Respiratory Medicine 113 (2016) 57–64

Contents lists available at ScienceDirect

Respiratory Medicine

journal homepage: www.elsevier.com/locate/rmed

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

Maria C. Mirabelli [a, *], John S. Preisser [b], Laura R. Loehr [c], Sunil K. Agarwal [d], R. Graham Barr [e], David J. Couper [b], John L. Hankinson [f], Noorie Hyun [b], Aaron R. Folsom [g], Stephanie J. London [h]

[a] Air Pollution and Respiratory Health Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health, Centers for Disease Control and Prevention, 4770 Buford Highway NE, Mailstop F–60, Atlanta, GA 30341, USA
[b] Department of Biostatistics, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, 3101 McGavran-Greenberg Hall, CB 7420, 135 Dauer Drive, Chapel Hill, NC 27599, USA
[c] Department of Epidemiology, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, 170 Rosenau Hall, CB 7400, 135 Dauer Drive, Chapel Hill, NC 27599, USA
[d] Department of Medicine, Johns Hopkins University, 2020 E. Monument Street, Room B-321, Baltimore, MD 21287, USA
[e] Department of Medicine, College of Physicians and Surgeons, Columbia University Medical Center, 630 West 168th Street, New York, NY 10032, USA
[f] Hankinson Consulting, Inc., 1860 Barnett Shoals Road, Suite 103, PMB 505, Athens, GA 30605-6821, USA
[g] Division of Epidemiology and Community Health, University of Minnesota, 1300 S. 2nd Street, Suite 300, Minneapolis, MN 55454, USA
[h] Epidemiology Branch, National Institute of Environmental Health Sciences, National Institutes of Health, Department of Health and Human Services, 111 TW Alexander Drive, PO Box 12233, MD A3-05, Research Triangle Park, NC 27709, USA

ARTICLE INFO

Article history:
Received 5 October 2015
Received in revised form
1 February 2016
Accepted 7 February 2016
Available online 11 February 2016

Keywords:
Aging
Epidemiology
Lung function tests
Respiratory
Spirometry

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 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.

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.

* Corresponding author.
E-mail address: mmirabelli@cdc.gov (M.C. Mirabelli).

http://dx.doi.org/10.1016/j.rmed.2016.02.003
0954-6111/Published by Elsevier Ltd.

Conclusions: Using methods accounting for dropout from an approximately 25-year health study, estimated rates of lung function decline varied by age, race, sex, and smoking status, with largest declines observed among current smokers at younger ages.

Published by Elsevier Ltd.

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