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Is there a Hispanic Health Paradox in Sensitivity to Air Pollution? Hospital Admissions for Asthma, Chronic Obstructive Pulmonary Disease and Congestive Heart Failure Associated with NO_2 and $PM_{2.5}$ in El Paso, TX, 2005–2010

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Abstract

Study Objective—Linkages between pollution and morbidity have been observed in numerous studies. But race/ethnicity has been underemphasized as a modifier of that association, and few studies have tested for a Hispanic Health Paradox in sensitivity to air pollution.

Methods—Daily asthma, chronic obstructive pulmonary disease (COPD) and congestive heart failure (CHF) hospital admissions in El Paso, Texas were studied in age groups and insurance groups. Daily $PM_{2.5}$ and NO_2 were calculated from pollution monitors and all models adjusted for apparent temperature and wind speed. Conditional logistic regression for the case-crossover design was used for a between-group comparison and for a within-group comparison for Hispanics.

Results—Hispanics were at lower risk than non-Hispanic whites and non-Hispanics of other races for NO_2-associated admissions, but at greater risk for $PM_{2.5}$-associated admissions. While Hispanics were generally protected with regards to NO_2, Hispanic children (vs. elderly) faced increased risk for asthma and uninsured Hispanics (vs. Private) faced increased risk for COPD admissions. While Hispanics were at increased risk of $PM_{2.5}$-associated admissions, certain characteristics heightened their risks: being a Hispanic child (vs. Elderly) for asthma; being a Hispanic with Medicare (vs. Private) for asthma; and being a Hispanic with private insurance (vs.

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all other insurance types) for CHF. The main effect of pollution on admissions was more significant for asthma and CHF than for COPD, which had the fewest cases.

Conclusions—There was heterogeneity in sensitivity to air pollution based on social characteristics and moderate evidence for a Hispanic Health Paradox in sensitivity to NO_2.

Keywords

Air pollution; Effect Modification; Hispanic Health Paradox; Case-crossover

INTRODUCTION

Myriad environmental epidemiology studies have determined linkages between daily levels of air pollutants and respiratory/cardiovascular morbidity and mortality[1–4]. This research has raised the question: do certain social groups face greater risks than others? To address this, researchers have examined patient attributes, like socio-economic status, as effect modifiers between air pollution and health outcomes[5–9]. They found that social characteristics do shape people's sensitivity to air pollution on a day-to-day basis[10–12]. Race/ethnicity has been underemphasized in the effect modification literature[11]. This is problematic, given the social science literature demonstrating racial/ethnic disparities in health[13] and the growing Hispanic population in the US. The Hispanic Health Paradox[14] would lead one to hypothesize that Hispanic ethnicity might be protective against the deleterious effects of air pollution. The Hispanic Health Paradox refers to the observation that for many Hispanics living in the United States (especially those born in Mexico), their health outcomes are equal to, or better than, those of non-Hispanic whites, despite higher poverty rates, less education, and worse access to health care[15]. To date, few studies have tested for a Hispanic Health Paradox in sensitivity to daily levels of air pollution[16–18].

This study makes three critical contributions to this literature. First, the focus on race/ethnicity and specifically Hispanic ethnicity, contributes to the nascent focus on the Hispanic Health Paradox in air pollution epidemiology. A limited number of previous studies have found that Hispanics are at lower risk than whites or blacks to respiratory conditions when pollution is high[16–18]. We also explore intra ethnic differences within the Hispanic population, which to the best of our knowledge, has not been examined. Second, in addition to asthma, we focus on Chronic Obstructive Pulmonary Disease (COPD) and Congestive Heart Failure (CHF). There are fewer air pollution epidemiology studies on COPD and CHF than the more commonly studied asthma, and effect modification by race/ethnicity and insurance status has rarely been investigated for these two diseases. COPD has been more consistently linked to air pollution[19–22] than has congestive heart failure[23]. However, there are mechanisms linking pollution exposure to cardiovascular diseases, which include translocation of particles into the circulatory system, release of proinflammatory mediators, and changes to the systemic autonomic nervous system balance[24]. Third, instead of using different lags in multiple models[23], we selected the best fitting lag-time in days between hourly exposure and the health effect by fitting a nonparametric distributed lag model with a continuous lag[25,26].

MATERIALS AND METHODS

Patient Covariates

We obtained hospital admissions records for asthma, COPD and CHF in El Paso County between 2005 and 2010 from the Texas Health Care Information Council in Austin, Texas (Texas Health Care Information Council, 2000). We extracted patients living in El Paso County that were hospitalized for asthma (ICD-9 code 493), COPD (ICD-9 codes 491, 492 and 496) or CHF (ICD-9 code 428) during the study period. We selected CHF because the linkage with traffic-related pollution is more robust for heart failure than other cardiovascular diseases[24,27]. The final data set included the following patient characteristics: race, ethnicity, age, insurance status, and date of admission.

