Journal of Pulmonary & Respiratory Medicine

Kinney et al., J Pulm Respir Med 2014, 4:6
DOI: 10.4172/2161-105X.1000220

Research Article Open Access

The Protective Effect of Hispanic Ethnicity on Chronic Obstructive Pulmonary Disease Mortality is Mitigated by Smoking Behavior

Gregory L Kinney[1]*, Deborah SK Thomas[2], Lisa Cicutto[3], Lee S Newman[4], Sharon Lutz[5] and John E Hokanson[1]

[1]Department of Epidemiology, Colorado School of Public Health, University of Colorado Denver, Aurora Colorado, USA
[2]Department of Geography and Environmental Sciences, University of Colorado Denver, Denver Colorado, USA
[3]National Jewish Hospital and University of Colorado Denver, Denver Colorado, USA
[4]Department of Environmental Health, Colorado School of Public Health, University of Colorado Denver, Aurora Colorado, USA
[5]Department of Biostatistics, Colorado School of Public Health, University of Colorado Denver, Aurora Colorado, USA

Abstract

Rationale: Studies in Hispanic/Latino populations have shown ethnicity to be either a predictive or protective factor for COPD mortality and it is unclear whether this disagreement is attributable to ethnic differences in smoking rates, smoking behavior or differences in genetic susceptibility.

Objectives: This study will examine the role of smoking behavior as a means of explaining differences in risk for COPD mortality between Hispanics and non-Hispanic whites.

Methods: Participants were recruited into a cohort study from the San Luis Valley in Colorado beginning in the early 1980's and followed for mortality until 2012. COPD and cardiovascular disease are often comorbid conditions and account for the competing risk of CVD in the analysis of COPD mortality. Mortality searches were conducted regularly and all ICD codes were collected for mortality event. Primary and secondary causes of each event were assessed using appropriate codes.

Results: Hispanic current smokers did not differ from NHW current smokers in years smoked (p=0.6) but Hispanic former smokers accumulated more years smoked compared to NHW former smokers (22 vs. 20, p=0.047). Hispanic ethnicity was significantly protective for COPD mortality adjusting for age, gender, pre-existing emphysema, hypertension and smoking status and accounting for the effect of CVD mortality (RR=0.58, 95% C.I. 0.34-0.99, p=0.035). Further adjustment for smoking behavior mitigated this effect.

Conclusions: The lower COPD mortality seen in Hispanic smokers may be due lower cumulative exposure to tobacco smoke. Thus, smoking behavior may play a key role in explaining differences in COPD mortality as they relate to Hispanic ethnicity.

Keywords: Chronic obstructive pulmonary disease; Smoking; Cardiovascular Disease (CVD); International classification of disease

Scientific Knowledge on the Subject

It is unclear whether Hispanic ethnicity is protective or predictive for mortality due to chronic obstructive pulmonary disease (COPD). Other causes of mortality are also strongly associated with smoking and they may compete as a cause of death with COPD. Smoking behaviors may also vary by ethnicity and this may affect specific mortality.

What this Study Adds to the Field

This study demonstrates that smoking behaviors differ between Hispanics and non-Hispanic whites and that controlling for those differences mitigates the protective effect observed for COPD mortality in Hispanics. Smoking is a complex behavior and may not be fully captured by commonly measured indicators.

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is the third leading cause of death in the United States and its incidence is increasing me [1]. COPD is associated with cardiovascular disease (CVD) and the combination of COPD and CVD represent roughly ½ of smoking attributable deaths in the U.S. [2]. It is unclear whether differences in COPD incidence and mortality are attributable to ethnic differences in smoking rates or smoking behavior or differences in genetic susceptibility. Brehm et al. [3] correctly point out that "Hispanic/Latino" is a heterogeneous group genetically and that what work there has been in Hispanic populations has had methodological flaws [3].

Chronic Lower Respiratory Diseases is the 7th leading cause of death in Hispanics [4]. Proyecto LatinoAmericano de Investigación en Obstrucción Pulmonar (PLATINO) found COPD prevalence in five major Latin American cities to be higher than expected (7.8-20% versus 4-10% expected) [5]. Samet et al. [6] found Hispanic ethnicity protective for COPD compared to non-Hispanic Whites (NHW) and attributed this to differences in smoking, however; Adams et al. found no difference in COPD [7] despite lower tobacco use in Hispanics. Lipton et al. report Hispanic ethnicity was a positive predictor of spatial "hot spots" of higher hospitalization related to COPD [8]. Recently Bruse et al. [9] found Hispanic ethnicity was protective for COPD and reduced pulmonary function and attributed this difference to genetic susceptibility beyond the effect of smoking.

