population-average estimates of FEV_1, FVC, and FEV_1/FVC at age 45 years, by race, sex, and baseline smoking status: the ARIC Study.

Characteristic	FEV_1, in ml	FVC, in ml	FEV_1/FVC, %
	Mean (95% CI)[a]	Mean (95% CI)[a]	Mean (95% CI)[a]
Black			
Female			
Current smoker	2565 (2510–2620)	3391 (3328–3454)	75.5 (74.5–76.4)
Former smoker	2680 (2620–2739)	3499 (3434–3565)	76.6 (75.6–77.6)
Never smoker	2695 (2643–2747)	3489 (3429–3548)	77.7 (76.9–78.5)
Male			
Current smoker	2874 (2806–2943)	3841 (3765–3917)	74.5 (73.5–75.6)
Former smoker	3052 (2982–3121)	3974 (3897–4051)	76.4 (75.4–77.4)
Never smoker	3143 (3075–3211)	3956 (3876–4036)	79.1 (78.1–80.0)
White			
Female			
Current smoker	2732 (2696–2768)	3651 (3611–3692)	74.3 (73.7–74.8)
Former smoker	3002 (2969–3035)	3908 (3871–3945)	77.0 (76.5–77.6)
Never smoker	3079 (3050–3108)	3940 (3906–3973)	78.7 (78.3–79.2)
Male			
Current smoker	3152 (3108–3195)	4372 (4324–4420)	72.6 (72.0–73.3)
Former smoker	3503 (3465–3540)	4637 (4595–4679)	76.1 (75.5–76.6)
Never smoker	3702 (3663–3741)	4751 (4706–4796)	78.7 (78.1–79.2)

[a]Adjusted for asthma history, body mass index, chronic obstructive lung disease other than asthma, height, follow-up time, and study center.

Table 3

Population-averaged rateof change in $FEV_{1,}$ and FVC in ml/year, at ages 45, 60, and 75 years, generated using inverse-probability-weighted independence estimating equations conditioning-on-being-alive, stratified by race, sex, and visit-specific smoking status: the ARIC Study.

Characteristic	Spirometry measure	Age, in years		
		45	60	75
		Mean (95% CI)[a]	Mean (95% CI)[a]	Mean (95% CI)[a]
Black				
Female				
Current smoker	FEV_1	−48.7 (−59.6, −37.8)	−25.6 (−33.4,−17.8)	−2.5 (−20.8, 15.8)
	FVC	−65.3 (−77.6,−53.1)	−29.6 (−38.5, −20.7)	6.1 (−14.7, 26.8)
Former smoker	FEV_1	−23.3 (−33.0, −13.6)	−27.1 (−30.6, −23.6)	−31.0 (−45.9, −16.0)
	FVC	−39.9 (−50.6,−29.1)	−28.7 (−32.5, −25.0)	−17.6 (−33.6,−1.5)
Never smoker	FEV_1	−24.8 (−32.5,−17.1)	−27.1 (−30.0, −24.1)	−29.4 (−41.4,−17.3)
	FVC	−39.5 (−48.2, −30.8)	−30.5 (−33.8,−27.1)	−21.5 (−35.2, −7.8)
Male				
Current smoker	FEV_1	−40.0 (−54.7, −25.2)	−25.5 (−34.3, −16.7)	− 11.0 (−34.6, 12.6)
	FVC	−60.6 (−78.1, −43.2)	−28.1 (−40.0, −16.2)	4.4 (−24.5, 33.2)
Former smoker	FEV_1	−24.8 (−38.7, −10.9)	−37.2 (−42.8, −31.6)	−49.6 (−71.3, −28.0)
	FVC	−43.2 (−59.2, −27.2)	−35.2 (−41.4, −29.0)	−27.2 (−51.9, −2.6)
Never smoker	FEV_1	−29.5 (−43.9, −15.0)	−40.4 (−47.7,−33.1)	−51.2 (−73.3, −29.2)
	FVC	−45.0 (−62.9, −27.0)	−39.1 (−48.2, −30.1)	−33.3 (−59.5,−7.1)
White				
Female				
Current smoker	FEV_1	−53.1 (−60.6, −45.7)	−22.0 (−26.5,−17.6)	9.1 (−4.1, 22.2)
	FVC	−58.9 (−67.2, −50.5)	−26.8 (−31.9, −21.6)	5.3 (−10.0, 20.6)
Former smoker	FEV_1	−37.4 (−44.0, −30.8)	−33.2 (−35.4, −31.0)	−29.1 (−39.2, −18.9)
	FVC	−45.9 (−53.0, −38.8)	−38.3 (−40.7, −36.0)	−30.7 (−41.6, −19.9)
Never smoker	FEV_1	−38.5 (−43.2, −33.9)	−32.8 (−34.4, −31.2)	−27.1 (−34.3, −19.9)
	FVC	−44.8 (−50.3, −39.3)	−39.4 (−41.4, −37.4)	−34.0 (−42.5, −25.4)
Male				
Current smoker	FEV_1	−67.0 (−76.6, −57.5)	−29.0 (−35.9,−22.1)	9.0 (−7.7, 25.7)
	FVC	−79.8 (−89.7, −69.9)	−28.8 (−36.1, −21.6)	22.1 (3.1, 41.2)
Former smoker	FEV_1	−50.0 (−57.1, −42.9)	−38.9 (−41.5, −36.3)	−27.8 (−38.8, −16.8)
	FVC	−68.1 (−75.8, −60.5)	−41.7 (−44.5, −38.9)	−15.3 (−27.0,−3.6)
Never smoker	FEV_1	−51.9 (−58.9, −44.8)	−39.2 (−42.0, −36.4)	−26.6 (−37.4, −15.7)
	FVC	−66.8 (−74.7, −59.0)	−42.5 (−46.0,−39.1)	−18.3 (−30.8, −5.8)