To prepare the data for analysis, the race and ethnicity variables were re-coded into three mutually exclusive racial/ethnic categories: non-Hispanic white henceforth called “white”, non-Hispanic other (this includes non-Hispanic black) henceforth called “other”, and Hispanics of all races, which is used as the reference group in the full model. We created the following age groups for asthma: age 3–17 (children); 18–49 (young adults); 50–74 (adult); and 75+ (elderly). We removed those under 3 since it is difficult to diagnose asthma in infants and toddlers[5]. For COPD and CHF, we created only two age categories: 45–74 years (adults) and 75 years and older (elderly) since there were few cases under age 45.

We then re-coded the primary insurance provider into Private (i.e., health insurance from a private company), Medicare (i.e., government insurance for people over 65 years old and the disabled), Medicaid (i.e., government insurance for the poor), and Uninsured; for asthma, we also have the category of Military (e.g., Civilian Health and Medical Program of Uniformed Services), but the counts are too low for it to be included in the COPD and CHF analysis. Counts for each group are provided in Table 1.

Weather data

We obtained weather data (i.e., average daily temperature, dew point, and average wind speed) from the US National Weather Service, based on observations at the El Paso International Airport (see Table 2). We combined temperature and dew point into an apparent temperature measure. Apparent temperature reflects a person’s perceived temperature and is calculated using metric units for temperature and dew point: $-2.653 + (0.994 * \text{Air Temperature}) + (0.0153 * \text{Dew Point}^2)$[28,29]. We transformed average wind speed into a categorical indicator. If a day had an average wind speed of less than or equal to two meters per second (the 10^{th} percentile), we designated it as low wind because peaks in evening levels of particulate matter in El Paso occurred 90% of the time that wind speed fell below 2 meters per second[30]. An indicator for wind speed exceeding 6 meters per second (90^{th} percentile) was used since high wind has been associated with hospitalizations in El Paso[31].

Pollution data

We examine the effects of $PM_{2.5}$ and NO_2 on asthma, COPD, and CHF. In terms of mechanistic associations, $PM_{2.5}$ induces the release of inflammatory cytokines and the

generation of free radicals, which may lead to oxidative stress, exacerbating some respiratory symptoms.[32] NO_2-induced effects in the lung involve direct as well as free-radical-mediated oxidation of biomolecules, resulting in damage to the membrane structures, disruption of cellular metabolism and cell damage or death[33]. Potential mechanisms by which $PM_{2.5}$ is associated with cardiovascular events include the activation by particulate matter of inflammatory pathways, production of reactive oxygen species, alterations in vascular tone, and decreased heart rate variability (a marker of cardiac autonomic dysfunction)[34]. Inhalation of NO_2 affects heart rate, heart rate variability, blood pressure, vascular tone, blood coagulability, and the progression of atherosclerosis; NO_2 can activate pro-inflammatory pathways and generate harmful reactive oxygen species[35].

We acquired $PM_{2.5}$ (i.e., particulate matter of 2.5 micrometers or less) and NO_2 (nitrogen dioxide) data for the study period from the Texas Commission on Environmental Quality continuous air monitoring stations in an hourly format disaggregated by pollution monitor. In the El Paso metropolitan area, the Commission operated two monitors that captured hourly $PM_{2.5}$ (i.e., AQS 481-141-0037 and 48-141-0053) and three monitors for hourly nitrogen dioxide (i.e. AQS 48-141-0037, 48-141-0055, and 48-141-0044)[1]. To create a daily variable from $PM_{2.5}$ hourly data, we computed the mean across monitors of the daily 24-hour average. For nitrogen dioxide, we identified the maximum 24-hour reading at each of the stations and then averaged them. These metameters for summarizing hourly pollutant data are in accordance with the National Ambient Air Quality Standards. Summary statistics are presented in Table 2.

METHODS

Lag selection

A lag-time must be specified between the exposure to the pollutant and the health outcome, because it takes time for the body to mount a response to the exposure. The difficulty in choosing a lag-time is exemplified by Belleudi et al.[23] as they specified eleven different lag-times (e.g., lag 1 and average of lags 0–6) to study the association between $PM_{2.5}$ and acute coronary syndrome hospitalizations. Instead, we incorporate hourly pollutant measures in a historical functional linear model[36] with a continuous lag. The strength of this approach is simultaneous estimation of a pollutant regression parameter at hourly lags from zero hours to any number of days, as specified by the analyst, which is possible by modeling the regression parameter in terms of a cubic spline function that is constrained to vary smoothly in the hourly lag[25,26]. The smoothness constraint is imposed by means of a second-order penalty on the coefficients of the spline function modulated by a smoothing parameter[37]. The historical functional linear model in this context is better known as a nonparametric distributed lag model[26], and was fit to the data using a modification of the COXPH function in the R software (survival package). The modification to the COXPH function (see online appendix) is needed to implement the second-order penalty on the coefficients of the spline function[38,39].

[1]See http://tceq4apmgwebp1.tceq.texas.gov/geotam3/index.html?region=06. (Note that 48-141-0053 has been deactivated (as of December 2012) and is not mapped).

