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Racial and Ethnic Minorities Have a Lower Prevalence of Airflow Obstruction than Non-Hispanic Whites

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\textbf{Abstract}

Racial and ethnic disparities in chronic obstructive pulmonary disease (COPD) are not well-studied. Our objective was to examine differences in limited COPD-related outcomes between three minority groups—African Americans (AAs), Hispanics, and American Indians (AIs) versus non-Hispanic Whites (NHWs), as the referent group, in separate cohorts. Separate cross-sectional evaluations were performed of three US-based cohorts of subjects at risk for COPD: COPDGene Study with 6,884 NHW and 3,416 AA smokers; Lovelace Smokers’ Cohort with 1,598 NHW and 378 Hispanic smokers; and Mining Dust Exposure in the United States Cohort with 2,115 NHW, 2,682 Hispanic, and 2,467 AI miners. Prebronchodilator spirometry tests were performed at baseline visits using standard criteria. The primary outcome was the prevalence of airflow obstruction. Secondary outcomes were self-reported physician diagnosis of COPD, chronic bronchitis, and modified Medical Research Council dyspnea score. All minority groups had a lower prevalence of airflow obstruction than NHWs (adjusted ORs varied from 0.29 in AIs to 0.85 in AAs; \textit{p}<0.01 for all analyses). AAs had a lower prevalence of chronic bronchitis than NHWs. In our study, all minority groups had a lower prevalence of airflow obstruction but a greater level of self-reported dyspnea than NHWs, and covariates did not explain this association. A better understanding of racial and ethnic differences in smoking-related and occupational airways obstruction may improve prevention and therapeutic strategies.

\textbf{Abbreviations}: AA: African Americans; AI: American Indians; ATS-DLD: American Thoracic Society-Diffuse Lung Disease; BMI: body mass index; CI: confidence interval; COPD: chronic obstructive pulmonary disease; CT: computed tomography; FEV\textsubscript{1}: forced expired volume in one second; FVC: forced vital capacity; GLI: Global Lung Initiative; GOLD: Global Obstructive Lung Disease; LSC: Lovelace Smokers’ Cohort; MIDUS: Mining Dust Exposure in the United States; mMRC: Modified Medical Research Council; NHANES: National Health and Nutrition Examination Survey; NHW: non-Hispanic Whites; OR: odds ratio; SD: standard deviation

\textbf{Introduction}

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death in the United States [1]. More than 11 million people in the United States have been diagnosed with COPD, but millions more may have the disease without even knowing it [1]. The two major phenotypes of COPD are chronic bronchitis and emphysema. Often, chronic bronchitis and emphysema phenotypes are concomitantly present. Although cigarette smoking is the most important risk factor for COPD in the United States, it is estimated that the occupational contribution to the population burden of COPD is about 15\% [2]. Some workers are at particularly high risk for developing COPD, such as those who work in dusty trades, including miners, but COPD in these workers is inadequately studied. In recent years, COPD prevalence has increased among racial and ethnic minorities [3]. COPD-related outcomes may differ by race and ethnicity, but disparities in these outcomes between minority and non-minority groups across the United States are not well-studied. These disparities constitute a critical gap in knowledge since current minorities in the United States are expected to become the majority in approximately 2044 [4]. Our objective was to examine differences in limited...
COPD-related outcomes between non-Hispanic Whites (NHWs, referent group) versus three racial and ethnic minority groups (African Americans (AAs), Hispanics, and American Indians (AIs)), by separately analyzing three at-risk cohorts, including smokers and/or miners without pooling of data across cohorts. Based upon our previous studies [5, 6], we hypothesized that minority groups have a lower prevalence of COPD-related outcomes than NHWs.

**Materials and methods**

**Study design**

Separate cross-sectional analyses of data were performed from baseline evaluations from three longitudinal US-based studies with significant proportions of minority smokers or miners, subjects at risk for developing COPD. NHW participants in each cohort study served as the referent group for separate comparison with minority participants in that cohort without pooling data across cohorts.

**Study population**

The COPDGene Study (www.copdgene.org) recruited 6,884 NHW and 3,416 AA smokers with ≥10 pack-years exposure, age 45–80 years, from 21 clinical centers across the United States between 2008 and 2011. Full inclusion and exclusion criteria and study design have been described previously [7].

