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Difference in airflow obstruction between Hispanic and non-Hispanic white female smokers

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Abstract

Rationale—Smoking-related respiratory diseases are a major cause of morbidity and mortality. However, the relationship between smoking and respiratory disease has not been well-studied among ethnic minorities in general and among women in particular.

Objective—The objective of this cross-sectional study was to evaluate the risk of airflow obstruction and to assess lung function among Hispanic and non-Hispanic white (NHW) female smokers in a New Mexico cohort.

Methods—Participants completed a questionnaire detailing smoking history and underwent spirometry testing. Outcomes studied included airflow obstruction, selected lung function

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parameters, and chronic mucus hyper-secretion. Chi square, logistic, and linear regression techniques were utilized.

Main findings—Of the 1,433 eligible women participants, 248 (17.3%) were Hispanic; and 319 had airflow obstruction (22.3%). Hispanic smokers were more likely to be current smokers, and report lower pack-years of smoking, compared to NHW smokers ($p < 0.05$ for all analyses). Further, Hispanic smokers were at a reduced risk of airflow obstruction compared to NHW smokers, with an O.R. of 0.51, 95% C.I. 0.34, 0.78 ($p = 0.002$) after adjustment for age, BMI, pack-years and duration of smoking, and current smoking status. Following adjustment for covariates, Hispanic smokers also had a higher mean absolute and percent predicted post-bronchodilator FEV_1/FVC ratio, as well as higher mean percent predicted FEV_1 ($p < 0.05$ for all analyses).

Principal conclusions—Hispanic female smokers in this New Mexico-based cohort had lower risk of airflow obstruction and better lung function than NHW female smokers. Further, smoking history did not completely explain these associations.

Keywords

Hispanic ethnicity; Smokers; Airflow obstruction; Pulmonary function; Chronic mucus hyper-secretion; Women

INTRODUCTION

Respiratory diseases related to cigarette smoking are a major cause of morbidity and mortality in the United States (U.S.). Although the epidemiology of smoking-related respiratory diseases among non-Hispanic whites (NHW) in the U.S. is well-described, less is known about the relationship between smoking and respiratory diseases among ethnic minorities in general and among women in particular.

Although both prevalence of smoking and mortality from chronic obstructive pulmonary disease (COPD) have been reported to be lower among Hispanics than among NHW nationwide [1–4], these findings have to be interpreted cautiously because of the heterogeneity of U.S. Hispanics with regard to racial ancestry, country of origin, place of birth, area of residence, pattern of tobacco use, and prevalence of obstructive airway diseases such as asthma [5–7]. Among Hispanic adults who participated in a nationwide survey in the U.S., current smoking was more frequently reported by men than women, and by Puerto Ricans than by Mexicans or by members of other Hispanic subgroups [6]. Consistent with these findings, a previous survey had reported a lower prevalence of self-reported chronic bronchitis among Mexican Americans than among Puerto Ricans in general and among women in particular [7]. To date, only one study has compared the prevalence of spirometric airflow obstruction between NHW and members of a specific Hispanic subgroup (Mexican Americans) among adults in the U.S. [8].

Hispanics in New Mexico comprise two different groups: those living in the southern part of the state are primarily of recent Mexican descent, while those residing in northern New Mexico trace their ancestry to the original Spanish conquistadores and colonists who settled several centuries ago [9]. Previous surveys suggest that Hispanics exhibit both a lower prevalence and mortality from self-reported chronic respiratory diseases than NHW in New Mexico, which may be explained by ethnic differences in smoking habits [2, 10–13]. Consistent with these findings, a 2006 survey of New Mexican residents showed that the proportion of never smokers was higher among Hispanics (59.7%; 95% C.I. 56.8–62.6%) than among NHW (50.9%; 95% C.I. 48.7–53.1%) [14].

The objective of the present study was to examine whether Hispanic ethnicity was associated with a reduced risk of objectively defined airflow obstruction and improved overall lung function among female smokers in a cohort study in New Mexico, particularly after adjusting for smoking.

