The Burden of Air Pollution: Impacts among Racial Minorities

R. Charon Gwynn[1,2] and George D. Thurston[1]

[1]Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, New York, USA; [2]Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Various epidemiologic investigations have shown that ambient air pollution levels are associated with acute increases in hospital admissions and mortality in the United States and abroad. The objectives of this investigation were a) to determine if racial minorities are more adversely affected by ambient air pollution than their white counterparts and b) to assess the contribution of socioeconomic status to any observed racial differences in pollution effect. Time-series regression methods were conducted to investigate these hypotheses for daily respiratory hospital admissions in New York City, New York. Pollutants considered included mean daily levels of particulate matter with a mass median aerodynamic diameter less than 10 μm (PM_{10}), ozone (O_3), strong aerosol acidity (H^+), and sulfates (SO_4^{2-}). The relative risk for respiratory hospital admission was calculated for each pollutant for a maximum minus mean increment in mean daily pollutant concentration. The greatest difference between the white and nonwhite subgroups was observed for O_3, where the white relative risk (RR) was 1.032 [95% confidence interval (CI): 0.977–1.089] and the nonwhite RR was 1.122 (95%CI: 1.074–1.172). Although not statistically different from each other, the various pollutants' RR estimates for the Hispanic nonwhite category in New York City were generally larger in magnitude than those for the non-Hispanic white group. When these analyses incorporated differences in the underlying respiratory hospitalization rates across races (that for nonwhites, was roughly twice that for whites), the disparities in attributable risks from pollution (in terms of excess admissions per day per million persons) were even larger for nonwhites versus whites. However, when insurance status was used as an indicator of socioeconomic/health coverage status, higher RRs were indicated for the poor/working poor (i.e., those on Medicaid and the uninsured) than for those who were economically better off (i.e., the privately insured), even among non-Hispanic whites. Thus, although potential racial differences in pollution exposures could not be explored as a factor, within-race analyses suggested that most of the apparent differences in air pollutant effects found across races were explained by socioeconomic and/or health care disparities. Key words: acidic aerosols, air pollution, epidemiology, ethnicity, ozone, particulate matter, poverty, race, respiratory hospital admissions, socioeconomic status, sulfate, time-series techniques. — Environ Health Perspect 109(suppl 4):501–506 (2001). http://ehpnet1.niehs.nih.gov/docs/2001/suppl-4/501-506gwynn/abstract.html

Among the extensive literature published in the field of environmental epidemiology, few studies have directly focused on the potential impacts of air pollution exposure on racial minorities. However, some researchers and policy makers have expressed concern that minority populations may be at an increased adverse health risk from air pollution exposure (1–4). The complexities surrounding these concerns are likely based on social issues that are beyond the scope of most environmental researchers and regulators. Furthermore, the lack of quality data and a general distrust of researchers on the part of communities often limit investigations into the potential impacts of air pollution on minority populations. Despite these obstacles, an understanding of the impacts of air pollution on racial minorities is necessary and may provide a valuable piece of the air pollution/health effect puzzle.

Studies suggest that disadvantaged non-white communities may experience higher than average exposures to air pollution (3,5). This may be because urban areas, where a large percentage of racial minorities reside, are prone to have higher levels of ambient air pollution due to heavy traffic. Furthermore, poorer communities are more likely than affluent communities to be located close to environmental hazards such as landfills, medical waste incinerators, diesel bus depots, and Superfund sites. For example, in New York City, New York, 6 of 19 bus depots are located in Harlem and Washington Heights, predominantly African–American and Hispanic communities (6). Additionally, in the United States in 1992, 62.2% of blacks and 71.2% of Hispanics lived in U.S. Environmental Protection Agency (U.S. EPA) nonattainment areas, compared to 52.5% of whites (4). Thus, as a result of potential higher exposures, nonwhites may experience increased health impacts from ambient air pollution.

Disparities in access to quality health care may also play a key role in the susceptibility of nonwhite populations to the effects of air pollution. In a study of asthmatic children in 1988, it was found that poor children were more likely to have routine and sick care in a neighborhood health center and hospital-based clinics and were less likely to receive care in a doctor's office (7). The same study also found that poor children were more likely to use the emergency department for sick care than nonpoor children. In another study among a random sample of first and sixth graders attending Baltimore, Maryland, city public schools, investigators found that black children were twice as likely as white children to use the emergency room as their primary source of care (8). Disparities in the access to routine preventive care among poorer, nonwhite communities may leave them more adversely affected by air pollution.