The Lovelace Smokers’ Cohort (LSC) recruited 1,598 NHW and 378 Hispanic smokers, mostly women, from Albuquerque, and surrounding New Mexico communities, aged 40–75 years, with ≥10 pack-years of smoking since March 2001. Full inclusion and exclusion criteria and study design have been described previously [5, 6].

The Mining Dust Exposure in the United States (MiDUS) Cohort recruited 2,115 NHW, 2,682 Hispanic, and 2,467 AI miners, mostly men, without regard to smoking history. Subjects were recruited from mobile screening clinics held at 20 largely rural communities in New Mexico by rotation from 1989 to 2016. Full inclusion and exclusion criteria and study design have been described previously [8, 9].

**Measurements**

Demographic information, including age, smoking history, environmental exposure history, and history of respiratory disease, was obtained using the adult American Thoracic Society (ATS)-Diffuse Lung Disease (DLD)-78 questionnaire [10]. Race and ethnicity were self-reported.

Prebronchodilator spirometry tests were performed by trained respiratory therapists using standard criteria [11]. The third National Health and Nutrition Examination Survey (NHANES III) predicted values for Caucasian Americans, AA, and Mexican Americans were used for the NHW, AA, and Hispanic study populations, respectively [12], and Crapo reference standard for AIs [13] was used. Dyspnea was quantified using the five-point modified Medical Research Council (mMRC) dyspnea scale, which asks respondents to rate dyspnea from 0 (absent) to 4 (dyspnea when dressing/undressing) [14].

**Predictor and outcome**

Self-reported race and ethnicity were examined as the predictor variable. The primary outcome was the baseline prevalence of airflow obstruction. The latter was defined in two ways, as determined by the values of the ratio of forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) of either less than 70% (absolute) or less than the statistically-defined lower limit of normal (LLN), corresponding to a z score of −1.645 [15]. Our secondary outcomes were self-reported physician diagnosis of COPD, chronic bronchitis, and modified Medical Research Council (mMRC) dyspnea score at baseline evaluation [14]. Chronic bronchitis was determined by the presence of self-reported cough and phlegm production for at least three months a year for two or more consecutive years. LLN was primarily defined using the National Health and Nutrition Examination Survey III (NHANES III) and was alternatively determined using the Global Lung Initiative (GLI) references.

**Covariates**

Standard covariates included age, body mass index (BMI), education, sex, current smoking, and pack-years of smoking. In addition, mining tenure in the MiDUS Cohort and self-reported dust and fume exposure in the remaining two cohorts were included as covariates. These covariates were chosen because of their biological relevance for the studied associations. Additional covariates included spirometric patterns and cardiovascular disease, the latter defined by self-reported physician diagnosis of hypertension, angina, myocardial infarction, congestive heart failure, or arrhythmias.

**Statistical approach**

Summary statistics, including means and standard deviations or adjusted standard errors of the means, were obtained for continuous variables, and proportions were obtained for categorical variables. Non-parametric tests (Wilcoxon rank-sum) were used for continuous variables, and chi-square tests and logistic regression were used for categorical variables, using Statistical Analysis Software Package Version 9.3 (SAS, Cary, NC). A two-sided p-value <0.05 was considered statistically significant.

**Institutional review board approval**

Each study site participating in the COPDGene Study obtained local institutional review board approval to enroll participants in this project. The LSC was approved by the Western Institutional Review Board (20031684, Olympia, WA). All subjects in the LSC and COPDGene Study provided informed consent to participate in the study. The
The MiDUS Cohort was approved (14-058) by the Human Research Protection Office at the University of New Mexico (Albuquerque, NM) with a research subject consent waiver.

**Data availability**

While COPDGene Study data are publicly available through the database of Genotypes and Phenotypes (dbGaP), data on the MiDUS Cohort and pooled data from the LSC can be shared with the appropriate institutional review board and institutional approvals by contacting the corresponding author.

**Results**

While AAs in the COPDGene Study and Hispanics in the remaining two cohorts were younger than the respective NHWs within each cohort, AIs were older than NHWs in the MiDUS Cohort (Table 1). The sex ratio was nearly balanced in the COPDGene Study, but LSC subjects (by study design) were mainly women, and MiDUS subjects (belonging to an occupational cohort of miners) were overwhelmingly men. Although minority groups in each cohort reported lower cumulative pack-years of smoking, AAs in the COPDGene Study were more likely, and AIs in the MiDUS Cohort were less likely to report current smoking than the respective NHWs within each cohort. All minority groups in each cohort showed a significantly lower educational status than the respective NHWs. In the MiDUS Cohort, AIs reported lower total mining tenure than the other groups, and AAs in the COPDGene Study were more likely to self-report exposure to dust/fumes than the respective NHWs within each cohort.