MATERIALS AND METHODS

Study Population

The study subjects were drawn from eligible women participants in a cohort study in New Mexico (Lovelace Smokers' Cohort), between March 2001 and July 2006. Since women are underrepresented in most studies of airflow obstruction, this large cohort of female ever-smokers was assembled to study the susceptibility of women to the adverse effects of cigarette smoking [15]. The catchment area for this cohort was Albuquerque, NM and its surrounding communities, comprising a population of approximately 700,000 persons. Most participants were recruited through newspaper or television advertisements and were paid a small stipend for their participation. This study was approved by the Western Institutional Review Board (Olympia, WA).

Inclusion Criteria

Women were included in the study if they were ages 40 to 75 years, former or current smokers with a minimum smoking history of 20 pack-years on initial screening, and able to understand English.

Study Measurements

All tests were conducted at Lovelace Scientific Resources (Albuquerque, NM). Information related to demographics, respiratory diseases, and smoking was obtained by self-report from all study participants via a questionnaire. The smoking-related variables studied were pack-years, age of onset, duration of smoking, time since smoking cessation in ex-smokers, and current smoking status at the time of testing. Body mass index (BMI) was measured using standardized methods [16].

Pre- and post-bronchodilator spirometry were obtained on all subjects by certified and registered respiratory therapists strictly adhering to the 1994 American Thoracic Society (ATS) guidelines [17]. After completion of baseline spirometry, all subjects were given two puffs of albuterol (90 mcg/spray metered dose inhaler) with a LiteAire® dual valve spacer (Thayer Medical Corporation, Tucson, AZ) and spirometry was repeated after 15 minutes. All patients were requested to not take any inhalers for the 4 hours prior to their appointment, unless medically necessary. Vmax Encore 22 (Viasys Respiratory Care, Yorba Linda, CA) and KoKo (Ferraris Respiratory, Louisville, CO) spirometers were used. Both machines met the 1994 ATS recommendations and were calibrated daily and checked at three different injection speeds, as per the ATS guidelines [17]. Additionally, respiratory therapists were monitored and periodically re-credentialed, as part of a standardized laboratory proficiency testing plan. Spirometric tests that did not meet the ATS criteria for within-maneuver and between-maneuver acceptability were excluded from the analyses [17].

Outcomes

Airflow obstruction—Airflow obstruction is defined as an FEV₁/VC ratio below the 5th percentile of the predicted value [18]. Since the FEV₁/VC reference standard is not readily available, a post-bronchodilator FEV₁/FVC ratio below the 5th percentile of the predicted value was used to define airflow obstruction. The reference standards were those from the

National Health and Nutrition Examination Survey (NHANES) III spirometric reference equations [19] - the Mexican-American reference standard was used for Hispanics and the Caucasian American reference standard for NHW.

Reversible and irreversible airflow obstruction—Airflow obstruction was considered reversible if there was an increase in FEV₁ of 12% and 200 mL from baseline after administration of an inhaled bronchodilator in a subject with airflow obstruction, and irreversible otherwise [18].

Absolute and percent predicted values of lung function—Absolute and percent predicted values of post-bronchodilator FEV₁/FVC and FEV₁ were also used as outcome measures.

Severity of lung impairment—Severity of lung impairment is typically determined using percent predicted FEV₁ [18]. This study used a modification of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria to classify the severity of impairment into the following stages: stage 1 (FEV₁ ≥ 80% of predicted value); stage 2 (FEV₁ 50 to < 80% of predicted value); and combined stages 3 and 4 (FEV₁ < 50% of predicted value) [20].

Chronic mucus hyper-secretion—Chronic mucus hyper-secretion was defined by the presence of at least two of the following three criteria: a) doctor diagnosis of chronic bronchitis; b) phlegm production over the previous 4 weeks; and c) phlegm production for most days for at least 3 consecutive months during the year. In addition, all participants who had cough productive of phlegm for at least 3 months a year for at least 2 consecutive years by self-report (*i.e.* who met the standard definition of chronic bronchitis) were included in this group.