Undetermined genetic differences across races may also contribute to differences in responses to air pollution. Sickle cell anemia is one example of a genetically transmitted disease that occurs in predominantly black populations. African Americans are also at increased risk for other diseases, such as lupus, hypertension, and diabetes, in which genetic and environmental factors may play key roles (9). Teasing out the contribution of genetic factors to the increased risk for these diseases is a complex, if not impossible, task. Yet genetic factors might plausibly result in differential susceptibility to environmental agents among racial subgroups.

Asthma prevalence and death rates are increasing in the United States, especially among blacks (10). As a result of these trends, many researchers have attempted to discern the role of these factors in the increasing prevalence of asthma among blacks in the United States. Socioeconomic status (SES) is the factor that has most consistently accounted for the observed differences in asthma prevalence rates between whites and blacks. For example, in New York City, asthma hospitalization rates by patient's ZIP code were correlated with the percentage of minorities in the population and inversely associated with the median household income (11). In Boston, Massachusetts, investigators

found that racial/ethnic differences in asthma prevalence in a cohort of 499 families were greatly reduced upon adjusting for SES (*12*). These results are consistent with those found in earlier work where, after adjustment for poverty, black and white children had equal asthma hospital discharge rates (*13*). And although the role of SES in asthma prevalence is less clear in other countries (perhaps because of differences in environmental exposure, government-funded health care systems, and/or centralized health statistic collection mechanisms), SES is apparently an important factor in the changing rates of asthma in the United States.

Collectively, these SES factors may leave racial minorities more affected by air pollution than the general population. While the reason(s) for this apparent heightened effect is not yet clear, the U.S. EPA is charged with protecting the health of the U.S. population from harmful levels of air pollution, especially among groups of persons considered to be especially sensitive (*14*). Furthermore, elevated air pollution effects in a sensitive population can provide further support for those effects observed in the general population. Thus, the differential impacts of air pollution on racial subgroups represent an important research priority.

In recent years, many investigators have assessed the impacts of various air pollutants on indices of morbidity and mortality in the United States and abroad. The general consensus is that ambient exposures to some key air pollutants result in excess adverse health effects in the general population. A limited number of investigations have attempted to assess the impacts of air pollution on minority groups. This is primarily because of the ecologic nature of the analyses conducted as well as limited data availability. Many of these investigations have been time-series analyses, in which daily variations in air pollution levels are correlated with daily measures of morbidity and mortality using regression models that control for confounders such as weather and season. Although time-series analyses are useful in assessing the acute health impacts of air pollution, large numbers of daily health observations are required to have enough power to test the air pollution–health effects association with confidence. Thus, power considerations are an important challenge to analyses of population subgroups, as the power of the analysis declines as the number of daily counts becomes smaller within subgroups. Hence, analyses of racial minorities using time-series analyses will be possible only when the largest cities are considered or when appropriate meta-analyses of multiple cities can be developed and applied.

Despite power limitations, several recent epidemiologic investigations have attempted

to characterize pollution associations among racial minorities. Ito and Thurston (*15*) examined the relationship between air pollution exposure and at-risk subpopulations in Cook County, Illinois. Race, gender, and cause-specific counts of mortality were regressed on air pollution and weather variables. In the race- and gender-specific analyses, black females had the highest risk estimates for total, respiratory, and cancer mortality. Other epidemiologic investigations have attempted to assess the health impacts of air pollution on African–American asthmatic children (*2,16*). Both of these studies indicated that air pollution has a significant impact on African–American asthmatic children. However, the latter two studies did not compare risk estimates across different racial/ethnic groups. Given the findings of these studies, further investigations directed toward explaining the apparently enhanced impact of air pollution on minority populations are necessary.

The size and diversity of the New York City population makes this setting conducive to studying the differing impacts of air pollution on racial subgroups. It is a densely populated metropolitan area with over 7 million residents. In 1990, this population was 52% white, 29% black, 24% Hispanic. However, although New York City is one of the richest cities in the country, the unequal distribution of resources reflects the larger social equity problems that face the nation. For example, the average household income in 1990 for whites in New York City was $48,000 compared to $30,000 for blacks and $28,000 for Hispanics (Figure 1). Additionally, the largest proportion of uninsured hospital admissions in New York City in 1990 was among Hispanic and nonwhite people (Figure 2). Thus, New York City is a large, diverse U.S. city with socioeconomic disparities that may result in a greater susceptibility among nonwhite residents.