All minority groups had a lower prevalence of self-reported physician diagnosis of COPD and spirometric airflow obstruction and higher FEV\(_1\)/FVC ratio values than the respective NHWs in each cohort (Table 2). AAs and AIs had a significantly lower prevalence of chronic bronchitis than the respective NHWs in each cohort. Further, compared to the respective NHWs, all minority groups in each cohort showed higher \(z\)-scores for FEV\(_1\) (using the NHANES III and GLI reference standards) and FVC (using the NHANES III reference standards). All minority groups reported greater levels of dyspnea on the mMRC scale than the respective NHWs in each cohort.

After adjustment for standard covariates, results were broadly similar to the univariate analysis (Table 3). However, the associations for self-reported physician diagnosis of COPD among Hispanics and chronic bronchitis among AI were no longer significantly different. Furthermore, the associations for FEV\(_1\) \(z\)-score among AIs in the MiDUS Cohort and FVC \(z\)-scores among Hispanics in the LSC and MiDUS Cohort were not significantly different than the respective NHWs. In addition, the Hispanic association was no longer significant using the GLI reference standards in the LSC. The greater levels of dyspnea (mMRC score) reported by all minority groups in each cohort were neither explained by standard covariates (Table 3) nor by differences in spirometric pattern distribution or prevalence of cardiovascular disease (Table 4).

**Discussion**

Compared to NHWs, minority groups in three separate cohorts of smokers or miners demonstrate a lower prevalence of airflow obstruction but greater self-reported dyspnea. AAs have a lower prevalence of chronic bronchitis. The study findings are unlikely to be explained by differences in age, smoking behavior, socioeconomic status...
Although AAs have lower prevalence and death rates from COPD than Caucasian Americans, several reports have demonstrated that AAs develop COPD with less intense cumulative smoking and at a younger age [16–18]. Additional reports indicate that COPD mortality rates have increased faster among AAs than Caucasian Americans [3]. Using quantitative CT scan analysis from the COPDGene substudy, AAs were shown to have less radiologic emphysema than NHWs but no significant difference in airway wall thickness [19]. Among severe COPD patients, AAs are disproportionately represented among those with early-onset disease.