Our guiding principle for lag selection was to select the lag with the highest relative risk for hospitalization. First, the lag for each pollutant by disease was selected by fitting single pollutant models adjusted for apparent temperature without any effect modification by patient covariates. We included apparent temperature on the previous day in the regression model with a linear regression spline (3 degrees of freedom) to control for lagged and nonlinear effects of apparent temperature[29,40]. Selection of the lag(s) in units of days associated with the highest relative risk of hospitalization was determined by plotting the log relative risk versus the continuous lag by disease. This is illustrated in Figure 1 for CHF for which the average of lags 0–3 of $PM_{2.5}$, and lags 0–1 of NO_2 were selected. In a similar fashion, the average of lags 0–1 of $PM_{2.5}$ and lags 0–2 of NO_2 for asthma, and lag 6 of $PM_{2.5}$ and the average of lags 3–6 of NO_2 for COPD were selected. Second, the lags for low and high wind speed were selected from among lags 0, 1, 2, and 3, examined one at a time, by disease and pollutant. Lag 0 for high wind speed was chosen for all pollutants and diseases. For low wind speed, lag 0 (COPD, CHF) and lag 3 (asthma) were selected for the models including $PM_{2.5}$ and lag 3 (asthma, CHF) and lag 0 (COPD) were selected for the models including NO_2.

Analysis Plan

We employed conditional logistic regression (as implemented in Proc PHREG in SAS Version 9.2 with strata= Calendar Day, and the Ties=Breslow option for handling tied failure times[41]) for the case-crossover design[42]. This is equivalent to Poisson regression for time-series modeling[43] except that seasonality is controlled for by design (i.e., by self-matching) instead of in the regression model. Exposures on the index date (i.e., date of hospitalization) were compared with exposures on referent dates selected to fall on the same day-of-week, month and year as the index date[23,44,45]. Others have instead matched on month, year, and apparent temperature, and then controlled for day-of-week using dummy variables, but both strategies yield similar results[46].

When using conditional logistic regression for the case-crossover design, the modifying effect of patient characteristics, such as race/ethnicity, on the association between air pollution and asthma, COPD or CHF cannot be estimated directly (as a main effect) because each patient serves as his/her own control. However, an advantage of this approach is that interactions between patient characteristics and the selected pollution lag can be estimated[31,46], as was done here, allowing for the investigation of effect modification by race/ethnicity, for example.

The analysis took place in two parts, one corresponding to the “between-group” comparison (interaction effects) and the other to the “within-group” comparison for Hispanics. A review article of best practices recommends that formal tests of interactions should be used when possible when comparing subgroups[47] and so we used interactions for our between-group comparison. Instead of using three-way interaction terms in a full model, we analyze Hispanics as a subgroup, despite some limitations (e.g., increased overall Type I error rates from multiple comparisons)[47].

We began by running full models looking at the interaction of race/ethnicity (other and white vs. Hispanic), age (younger adults, older adults and elderly vs. children for asthma;

and elderly vs. older adult for COPD and CHF), and insurance (Medicaid, Medicare, Uninsured and Military vs. Private for asthma; military is removed for COPD and CHF) with each of the two pollutants for the three diseases. We worked with single pollutant models because of the strong correlation between daily NO_2 and $PM_{2.5}$ metameters (i.e., Pearson correlation coefficient of 0.52). We present results of the full models in two ways. One, we present the parameter estimate, standard error and statistical significance in tabular form and two, we computed estimates of relative risk of hospitalization for the social categories (e.g., white) in comparison to the reference group (e.g., Hispanic) when each pollutant is at the 98th percentile adjusting for apparent temperature and wind speed, which are presented in graphical form. We used the 98th percentile to represent days when pollution is a serious health risk and any disparities would likely be amplified. In discussion of the results, we focus on relative risk ratios at 2 or over, and at 0.5 or under, which represent substantive effects.

Then, we report results from an exploratory subgroup analysis for Hispanics. Specifically, we report the parameter estimate, standard error and statistical significance as well as the relative risk of hospitalization (when pollution is at the 98th percentile) for the insurance-based groups by disease. In the COPD and CHF models, age was removed because it was too closely related to insurance status; this problem did not exist in the asthma model due to the greater age range of the patients. This subgroup analysis allows us to determine if there are social inequalities within the Hispanic racial/ethnic category. As a point of reference, we also ran subgroup models for white and other (tables not shown).

RESULTS

Full Model

Table 3 reports parameter estimates and standard errors for the six full models. The statistically significant findings include the following. There were five significant ($p<.10$) findings out of 20 tested for **asthma**. Those with Medicare ($p<.05$) had a higher risk of being hospitalized for asthma when $PM_{2.5}$ increased than did those on private insurance. Adults ($p<.05$) and the elderly ($p<.05$) had decreased risks as compared to children when NO_2 increased. Those with Medicare ($p<.10$) had a higher risk of being hospitalized for asthma when NO_2 increased than did those on private insurance. Increasing NO_2 also had a direct effect on asthma hospitalizations ($p<.05$). There was one significant finding out of 14 tested for **COPD**. Those with other race ($p<.10$) had a significantly higher risk of being hospitalized for COPD when NO_2 increased than did Hispanics. For **CHF**, there were three significant findings out of 14 tested. Those on Medicare ($p<.10$) were at lower risk than those on private insurance when $PM_{2.5}$ increased. $PM_{2.5}$ had direct effect on CHF ($p<.05$). Those with other race ($p<.10$) had a higher risk of being hospitalized for CHF when NO_2 increased than did Hispanics.