The primary objective of this study is to investigate, via time-series analyses, whether racial minorities in New York City are at an increased risk of hospitalization because of

respiratory illness attributable to air pollution than are white residents of the same city. The secondary objective of this study is to assess the role of SES in these potential differences.

Materials and Methods

Hospital Admissions Data

Daily counts of respiratory hospital admissions were obtained from the Statewide Planning and Research Cooperative System, a division of the New York State Department of Health for 1988, 1989, and 1990. Respiratory admissions considered were acute bronchitis/bronchiolitis, pneumonia, chronic obstructive pulmonary disorder, or asthma [International Classification of Diseases, 9th Revision, codes 466, 480–486, 490–493, and 496 (*17*)]. Unscheduled daily admissions were aggregated for white and nonwhite subgroups for the five New York City boroughs: Bronx, Kings, Queens, Richmond, and New York. In this data set, the nonwhite subgroup was composed of admissions where race was classified as black, Asian/Pacific Islander, Alaskan/Native American, or other, and/or ethnicity was classified as Hispanic, whereas the white subgroup was composed of admissions where race was classified as white and ethnicity was classified as non-Hispanic.

Environmental Data

The environmental data used in this investigation consist of pollution and weather data collected from various sources. The pollutants considered in these analyses were particulate matter with an aerodynamic diameter under 10 μm (PM_{10}), ozone (O_3), sulfate (SO_4^2), and strong aerosol acidity (H^+). These pollutants demonstrated significant health impacts in the general population of cities in New York State in other published work (*1,18*). Daily values of the U.S. EPA criteria air pollutants PM_{10} and O_3 were obtained from the Aerometric Information Retrieval System (Air*DATA*) (*19*). Daily concentrations of Coefficient of Haze (CoH), an index of elemental carbon concentration in the air,
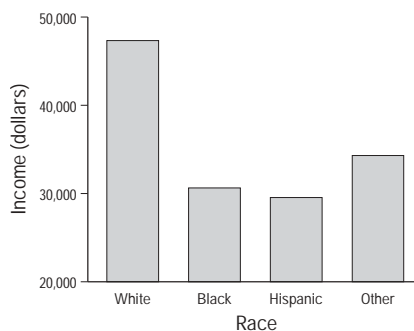

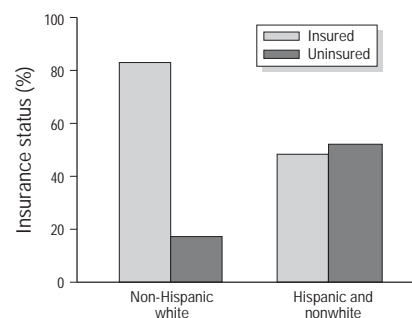**Figure 1.** 1990 Average household income by race in New York City, New York.

Figure 2. 1990 insurance status by race/ethnicity in New York City, New York.

were obtained from the New York State Department of Environmental Conservation. Values from selected sites were averaged to obtain daily mean values for each metropolitan area. As PM$_{10}$ was collected only every sixth day, missing values were filled in using regression imputation, with available particulate matter (PM) indicators (SO$_4^2$ and CoH) as independent variables (predicted vs observed R = 0.81).

Daily measurements of H$^+$ and SO$_4^2$ were collected by researchers at the New York University School of Medicine (NYUSM) from 19 May 1988 to 4 July 1990 at a monitor located in White Plains, New York (a suburb of New York City), using the sequential acid aerosol system designed at NYUSM (*20*). The availability of these unique daily data during this time defines the period of this study. Daily weather data were collected at LaGuardia Airport in New York City by the National Oceanic and Atmospheric Administration (NOAA). Table 1 provides summary statistics for the health and environmental variables used in this analysis. Time-series plots of these variables are shown in Figure 3.