### Table 2. Univariate analysis of racial and ethnic differences in respiratory outcomes in the three cohorts.

<table>
<thead>
<tr>
<th>Respiratory outcome</th>
<th>COPDGene Study</th>
<th>LSC</th>
<th>MIDUS Cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>African-American (n = 3,416)</td>
<td>Hispanic (n = 1,598)</td>
<td>Hispanic (n = 2,682)</td>
</tr>
<tr>
<td>Mean ± SD or %</td>
<td>NHW (n = 6,884)</td>
<td>NHW (n = 6,884)</td>
<td>NHW (n = 1,598)</td>
</tr>
<tr>
<td>Airflow obstruction (GOLD definition FEV1/FVC &lt; 70%)</td>
<td>35.7%</td>
<td>55.5%*</td>
<td>14.8%</td>
</tr>
<tr>
<td>Airflow obstruction (FEV1/FVC &lt; LLN (or z score −1.645)) NHANES III</td>
<td>28.9%</td>
<td>43.1%*</td>
<td>16.1%</td>
</tr>
<tr>
<td>Airflow obstruction (FEV1/FVC &lt; LLN (or z score −1.645)) GLI</td>
<td>27.01%</td>
<td>41.68%*</td>
<td>10.85%</td>
</tr>
<tr>
<td>Absolute FVC (Ls.)</td>
<td>3.1 ± 0.9</td>
<td>3.4 ± 1.0*</td>
<td>3.4 ± 0.9</td>
</tr>
<tr>
<td>FVC z score, NHANES III</td>
<td>−0.73 ± 1.26</td>
<td>−1.10 ± 1.43*</td>
<td>−0.34 ± 1.17</td>
</tr>
<tr>
<td>FVC z score, GLI</td>
<td>−0.75 ± 1.22</td>
<td>−0.76 ± 1.25</td>
<td>−0.3 ± 1.05</td>
</tr>
<tr>
<td>Absolute FEV1 (Ls.)</td>
<td>2.3 ± 0.9</td>
<td>2.2 ± 1.0*</td>
<td>2.6 ± 0.7</td>
</tr>
<tr>
<td>FEV1 z score, NHANES III</td>
<td>−1.09 ± 1.44</td>
<td>−1.82 ± 1.82*</td>
<td>−0.60 ± 1.27</td>
</tr>
<tr>
<td>FEV1 z score, GLI</td>
<td>−1.13 ± 1.41</td>
<td>−1.57 ± 1.60*</td>
<td>−0.53 ± 1.18</td>
</tr>
<tr>
<td>FEV1/FVC ratio absolute</td>
<td>72.0 ± 13.8</td>
<td>64.3 ± 16.6*</td>
<td>76.6 ± 9.0</td>
</tr>
<tr>
<td>Ratio z score, NHANES III</td>
<td>−1.22 ± 2.08</td>
<td>−2.04 ± 2.72*</td>
<td>−0.59 ± 1.52</td>
</tr>
<tr>
<td>Ratio z score, GLI</td>
<td>−0.90 ± 1.61</td>
<td>−1.57 ± 1.74*</td>
<td>−0.43 ± 1.1</td>
</tr>
<tr>
<td>Self-reported physician diagnosis of COPD</td>
<td>11.6%</td>
<td>19.0%*</td>
<td>7.7%</td>
</tr>
<tr>
<td>Chronic bronchitisa</td>
<td>13.7%</td>
<td>20.6%*</td>
<td>27.2%</td>
</tr>
<tr>
<td>mMRC score</td>
<td>1.5 ± 1.5</td>
<td>1.3 ± 1.4*</td>
<td>2.1 ± 1.4</td>
</tr>
<tr>
<td>Score 0</td>
<td>42.3%</td>
<td>45.9%*</td>
<td>19.0%</td>
</tr>
<tr>
<td>Score 1</td>
<td>11.6%</td>
<td>14.8%*</td>
<td>23.3%</td>
</tr>
<tr>
<td>Score 2</td>
<td>13.5%</td>
<td>12.6%</td>
<td>2.4%</td>
</tr>
<tr>
<td>Score 3</td>
<td>20.2%</td>
<td>17.5%*</td>
<td>42.3%</td>
</tr>
<tr>
<td>Score 4</td>
<td>12.4%</td>
<td>9.0%*</td>
<td>13.0%</td>
</tr>
<tr>
<td>Prevalent self-reported cardiovascular disease</td>
<td>7.6%</td>
<td>12.5%*</td>
<td>26.7%</td>
</tr>
</tbody>
</table>

Note: **p < 0.05, *p < 0.01; for the MIDUS cohort, the p values refer to three-group comparison; non-parametric tests (Wilcoxon rank-sum) were used for continuous variables, and chi-square test was used for categorical variables.

Abbreviations: COPD, chronic obstructive pulmonary disease; FVC, forced vital capacity; FEV1, Forced expiratory volume in one second; GLI, Global Lung Initiative; GOLD, Global Obstructive Lung Disease; LSC, Lovelace Smokers’ Cohort; MIDUS, Mining Dust Exposure in the United States; mMRC, Modified Medical Research Council; NHANES, National Health and Nutrition Examination Survey.

*aStandard definition of chronic bronchitis was defined by cough and phlegm production for at least three months a year for two or more consecutive years.

(measured by proxy variable education), or duration of mining dust exposure since these covariates were adjusted for in the multivariable analysis.

Although AAs have lower prevalence and death rates from COPD than Caucasian Americans, several reports have demonstrated that AAs develop COPD with less intense cumulative smoking and at a younger age [16–18]. Additional
occurring before 55 years) than those with late-onset disease
than those with late-onset disease. After additional adjustment for spirometric pattern and self-reported cardiovascular disease.

Table 4.