Smoking is the leading preventable cause of morbidity and mortality [10] and is responsible for one in five deaths and over $193 billion dollars in health care costs in the U.S. Smoking causes COPD [11] and CVD, and COPD with comorbid CVD substantially increases clinical and economic burden [12]. Smoking is decreasing in both

***Corresponding author**: Gregory L Kinney, Department of Epidemiology, Colorado School of Public Health, University of Colorado Denver, Aurora Colorado, USA, Tel: 303-724-4437; E-mail: Greg.Kinney@UCDenver.edu

Received October 20, 2014; **Accepted** November 20, 2014; **Published** November 25, 2014

Citation: Kinney GL, Thomas DSK, Cicutto L, Newman LS, Lutz S, et al. (2014) The Protective Effect of Hispanic Ethnicity on Chronic Obstructive Pulmonary Disease Mortality is Mitigated by Smoking Behavior. J Pulm Respir Med 4: 220. doi:10.4172/2161-105X.1000220

NHW and Hispanics l [13] however a concomitant decrease in COPD incidence and mortality has not been observed. This is likely due to lag time between smoking exposure and diagnosis of COPD.

This study examines the role of Hispanic ethnicity on COPD mortality in a stable cohort of Hispanics and NHWs. The relationship between COPD mortality, Hispanic ethnicity and smoking behavior was examined accounting for the competing risk of CVD in order to assess whether smoking behavior varies by ethnicity and whether this influences COPD mortality.

Methods

Study population

The San Luis Valley Diabetes Study (SLVDS) was designed to study diabetes and its complications) in Hispanics and NHWs [14]. This cohort (n=1890) was 43.5% Hispanic, with X% having diabetes. All participants were 20-74 years of age at the inception of the study.

An intensive follow-up of vital status was conducted on this cohort in 1998. This follow-up consisted of telephone contact, death certificate matching and tracking of posted obituaries and it identified 316 deceased individuals. Coroners' reports, autopsy results and medical records were obtained for 98% of those deceased at that time. Another mortality follow-up was conducted in 2002 identifying a further 149 decedents and all International Classification of Disease (ICD9 if death occurred before 1999 or ICD10 beginning in 1999) codes (primary and secondary) were recorded for each death. Further mortality follow-ups were performed in 2007 and 2010 using participant social security numbers within the state of Colorado.

Mortality assessment

A broad definition of COPD was used that included all primary and secondary causes of death using International Classification of Disease (ICD9 or ICD10) codes; 490-496 (ICD-9), J40-J47 (ICD-10). ICD codes were stripped to 3 digits identifying these broad mortality classifications; 490: bronchitis NOS, 491: chronic bronchitis, 492: emphysemas, 493: asthma, 494: bronchiectasis, 495: extrinsic allergic alveolitis, 496: COPD. A primary code of Asthma with no secondary code of emphysema, bronchiectasis or extrinsic allergic alveolitis was classified as not having COPD and a primary code of Asthma with a secondary code matching any of the above was included as COPD. Primary bronchiectasis or primary extrinsic allergic alveolitis were included as COPD. Primary bronchitis or chronic bronchitis with a secondary code suggesting COPD was included as COPD. A similar approach was used to assigning CVD mortality using ICD9 and ICD10 codes; ICD-9 codes 390.x to 459.x or ICD-10 codes I00.x to I99.x identified a mortality event as CVD for this study.

Smoking related covariates

Current and former smoking status was assessed at the baseline visit using a standard questionnaire. Pack years were calculated as # cigarettes/day multiplied by # years smoked divided by 20. Smoking behavior was assessed using a standard set of questions that recorded the age of initiation and termination of smoking, whether the participant inhaled the smoke, during how many puffs, how deeply, how much of each cigarette was generally allowed to burn in the ashtray and how far down cigarettes were usually smoked. Age of initiation and termination of smoking were continuous values and the remaining behavioral variables were categorical. Emphysema and chronic bronchitis were assessed using self-reported occurrence of a doctor's diagnosis and the age of that diagnosis.

Statistical methods

All demographic and univariate analyses were performed using SAS 9.2 (SAS Institute Inc, Cary NC, USA). Continuous variables are expressed as mean and standard deviation and were compared using Student's T-test. Categorical variables are expressed as counts and percentages and were compared using chi square. All p values are 2 tailed and $p<0.05$ was considered significant.

Because of the strong, positive association between smoking and COPD as well as between smoking and CVD, competing risks must be considered when assessing COPD mortality. The cuminc macro [15] in SAS was used to estimate the hazard of the subdistribution or cumulative incidence function (CIF) using the method of Fine and Gray [16] and its variance using the Delta method [17]. In this approach subjects experiencing the competing risk of interest are always in the risk set and contribute density fully until experiencing the competing risk. At that point they continue to contribute density in a weighted function that is reduced over time. Where competing risks do not occur this weighted likelihood function is identical to the Cox Proportional Hazards model. The CIFs were compared for smoking status, ethnicity and a combination of both using the method of Pepe and Mori [18] which directly compares the area between the curves of each CIF weighted for the occurrence of the competing risk.