Statistical Analysis

Summary statistics, including means, standard deviations (S.D.), medians, and interquartile ranges for the continuous variables and proportions for the categorical variables, were obtained. Chi-square and Fisher's exact tests were used for the bivariate analysis of categorical variables, while the two-sample t-test and Kruskal-Wallis test were used for continuous variables. Logistic regression was used to develop models for binary outcomes. Linear regression was similarly used for continuous outcomes. Ethnicity was included as the only predictor variable in the unadjusted model. Covariates included in the adjusted models were age, pack-years and duration of smoking, current smoking, and BMI. Additionally, height was included as a covariate in the analysis of absolute lung function values. Age, pack years and duration of smoking, and height were analyzed as continuous variables, whereas current smoking status and BMI were treated as categorical variables. All three time-related variables (*i.e.* age, pack-years of smoking, and duration of smoking) were included in the model since there was a better statistical fit with all three rather than two variables. Further, multiple smoking-related covariates were used to minimize the possible effect of residual confounding from smoking on the study results. Although not generally a confounder, age was included in the model since it affected the results of the analysis of absolute values of FEV₁. Results from these models were expressed as differences in the means between the two ethnic groups. Interactions between ethnicity and various covariates (*e.g.* current smoking status) were tested after main effects models had been developed. All analyses were conducted in SAS 9.1 (Cary, NC). A two-sided p-value of < 0.05 was considered statistically significant.

RESULTS

Of the 1,433 eligible women participants in the Lovelace Smokers' Cohort, 248 (17.3%) were Hispanic, 830 (57.9%) were current smokers, 517 (36.1%) smoked ≥ 40 pack-years, and 422 (29.4%) were obese (BMI ≥ 30 kg/m²). Hispanics were significantly more likely to be younger, to be overweight or obese, to have an annual household income of $\leq$ \$40,000, and to have at most a high school education than NHW ($p < 0.05$ for all analyses, Table 1). In addition, Hispanics had a higher prevalence of current smoking but a lower intensity of smoking (fewer pack-years) than NHW ($p < 0.001$ for both analyses). There was no significant difference in duration or age of onset of smoking, or time since smoking cessation for ex-smokers between the two groups.

Hispanics had a lower prevalence of airflow obstruction than NHW and Hispanic ethnicity was associated with reduced odds of airflow obstruction (Tables 2 and 3, and Figure 1). Although the absolute values of FEV₁ did not significantly differ among the two ethnic groups (Figure 2), Hispanics were more likely than NHW to have a higher value for each of the following lung function measures - FEV₁ percent predicted, FEV₁/FVC percent predicted, and FEV₁/FVC ratio (Table 2). In spite of their better lung function, Hispanics were more likely to report chronic mucus hyper-secretion than NHW. There was however no significant difference in the distribution of reversible and irreversible obstruction between Hispanics and NHW subjects with airflow obstruction.

Variables significantly associated with airflow obstruction in univariate analyses included age (OR = 1.53 per every 10 year-increase, 95% CI = 1.36, 1.73, $p < 0.001$), BMI (OR = 0.55, 95% CI = 0.41, 0.73 and OR = 0.38, 95% CI = 0.27, 0.53 for overweight and obese, respectively, $p < 0.001$), pack-years (OR = 1.25 per every 10 pack-year increment, 95% CI = 1.19, 1.32, $p < 0.001$), and duration of smoking (OR = 1.96 per every 10-year increment, 95% CI = 1.73–2.22, $p < 0.001$). Current smoking and time since smoking cessation in ex-smokers was not significantly associated with airflow obstruction.