Figure 2 shows relative risk estimates and 95% Wald confidence intervals for hospital admissions for the comparisons between the race/ethnicity, age, and insurance-based categories when the pollutant is at the 98th percentile. Substantive social disparities (i.e., RR ≥ 2 or RR≤ 0.5) were noted for the following pollutants and diseases. People on Medicare had a 2.29 (1.14–4.58) times greater risk than the privately insured for an **asthma** admission

when $PM_{2.5}$ levels were high. Adults and the elderly had 0.44 (0.22–0.89) and 0.38 (0.15–0.99) times lower risk, respectively, for **asthma** admission than children when NO_2 levels were high. People on Medicare had a 2.01 (0.92–4.39) times higher risk than the privately insured for **asthma** hospitalizations when NO_2 levels were high. Those without health insurance had 3.46 (0.75–15.99) times higher risk than those on private insurance for a **COPD** admission when NO_2 levels were high. Others (which includes African Americans) had 2.64 (0.98–7.14) times greater risk than Hispanics for a **COPD** admission when NO_2 levels were high.

Hispanic subgroup analysis

Table 4 reports parameter estimates, standard errors, and relative risk ratios for intra-ethnic social categories (e.g., insurance status) for Hispanics for each pollutant and disease (a total of 6 models). For asthma, there were four relative risk findings of note: Hispanic elderly had 0.43 (95% CI: 0.16–1.14) times lower risk than Hispanic children when $PM_{2.5}$ levels were high; Hispanics on Medicare had a 2.00 (0.88–4.56) times greater risk than those that were privately insured when $PM_{2.5}$ levels were high; Hispanic adults and the elderly had 0.50 (0.22–1.15) and 0.39 (0.13–1.18) times lower risk, respectively, than children when NO_2 levels were high. There was one notable finding for COPD and one for CHF: Uninsured Hispanics were 3.66 (0.56–24.03) times more like than privately insured Hispanics for a COPD admission when NO_2 levels were high; and Hispanics with Medicaid, Medicare, and the uninsured were at 0.35 (0.13–0.96), 0.41 (0.20–0.82), and 0.44 (0.18–1.11) times lower risk, respectively, than were Hispanics on private insurance for CHF admission when $PM_{2.5}$ levels were high.

Limitations

The analysis would be improved by larger numbers of non-Hispanics in the study area, especially black Americans so that they could be analyzed separately. Better geographic coverage of the state's air pollution monitoring network would have potentially improved the accuracy of our aggregated pollution values. Pollution values were assumed to map to individual exposure, an assumption commonly made in studies like this, although, this is not necessarily the case. It was assumed, as per the historical functional linear model, that the hospitalization occurred at the end of the day, because the exact time of hospital admission was not known. Although significant findings are highlighted here, the overall Type I error rate is inflated from the multiple comparisons, so this is an exploratory analysis attempting to highlight patterns in the data. Lastly, we had greater statistical power to detect direct effects and interaction effects for NO_2 than $PM_{2.5}$ due to the larger standard deviation for the NO_2 metameter.

CONCLUSIONS

This study provided modest evidence of a Hispanic Health Paradox (HHP) for NO_2-related sensitivity, but not $PM_{2.5}$ in El Paso, Texas. Hispanics were at lower risk than others and whites for admissions associated with all three diseases as NO_2 increased (Table 3) and was at peak levels (Figure 2). The other vs. Hispanic interaction with NO_2 was significant in the COPD and CHF models, reflecting this paradox. However, NO_2 still impacted Hispanics'

health as this pollutant had a significant main effect on asthma and CHF admissions within the Hispanic subgroup (Table 4). Alternatively, when the daily average of $PM_{2.5}$ increased (Table 3) and was at peak levels (Figure 2), Hispanics were at greater risk than whites for hospital admission associated with all three diseases, and Hispanics were at greater risk than others for COPD and CHF (but not asthma) although these findings were not significant and the effects were not particularly strong. There was a significant main effect of $PM_{2.5}$ on CHF admissions among Hispanics, but not for the white or other subgroups (which had negative, insignificant parameter estimates; tables not shown). This corroborates Hispanics' sensitivity to $PM_{2.5}$.

In terms of why Hispanics would be more sensitive to changes in $PM_{2.5}$ but not NO_2, a genetic explanation seems implausible. A recent study revealed the incredible genetic variability within the Mexican population, which is as extensive as the variability between Asians and Europeans[48]. This suggests that considering Hispanics or even Mexican-origin Hispanics as a single biological group is untenable. Alternatively, social and cultural explanations are more likely, but additional research is needed to understand the mechanisms behind such explanations. Higher rates of underlying chronic health conditions (related to lifestyle and socioeconomic deprivation) may contribute to Hispanics' sensitivity to $PM_{2.5}$ in El Paso. Specifically, the risk of diabetes is especially pronounced for US born Mexican-Americans[49] which comprise the majority of El Paso's Hispanic population. US-born Mexican-Americans also have relatively high rates of cardiovascular disease[50]. Both diabetes and cardiovascular disease may intensify the association between $PM_{2.5}$ and cardiovascular events[34], but were unmeasured in our models due to the lack of available data. While it is unclear why Hispanics may be less sensitive to NO_2 than other groups, it is the case that two previous time-series studies found that Hispanics had lower risk of asthma admissions than did whites and blacks associated with nitrogen dioxide/oxides, which we also found here[16,17]. That Hispanics would experience paradoxically better health outcomes when exposed to NO_2,, but not $PM_{2.5}$ needs to be confirmed by additional studies.

