Chronic Obstructive Pulmonary Disease in Hispanics

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Hispanics are individuals whose ancestry can be traced to Spain and/or areas previously under Spanish control (e.g., Mexico, Puerto Rico). They are a rapidly growing subset of the population of the United States and are quite diverse in their racial ancestry, country of origin, area of residence, socioeconomic status, tobacco use, and access to health care. Current evidence suggests that the prevalence and morbidity of chronic obstructive pulmonary disease (COPD) vary widely among Hispanic-American nations, with similar but limited findings among Hispanic subgroups in the United States. Potential reasons for such variation include differences in racial ancestry and genetic susceptibility, exposure to tobacco smoke and/or biomass smoke, access to health care, and disease management. Future studies of COPD in Hispanics should include large samples of subgroups that are well defined with regard to self-reported ethnicity, country of origin, area of residence, tobacco use, and socioeconomic status. Areas that need to be carefully examined include validation of COPD diagnoses for epidemiologic studies (e.g., by radiologic assessment), COPD in high-risk groups (e.g., Puerto Ricans), impact of biomass smoke (in rural areas) and air pollution (in urban areas) on COPD morbidity, effects of migration and acculturation on COPD prevalence and morbidity among Hispanic subgroups in the United States, development of reference values for spirometry, smoking cessation, and overcoming barriers to management. Public health measures, such as effective smoking prevention and cessation programs, reduction of air pollution and exposure to biomass smoke, and improved access to health care, would help reduce the burden of COPD among Hispanics in the United States and Latin America.

Keywords: chronic obstructive pulmonary disease; genetics; Hispanics; risk factors

Chronic obstructive pulmonary disease (COPD) is a major public health problem in the United States, where the age-specific death rates attributable to this illness doubled between 1970 and 2002 (1).

Hispanics are the fastest-growing minority group in the United States (2). The term Hispanic or Latino refers to individuals whose ancestry can be traced back to Spain and/or Hispanic America (a vast area of Latin America that was under Spanish control and comprises Mexico, large parts of Central and South America, and some Caribbean countries) (3). Because of admixture (or lack thereof) among racially diverse groups (e.g., Europeans, Amerindians [people native to the Americas], and Africans) and migration to and from the large territory encompassed by the old Spanish empire, contemporary Hispanics are diverse with regard to racial ancestry (reviewed in Reference 4), country of origin, current area of residence and environmental exposures, socioeconomic status, tobacco use, and access to health care. These underlying differences are likely to explain the variation in the prevalence of respiratory diseases such as asthma (4) among and within Hispanic subgroups. Table 1 shows the marked variation in exposure to cigarette smoking and in selected demographic and health care characteristics among Hispanic-American nations. According to the 2000 U.S. Census, the ancestry of Hispanics living in the United States can be traced to Mexico (59.3%), Puerto Rico (9.7%), Central and South America (9.1%), and Cuba (3.5%) in 81.6% of the members of this ethnic group, with the remainder grouped as "other Hispanics" (e.g., Dominicans, Spaniards, and some Hispanics in New Mexico) (3).

In this perspective, we examine recent evidence and review previous studies on COPD in Hispanics, including data on prevalence, morbidity, and mortality; known and potential risk factors for COPD and/or COPD morbidity, including cigarette smoking, exposure to biomass smoke, air pollution, and genetic susceptibility; diagnosis; and management. On the basis of this review, we contend that new studies of COPD in Hispanics are essential, and that these studies should target well-defined Hispanic subgroups, both at presumably high (e.g., Puerto Ricans) and low (e.g., Mexican Americans) risk for COPD and/or COPD morbidity.

COPD PREVALENCE IN HISPANICS

Until recently, there were no adequate data on COPD prevalence in Hispanic America. The Latin American Project for the Investigation of Obstructive Lung Disease (PLATINO) examined the prevalence of and risk factors for COPD (defined as a post-bronchodilator $FEV_1/FVC < 0.70$) (5) among 5,315 adults ($\geq$40 yr) in five large cities in Hispanic America and Brazil: Santiago, Chile; Mexico City, Mexico; Montevideo, Uruguay; Caracas, Venezuela; and São Paolo, Brazil (6) (Table 2). Estimates of COPD prevalence (adjusted for pack-years of smoking and other covariates) were lowest in Mexico City (11.9%) and highest in Montevideo (19.4%), with similar trends after the analysis was restricted to subjects who self-identified as white. When a more stringent definition of COPD ($FEV_1/FVC < 0.70$ and $FEV_1 < 80\%$ of predicted) was used, the estimated prevalence of COPD ranged from 2.6% in Mexico City to 7.1% in Montevideo. Despite potential misclassification of asthma as COPD (particularly among nonsmokers), these results suggest that COPD is an important public health problem in Hispanic America, where the prevalence of this illness varies markedly among major cities in different nations. The PLATINO findings could be explained by larger airways relative to lung size (and thus a higher FEV_1/FVC) among individuals living at high altitude, differences in the frequency of genetic variants that confer susceptibility for COPD, "reverse causation" (e.g., if individuals with moderate to severe COPD preferred to live at