COPD Gene Study-African American

<table>
<thead>
<tr>
<th>Respiratory outcomes</th>
<th>LSC-Hispanic</th>
<th>MIDUS Cohort-Hispanic</th>
<th>MIDUS Cohort-American Indian</th>
</tr>
</thead>
<tbody>
<tr>
<td>Categorical outcomes</td>
<td>Odds ratio (95% CI)</td>
<td>Odds ratio (95% CI)</td>
<td>Odds ratio (95% CI)</td>
</tr>
<tr>
<td>Airflow obstruction (GOLD definition FEV1/ FVC &lt;70%)</td>
<td>0.72 (0.65, 0.80)*</td>
<td>0.45 (0.31, 0.65)*</td>
<td>0.50 (0.32, 0.78)*</td>
</tr>
<tr>
<td>Airflow obstruction (FEV1/ FVC &lt;LLN (or z score −1.645)), NHANES III</td>
<td>0.85 (0.76, 0.94)*</td>
<td>0.64 (0.45, 0.91)*</td>
<td>0.59 (0.50, 0.71)*</td>
</tr>
<tr>
<td>Airflow obstruction (FEV1/ FVC &lt;LLN (or z score −1.645)), GLI</td>
<td>0.80 (0.72,0.89)*</td>
<td>0.44 (0.27, 0.69)*</td>
<td>0.47 (0.39, 0.57)*</td>
</tr>
<tr>
<td>Self-reported physician diagnosis of COPD</td>
<td>0.56 (0.49, 0.65)*</td>
<td>0.79 (0.48, 1.24)</td>
<td>0.52 (0.26, 1.06)</td>
</tr>
<tr>
<td>Chronic bronchitis*</td>
<td>0.50 (0.44, 0.56)*</td>
<td>0.87 (0.63, 1.18)</td>
<td>1.42 (0.92, 2.19)</td>
</tr>
<tr>
<td>mMRC score &lt;2</td>
<td>0.61 (0.55, 0.67) *</td>
<td>0.59(0.45, 0.78)*</td>
<td>0.68 (0.59, 0.80)*</td>
</tr>
<tr>
<td>mMRC score ≥2</td>
<td>1.65 (1.49, 1.82)*</td>
<td>1.69 (1.28, 2.23)*</td>
<td>1.33 (1.15, 1.53)*</td>
</tr>
</tbody>
</table>

Continuous outcomes

<table>
<thead>
<tr>
<th>Respiratory outcomes</th>
<th>Difference (95% CI)</th>
<th>Difference (95% CI)</th>
<th>Difference (95% CI)</th>
<th>Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC z score, NHANES III</td>
<td>0.25 (0.19, 0.32)**</td>
<td>0.15 (0.00, 0.30)**</td>
<td>0.09 (−0.02, 0.19)</td>
<td>−0.19 (−0.30, −0.07)*</td>
</tr>
<tr>
<td>FVC z score, GLI</td>
<td>−0.06 (−0.12, −0.01)</td>
<td>−0.04 (−0.18, 0.11)</td>
<td>−0.01 (−0.01, 0.08)</td>
<td>−0.15 (−0.25, −0.05)*</td>
</tr>
<tr>
<td>FEV1 z score, NHANES III</td>
<td>0.43 (0.36, 0.51) *</td>
<td>0.26 (0.09, 0.42) *</td>
<td>0.23 (0.12, 0.33)*</td>
<td>0.05 (−0.06, 0.17)</td>
</tr>
<tr>
<td>FEV1 z score, GLI</td>
<td>0.18 (0.11, 0.25)</td>
<td>0.17 (0.01, 0.34)**</td>
<td>0.21 (0.11, 0.30)*</td>
<td>0.02 (−0.08, 0.13)</td>
</tr>
<tr>
<td>FEV1/FVC ratio z score, NHANES III</td>
<td>0.15 ± (0.04, 0.26)</td>
<td>0.28 (0.08, 0.48)*</td>
<td>0.33 (0.21, 0.45)*</td>
<td>0.44 (0.31, 0.57)*</td>
</tr>
<tr>
<td>FEV1/FVC ratio z score, GLI</td>
<td>0.23 (0.16, 0.31) *</td>
<td>0.34 (0.18, 0.50)*</td>
<td>0.41 (0.30, 0.53)*</td>
<td>0.31 (0.19, 0.44)*</td>
</tr>
<tr>
<td>mMRC (mean)</td>
<td>0.33 (0.26, 0.39)*</td>
<td>0.27 (0.10, 0.43)*</td>
<td>0.17 (0.06, 0.28)*</td>
<td>0.76 (0.64, 0.88)*</td>
</tr>
</tbody>
</table>

Note: NHWs served as the referent group for separate comparisons in each cohort without pooling data across cohorts. All analyses were adjusted for age, baseline BMI, and pack-years of smoking as continuous variables, as well as high school education, sex, and current smoking status at baseline as categorical variables. In addition, total mining tenure was included as a continuous covariate in the MIDUS cohort and exposure to dust and fumes as a categorical covariate in the other two cohorts. **p<0.05, *p<0.01. Categorical mMRC scores were analyzed using logistic regression. For the mean of continuous mMRC score in the LSC, Hispanics showed a p-value of 0.07.