Examination of intra-Hispanic disparities provides another angle from which to examine the HHP to determine if Hispanics share similar advantages (or risks, in the case of $PM_{2.5}$ exposure) based on their social characteristics. While Hispanics were generally protected with regards to NO_2, Hispanic children (vs. elderly) faced increased risk for asthma, and uninsured Hispanics (vs. Private) faced increased risk for COPD. In a multicenter cohort study of 24 emergency rooms, Hispanics with COPD were less likely to have a primary care physician and to be insured than were whites[51], meaning that Hispanics, especially those that are uninsured, receive less preventative care for their COPD. This makes them potentially more vulnerable to a NO_2-induced exacerbation. The directionality of these two effects was the same (only weaker) for the white and other subgroups, except for uninsured others, who had lower risk than privately insured others (tables not shown). While Hispanics were at increased risk of admissions when $PM_{2.5}$ was high, certain characteristics heightened risks even more: being a Hispanic child (vs. Elderly) for asthma; being a Hispanic with Medicare (vs. Private) for asthma; and being a privately insured Hispanic (vs. other insurance-types) for CHF. If it is the case that the privately insured Hispanics are more likely to be multigenerational Americans, then this CHF finding may align with the Hispanic Health Paradox. This significant risk for the privately insured was not found for white or

other subgroups and the direction of the associations was opposite (private had lower risk, tables not shown). Like Hispanic children, other children were at increased risk for asthma associated with $PM_{2.5}$; the association was opposite for white children with white elderly having two times greater risk. Lastly, the increased risk for asthma admissions for those on Medicare was found for all three subgroups.

This study contributes to the growing literature on air pollution epidemiology for COPD and CHF, which are less often studied than asthma. This was one of the first studies to consider how social factors modify associations between air pollution and COPD and CHF. Also, we found that the impact of traffic related pollution was more likely to be statistically significant for asthma (N=4953) and CHF (N=7946) admissions than it was for COPD (N=3702) admissions. The lack of significant findings for NO_2 and $PM_{2.5}$ in the COPD models might be explained by the relatively fewer number of cases. These exploratory findings should be compared with findings from other cities in the future.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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HIGHLIGHTS

- Hispanics were at lower risk of NO_2-associated admissions than whites and others
- Hispanics were at greater risk of $PM_{2.5}$-associated admissions than whites and others
- Some Hispanic subgroups faced increased risk of $PM_{2.5}$-and NO_2-associated admissions