The Competing Risks Regression (CRR) function in the R [19] cmprsk package was used to calculate the hazard for the risk of COPD mortality accounting for the competing risk of CVD mortality controlling for covariates. The approach proposed by Fine and Gray and implemented in R for multivariate modeling in the presence of competing risks is a model for the subdistribution hazard. The subdistribution hazard is defined as the hazard of failing from a given cause in the presence of competing events, given that a subject has survived or has already failed due to different causes. The relative risk reported in these analyses is the ratio of subdistribution hazards for the group of interest with respect to the baseline group, holding all other covariates equal.

Results

A total of 1887 participants were included in this analysis. There were similar proportions of Hispanic and NHW (49% vs. 51%) and gender (47.5% Male Hispanic vs. 52.8% Male NHW, p=0.1) and did not differ by age (54.0 vs. 54.3, p=0.6). Hispanic participants had lower BMI (26.5 verses 27.2, p=0.0007) and were more likely to be hypertensive (41.3% vs. 36.2%, p=0.025). Hispanic participants differed from NHWs in their marital status (more likely to be married, less likely to be divorced or widowed), education completed (less likely to have completed greater than 12 years), working status (less likely to be currently working) and income (more likely to earn <$20,000/year) ($p<0.0001$ in all cases). Smoking status was distributed differently by ethnicity ($p<0.0001$) where Hispanics were more likely to be current smokers (29% vs. 20%) and less likely to be never smokers (41% vs. 49%) (Table 1).

Smoking behavior was compared between Hispanic and NHW participants in current and former smokers. The age of initiation of smoking was similar between Hispanic current smokers and NHW current smokers as well as for the same comparison in former smokers. Hispanic former smokers tended to have stopped smoking at a later age (42 vs. 39, p=0.005). Hispanic current and former smokers accumulated less pack years (20 and 19 vs. 33 and 24, $p<0.0001$ and p=0.004 respectively) and these pack years were accumulated differently compared to NHW current and former smokers with

Hispanic participants smoking less packs per day (0.6 and 0.7 vs. 1.0 and 1.0, p<0.0001 for both comparisons). Hispanic current smokers did not differ from NHW current smokers in years smoked (p=0.6) but Hispanic former smokers accumulated more years smoked compared to NHW former smokers (22 vs. 20, p=0.047). Hispanic former smokers were less likely to report inhaling when they smoked (76% vs. 83%, p=0.04) and of those reporting inhaling, current Hispanic smokers were less likely to inhale every puff than current NHW smokers (54% vs. 74%, overall chi-square comparison for the three categories, p=<0.0001). Health related outcomes of smoking; chronic bronchitis age and emphysema, were similar between ethnicities though the age at diagnosis of chronic bronchitis in former smokers was older in Hispanics compared to NHW (67 vs. 33, p=0.004) and the percent reporting emphysema was lower in Hispanics compared to NWH in current smokers (1% vs. 3%, p=0.01) (Table 2).

Figure 1 through 3 show the cumulative incidence functions for COPD mortality accounting for the effect of the competing risk of CVD mortality for three categories, smoking, ethnicity and the

		Hispanic N=929		NHW N=958		p
Age (years, SD)		54	12.5	54.3	12	0.64
Sex (n,%)						0.104
	Male	410	47.2	458	52.8	
	Female	521	51	501	49	
BMI (kg/m^2, SD)		26.5	4.8	27.2	5.1	0.0007
Hypertension (n,%)						0.025
	Hypertension	355	41.3	336	36.2	
	No Hypertension	504	58.7	593	63.8	
Marital Status (n,%)						<0.0001
	Never Married	45	4.9	31	3.2	
	Married	680	73.4	791	82.6	
	Divorced	67	7.2	46	4.8	
	Widowed	93	10	73	7.6	
	Other	41	4.4	17	1.8	
Education Completed (n,%)	(years)					<0.0001
	<8	209	22.5	17	1.8	
	8 - 11	284	30.5	124	12.9	
	12	253	27.2	373	38.9	
	13+	185	19.9	445	46.4	
Working Status (n,%)						<0.0001
	Not Working	534	57.6	382	39.9	
	Working	393	42.3	576	60.1	
Income (n,%)	($/year)					<0.0001
	<20,000	697	74.9	414	43.2	
	20,000 - 50,000	200	21.5	447	46.6	
	>50,000	34	3.7	98	10.2	
Cigarette Smoking (n,%)						<0.0001
	Current Smoker	267	28.7	193	20.2	
	ex-Smoker	277	29.8	298	31.1	
	Never Smoker	385	41.4	467	48.8	