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TABLE 1. SELECTED DEMOGRAPHIC AND HEALTH CARE DATA FROM SOME HISPANIC-AMERICAN COUNTRIES

	Per Capita GDP*	Life Expectancy† (*yr*)	Prevalence of Tobacco Use‡ (%)	Health Spending§	Age-adjusted COPD Deaths‖
Argentina	$15,200	76.32	39.8	$383	11.4
Chile	$12,600	76.96	40	$359	17.3
Colombia	$8,600	72.27	18.9	$168	43.1
Costa Rica	$12,500	77.21	19.4	$290	26.1
Guatemala	$5,000	69.69	26.8	$127	107.8
Mexico	$10,700	75.63	32	$424	26.8
Nicaragua	$3,100	70.92	51 (men) 16 (women)	$67	21.5
Peru	$6,600	70.14	33.8	$104	8.8
Puerto Rico	$19,300	78.54	13.1		28.7

Definition of abbreviations: COPD = chronic obstructive pulmonary disease; GDP = gross domestic product.

* Per capita GDP is total market value of all final goods and services produced in a country in a given year, equal to total consumer, investment, and government spending, plus the value of exports, minus the value of imports in U.S. dollars. Source: CIA World Factbook (68).

† Life expectancy is the average number of years to be lived by a group of people born in the same year, if mortality at each age remains constant in the future. Source: CIA World Factbook (68).

‡ Prevalence of current smokers, with the definition of current varying from country to country (15).

§ Health spending is health care expenditures per capita in current U.S. dollars. Source: World Bank (69).

‖ Age-adjusted COPD deaths per 100,000 people. Source: World Health Organization (70) and Puerto Rico Department of Health, personal communication, 12/12/2007.

low altitude), residual confounding by cigarette smoking (which was assessed by questionnaire only) and/or air pollution, and/or underlying differences in asthma prevalence among Hispanic subgroups (4).

Results of a few studies with limited phenotypic assessment suggest variation in COPD prevalence among Hispanic subgroups in the United States. Among adolescents and adults (ages 12–74 yr) who participated in the Hispanic Health and Nutrition Examination Survey between 1982 and 1984, the estimated age-adjusted prevalence of self-reported physician-diagnosed chronic bronchitis was higher in Puerto Ricans (2.9%) than in Mexican Americans (1.7%) or Cubans (1.7%) (7, 8). Smoking was a significant risk factor for chronic bronchitis in Puerto Ricans and Cubans but not in Mexican Americans, likely because of lower tobacco use in the latter group. Among adults living in New Mexico, Hispanic ethnicity has been associated with reduced risks of physician-diagnosed COPD (chronic bronchitis or emphysema) and chronic airflow obstruction (defined as $FEV_1 < 65\%$ of predicted and $FEV_1/FVC < 0.80$), a finding attributed to lower cigarette consumption in Hispanics compared with non-Hispanic whites in New Mexico (9). Hispanics in New Mexico are a heterogeneous group composed of long-term residents in the state and more recent migrants from Mexico, and thus these results should be interpreted cautiously and not extrapolated to other Hispanic subgroups in the United States.

TABLE 2. PREVALENCE OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN THE PLATINO STUDY*

	COPD Prevalence (%)§		
	Crude	Adjusted†	Whites Only
Mexico City (Mexico)	7.8 (5.9–9.7)	11.9 (11.3–12.5)	12.7 (7.6–17.7)
Caracas (Venezuela)	12.1 (10.3–13.9)	13.0 (12.3–13.6)	13.5 (10.2–16.7)
São Paulo (Brazil)	15.8 (13.5–18.1)	14.9 (14.1–15.7)	16.2 (13.1–19.3)
Santiago (Chile)	16.9 (14.7–19.1)	14.5 (13.8–15.1)	17.8 (15.0–20.3)
Montevideo (Uruguay)	19.7 (17.2–22.1)	19.4 (18.4–20.3)	20.3 (17.8–22.8)
P value‡	0.0001	0.0001	0.01

Definition of abbreviation: COPD = chronic obstructive pulmonary disease; PLATINO = Latin American Project for the Investigation of Obstructive Lung Disease.