After adjustment for covariates (age, BMI category, pack-years and duration of smoking, and current smoking status), Hispanic ethnicity was associated with halving of the odds of airflow obstruction (Table 3). Interestingly, the inverse association between Hispanic ethnicity and airflow obstruction was accentuated when an alternative definition of airflow obstruction, suggested by GOLD (*i.e.* FEV₁/FVC $< 70\%$), was used (Table 3) [20]. This was likely related to the differential effect of the GOLD definition on the classification of obstruction among older NHW [21].

After adjustment for smoking history and other covariates, Hispanics had significantly higher measures of lung function (mean percent predicted FEV₁ and FEV₁/FVC, and mean FEV₁/FVC) than NHW (Table 4). There was no significant difference in the risk of chronic mucus hyper-secretion between the two ethnic groups when current smoking status was added to the statistical model.

Interactions between ethnicity and each of the various covariates were also explored. However, these interactions did not explain the described protective effect of Hispanic ethnicity on the outcome measures in this study. For example, the only statistically significant interaction was between ethnicity and current smoking status in modeling percent predicted FEV₁/FVC ratio; $p < 0.001$. The difference in percent predicted FEV₁/FVC ratio between NHWs and Hispanics varied by only 7% between current and former smokers, indicating that difference in current smoking did not explain the described protective effect of Hispanic ethnicity (Table 4).

Additionally, in order to study the overlap with ‘asthma’, we evaluated whether exclusion of those with reversible spirometric airflow obstruction would materially change the results of this study. This approach allowed for greater objectivity than a self-report of asthma diagnosis. Exclusion of those with reversible airflow obstruction from the analysis did not significantly change the results shown in this study (data not shown).

DISCUSSION

Among female smokers enrolled in a cohort study in New Mexico, Hispanic ethnicity was associated with reduced risk of airflow obstruction and better overall lung function, even after adjustment for current smoking, pack-years and duration of smoking, among other covariates. Although Hispanic ethnicity was associated with an increased risk of chronic mucus hyper-secretion, this association was no longer significant after adjustment for current smoking. To our knowledge, this is the first study demonstrating a protective relationship between membership in a Hispanic subgroup and airflow obstruction among smokers.

In a study of 248 elderly subjects in San Antonio, Texas, Mexican-American ethnicity was associated with reduced intensity of smoking but similar severity of airflow obstruction [8]. Interpretation of those findings is limited by potential misclassification of asthma as COPD (since 43% of Mexican Americans were never smokers), lack of multivariate and/or gender-specific analyses, non-availability of post-bronchodilator spirometric measures, and lack of information on adherence to the ATS guidelines (a key issue for an elderly cohort using a hand-held spirometer in a home-based assessment). Our findings are generally consistent with those of previous studies in New Mexico that showed a lower prevalence of and mortality from self-reported chronic respiratory diseases among Hispanics [2, 10–13]. Whereas previous findings were attributed to ethnic differences in smoking in New Mexico, our results for airflow obstruction and lung function cannot be solely explained by smoking history.

Our findings may be explained by ethnic differences in the metabolism of smoke products, patterns of smoking, accuracy of assessment of smoking, or exposure to environmental tobacco smoke (ETS). For example, one study showed that whites and Mexican Americans had similar serum cotinine levels when they smoked up to 5 cigarettes per day, but that serum cotinine levels increased significantly more in whites than in Mexican Americans with each additional cigarette smoked [22]. Potential ethnic differences in smoking patterns include the type of cigarette smoked (length of cigarette, menthol or non-menthol, filter or non-filter, and nicotine yield), and the method of smoking cigarettes (blocking ventilation holes by fingers or lips, and frequency and depth of inhalation). For example, fewer Mexican Americans in the Hispanic Health and Nutrition Examination Survey smoked mentholated cigarettes [23] than has been reported among whites in several large studies. Some studies suggest that menthol may allow for greater inhalation of smoke either because of its ‘cooling sensation’ or local anesthetic properties and may therefore, allow greater exposure to nicotine, for the same number of cigarettes smoked [24–28].