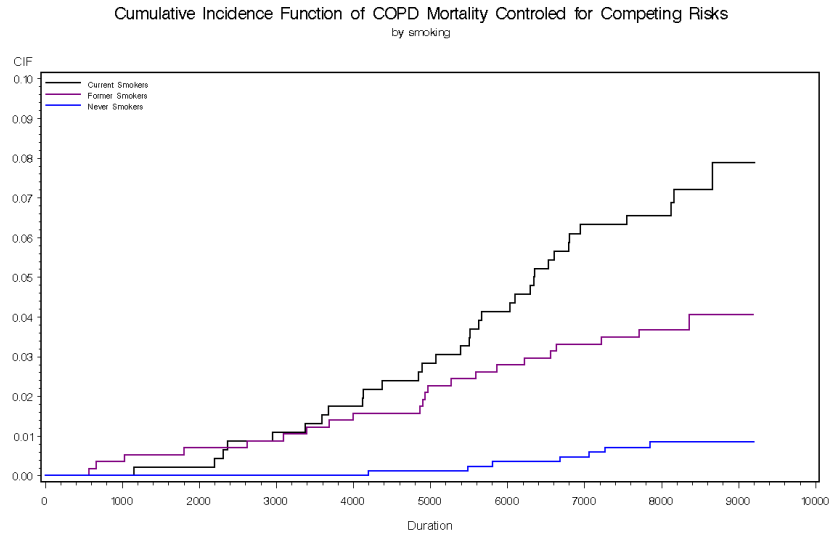
Table 1: Demographic characteristics of study participants.

	Hispanic		NHW			
	Current n=267	Former n=277	Current n=193	Former n=298	P (current)	P (former)
Start Age (years ± SD)	21.1 ± 9.9	19.8 ± 8.0	19.7 ± 7.3	18.7 ± 4.6	0.088	0.073
Stop Age (former only) (years ± SD)	NA	42.1 ± 14.3	NA	38.7 ± 14.0	NA	0.005
Pack Years (packs/year, SE)	19.6, 1.4	18.6, 1.4	32.5 ± 1.7	24.3 ± 1.4	<0.0001	0.004
Years Smoked (years, SE)	31.7, 0.9	22.3, 0.9	32.4 ± 1.0	19.9 ± 0.8	0.59	0.047
Packs per Day (packs, SE)	0.6, 0.04	0.7, 0.04	1.0 ± 0.05	1.0 ± 0.4	<0.0001	<0.0001
Inhaled when smoke (% Yes)	82.1%	76.4%	88.8%	83.3%	0.05	0.04
Inhale puffs every/few/v-few (%)	54/27/18	65/26/9	74/20/6	74/21/5	<0.0001	0.088
Chronic Bronchitis n (%)	13 (2.8)	9(1.6)	18 (3.9)	11(1.9)	0.17	0.95
Chronic Bronchitis age (yrs)	51.6, 7.7	67.4, 8.7	34.4, 6.7	32.7, 7.9	0.09	0.004
Emphysema n (%)	4 (0.9)	9 (1.6)	13 (2.8)	12 (2.1)	0.01	0.72
Emphysema age	42.8, 4.7	63.0, 3.5	51.6, 3.4	55.3, 3.2	0.14	0.11

1: "Current" compares current Hispanic smokers to current Non-Hispanic White smokers, "former" compares former Hispanic smokers to former Non-Hispanic White smokers.

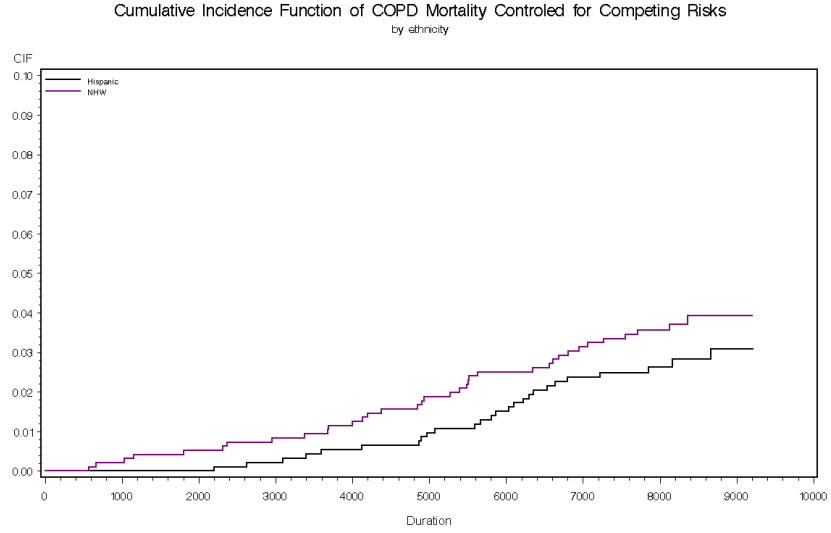
Table 2: Smoking behavior variables in Hispanics and Non-Hispanic Whites by smoking status.