Health Effects Analysis

The approach used to assess the impact of each air pollutant taken in these analyses is similar to that analyzed in previously published work (*18*). A negative binomial regression model was used to model daily variations in respiratory hospital admissions. Day-of-week and holiday patterns observed in the time-series plots for many of the health outcomes were addressed by creating indicator variables for each day of the week and each of the eight major federal holidays and included in the regression models. A 31-day weighted moving average filter similar to the 19-day filter used by Shumway et al. (*21*) was used to adjust for seasonal variations in both the hospital admissions and environmental outcomes. The nonlinear, potentially synergistic, effects of weather on human health were addressed by including three-dimensional (3-D) LOESS surfaces of respiratory hospital admissions versus temperature and relative humidity in the regression model.

The resulting pollutant regression coefficients were used to calculate the relative risk (RR) of respiratory hospital admissions for the white and nonwhite subgroups. Relative

risks and 95% confidence intervals (95% CI) were computed using the most significant lag, as determined for the entire population, of each pollutant as follows:

$$RR = \exp(\Delta Conc \times \beta_{pollutant})$$
$$LL = \exp(\Delta Conc\,(\beta_{pollutant} - 1.96 \times se_{pollutant}))$$
$$UL = \exp(\Delta Conc\,(\beta_{pollutant} + 1.96 \times se_{pollutant}))$$

where $\Delta Conc$ is the maximum concentration of the pollutant minus its mean concentration.

Age-adjusted attributable risks (ARs) were also calculated in this analysis by multiplying the relative risk by the baseline rate of hospital admissions for each subgroup. The ARs were computed for each pollutant to demonstrate the total number of people in each subgroup affected by the respective increased relative risks. When comparing risks across subgroups, it may be important to consider the underlying baseline rate of hospital admission in each subgroup. These different baseline rates should be considered when comparing the risk impacts of air pollution on the various subgroups. For example, nonwhites have a larger rate of daily respiratory hospital admissions than whites in this city, either because of a higher prevalence of respiratory disease or a larger reliance upon the hospital for care, or both. Similar relative risks for white and nonwhite groups would appear to suggest that each population is affected equally by air pollution, but because these groups have different baseline rates, they are not affected equally in absolute terms (e.g., number of excess daily admissions per million persons). Thus, white and nonwhite groups with identical relative risks but different baseline rates have different attributable risks, a metric more revealing of absolute differences in individual changes in risk.

To investigate the impact of socioeconomic factors on the risk estimates, we further categorized the race-specific respiratory hospital admissions data by health insurance status. In this situation, health insurance status can provide a crude indicator for SES, allowing this factor to be addressed in the regression analysis. The insurance categories were created so that the insured category included those privately insured and those with Medicare, whereas the uninsured category included those having Medicaid or no health insurance. Thus, the insurance categories were constructed so that these two groups represented admissions among the upper- and middle-income persons and the poor and working poor, respectively. These counts were then used as dependent variables in the regression analysis, allowing investigation of the possible roles of insurance status and SES in racial variations in air pollution effects.

Table 1. Summary statistics for environmental and health outcome variables for New York City, New York, 1988–1990.

	n	Minimum	25th Percentile	Mean	75th Percentile	Maximum
Respiratory hospital admissions (admissions per day)	1,096	86	156	197.1	232	391
Non-Hispanic white	1,096	26	52	64.1	74	133
Hispanic and nonwhite	1,096	52	103.8	133	159	258
H$^+$ (nmole/m^3)	763	1.63	16.3	35.7	44.6	286.7
SO$_4^2$ (nmole/m^3)	731	0.78	24.3	57.8	71.6	398.7
PM$_{10}$ (µg/m^3)	184	12	25.4	37.3	45.65	91
Filled PM$_{10}$ (µg/m^3)	776	12	28.3	37.4	43.35	97.98
Mean daily O$_3$ (ppb)	1,096	3.33	13.7	22.1	27.8	80.7
Mean daily temperature (°F)	1,096	11.4	42.4	55.3	70.1	89
Mean daily relative humidity (%)	1,096	28.3	52.3	63.8	74.7	100
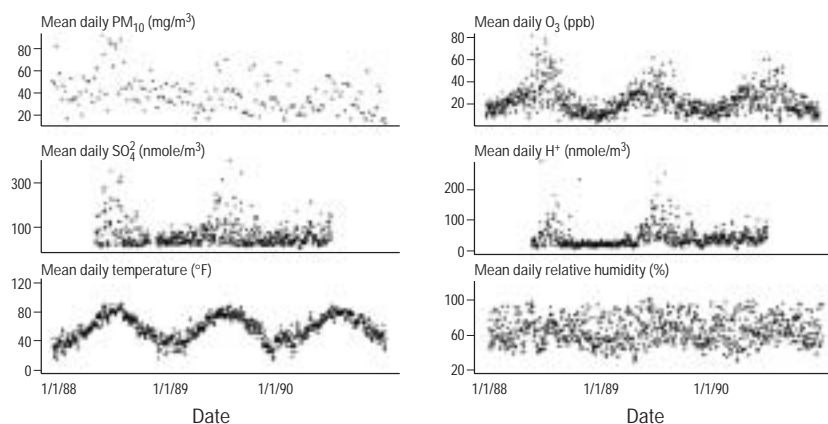
Figure 3. Time-series plots of environmental variables in New York City, New York, 1988–1990.