A potential ethnic difference in the accuracy of assessment of cigarette smoking is suggested by some (but not all) studies that show that Mexican Americans underreport their cigarette consumption. In addition, there may be increased residential and occupational exposure to ETS and wood-burning stoves among Hispanics [29–33]. Further, low socioeconomic status is associated with greater nicotine use [34–36]. However, all these factors would introduce information bias in a direction that would increase the odds of abnormal lung function among the Hispanic population and therefore do not explain the observed association.

Obesity (which was more common among Hispanic women in this study) causes a loss of expiratory reserve volume, which may cause an increase in FEV₁/FVC ratio. Statistical adjustment for BMI category may not be wholly able to account for this effect of obesity. Further, an ethnic difference in spirometry technique, whereby Hispanic women did not exhale as long as NHW women, may also cause an artefactual increase in FEV₁/FVC ratio among Hispanic participants. However, these explanations do not account for the observed difference in percent predicted FEV₁ between the two groups. Further, this study followed a stringent spirometry quality control plan and excluded all tests that did not meet the ATS criteria.

Another alternative explanation for our findings is ethnic differences in genetic susceptibility to the adverse effects of tobacco smoke. Individuals who carry variant alleles of the CYP2A6 gene are poor metabolizers of nicotine. However, no significant ethnic difference in human cytochrome P450 enzyme activity has been previously described [37]. The best studied genetic defect leading to airflow obstruction is alpha-1 antitrypsin (AAT) deficiency, although a similar relationship with genetic variation in beta-defensin-1 has been recently described [38]. In general, there is lack of molecular and genetic studies in Hispanics with airflow obstruction to allow for any valid conclusion to be drawn about a difference in genetic sensitivity to cigarette smoke between Hispanic and NHW smokers.

The strengths of this study include its focus on women, use of post-bronchodilator spirometry to define obstruction, strict adherence to the 1994 ATS guidelines in the performance of spirometry, use of NHANES III reference standards, and use of statistically-defined lower limit of predicted FEV₁/FVC ratio [18], instead of a fixed ratio (*i.e.* FEV₁/FVC < 70%) [20]. The fixed ratio, although simple to use, has not been clinically validated and its use is particularly problematic in patients with milder disease who are elderly [21, 39].

We also recognize several limitations to our study. We cannot exclude ethnic differences in AAT deficiency as an alternative explanation to our findings. However, severe AAT deficiency only accounts for 1–2% of cases of COPD [40]. We did not adjust for occupational and environmental exposures, patterns of tobacco use, and dietary habits. We also lack data on the place of birth and country of origin of the study participants. Thus, we are unable to differentiate Mexican Americans from New Mexican Hispanics who have lived in the state for centuries. However, new immigrants of five years or less duration account for only 4% of the Hispanics in the Albuquerque area, 86% of whom are born in the United States [41]. This study, established in 2001, used the 1994 ATS spirometry guideline and not the more stringent 2005 ATS/ERS guideline. Similarly, the post-bronchodilator spirometry was performed after two (and not the currently recommended four) metered-dose inhaler inhalations of a rapidly acting beta-agonist. We cannot completely exclude asthma as the cause of airway obstruction in this study. However, the low prevalence of bronchodilator reversibility in this study (about 11%), use of post-bronchodilator lung function measures, and an inclusion criterion of minimum smoking history of 20 pack-years makes this possibility less likely. Finally, our study cohort may not be representative of all Hispanic and NHW female smokers in New Mexico and in other parts of the United States. However, the smoking behavior observed in our study is consistent with that observed in representative surveys of the state of New Mexico which show that among ever smokers [14], Hispanic adults are more likely to be current smokers and have a lower smoking intensity as compared to non-Hispanic whites ($p < 0.001$ for both analyses). Further, inclusion of English-speaking Hispanics may introduce a selection bias in this study. However, this bias is likely to be small since (unlike other large Hispanic populations in the United States) most Hispanics in the Albuquerque area of New Mexico speak English [42]. Finally, while possible, selection bias is an unlikely explanation for our findings because

participants would have had to enter the study for reasons related to both ethnicity and lung function (which had not been previously measured in most participants).