combined effect of smoking and ethnicity. As expected Figure 1 shows that current and former smokers have significantly increased COPD mortality compared to never smokers (Cumulative Incidence? Current=0.079, Former 0.040, Never 0.009, p<0.0001 Current vs. Never, p=0.0004 Former vs. Never). Current and Former smokers had similar COPD mortality experiences (p=0.224). Figure 2 shows the CIF for Hispanic participants compared to NHW participants accounting for the competing risk of CVD mortality without considering smoking. Overall, ethnicity has no significant effect on COPD mortality (CI Hispanic=0.031 vs. NHW 0.039, p=0.071). Figure 3 combines smoking status and ethnicity and shows that after accounting for the competing risk of CVD mortality, Hispanic Current smokers have significantly lower incidence of COPD mortality compared to NHW Current smokers (CI Hispanic=0.061 vs. NHW 0.1, p=0.037) The risk for Hispanic Current smokers is also not different compared to the risk of COPD mortality for NHW former smokers or Hispanic former smokers (CI Hispanic Current=0.061 vs. NHW Former 0.044, p=0.550; vs. Hispanic Former p=0.771). Though the comparison between NHW Current smokers and NHW Former smokers was larger than that comparison in Hispanics it did not reach statistical significance (CI NHW Current=0.1 vs. NHW Former=0.044, p=0.132).



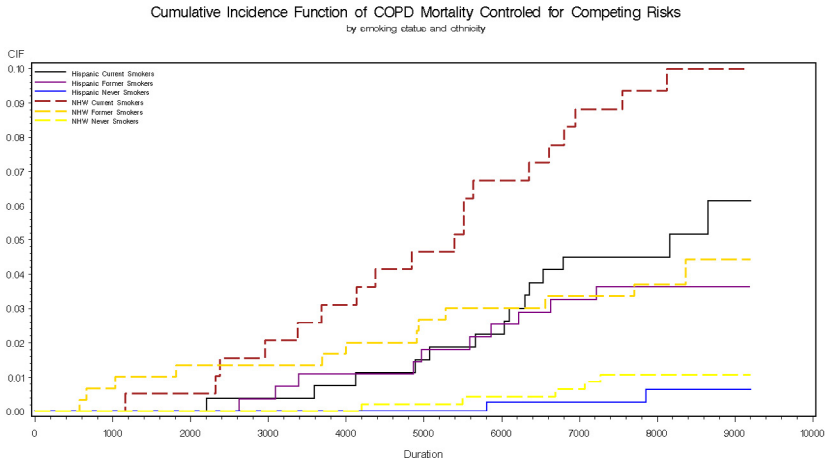
Current Smokers verses Never Smokers, p<0.0001
Former Smokers verses Never Smokers, p=0.0036
Current Smokers verses Former Smokers, p=0.211

Figure 1: The Cumulative Incidence Function (CIF) for COPD mortality accounting for CVD mortality by smoking category. Current Smokers are plotted in black, Former Smokers are plotted in purple and Never Smokers are plotted in blue.
Current Smokers are at increased risk for COPD mortality compared to Never Smokers (p<0.0001) but they have a similar CIF compared to Former Smokers (p=0.224). Former Smokers are at increased risk for COPD mortality compared to Never Smokers (0.00041).



Non-Hispanic White versus Hispanic, p=0.071

Figure 2: The Cumulative Incidence Function (CIF) for COPD mortality accounting for CVD mortality by ethnicity. Hispanic ethnicity is plotted in black and Non-Hispanic White ethnicity is plotted in Purple. Non-Hispanic White participants have a similar CIF for COPD mortality compared to Hispanic participants (p=0.071).



Hispanic Current Smokers versus NHW Current Smokers, p=0.037
Hispanic Former Smokers versus NHW Former Smokers, p=0.392
Hispanic Non-Smokers versus NHW Non-Smokers, p=0.267

Figure 3: The Cumulative Incidence Function (CIF) for COPD mortality accounting for CVD mortality by smoking category and ethnicity. Solid lines indicate Hispanic ethnicity and dotted lines indicate Non-Hispanic White ethnicity. Hispanic Current Smokers are plotted in black, Hispanic Former Smokers are plotted in purple and Hispanic Never Smokes are plotted in glue. NHW Current Smokers are plotted in red, NWH Former Smokers are plotted in orange and NHW Never Smokers are plotted in yellow. NHW Current Smokers are at increased risk for COPD mortality compared to Hispanic Current Smokers (p<0.037). Hispanic Former Smokers have a similar CIF compared to NHW Former Smokers (p=0.392) and Hispanic Never Smokers are also similar to NHW Never Smokers (p=0.267).