Results

Figure 4 presents race-specific respiratory hospital admissions relative risk estimates in New York City for filled PM_{10}, H^+, O_3, and SO_4^2. The numeric values for white and nonwhite estimates are shown in Table 2, along with the pollutant relative risks for the entire population as a whole. Positive associations were generally found between air pollution and increased hospital admissions in all categories. The greatest difference between white and nonwhite subgroups was observed for O_3; the white RR was 1.032 (95% CI: 0.977–1.089) and the nonwhite RR was 1.122 (95% CI: 1.074–1.172). Although not statistically different from each other, the pollutant relative risk estimates for the Hispanic nonwhite category in New York City tended to be slightly larger in magnitude than those for the non-Hispanic white category.

Table 3 shows the respiratory hospital admissions attributable risks for the pollutants considered. Although the relative risk estimates for respiratory hospital admissions are slightly higher for the nonwhite group than for the white group, the underlying rate of admission for the nonwhite group is also much higher than that of the white group. For example, the

nonwhite O_3 RR is 1.122 for a maximum minus mean increment in O_3 mean daily concentration, but when the baseline rate for hospital admissions among this group is considered (32.0 admissions/day/10^6) the attributable risk is 3.90 admissions/day/10^6. This was about 7-fold greater attributable risk than that found for the white group, which had an attributable risk of 0.53 (RR = 1.032). The attributable risk comparison therefore suggests that, in terms of number of admissions per day per unit population, the impacts of these air pollutants on the nonwhite subgroup (vs on the white subgroup) are much higher than were indicated by a comparison of their raw relative risks.

Figures 5 and 6 illustrate the PM_{10} and O_3 respiratory hospital admissions relative risks by insurance status for the white and nonwhite subgroups as well as for the entire population. For the filled PM_{10}, the RR for the entire uninsured population was 1.045 (95% CI: 1.000–1.091), which was essentially the same as the RR for nonwhite uninsured—1.045 (95% CI: 0.998–1.094). Similarly, for O_3, the RR for the entire uninsured population was 1.138 (95% CI: 1.084–1.194); whereas the nonwhite uninsured had an RR of 1.140 (95% CI: 1.085–1.198). These results suggest that a large portion of the apparent difference in pollutant risk estimates between racial subgroups can be explained by socioeconomic factors such as insurance status and poverty.

Discussion

The first objective of this investigation was to assess the impacts of ambient air pollution, as

represented by O_3, PM_{10}, SO_4^2, and H^+. These analyses yielded statistically significant overall associations between air pollution and increased hospital admissions. Moreover, they showed that the relative risk estimates due to increases in ambient pollutant levels for the Hispanic nonwhite population of New York City generally are larger than, although not statistically significantly different from, those of the non-Hispanic white population. In the race-specific analyses, significant pollutant effects were found among nonwhites but not among whites. The lack of statistically significant differences in the between-group comparisons may be a result of several study limitations including the smaller numbers of admission counts in these various subpopulations, which limits the overall power of the analysis; the limited study period length; and/or the inability to differentiate air pollution exposures between the subgroups.

To understand the total impact on population subgroups, we examined the attributable risks, rather than the relative risks. Using the attributable risk to interpret the health impact of air pollution on the subgroups, the differences between the two subgroups become more exaggerated. The nonwhite subgroup appears to be more adversely affected by the effects of air pollution in terms of the number of people per day being admitted to the hospital. This is because the nonwhite subgroup has a larger baseline rate of being admitted to the hospital for respiratory causes than the white subgroup. As discussed earlier, this may be due partly to the lack of access to adequate preventive
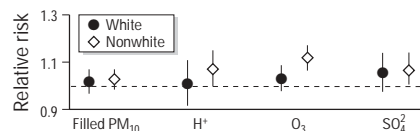
Figure 4. New York City, New York, race-specific relative risks (based upon the maximum minus mean increment) and their 95% CI for hospital admissions outcomes.