In summary, Hispanic ethnicity was associated with lower risk of airflow obstruction and better lung function among female smokers in New Mexico. This finding was not completely explained by smoking history. Possible explanations for this finding include ethnic differences in the metabolism of smoke products, patterns of tobacco use, accuracy of assessment of smoking, body habitus, genetic susceptibility to smoking, and/or unmeasured occupational and environmental exposures other than tobacco smoke.

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ABBREVIATION LIST

NHW	Non-Hispanic white
NHANES III	Third National Health and Nutrition Examination Survey
BMI	Body mass index
ATS	American Thoracic Society
ERS	European Respiratory Society
FEV₁	Forced expiratory volume in one second
FVC	Forced vital capacity
GOLD	Global Initiative for Chronic Obstructive Lung Disease
COPD	Chronic Obstructive Pulmonary Disease
CYP	Cytochromal
U.S	United States
ETS	Environmental tobacco smoke
AAT	Alpha 1-antitrypsin

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Figure 1.
Distribution of airflow obstruction and ethnicity in the cohort

Figure 2.

Boxplot showing the distribution of absolute values of FEV₁ among the ethnic groups, among all participants and those with airflow obstruction, as defined by NHANES-III fifth percentile of FEV₁/FVC ratio

For comparison of NHW and Hispanics, unadjusted for other covariates and using the Kruskal-Wallis test: $p = 0.65$ (all participants) and $p = 0.41$ (participants with airflow obstruction).

Table 1

Distribution of potential risk factors for airflow obstruction among Hispanic and non-Hispanic White (NHW) female smokers.

Characteristics		Hispanic (n = 248)	NHW (n = 1,185)	p value
Age (in years)	Mean (S.D.)	53.6 (8.9)	56.5 (9.5)	<0.001
Body mass index (BMI) category	BMI < 25 kg/m ² - Normal or Underweight (%) [✱]	28.2	37.2	0.03
	BMI 25 to < 30 kg/m ² - Overweight (%)	39.1	34.0	
	BMI ≥ 30 kg/m ² - Obese (%)	32.7	28.8	
Education (% > high school) [†]		47.0	73.3	<0.001
Annual income (% > \$ 40,000) [‡]		33.2	41.2	0.04
Current smokers (%)		78.6	53.6	< 0.001
Pack-years of smoking [*]	Median (25 th , 75 th percentiles)	29.0 (21.9, 38.3)	35.0 (26.0, 49.5)	< 0.001
Duration of smoking (in years) [*]	Mean (S.D.)	33.5 (8.8)	33.3 (9.9)	0.85
Age of start of smoking (in years)	Mean (S.D.)	16.9 (4.0)	17.1 (3.8)	0.41
Time since smoking cessation for ex-smokers (in years)	Mean (S.D.)	9.2 (6.8)	10.6 (8.8)	0.17

[†] 113 participants had missing data on education

[‡] 289 participants had missing data on income

^{*} 1 participant had missing data on duration and pack years of smoking

[✱] 25/1,433 (1.7%) subjects were underweight (BMI < 18 kg/m²)

Table 2

Distribution of pulmonary function test and chronic mucus hyper-secretion variables among Hispanic and non-Hispanic white (NHW) female smokers.