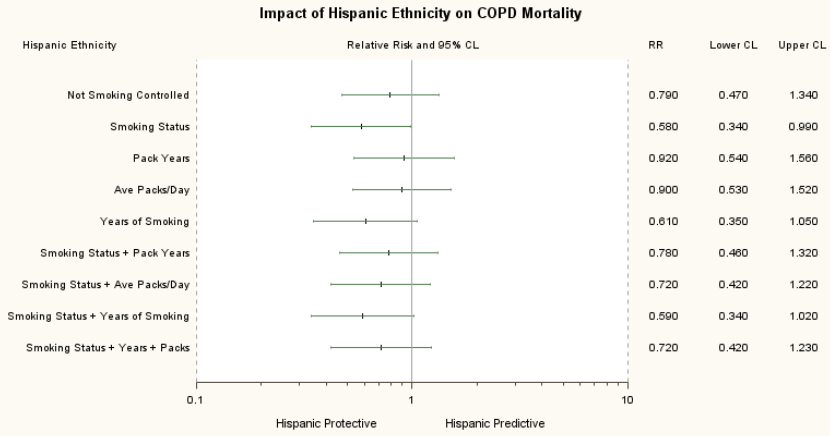


Figure 4: The relative risk (sub-distribution hazards ratio) for Hispanic participants compared to Non-Hispanic Whites for each of five distinct competing risks regression models of COPD mortality accounting for CVD mortality. Each model is controlled for age, gender, pre-existing emphysema and hypertension with smoking assessed using different measures.

Competing risks regression models predicting COPD mortality, accounting for the effect of CVD mortality are reported in Figure 4. Competing risks regression reports a relative risk (ratio of the sub-distribution of the hazards) and 95% confidence intervals for these models. This figure shows the risk for Hispanic ethnicity compared to NHW ethnicity controlled for age, gender, pre-existing emphysema, hypertension and controlling for smoking using several approaches in separate models. Individual models are; no smoking control, controlled for smoking status (Current Smoker or Former Smoker compared to Never Smoker), controlled for ATS packyears of smoking as a continuous variable and controlled for the components of packyears; years smoked and packs per day both as continuous variables. With no control for smoking, Hispanic ethnicity is not associated with COPD mortality (RR=0.79, 95% C.I. 0.47-1.34, p=0.29). Controlling for smoking status as above shows that Hispanic ethnicity is significantly protective for COPD mortality (RR=0.58, 95% C.I. 0.34-0.99, p=0.035). Controlling for packyears of smoking or the elements of packyears (average packs per day, years smoked) did not result in a significant association between Hispanic ethnicity and COPD mortality (packyears p=0.59, average packs per day, p=0.54, years smoked p=0.06); however, all RR estimates for Hispanics compared to NHWs are <1.0.

Discussion

Hispanics have lower COPD mortality compared to NHW after accounting for smoking status and competing risks from cardiovascular mortality. Measures of smoking were assessed in this population and a significant protective effect of ethnicity was found only when controlling for smoking status, but that effect was reduced when controlling for differences in smoking behavior as measured by packyears, average packs per day smoked or years of smoking exposure. We found significant differences between Hispanic and NWH smokers that are

associated with the intensity of the smoking experience. Hispanic ethnicity was associated with less intense smoking where smokers reported inhaling smoke at every puff less frequently. This difference in mortality may be mediated by differences between Hispanic and NHW smoking behaviors that are not captured by commonly collected smoking questionnaires. Our study also examined smoking behavior in more detail than is typically reported, e.g. measuring smoking intensity, depth of inhalation and regularity of inhalation rather than reporting smoking status and pack-years only.

Smoking is a complex behavior and some aspects of this behavior that differ by ethnicity are clear in this study; while overall exposure (pack years) is significantly lower in Hispanic participants, they were also exposed to less smoke due to a different, less intense pattern of smoking exposure. This indicates that positive smoking status measures a less intense smoking exposure in Hispanics compared to NHW. It also indicates that pack years, while lower in Hispanics, represents a lower exposure to smoking intensity than in NHW due to less inhalation. This observation should be confirmed in future studies with complete data collection for those variables. Taken together, these findings suggests that the risk of COPD mortality in Hispanic compared to NHW subjects is mediated by these commonly unmeasured aspects of smoking behavior. This may explain the protective effect attributed to Hispanic ethnicity on COPD reported in other studies that did not account for detailed smoking behavior.

We also observed that Hispanic former smokers who report having chronic bronchitis do so at a significantly older age compared to NHW former smokers. We speculate that this may be related to access to healthcare and the establishment of a subsequent diagnosis in Hispanics at an older age. We also observe that several participants who quit smoking also reported physician diagnosed emphysema or chronic bronchitis. Of those reporting emphysema, 70% reported quitting smoking within 3 years of that diagnosis and 50% reported quitting smoking within 3 years of a diagnosis of chronic bronchitis. The proximity of quitting smoking to a diagnosis may suggest that those participants identified as former smokers were experiencing more severe disease compared to people identified as current smokers.