Table 2. Respiratory hospital admissions race-specific regression results for specific New York City pollutants considered, 1988–1990.

Pollutant	Racial subgroup	Coefficient (β)	Standard error	t-statistic	RR (based upon the max. minus mean increment) (95% CI)
Filled PM_{10}	White	3.50 E-4	4.15 E-4	0.84	1.021 (0.971–1.074)
(1-day lag)	Nonwhite	4.48 E-4	3.27 E-4	1.37	1.027 (0.988–1.069)
H^+	White	4.10 E-5	1.79 E-4	0.22	1.010 (0.923–1.106)
(0-day lag)	Nonwhite	2.64 E-4	1.41 E-4	1.87	1.069 (0.987–1.079)
O_3	White	5.33 E-5	4.61 E-5	1.16	1.032 (0.987–1.079)
(1-day lag)	Nonwhite	1.96 E-4	3.74 E-4	5.24	1.122 (1.074–1.172)
SO_4^2	White	8.42 E-5	5.44 E-5	1.55	1.058 (0.984–1.136)
(1-day lag)	Nonwhite	1.03 E-4	4.33 E-5	2.38	1.072 (1.011–1.136)

Table 3. Race-specific attributable risk estimate for respiratory hospital admission calculated from differing baseline rates.

Pollutants	Hispanic and nonwhite BR = 32.0 admissions/day/10^6		Non-Hispanic white BR = 16.7 admissions/day/10^6	
	Relative risk[a]	Attributable risk[b]	Relative risk[a]	Attributable risk[b]
Filled PM_{10}	1.027	0.86	1.021	0.35
H^+	1.069*	2.21	1.010	0.17
O_3	1.122*	3.90	1.032	0.53
SO_4^2	1.072*	2.30	1.058	0.97

BR, baseline rates.
[a]Computed for maximum minus mean increase in pollutant concentration. [b]Number per day per million persons. *$p < 0.05$.
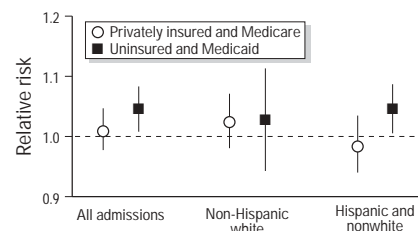
Figure 5. White and nonwhite filled PM_{10} relative risks (based upon the maximum minus mean increment) and their 95% CI for respiratory hospital admissions by insurance status in New York City, New York.
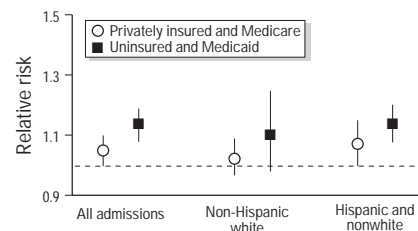
Figure 6. White and nonwhite O_3 relative risks (based upon the maximum minus mean increment) and their 95% CI for respiratory hospital admissions by insurance status in New York City, New York.

healthcare experienced by nonwhites. Thus, the attributable risk may provide a more realistic comparison of the varying impacts of air pollution on racial subpopulations.

The importance of also examining the attributable risk, rather than relative risk alone, when making across-race comparisons, can be seen by examining the results of other published analyses that have used relative risks only. Zanobetti and Schwartz (22), for example, looked at PM_{10} air pollution mortality relative risks by race, and found that whites actually had slightly higher relative risks estimates than blacks. However, if the mortality attributable risk had been calculated for each race, different conclusions could have been reached. For example, computing attributable risks from the Zanobetti and Schwartz race-specific PM_{10} relative risks for Chicago (applied to the population > 64 years of age, the age group in which most deaths occur), it is found that the attributable risks for older blacks is 2.6 deaths/million/day/10 $\mu g/m^3$, and only 1.75 for whites. Thus, the PM_{10} attributable risk for this age group is approximately 50% larger for blacks than for whites. Therefore, it is important to examine not only relative risks, but also to consider age-adjusted attributable risks when evaluating risks across different racial populations that likely have very differing age distributions and baseline rates.