Abbreviations: COPD, chronic obstructive pulmonary disease; FVC, forced vital capacity; FEV1, forced expiratory volume in one second; GLI, Global Lung Initiative; GOLD, Global Obstructive Lung Disease; LSC, Lovelace Smokers’ Cohort; MIDUS, Mining Dust Exposure in the United States; NHANES, National Health and Nutrition Examination Survey.

COPD Gene Study-African American

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<td>Odds ratio (95% CI)</td>
</tr>
<tr>
<td>mMRC &lt; 2</td>
<td>0.50 (0.45, 0.56)*</td>
<td>0.52 (0.39, 0.69)*</td>
<td>0.65 (0.55, 0.76)*</td>
</tr>
<tr>
<td>mMRC ≥ 2</td>
<td>1.99 (1.78, 2.22)*</td>
<td>1.93 (1.45, 2.58)*</td>
<td>1.36 (1.17, 1.58)*</td>
</tr>
</tbody>
</table>

Continuous outcomes

<table>
<thead>
<tr>
<th>Respiratory outcomes</th>
<th>Difference (95% CI)</th>
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<th>Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>mMRC (mean)</td>
<td>0.40 (0.34, 0.46)*</td>
<td>0.33 (0.17, 0.49)*</td>
<td>0.21 (0.10, 0.32)*</td>
<td>0.71 (0.59, 0.83)*</td>
</tr>
</tbody>
</table>

Note: The analyses were adjusted for age, baseline BMI, total mining tenure, and pack-years of smoking as continuous variables, as well as high school education, sex, current smoking status, spirometric patterns (restrictive or obstructive), and cardiovascular disease at baseline as categorical variables. In addition, total mining tenure was included as a continuous covariate in the MIDUS cohort and exposure to dust and fumes as a categorical covariate in the other two cohorts. **p<0.05, *p<0.01.

Abbreviations: COPD, chronic obstructive pulmonary disease; LSC, Lovelace Smokers’ Cohort; MIDUS, Mining Dust Exposure in the United States; mMRC, Modified Medical Research Council.

Table 3. Multivariable analysis of racial and ethnic differences in respiratory outcomes in the three cohorts.

The present study

(occuring before 55 years) than those with late-onset disease
[20]. It has been previously reported that AAs have worse dyspnea and health-related quality of life than Caucasians after adjustment for lung function. One possible explanation may be their use of fewer respiratory medications, lower utilization of medical care, and poorer access to care [21, 22]. Another possible explanation is that AAs are twice as likely as NHWs to report a history of asthma [23]. COPD patients with concomitant asthma experience poorer quality of life and greater dyspnea than those without asthma. The presence of comorbidities such as gastroesophageal reflux has a greater negative impact on dyspnea in AAs than

NHWs, which may also help explain their greater report of dyspnea [24].

In a study utilizing the NHANES III data, an obstructive pattern was less common in Mexican Americans (8.2%) than NHWs (15.1%) or AAs (12.4%) [25]. Several studies have demonstrated reduced prevalence and mortality of COPD, higher baseline lung function, and a slower decline in lung function in Hispanic groups, than NHWs [5, 6, 26–29]. These findings may contribute to the “Hispanic paradox” referring to the fact that despite their lower socioeconomic and higher comorbidity indices, Hispanics have a lower all-cause mortality rate than NHWs [30]. The present study
indicates that the Hispanic “protective effect” for airflow obstruction that our group first described in urban New Mexico women smokers [5, 6] is also true for rural male New Mexico miners. While some investigators have attributed the Hispanic “protective” effect to their lower cumulative exposure to tobacco smoke [26], others demonstrate a protective effect of the AI ancestry component in racially admixed Hispanic populations of New Mexico and Costa Rica [5, 31]. Studies have also identified unique genetic loci that may play a role in COPD pathogenesis in Hispanic populations [32]. Additionally, the greater dyspnea score in Hispanics than NHWs is consistent with their lower health-related quality of life previously reported in the LSC [33] and is not explained by differences in either lung function or CT measure of lung structure [34]. It is possible that, like in AAs, the disparities in access to healthcare, health literacy, and health behavior may explain this difference in Hispanics [33].