Other studies have found that Hispanics compared to NHWs report more current smoking, similar numbers of packyears, less packs per day and more years of smoking. In our study, Hispanic ethnicity is associated with higher current smoking but lower pack years due to lower packs per day smoked. Smoking rates in Hispanic populations vary considerably [20] though this study represents only a single Hispanic population. Consistent with these findings, Samet et al. compared COPD mortality rates between Hispanic and Non-Hispanic Whites in New Mexico and found that Hispanics had lower rates of COPD mortality between 1958 and 1982 [21] again suggesting a protective effect of Hispanic ethnicity though this study did not consider competing risks for mortality. Bruse et al., also working in New Mexico, reported similar findings and identified Native American ancestry, measured using ancestrally informative markers (AIMs), as being independently protective for COPD and rapid decline in FEV19. They also observed that Hispanic smokers accumulated similar numbers of pack years to NHWs but that differences were noted for an increased number of years of smoking with decreased packs smoked per day for Hispanics. Our study observed similar differences between Hispanic and NHW participants as they relate to smoking and considered these differences when applied those differences when modeling COPD mortality. We observed a longer (more pack years) and less intense smoking exposure (less packs per day) in Hispanics in the San Luis Valley, which has a similar history of settlement and migration to the study area in New Mexico. In our case, more detailed measures of smoking behavior appear to explain the protective effect of Hispanic ethnicity adjusting for smoking status. These similarities between Hispanic smokers in New Mexico and Colorado suggest that our findings may applicable to Hispanic smokers in New Mexico.

This study has several strengths and limitations. Limitations include: 1) death certificate data were used for cause specific mortality rather than hospital records; however, we considered any mention of COPD on the death certificate, not just as the primary cause of death. 2) This study reports on common questions related to smoking behavior (packs per day, years smoked, current smoking status) but did not collect more detailed behavioral questions (frequency and depth of inhalation) for all participants. Future studies of smoking related disease should include more detailed surveys that capture current and past smoking behaviors with more granularity. 3) Addiction to smoking was not assessed in this study, nor were known nicotine addiction genes. If addiction occurs at a different rate in Hispanics compared to NHWs this could partially explain differences in smoking behavior between the groups. Pérez-Stable et al. reported that nicotine is not metabolized at a different rate in Hispanics compared to NHW [22], suggesting that addictive behaviors might be similar. 4) Ethnicity was self-reported. Parra et al. reported the average admixture for the Hispanic population of this study as 65% European, 35% Native American and less than 1% African and that the average time to unadmixed ancestors was greater than seven generations [23]. 5) Socioeconomic factors such as access to care were not evaluated in this study which may influence COPD mortality.

Strengths of this study include: 1) use of competing risks regression to adjust for the most common smoking related mortality risk, cardiovascular disease which has been shown to be significantly lower in male Hispanics with diabetes in this cohort [24]. Lung cancer did not play a large role in this study and the 17 decedents with report of lung cancer were evenly distributed between Hispanics and NHWs. This suggests that lung cancer is not acting as a competing risk that differs by ethnicity. 2) This study was conducted in a well characterized, population based sample of a region of the country that was 46.6% Hispanic at the time of baseline data collection. 3) This study has very complete, long term (35,789 person years) follow-up of its participants and death certificates or coroners reports were obtained for 98% of decedent participants. 4) A thorough smoking assessment was performed that describes smoking behavior beyond smoking status and pack years on a subset of the population.

The Hispanic population of the United States is increasing; between 2000 and 2010 U.S. Census reports an increase by 35.3 million to 50.5 million Hispanic people (13% to 16% of the total population of the US) and differences in specific disease etiology would play in increasingly important role in the public health impact of smoking related disease. Hispanic ethnicity represents a diverse population in terms of ancestry, culture and behavior. In some cases this may result in true protection from COPD or COPD mortality, but this protective effect may be explained by smoking behavior which is not typically assessed on a population basis. Care should be taken in evaluating studies that describe Hispanic ethnicity as a factor related to COPD mortality as a perceived protective effect of ethnicity may result in decreased emphasis on smoking prevention and early disease detection in this large population due to a perceived decreased risk.

Conclusions

The lower COPD mortality seen in Hispanic smokers may be due

to smoking behavior. Hispanic smokers inhale less, possibly resulting in lower cumulative exposure to tobacco smoke. Thus, smoking behavior may play a key role in explaining differences in COPD mortality as they relate to Hispanic ethnicity.

Author Contributions

GLK, DSKT, LC, LSN, SL and JEH made substantial contributions to conception and design; GLK, SL and JEH made substantial contributions to acquisition of data or analysis and interpretation of data. All authors made substantial contributions to drafting the article or revising it critically for important intellectual content and final approval of the version to be published.

Sources of Support

This work was supported by DK-30747 and CRC-RR00051

Acknowledgments

The authors would like to acknowledge Richard F Hamman, MD, DrPH, Julie Marshal, PhD, Judy Baxter, MA and Sharon Scarbro, MS.