* Adapted from Reference 6. COPD was defined as $FEV_1/FVC < 0.70$.

† Rates adjusted for age, sex, ethnic origin, education, pack-years of smoking, exposure to domestic biomass and coal pollution, occupational exposure to dust, and body mass index.

‡ Wald test, taking account of sampling strategy.

§ Values in parentheses represent 95% confidence interval.

MORBIDITY OF COPD IN HISPANICS

There are limited data on morbidity from COPD in well-characterized Hispanic subgroups. Among 721 Mexican-American and white elderly subjects (≥65 yr of age) in San Antonio, Texas, Mexican Americans had lower intensity of cigarette smoking but a severity of COPD (using prebronchodilator spirometric measures and the GOLD [Global Initiative for Chronic Obstructive Lung Disease] staging system) similar to that found among whites (10). Interpretation of these findings is limited by potential misclassification of asthma as COPD, possible underreporting of cigarette smoking in Mexican Americans (43% of whom reported having never smoked), lack of multivariate analyses and data on post-bronchodilator spirometric measures, and limited information on the protocol used for spirometry.

MORTALITY FROM COPD IN HISPANICS

In Mexico, the crude and age-adjusted mortality rates from COPD for both men and women (age, ≥35 yr) increased gradually from 1980 until about 1998, after which there was a slow decrease until 2002 (11). Overall, the age-adjusted mortality rates from COPD in Mexico increased by 72% in men and 50% in women from 1980 to 2002. The age-adjusted mortality rates from COPD in 2002 were calculated as 29 per 100,000 in men and 16 per 100,000 in women. Although temporal trends may reflect changes in diagnostic patterns for COPD, these results and others (12) suggest that this respiratory disease is an important cause of mortality in Mexico and that COPD mortality rates vary among Hispanic-American nations (12).

Few studies have examined COPD mortality rates in specific Hispanic subgroups in the United States. From 1958 to 1982, the age-adjusted annual mortality rates for COPD increased among Hispanic males (from 5.0 to 30.1 per 100,000) and females (from 2.9 to 13.7 per 100,000) in New Mexico (13). However, Hispanics had lower mortality rates for COPD than non-Hispanic whites at every time point in the study. Although Hispanic heritage was ascertained using surnames in death certificates (an approach previously validated in a similar population), the aforementioned heterogeneity of Hispanics in New Mexico prevents drawing conclusions about specific subgroups. In another study conducted from 1986 to 1988 in 18 states in the United States and the District of Columbia, the age-adjusted mortality rates for COPD (per 100,000 individuals) among men were 47.8 in whites, 34.1 in blacks, 32.8 in Puerto Ricans, 15.2 in Cubans, and 12.0 in Mexicans (14). Among women in the United States during the same period, Puerto Ricans had the second-highest (15.5/100,000) and Mexicans the third-lowest (6.4/100,000) age-adjusted mortality rate for COPD of all ethnic groups.

KNOWN AND POTENTIAL RISK FACTORS FOR COPD IN HISPANICS

Cigarette Smoking

Cigarette smoking, the most important risk factor for COPD worldwide (5), is common among residents of Hispanic-American

nations. Although an adequate comparison of tobacco use across countries is not possible because of likely differences in study methodology and accuracy of exposure assessment, we show estimates of the prevalence of current smoking in selected Hispanic-American nations in Table 1. The estimated mean annual cigarette consumption per capita (including smokers and nonsmokers) in Hispanic-American nations ranges from 160 in Peru to 712 in Mexico to 1,418 in Argentina (15).

Recent data suggest that Mexican smokers prefer "regular" (nonlight) brands of cigarettes (16). In a study of 2,373 smokers in four nations, the proportion of smokers who reported consuming "light" cigarettes was lower in Mexico (17.6%) than in China (23.9%), Poland (56.7%), and Brazil (71.9%) (16). In that study, Mexican smokers who consumed regular brands smoked more cigarettes and thus had higher concentrations of salivary cotinine than those who consumed light brands.