Studies on AIs and COPD are limited, and none have looked at participation in dusty trades to the best of our knowledge. Consistent with our findings, county-level mortality rates from COPD in AI communities of New Mexico are generally low [35]. One study on First Nations people from Saskatchewan, Canada, of mean age of 34.8 ± 14.5 years, describes a 7–9% prevalence of chronic bronchitis [36], significantly lower than the prevalence rates in the present study. Other studies similarly report low prevalence rates for chronic bronchitis [37, 38]. Our study results suggest that the relatively lower level of chronic bronchitis in AIs in the univariate analysis is likely explained by covariates.

The effect of different diagnostic criteria on COPD prevalence has been previously described. For example, using the LLN criteria in older subjects is associated with a lower prevalence of COPD than the GOLD guidelines [39]. While the most used reference equations in the United States remain those reported by Hankinson et al. in 1999, based on the third National Health and Nutrition Examination Survey, there has been a movement worldwide toward adopting the GLI 2012 reference equations [40]. The use of LLN using the GLI equations may be related to a lower prevalence of airflow obstruction in our study than the NHANES III standards. As previously published, the differences in the diagnosis and severity categorization of obstructive lung disease in individual subjects, particularly older, taller/shorter subjects, can be significant and may result in differences in clinical management [41]. Therefore, while one prediction equation cannot be endorsed over the other, generally, they should not be used interchangeably.

The present study has several strengths, including analyzing three minority groups in three separate cohorts without pooling data across cohorts. We have reproduced the findings described in Hispanic smokers in previous studies [5, 6, 26–29] in an occupational cohort of miners, a group at risk for COPD but inadequately studied. Our inclusion of AIs covers a critical gap in this field of research. It helps support our innovative hypothesis that the AI ancestry component in racially admixed Hispanics of the Southwestern United States may explain their “protection” from airflow obstruction [5]. Although the present study used rigorous spirometric criteria, potential limitations are noted. First, an obstructive spirometric pattern may not confirm the presence of COPD since it may also be seen in asthma, particularly given that the spirometry was not specifically obtained after bronchodilator administration. Further, the study does not address other COPD-related outcomes, which include hospitalizations, respiratory exacerbations, progression of dyspnea, onset of respiratory failure, and mortality. Self-reported race and ethnicity may not be accurate, potentially leading to misclassification in the use of spirometric reference equations. However, in our previous study, self-reported ethnicity was highly correlated with genetic ancestry information markers [5]. No well-validated spirometric reference equations currently exist for AIs. Although differences in genetic predisposition might underlie our findings, the present study may have been limited by differences in cultural, geographic, dietary, psychosocial stress, and socioeconomic factors between and within the three racial and ethnic categories [42–44]. In addition, smoking behavior is a complex variable that may not be entirely captured by measures such as pack-years of exposure and current smoking status. It is difficult to account for factors such as type of cigarettes, depth of inhalation, or number of puffs per cigarette, which may differ between minority and non-minority populations [5]. However, previous reports provide evidence that Hispanics may underestimate the amount of cigarette smoking, which, if underestimated in our study, would only strengthen the observed protective nature of Hispanic ethnicity for airflow obstruction [45]. The study analyzed only baseline data of three longitudinal cohorts. Longitudinal analyses in the future could provide further information to better evaluate/interpret the obtained cross-sectional results. Finally, findings may not be representative of racial/ethnic minorities in all parts of the United States because participants in the COPDGene Study were recruited from select clinical centers across the United States, and the LSC and the MiDUS Cohort were recruited from communities in New Mexico.

**Conclusions**

Our finding of a lower prevalence of airflow obstruction but greater self-reported dyspnea in all minority groups relative to NHWs suggests that there is insufficient mechanistic research into racial and ethnic disparities in COPD to disentangle biological, social, and environmental effects on airway diseases. Genomic, proteomic, metabolomic, and behavioral studies in the future may help better understand the pathophysiology of racial and ethnic differences. This, in turn, may result in new therapeutic strategies and drug targets. As COPD patients of the future will progressively include more racial and ethnic minorities, targeted preventive strategies and new therapeutics are needed among smokers and workers engaged in dusty trades.
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Disclosure statement

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