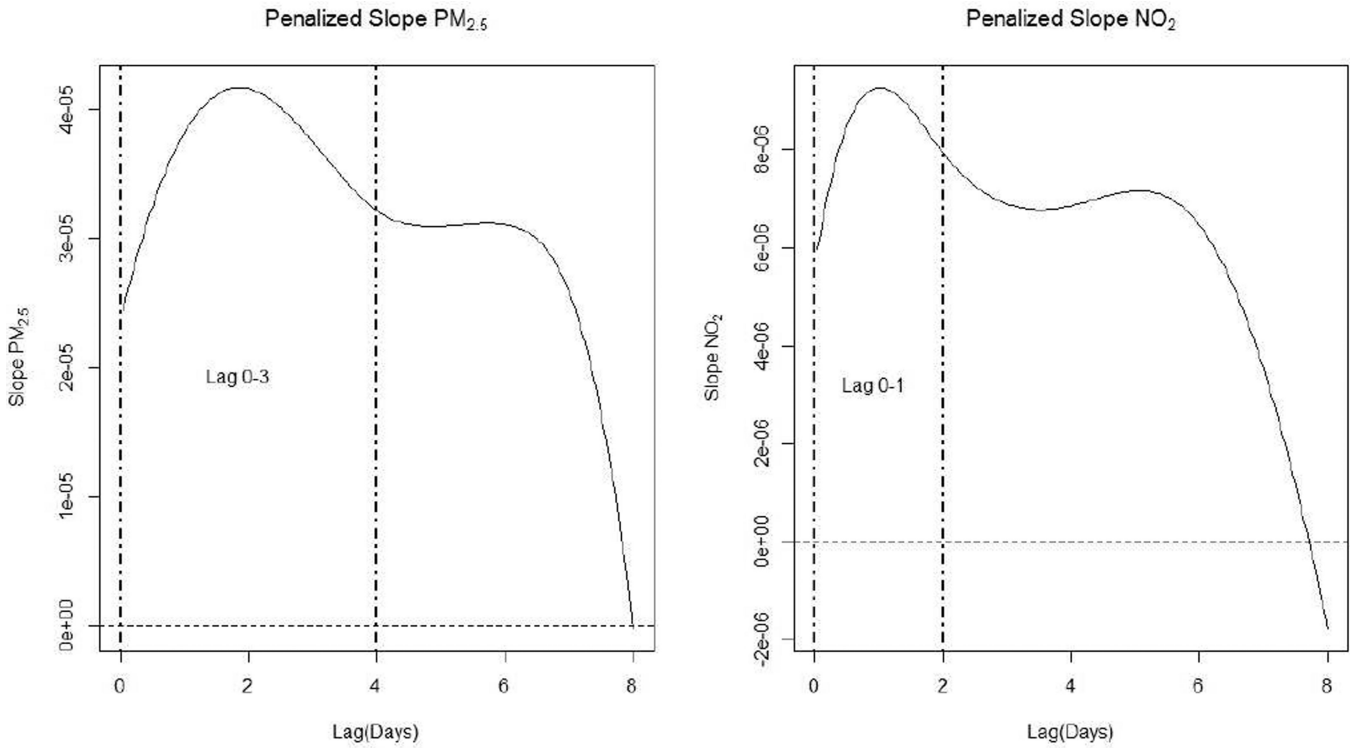

Figure 1.

Log relative risk of congestive heart failure plotted against lags for single pollutant models as estimated using a nonparametric distributed lag model[25,26,36]

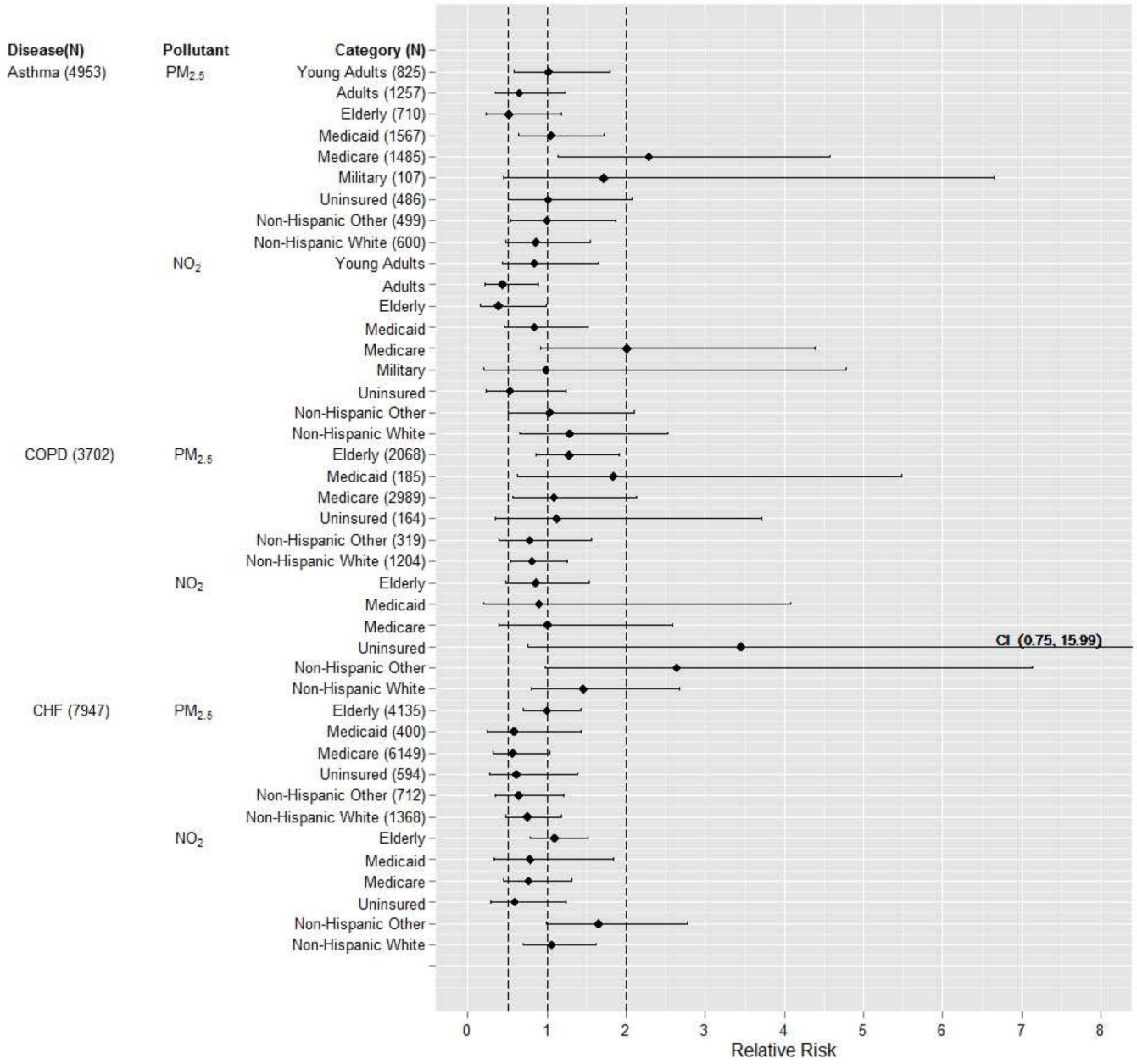

Figure 2.

Relative risk estimates and 95% Wald confidence intervals for hospital admissions for the comparisons between age, insurance, and race/ethnicity when the pollutant is at the 98th percentile (based on results from Table 3).