Tobacco use varies among and within Hispanic subgroups in the United States. In a nationwide survey conducted from 1999 to 2001 among 133,081 adults (≥18 yr) representing 14 ethnic groups, cigarette use in the preceding month was reported by 19.2% of Cubans, 21.3% of Central or South Americans, 22.8% of Mexicans, 25.7% of blacks, 27.4% of whites, and 30.4% of Puerto Ricans (17). Puerto Ricans reported the highest prevalence of recent cigarette use among males (34.2%) and females (27.3%) of all ethnic groups. Recent cigarette use was reported more often by males than by females across Hispanic subgroups (e.g., 29.8 vs. 15.6% in Mexicans), with narrower gender gaps observed in Puerto Ricans (34.2 vs. 27.3%) and Cubans (21.1 vs. 17.5%). In adjusted analyses of data from a separate survey of 8,882 Hispanics in five Hispanic subgroups in the U.S. mainland, Puerto Ricans and Cubans were more likely to be heavy smokers than Mexicans (18). Although Puerto Rican and Cuban females were more likely to be current smokers than Mexican females, there was no significant difference in current smoking in males across Hispanic subgroups.

Several studies have examined the relation between acculturation and tobacco use among Hispanics in the United States, yielding conflicting results (18–21). A population-based survey in New Mexico conducted from 1984 to 1985 showed no clear pattern of smoking based on language preference, which was used as a surrogate for acculturation (19). In contrast to these findings, a survey of Hispanic women in California showed that those who spoke predominantly English were consistently more likely to be current daily smokers than those who spoke predominantly another language (20). In a cross-sectional study of Hispanics in the U.S. mainland, acculturation (as measured by questionnaire) was directly associated with current smoking in women but inversely associated with current smoking in men (18). Because of limited statistical power, no results were reported for specific Hispanic subgroups. In a study of 5,030 adults of Mexican descent in Houston, Texas, males were more likely to report ever smoking than females regardless of their country of birth and the country in which they began to smoke (21). However, this gender gap in smoking was more pronounced in adults who were born in and began to smoke in Mexico, likely reflecting cultural differences in acceptability of smoking among women between the United States and Mexico. In that study, increased acculturation was associated with lifetime smoking in women but not in men.

Exposure to Biomass Smoke

Smoke from wood (commonly used as a cooking fuel in rural areas of Hispanic America) (22) contains particles, polycyclic aromatic hydrocarbons, and carbon monoxide (23). Results from studies limited by a cross-sectional design and potential selection bias suggest that exposure to biomass and wood smoke is associated with COPD in Hispanic America (24, 25). In a hospital-based, case-control study of adult (≥40 yr) Mexican women, exposure to wood smoke was associated with an increased risk of chronic bronchitis (with and without chronic airflow obstruction), and there was a linear relationship between intensity of wood smoke exposure (hour-years of cooking with a wood stove) and chronic bronchitis (24). In analyses adjusting for age and intensity of cigarette smoking, exposure to more than 200 hour-years of biomass smoke was associated with a 75-fold increased odds of chronic bronchitis with chronic airflow obstruction (95% confidence interval for odds ratio, 18–306). A subsequent cross-sectional study of 845 adult Mexican women in a rural town showed that current biomass smoke exposure was associated with respiratory symptoms (cough and phlegm) and a mild reduction in FEV_1/FVC (25). All women with moderate to severe airflow obstruction reported current exposure to a biomass stove. Among women who cooked with biomass stoves and had an assessment of indoor particle concentrations in their kitchen ($n = 410$), exposure to PM_{10} (particulate matter with a diameter ≤ 10 μm) of more than 2.6 mg/m^3 was associated with an 81-ml reduction in FEV_1.

COPD resulting from exposure to biomass smoke and COPD due to tobacco use have different clinical presentations but may have the same overall prognosis. Among 481 adults attending a COPD clinic in Mexico City, 136 (28.3%) were nonsmokers with history of exposure to biomass smoke (26). Compared with adults with COPD due to tobacco use, those with COPD due to biomass smoke exposure were more likely to be women (84 vs. 24%) and to have better spirometric measures of lung function but lower oxygen saturation on room air and higher Pa_{CO_2} at baseline. There was no significant difference in symptom severity or health-related quality of life between adults with COPD due to biomass smoke and those with COPD due to tobacco use at baseline. After adjustment for age and other covariates, adults with COPD due to exposure to biomass smoke had a mortality rate at follow-up (median time, 83 mo) similar to those with COPD due to tobacco use.