Outcomes		Hispanic (n = 248)	NHW (n = 1,185)	p value
FEV ₁ percent predicted	Mean (S.D.)	90.8 (16.5)	86.3 (18.7)	<0.001
FEV ₁ absolute (liters) *	Mean (S.D.)	2.35 (0.57)	2.32 (0.63)	0.49
FEV ₁ /FVC percent predicted	Mean (S.D.)	80.3 (2.0)	78.8 (2.0)	<0.001
FEV ₁ /FVC absolute	Mean (S.D.)	77.5 (7.8)	73.3 (12.3)	<0.001
Airflow obstruction (%)		12.9	24.2	<0.001
Irreversible airflow obstruction, for those with obstruction (%)		87.5	89.9	0.76
Airflow obstruction, using <70% FEV ₁ /FVC ratio criterion (%)		11.7	28.1	<0.001
Irreversible airflow obstruction, for those with obstruction, using <70% FEV ₁ /FVC ratio criterion (%)		89.6	84.6	0.50
Severity of lung impairment among those with airflow obstruction (%)	Stage 1	40.6	25.1	0.17
	Stage 2	43.8	56.1	
	Stages 3 & 4	15.6	18.8	
Severity of lung impairment among those with airflow obstruction, using <70% FEV ₁ /FVC ratio criterion (%)	Stage 1	30.9	34.5	0.89
	Stage 2	52.9	48.3	
	Stages 3 & 4	16.2	17.2	
Chronic mucus hyper-secretion (%)		40.4	32.1	0.01
Chronic mucus hyper-secretion or airflow obstruction (%)		45.7	46.6	0.80
Chronic mucus hyper-secretion or airflow obstruction, using <70% FEV ₁ /FVC ratio criterion (%)		44.5	48.8	0.22

* Distribution of absolute FEV₁ among the ethnic groups is shown in Figure 2

Table 3

Odds ratios for airflow obstruction and chronic mucus hyper-secretion for Hispanic (n=248) relative to non-Hispanic white (n=1,185) female smokers.

Outcomes	Unadjusted analysis			Adjusted analysis [†]		
	Odds Ratio	95% C.I.	p value	Odds Ratio	95% C.I.	p value
Airflow obstruction	0.46	0.31, 0.69	<0.001	0.51	0.34, 0.78	0.002
Airflow obstruction (using <70% FEV ₁ /FVC ratio criterion)	0.34	0.22, 0.50	<0.001	0.37	0.24, 0.58	<0.001
Chronic mucus hyper-secretion	1.44	1.08, 1.91	0.01	1.13	0.83, 1.54	0.43
Chronic mucus hyper-secretion or airflow obstruction	0.96	0.73, 1.27	0.80	0.85	0.63, 1.14	0.28

[†]Adjusted for age, BMI category, pack-years and duration of smoking, and current smoking status. Similar results were obtained when time since smoking cessation was included in the model instead of duration of smoking.

Note: For Chronic mucus hyper-secretion, the ethnicity effect is no longer significant after current smoking status is added to the model.

Table 4

Differences in least squares means of selected pulmonary function parameters between Hispanic (n=248) and non-Hispanic white (n=1,185) female smokers.

Outcomes	Unadjusted analysis			Adjusted analysis [‡]		
	Difference (NHW – Hispanic)	95% C.I.	p value	Difference (NHW – Hispanic)	95% C.I.	p value
Mean percent predicted FEV ₁	– 4.51	–7.02, –1.99	< 0.001	–2.74	–0.30, 5.18	0.03
Mean absolute FEV ₁ (in liters)	–0.03	–0.11, 0.06	0.52	–0.05	–0.11, 0.02	0.13
Mean percent predicted FEV ₁ /FVC ratio *100	–1.51	–1.78, –1.23	< 0.001	–0.89 [*]	–0.89, –0.88	< 0.001
Mean absolute FEV ₁ /FVC ratio *100	–4.21	–5.81, –2.62	< 0.001	–2.78	–4.34, –1.21	< 0.001

[‡] Adjusted for age, BMI category, pack-years and duration of smoking, and current smoking status. Additionally, absolute lung function values were adjusted for height (similar results were obtained when height² was added to the model). Similar results were obtained when time since smoking cessation was included in the model instead of duration of smoking.

^{*} There was a significant interaction between ethnicity and current smoking status in modeling percent predicted FEV₁/FVC ratio; p < 0.001. The adjusted difference in mean percent predicted FEV₁/FVC ratio between non Hispanic whites (NHW) and Hispanics in current smokers was –0.90% (–0.91, –0.897%) and in ex-smokers was –0.84% (–0.85, –0.83).