Notes: “Child” is the reference group for age comparisons in asthma models; “Adult” is the reference group for age comparisons in the COPD and CHF models. For all three diseases, “Hispanic” is the reference group in the race/ethnicity comparisons and “private insurance” is the reference group for insurance-based comparisons.

Table 1

Counts by Disease for El Paso, Texas (2005–2010) (NA=Not Applicable)

	Sex			Age					Insurance Status					
	Female	**Male**	**Total**	**Children**	**Young Adults**	**Adults**	**Elderly**	**Total**	**Medicaid**	**Medicare**	**Military**	**Private**	**Uninsured**	**Total**
Chronic Obstructive Pulmonary Disease (COPD) Admissions														
Hispanics	1049	1130	2179	NA	NA	950	1229	2179	125	1738	NA	204	112	2179
Non-Hispanic Whites	634	570	1204	NA	NA	539	665	1204	39	987	NA	145	33	1204
Non-Hispanic Others	149	170	319	NA	NA	145	174	319	21	264	NA	15	19	319
Total	1832	1870	3702	NA	NA	1634	2068	3702	185	2989	NA	364	164	3702
Congestive Heart Failure (CHF) Admissions														
Hispanics	3074	2793	5867	NA	NA	2901	2966	5867	355	4462	NA	543	506	5866
Non-Hispanic Whites	657	711	1368	NA	NA	529	839	1368	24	1111	NA	188	45	1368
Non-Hispanic Others	345	367	712	NA	NA	382	330	712	21	576	NA	72	43	712
Total	4076	3871	7947	NA	NA	3812	4135	7947	400	6149	NA	803	594	7946
Asthma Admissions														
Hispanics	2144	1710	3854	1840	617	883	514	3854	1337	1065	55	1001	396	3854
Non-Hispanic Whites	369	231	600	135	104	215	146	600	82	266	27	188	37	600
Non-Hispanic Others	304	195	499	186	104	159	50	499	148	154	25	119	53	499
Total	2817	2136	4953	2161	825	1257	710	4953	1567	1485	107	1308	486	4953

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Table 2

Descriptive Statistics for Pollution and Weather Variables: Daily levels in El Paso, TX (2005–2010)

Variables (Unit)	N	Min	5th centile	25th centile	Median	Mean	75th centile	95th centile	Max	SD[b]
Apparent temperature (Celsius)	2191	−3.33	3.74	10.28	17.89	17.58	25.69	29.61	32.36	8.64
Wind speed (m/s)	2191	0.41	1.66	2.55	3.37	3.76	4.51	7.43	13.06	1.78
Temperature (Celsius)	2191	−2.96	5.02	11.97	19.77	18.93	25.97	30.86	34.58	8.29
Dew Point (Celsius)	2191	−21.16	−12.96	−6.11	−0.07	1.13	9.49	15.95	18.77	9.25
Nitrogen dioxide (ppb)	2191	5.37	14.41	24.91	33.67	33.86	41.97	54.28	91.73	12.35
Nitrogen dioxide L0-1[a] (ppb)	2190	8.08	17.39	26.35	33.47	33.86	40.85	51.27	76.83	10.5
Nitrogen dioxide L0-2[a] (ppb)	2189	9.06	18.87	27.15	33.85	33.86	40.33	49.6	69.41	9.43
Nitrogen dioxide L3-6[a] (ppb)	2185	9.62	19.81	27.63	33.81	33.86	39.99	48.12	66.19	8.75
PM2.5 (μg/m3)	2191	2.21	5.11	7.86	10.35	11.41	13.53	21.47	62.04	5.28
PM2.5 L0-1* (μg/m3)	2190	2.57	5.75	8.29	10.6	11.4	13.63	20.2	42.9	4.55
PM2.5 L0-3* (μg/m3)	2188	3.49	6.34	8.64	10.91	11.4	13.35	18.35	31.73	3.81
PM2.5 L6* (μg/m3)	2185	2.21	5.11	7.86	10.33	11.4	13.53	21.47	62.04	5.28

[a] La-b=average over lags a–b

[b] SD=standard deviation

Table 3

Parameter estimates for the six full models, which are single pollutant models for NO_2[a] and $PM_{2.5}$[b] predicting Asthma, Chronic Obstructive Pulmonary Disease (COPD) and Congestive Heart Failure