References

1. Minino A, Xu J, Kochanek KD (2011) Deaths: Preliminary Data for 2008. National Vital Statistics Reports 59.
2. Adhikari B, Kahende D, Malarcher A (2008) Smoking-Attributable Mortality, Years of Potential Life Lost, and Productivity Losses --- United States, 2000--2004. Morbidity and Mortality Weekly Report 57: 1226-1228.
3. Brehm JM, Celedón JC (2008) Chronic obstructive pulmonary disease in Hispanics. Am J Respir Crit Care Med 177: 473-478.
4. Heron M (2011) Deaths: leading causes for 2007. Natl Vital Stat Rep 59: 1-95.
5. Menezes AM, Perez-Padilla R, Jardim JR, Muiño A, Lopez MV, et al. (2005) Chronic obstructive pulmonary disease in five Latin American cities (the PLATINO study): a prevalence study. Lancet 366: 1875-1881.
6. Samet JM, Wiggins CL, Key CR, Becker TM (1988) Mortality from lung cancer and chronic obstructive pulmonary disease in New Mexico, 1958-82. Am J Public Health 78: 1182-1186.
7. Adams SG, Anzueto A, Pugh JA, Lee S, Hazuda HP (2006) Mexican American elders have similar severities of COPD despite less tobacco exposure than European American elders. Respir Med 100: 1966-1972.
8. Lipton R, Banerjee A (2007) The geography of chronic obstructive pulmonary disease across time: California in 1993 and 1999. Int J Med Sci 4: 179-189.
9. Bruse S, Sood A, Petersen H, Liu Y, Leng S, et al. (2011) New Mexican Hispanic smokers have lower odds of chronic obstructive pulmonary disease and less decline in lung function than non-Hispanic whites. Am J Respir Crit Care Med 184: 1254-1260.
10. U.S.Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. U.S. Department of Health and Human Services. How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease: A Report of the Surgeon General. A Report of the Surgeon General 2010.
11. Calverley PM, Walker P (2003) Chronic obstructive pulmonary disease. Lancet 362: 1053-1061.
12. Dalal AA, Shah M, Lunacsek O, Hanania NA (2011) Clinical and economic burden of patients diagnosed with COPD with comorbid cardiovascular disease. Respir Med 105: 1516-1522.
13. King B, Dube S, Kaufmann R (2010) Vital Signs: Current Cigarette Smoking Among Adults Aged Greater than or Equil too 18 Years -United States, 2005-2010. Morbidity and Mortality Weekly Report 60: 1207-1211.
14. Hamman RF, Marshall JA, Baxter J, Kahn LB, Mayer EJ, et al. (1989) Methods and prevalence of non-insulin-dependent diabetes mellitus in a biethnic Colorado population. The San Luis Valley Diabetes Study. Am J Epidemiol 129: 295-311.
15. Pintilie M (2007) Analysing and interpreting competing risk data. Stat Med 26: 1360-1367.
16. Fine JP, Gray RJ (1999) A proportional hazards model for the subdistribution of a competing risk. Journal of the American Statistical Association 94: 496-509.
17. Marubini E, Morabito A, Valsecchi MG (1983) Prognostic factors and risk groups: some results given by using an algorithm suitable for censored survival data. Stat Med 2: 295-303.
18. Pepe MS, Mori M (1993) Kaplan-Meier, marginal or conditional probability curves in summarizing competing risks failure time data? Stat Med 12: 737-751.
19. Dean CB, Nielsen JD (2007) Generalized linear mixed models: a review and some extensions. Lifetime Data Anal 13: 497-512.
20. Samet JM, Wiggins CL, Key CR, Becker TM (1988) Mortality from lung cancer and chronic obstructive pulmonary disease in New Mexico, 1958-82. Am J Public Health 78: 1182-1186.
21. Pérez-Stable EJ, Herrera B, Jacob P 3rd, Benowitz NL (1998) Nicotine metabolism and intake in black and white smokers. JAMA 280: 152-156.
22. Parra EJ, Hoggart CJ, Bonilla C, Dios S, Norris JM, et al. (2004) Relation of type 2 diabetes to individual admixture and candidate gene polymorphisms in the Hispanic American population of San Luis Valley, Colorado. J Med Genet 41: e116.
23. Swenson CJ, Trepka MJ, Rewers MJ, Scarbro S, Hiatt WR, et al. (2002) Cardiovascular disease mortality in Hispanics and non-Hispanic whites. Am J Epidemiol 156: 919-928.
24. Kaplan RC, Bangdiwala SI, Barnhart JM, Castañeda SF, Gellman MD, et al. (2014) Smoking Among U.S. Hispanic/Latino Adults The Hispanic Community Health Study/Study of Latinos. American Journal of Preventive Medicine 46: 496-506.