The second objective of this study was to evaluate the impact of SES on the air pollution risk estimates. We used insurance status as a crude indicator of SES that may reflect a greater reliance on the emergency room, lack of good nutritional practices, inadequate living conditions, etc. We found that, after adjusting for insurance status, the O_3 relative risk for the nonwhite insured subgroup became nonsignificant, whereas the O_3 relative risk for the uninsured nonwhite subgroup remained elevated and statistically significant. Similar effects were seen for the PM_{10} relative risk; however, the magnitude of this effect was on a smaller scale. Although relative risk effect estimates above 1.0 were found for all groups, it was apparent that the overall air pollution–hospital admissions association was driven largely by the minority Medicaid and uninsured population (i.e., the minority poor and working poor).

The use of insurance status as an indication of SES has inherent limitations such as the potential for misclassification of the very wealthy self-insured individuals into the uninsured group made up largely of poor individuals. One previous study found that the air pollution–asthma admissions association in Los Angeles, California, was stronger for the those having MediCal (California Medicaid) than for the uninsured, suggesting that low family income, rather than lack of

insurance, is a better predictor of a pollution-related asthma exacerbation (23). However, assuming that the decision to admit a patient to the hospital is not related to their insurance status, this nondifferential misclassification would result in underestimation of the effect. An additional limitation was the lack of pollution exposure estimates for each group. It cannot be precluded that the differences between these groups could be due partly to higher pollution exposures among the poor. However, in the case of a regional pollutant such as O_3, this seems relatively unlikely as an explanatory factor. Despite such possible limitations, our results appear to be consistent with the hypothesis that racial minorities can be more affected by air pollution because these groups are more likely to be socioeconomically disadvantaged.

Our analysis of hospital admissions indicates that the greatest effects of air pollution occur among the poor and working poor; however, this may to some extent reflect the use of hospital admissions, a very severe outcome, as the analysis health end point. For example, it is likely that persons with private health insurance are also affected by air pollution, but can afford to visit a private physician before the effects become more severe to necessitate a hospital admission. Indeed, recent papers have reported air pollution associations with doctors' visits among the general population (24,25). Unfortunately, private doctor visits counts are not publicly available for New York City, so we could not test for wider, less severe, effects in this population as part of this work. However, it remains that the most severe morbidity effects, as represented by hospital admissions, occur disproportionately among the poor and working poor in this city.

Although in the present study the largest air pollution effects were found among nonwhites, the apparent enhancement of the adverse effects of air pollution by SES was observed across all races and is not unique to minorities. However, poverty is clearly more prevalent among nonwhites than among whites, and we conclude that it is largely this socioeconomic disparity that is making the burden of air pollution greatest among nonwhite populations. The implications that people of lower SES are more susceptible to the adverse effects of air pollution that consistently have been demonstrated for the general populations are far reaching. Health risks from ambient air pollution are not the result of a personal decisions (unlike that from smoking) and have no direct benefit to an individual. Arguably, poverty is also not the direct result of an individual's personal decision. Thus, a large subset of the U.S. population may be at increased health risk from exposure to environmental agents over which

they have little control, merely because of their social status. Theoretically, the problem of poverty in the United States is a tangible one, and thus measures can be taken to reduce or eliminate it. Thus, the impacts of SES on the air pollution-health associations demonstrated here can be added to the mounting evidence of the effects of poverty on other health risks, such as asthma.

In summary, our results strongly suggest that, although the differences between the white and nonwhite respiratory hospital admissions–air pollution effect estimates could not be shown to be statistically significant in a two-way test, the nonwhite risk estimates generally were larger than those of the whites. Additionally, when their respective baseline hospital admission rates were incorporated, differences between each group's attributable risks became even more disparate. Finally, within-race analyses by insurance coverage suggested that healthcare and socioeconomic factors likely were driving many of the apparent differences between racial subpopulations, rather than any race-derived biologic difference in air pollution sensitivity.