Air Pollution

Results of studies limited by an ecological design suggest that air pollution leads to increased COPD mortality in some urban areas of Hispanic America. Salinas and Vega compared standardized mortality ratios from COPD among zones of greater Santiago (the capital of Chile) from 1988 to 1991 (27). The central zones (which had heavier vehicular traffic and where "safe" thresholds for air quality standards for carbon monoxide, ozone, and suspended particles were frequently exceeded) had higher standardized mortality ratios for COPD. In another study, Téllez-Rojo and colleagues showed that each 3-day lag increase of 10 μg/m^3 in the daily average level of PM_{10} was associated with a 4.1% (95% confidence interval, 1.3–6.9%) increase in COPD mortality (outside of a medical unit) among elderly residents of Mexico City in 1994 (28). For COPD mortality in medical units, the observed effects of PM_{10} were weaker and less consistent. These discrepant findings may be partly explained by unmeasured confounding by socioeconomic status and concurrent illnesses among individuals with COPD (29). It has been estimated that if pollution control policies were implemented in Mexico City (Mexico), Santiago (Chile), and São Paulo (Brazil), 48,000 cases of chronic bronchitis could be prevented over a 20-year period (30).

Genetics

In non-Hispanic populations, several studies have shown a significant genetic contribution to COPD (31–33) and identified

genomic regions (34, 35) and genes (other than α_1-antitrypsin) (36, 37) that may confer susceptibility to the detrimental effects of cigarette smoking.

Little is known about COPD genetics in Hispanics. Lung function (an intermediate phenotype of COPD that is usually assessed by spirometry) has been shown to be significantly heritable in a population-based study of Hispanic families in New Mexico (38) and in a study of families of Costa Rican children with asthma (39). However, there have been no studies of the heritability of lung function in families of Hispanics with COPD.

Although the estimated prevalence of α_1-antitrypsin deficiency in Spain is among the highest in Europe (40), there are no sufficient data on the prevalence of α_1-antitrypsin deficiency in Hispanic America (41). To date, there has been no genomewide study of linkage or association for COPD susceptibility genes in any Hispanic subgroup. Only one study has examined candidate genes (surfactant protein genes) for COPD in a Hispanic population (101 cases and 81 control subjects from Mexico) (42). Limitations of that study include small sample size, lack of adjustment for multiple testing, and no control for population stratification (confounding by differences in allelic frequencies and disease prevalence in the ancestral populations of a racially admixed group such as Mexicans).

DIAGNOSIS OF COPD IN HISPANICS

According to the GOLD, a diagnosis of COPD should be considered and confirmed by spirometry in any adult ($\geqslant$40 yr) at risk (e.g., cigarette smoker) with symptoms of cough, sputum production, or dyspnea (5). Common reasons for misdiagnosis of COPD in Hispanics may include lack of reference values for spirometric measures for most Hispanic subgroups other than Mexican Americans (43), language barriers, and inadequate access to health care (which may preclude spirometry). In 2003, approximately one-third of the 41.2 million uninsured individuals in the United States were Hispanic (44).

English-speaking practitioners need to be aware of variation in descriptive terms for dyspnea in Spanish among and within Hispanic subgroups. Vázquez-García and colleagues described seven clusters of descriptors for dyspnea in patients with COPD who spoke Mexican Spanish (45). Because some descriptors of dyspnea (e.g., *agitación*) had no adequate English translation, the authors raised the possibility of missing this symptom even when using an interpreter. A separate study in New York City showed similar difficulties translating the word "wheeze" into Spanish (46). In a study of chronic bronchitis in Nicaragua, a Spanish questionnaire translated directly from the British Medical Research Council questionnaire had a lower positive predictive value (57%) than a translated questionnaire adapted to local Spanish (90%) (47).

Obesity is a risk factor for obstructive sleep apnea (OSA) (48) and a common entity in Mexican Americans (49) and Puerto Ricans (50). OSA and COPD are common diseases that can coexist in a significant proportion of patients (51, 52). Diagnosing and treating OSA may result in decreased severity of nocturnal hypoxemia in Hispanics with COPD (52).

TREATMENT OF COPD IN HISPANICS

The cornerstones of COPD treatment are smoking cessation, oxygen therapy (when appropriate), management of chronic symptoms, pulmonary rehabilitation, and treatment of acute exacerbations. Treatment of COPD in Hispanics may be impaired by lack of access to health care, language barriers, and cultural beliefs.

There is insufficient evidence to support or refute the efficacy of ethnicity-specific smoking cessation programs in Hispanics, particularly in the absence of interventions tailored to specific Hispanic subgroups (53). English-speaking Hispanics are significantly less likely to use nicotine replacement therapy or antidepressants when attempting to quit smoking compared with whites (54). Cultural beliefs that may explain this finding include viewing smoking as a weakness rather than an illness, general avoidance or mistrust of pharmaceuticals (including bupropion and nicotine replacement therapy), and misconceptions regarding both smoking and smoking cessation (55).