[a]Estimated for participants aged 45–64 years at baseline and adjusted for baseline characteristics (age, height, study center) and time-varying characteristics collected at baseline and updated at visits 2 and 5 (asthma history, body mass index, chronic obstructive lung disease other than asthma, follow-up time).

Table 4

Estimated annual declines in FEV_1 and FVC, in ml/year, at ages 45, 60, and 75 years, generated using unweighted generalized estimating equations, stratified by race, sex, and smoking status: the ARIC Study.

	Spirometry measure	Age, in years		
		45	60	75
		Mean (95% CI)[a]	Mean (95% CI)[a]	Mean (95% CI)[a]
Black				
Female				
Current smoker	FEV_1	−38.1 (−47.0, −29.1)	−28.0 (−35.3, −20.8)	−18.0 (−33.7, −2.4)
	FVC	−54.9 (−64.9, −44.9)	−32.3 (−40.6, −24.0)	−9.7 (−27.6, 8.2)
Former smoker	FEV_1	−19.6 (−27.2,−12.0)	−28.2 (−31.3, −25.0)	−36.7 (−48.4,−25.1)
	FVC	−34.7 (−43.4, −25.9)	−30.2 (−33.7, −26.7)	−25.8 (−38.8, −12.7)
Never smoker	FEV_1	−22.5 (−28.4, −16.5)	−27.7 (−30.3, −25.0)	−32.8 (−42.5, −23.2)
	FVC	−36.4 (−43.4, −29.5)	−31.2 (−34.2, −28.2)	−26.0 (−37.2, −14.8)
Male				
Current smoker	FEV_1	−28.2 (−40.4, −16.0)	−29.0 (−37.7, −20.3)	−29.9 (−50.1, −9.7)
	FVC	−51.5 (−65.6, −37.5)	−30.8 (−42.1, −19.5)	−10.0 (−34.4, 14.3)
Former smoker	FEV_1	−17.7 (−28.5,−6.8)	−37.1 (−41.7, −32.6)	−56.6 (−73.2, −39.9)
	FVC	−37.6 (−49.9, −25.2)	−35.0 (−40.3, −29.7)	−32.4 (−51.5,−13.2)
Never smoker	FEV_1	−24.0 (−35.6, −12.4)	−40.0 (−47.3, −32.8)	−56.1 (−73.8, −38.5)
	FVC	−41.5 (−55.4, −27.7)	−38.2 (−46.9, −29.5)	−34.8 (−55.3, −14.4)
White				
Female				
Current smoker	FEV_1	−47.2 (−53.4, −41.0)	−22.8 (−27.0, −18.5)	1.7 (−9.9, 13.3)
	FVC	−53.3 (−60.2, −46.4)	−26.7 (−31.5, −21.9)	−0.1 (−13.2, 13.0)
Former smoker	FEV_1	−37.6 (−42.8, −32.5)	−31.8 (−33.6, −30.0)	−25.9 (−33.8,−18.1)
	FVC	−45.3 (−50.9, −39.7)	−36.8 (−38.8, −34.9)	−28.4 (−36.9, −19.9)
Never smoker	FEV_1	−40.4 (−43.8,−37.1)	−31.2 (−32.6, −29.8)	−22.0 (−27.3, −16.6)
	FVC	−46.5 (−50.4, −42.7)	−37.3 (−39.0, −35.7)	−28.2 (−34.3, −22.0)
Male				
Current smoker	FEV_1	−59.6 (−67.3, −51.9)	−30.4 (−36.9, −23.8)	− 1.2 (−16.0, 13.7)
	FVC	−72.5 (−80.9, −64.2)	−30.5 (−37.6, −23.4)	11.6 (−5.0, 28.2)
Former smoker	FEV_1	−47.6 (−53.0, −42.2)	−37.0 (−39.2, −34.8)	−26.4 (−34.7, −18.0)
	FVC	−64.3 (−70.2, −58.5)	−40.4 (−42.9, −37.9)	−16.5 (−25.7, −7.3)
Never smoker	FEV_1	−51.6 (−56.9, −46.3)	−37.6 (−40.1,−35.1)	−23.6 (−31.9,−15.2)
	FVC	−65.3 (−71.3, −59.3)	−40.6 (−43.7, −37.5)	−16.0 (−25.7, −6.2)

[a] Estimated for participants aged 45 years at baseline and adjusted for baseline characteristics (age, height, study center) and time-varying characteristics collected at baseline and updated at visits 2 and 5 (asthma history, body mass index, chronic obstructive lung disease other than asthma, follow-up time).