REFERENCES AND NOTES

1. Thurston GD, Ito K, Kinney PL, Lippmann M. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. J Expo Anal Environ Epidemiol 2:429–450 (1992).
2. Olden K. Effects of air pollution on African-American and other minority populations. Otolaryngol Head Neck Surg 114:255 (1996).
3. Sexton K, Gong H Jr, Bailar JC III, Ford JG, Gold DR, Lambert WE, Utell MJ. Air pollution health risks: do class and race matter? Toxicol Ind Health 9:843–878 (1993).
4. Brown P. Race, class, and environmental health: a review and systematization of the literature. Environ Res 69:15–30 (1995).
5. Centers for Disease Control and Prevention. Children at risk from ozone air pollution—United States, 1991-1993. JAMA 273:1484–1485 (1995).
6. King M, Orcutt J, Hersh R. Just transportation? In: Mobilizing the Region: a Weekly Bulletin from the Tri-State Transportation Campaign. New York: 1998:2.
7. Halfon N, Newacheck PW. Childhood asthma and poverty: differential impacts and utilization of health services. Pediatrics 91:56–61 (1993).
8. Mak H, Johnston P, Abbey H, Talamo RC. Prevalence of asthma and health service utilization of asthmatic children in an inner city. J Allergy Clin Immunol 70:367–372 (1982).
9. Bowman JE. Genetic Variation and Disorders in Peoples of African Origin. Baltimore:Johns Hopkins University Press, 1990.
10. Centers for Disease Control and Prevention. Asthma—United States, 1982-1992. JAMA 273:451–452 (1995).
11. Claudio L, Tulton L, Doucette J, Landrigan PJ. Socioeconomic factors and asthma hospitalization rates in New York City. J Asthma 36:343–350 (1999).
12. Litonjua AA, Carey VJ, Weiss ST, Gold DR. Race, socioeconomic factors, and area of residence are associated with asthma prevalence. Pediatr Pulmonol 28:394–401 (1999).
13. Wissow LS, Gittelsohn AM, Szklo M, Starfield B, Mussman M. Poverty, race, and hospitalization for childhood asthma. Am J Public Health 78:777–782 (1988).
14. The Clean Air Act of 1970. Vol. 42:U.S. Code 91-604, 1970.
15. Ito K, Thurston GD. Daily PM10/mortality associations: an investigation of at-risk subpopulations. J Expo Anal Environ Epidemiol 6:79–95 (1996).
16. White MC, Etzel RA, Wilcox WD, Lloyd C. Exacerbations of childhood asthma and ozone pollution in Atlanta. Environ Res 65:56–68 (1994).
17. U.S. Health Care Financing Administration. The International Classification of Diseases, Ninth Revision. Third Edition.

U.S. Department of Health and Human Services, Washington, D.C. (1989).

18. Gwynn RC, Burnett RT, Thurston GD. A time-series analysis of acidic particulate matter and daily mortality and morbidity in the Buffalo, New York, region. Environ Health Perspect 108:125–133 (2000).

19. U.S. EPA. Aerometric Information Retrieval System. U.S. Environmental Protection Agency:Research Triangle Park, NC. Available: *http://www.epa.gov/air/data*.

20. Thurston GD, Gorczynski JE Jr, Jaques P, Currie J, He D. An automated sequential sampling system for particulate acid aerosols: description, characterization, and field sampling results. J Expo Anal Environ Epidemiol 2:415–428 (1992).

21. Shumway RH, Tai R, Pawitan Y. Statistical Analysis of Daily London Mortality and Associated Weather and Pollution Effects. Davis, CA:Division of Statistics, University of California, 1983;53.

22. Zanobetti A, Schwartz J. Race, gender, and social status as modifiers of the effects of PM10 on mortality. J Occup Environ Med 42:469–474 (2000).

23. Nauenberg E, Basu K. Effect of insurance coverage on the relationship between asthma hospitalizations and exposure to air pollution. Public Health Rep 114:135–148 (1999).

24. Medina S, Le Tertre A, Quenel P, Le Moullec Y, Lameloise P, Guzzo JC, Festy B, Ferry R, Dab W. Air pollution and doctors' house calls: results from the ERPURS system for monitoring the effects of air pollution on public health in Greater Paris, France, 1991-1995. Evaluation des Risques de la Pollution Urbaine pour la Sante. Environ Res 75:73–84 (1997).

25. Hajat S, Haines A, Goubet SA, Atkinson RW, Anderson HR. Association of air pollution with daily GP consultations for asthma and other lower respiratory conditions in London. Thorax 54:597–605 (1999).