Treating acute exacerbations of COPD (AECOPDs) in a cost-effective manner is an important goal in Hispanic-American countries with limited health care budgets. A pharmacoeconomics study using unpublished data from six Hispanic-American countries and Brazil concluded that initial treatment failure (resulting in repeat clinic visits, emergency department visits, or hospitalizations) constituted over half of the cost of treatment in AECOPDs (56) and speculated that use of broad-spectrum antibiotics as first-line treatment of AECOPDs may reduce overall treatment costs. This latter suggestion is supported by evidence of increased resistance to less expensive, earlier-generation antibiotics by common bacterial pathogens in AECOPDs, possibly due to overuse resulting from the availability of these medications without a prescription in Hispanic America (57, 58).

FUTURE DIRECTIONS

Many published studies of COPD in Hispanics make the implicit assumption that Hispanics are a homogeneous group despite significant evidence to the contrary. New studies of the prevention, epidemiology, genetics, diagnosis, and treatment of COPD in Hispanics should focus on subgroups that are carefully characterized with regard to self-reported ethnicity, place of birth, country of origin, smoking habits, biomass smoke exposure, and socioeconomic status.

Studies of genetic susceptibility to the detrimental effects of cigarette smoking in Hispanics should take into account and adjust for potential population stratification. This can be accomplished either by genomic control methods (59) for case-control studies or by using a family-based design (which protects against bias due to population stratification). On the other hand, racial admixture can be properly leveraged in studies of complex diseases such as COPD in Hispanics. Admixture mapping aims to localize disease-susceptibility genes in racially admixed populations in which the ancestral populations have differing genetic risk (60–62). Very recently, genomewide panels of single-nucleotide polymorphisms were designed to differentiate Amerindian ancestry from others (primarily European) in Mexican Americans (63) and in other Hispanic subgroups of predominantly Spanish and Amerindian ancestry (64, 65). Although admixture mapping may prove helpful for future studies of COPD and other complex diseases in the aforementioned Hispanic subgroups, there is uncertainty as to whether available single-nucleotide polymorphism panels will perform well in admixture mapping of complex diseases in Hispanic populations with substantial African ancestry (e.g., Puerto Ricans) (65).

Although PLATINO represents a critical first step toward understanding the epidemiology of COPD in Hispanic America, further research is needed with regard to the prevalence of this disease in other countries and in rural areas, as well as validation of COPD diagnoses made by questionnaire and/or spirometry (e.g., by radiologic assessment). Because of previous data on chronic bronchitis, high prevalence of asthma, and patterns of tobacco use (particularly in women), studies of COPD

prevalence and morbidity are urgently needed among Puerto Ricans, both in the U.S. mainland and in Puerto Rico.

Because of concerns regarding underreporting of smoking in some Hispanic subgroups (e.g., Mexican Americans) (66), future studies of smoking should assess tobacco use by objective measures (e.g., urinary cotinine) to validate exposure assessment by questionnaires. Given evidence of a positive association between acculturation and smoking in Mexican women, studies of the impact of migration and acculturation on COPD are needed among individuals of Mexican descent and members of other Hispanic subgroups.

Because the use of spirometric reference values developed for other ethnic groups can result in misclassification of airflow obstruction in Hispanics (67), reference values for spirometric measures of lung function need to be developed for most Hispanic subgroups in the United States (e.g., Puerto Ricans) and Hispanic America. Rigorous evaluation of the efficacy of ethnicity-specific prevention programs and therapeutic interventions (e.g., smoking cessation) and improved cultural competence among non-Hispanic physicians should be undertaken.

Despite the need for further research, definite actions can now be taken to improve the care of Hispanics with COPD. These include vigorous programs to curtail smoking, reducing exposure to air pollution (in urban areas) and biomass smoke (in rural areas), and improving access to health care. Providers caring for Hispanic patients should have a high index of suspicion for tobacco use and COPD in Hispanics in general and among certain subgroups (e.g., Puerto Ricans) in particular, be aware of nonsmoking causes of COPD (e.g., exposure to biomass smoke), provide advice against smoking, and facilitate access to smoking cessation programs whenever appropriate. Finally, English-speaking physicians caring for Hispanics with COPD should have access to translators who are familiar with the dialect(s) and cultural beliefs of the predominant Hispanic subgroups living in their communities.

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