Full Model with Interactions	ASTHMA	COPD	CONGESTIVE HEART FAILURE
	Parameter Estimate (std error)	Parameter Estimate (std error)	Parameter Estimate (std error)
$PM_{2.5}$*Young Adult (ref: child)	0.001(0.011)	NA	NA
$PM_{2.5}$*Adult (ref: child/ref: adult)[c]	−0.017(0.013)	NA	NA
$PM_{2.5}$*Elderly (ref: child)	−0.027(0.017)	0.009(0.021)	0.000(0.008)
$PM_{2.5}$*Medicaid (ref: private)	0.002(0.010)	0.023(0.013)	−0.025(0.021)
$PM_{2.5}$*Medicare (ref: private)	0.033(0.014)**	0.003(0.023)	−0.027(0.014)*
$PM_{2.5}$*Military (ref: private)	0.022(0.028)	NA	NA
$PM_{2.5}$*Uninsured (ref: private)	0.001(0.015)	0.004(0.008)	−0.023(0.019)
$PM_{2.5}$*Non-Hispanic Other (ref: Hispanic)	0.001(0.013)	−0.010(0.014)	−0.021(0.015)
$PM_{2.5}$*Non-Hispanic White (ref: Hispanic)	−0.006(0.012)	−0.008(0.021)	−0.014(0.011)
$PM_{2.5}$	0.007(0.008)	−0.004(0.013)	0.041(0.014)**
NO_2*Young Adult (ref: child)	−0.003(0.006)	NA	NA
NO_2*Adult (ref: child)	−0.015(0.007)**	NA	NA
NO_2*Elderly (ref: child/ref: adult)[c]	−0.017(0.009)**	−0.003(0.006)	0.002(0.003)
NO_2*Medicaid (ref: private)	−0.003(0.005)	−0.002(0.014)	−0.004(0.008)
NO_2*Medicare (ref: private)	0.013(0.007)*	0.000(0.009)	−0.005(0.005)
NO_2*Military (ref: private)	−0.000(0.015)	NA	NA
NO_2*Uninsured (ref: private)	−0.011(0.008)	0.023(0.015)	−0.009(0.007)
NO_2*Non-Hispanic Other (ref: Hispanic)	0.001(0.007)	0.018(0.01)*	0.009(0.005)*
NO_2*Non-Hispanic White (ref: Hispanic)	0.005(0.006)	0.007(0.006)	0.001(0.004)
NO_2	0.012(0.005)**	−0.001(0.009)	0.002(0.003)

[a] The asthma model uses the average over lags 0, 1 and 2; the COPD model uses the average over lags 3, 4, 5 and 6; the CHF model uses the average of lags 0 and 1.

[b] The asthma model uses the average over lags 0 and 1; the COPD model uses lag 6; the CHF model uses the average of lags 0, 1, 2 and 3.

[c] The reference category is “child” for the asthma models and “adult” for the COPD and CHF models.

** $p<0.05$;

* $p<0.10$

Table 4

Parameter estimates and relative risk estimates (when $PM_{2.5}$[a] and NO_2[b] are at the 98th percentile) for Asthma (A), Chronic Obstructive Pulmonary Disease (B) and Congestive Heart Failure (C) for the Hispanic Subgroup

Hispanic Subgroup Analysis	Models including $PM_{2.5}$		Models including NO_2	
	Parameter Estimate (Std Error)	Relative Risk at 98th percentile and 95% CI[a]	Parameter Estimate (Std Error)	Relative Risk at 98th percentile and 95% CI[b]
A) Asthma				
Young Adults vs Children	−0.004(0.013)	0.89(0.46,1.70)	−0.002(0.007)	0.856(0.40,1.84)
Adults vs Children	−0.019(0.016)	0.61(0.29,1.26)	−0.012(0.008)	0.50(0.22,1.15)
Elderly vs Children	−0.034(0.021)*	0.43(0.16,1.14)	−0.017(0.010)*	0.39(0.129,1.18)
Medicaid vs Private	−0.006(0.011)	0.87(0.50,1.52)	−0.004(0.006)	0.82(0.44,1.58)
Medicare vs Private	0.028(0.017)*	2.00(0.88,4.56)	0.009(0.009)	1.68(0.66,4.24)
Military vs Private	0.001(0.037)	1.02(0.17,6.02)	0.009(0.019)	1.65(0.20,13.28)
Uninsured vs Private	0.001(0.016)	1.02(0.47,2.22)	−0.009(0.009)	0.60(0.23,1.54)
Pollution variable	0.014(0.011)	NA	0.011(0.005)**	NA
B) Chronic Obstructive Pulmonary Disease (COPD)				
Medicaid vs Private	−0.001(0.028)	0.98(0.23,4.20)	0.004(0.018)	1.21(0.19,7.63)
Medicare vs Private	0.019(0.017)	1.64(0.68,4.00)	−0.006(0.012)	0.72(0.21,2.44)
Uninsured vs Private	−0.004(0.028)	0.90(0.21,3.85)	0.024(0.018)	3.66(0.56,24.03)
Pollution variable	−0.011(0.016)	NA	0.002(0.011)	NA
C) Congestive Heart Failure (CHF)				
Medicaid vs Private	−0.049(0.024)**	0.35(0.13,0.96)	−0.009(0.009)	0.59(0.23,1.52)
Medicare vs Private	−0.042(0.017)**	0.40(0.20,0.82)	−0.008(0.006)	0.64(0.34,1.20)
Uninsured vs Private	−0.038(0.022)*	0.44(0.18,1.11)	−0.011(0.008)	0.53(0.23,1.23)
Pollution variable	0.056(0.016)**	NA	0.009(0.005)*	NA

[a] PM2.5=24.77 μg/m3 (asthma); 26.34 μg/m3 (COPD) and 21.38 μg/m3 (CHF)

[b] NO2=55.41 ppb (asthma) 53.34 ppb (COPD) and 56.23 ppb (CHF)

** p<0.05;

* p<0.10