The Effect of Ozone on Inner-City Children with Asthma

Identification of Susceptible Subgroups

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ABSTRACT

Within a cohort of 846 inner-city asthmatic children aged 4 to 9 yr, we looked for subgroups that were more susceptible to the effects of summer ozone. Daily diaries were compared with ambient ozone levels to evaluate effect modification by demographic and environmental characteristics. Children born > 3 wk prematurely or weighing < 5.5 lb. had greater declines in morning peak expiratory flow rate (PEFR) (1.8% versus 0.3% per 15 ppb ozone, p < 0.05) and a higher incidence of morning symptoms (odds ratio = 1.42 versus 1.09 per 15 ppb ozone, p < 0.05) than did children who had been full-term infants of normal birthweight. Among children who had been of low birthweight (LBW) or had been premature infants, greater declines were seen among those whose reported baseline medication category was "no medication" (3.2% decline) or "steroids" (2.7%) as opposed to beta agonists or xanthines (0.8%) or cromolyn without steroids (0.1%). Among the children who had been normal birthweight and full-term infants, the cromolyn without steroids group had the greatest declines in %PEFR (1.3%, versus < 0.5% in each of the other three groups). Nonatopic children also had greater responses to ozone. We conclude that among an asthmatic cohort, children who had had an LBW or a premature birth showed the greatest responses to ozone.

INTRODUCTION

The U.S. Clean Air Act mandates that the National Ambient Air Quality Standards for criteria pollutants be set low enough to protect the health of all susceptible groups within the population (1, 2). "Susceptible groups" are defined as those who share one or more characteristics that increase their risk for adverse health events over that of persons without those characteristics. In particular, asthmatic individuals have been shown to have lower peak expiratory flow rates (PEFR) and increased symptom reports on days with increased pollution levels (3). Analyses of asthmatic cohorts have found heterogeneity in the response to ambient pollutants, which suggests that not all asthmatic individuals have similar susceptibility. Medication use and symptom status have been shown to modify asthmatic children's response to air pollution (3, 6). Heavy personal tobacco use has also been identified as an effect modifier among adult asthmatic individuals (7). Asthma is a multifactorial disease with a complex etiology, and host characteristics such as birthweight and atopy, as well as exposures in the home environment, may therefore also modify the response of asthmatic individuals to air pollution. Few studies have had sufficient data on these characteristics to evaluate differential responses to ambient air pollution in subgroups of asthmatic individuals.

We used data collected as part of a large multicenter study of inner-city children with asthma to evaluate a series of demographic, host, and home environment characteristics that may modify the symptomatic and pulmonary functional response of asthmatic children to urban air pollution. This report describes subgroups of asthmatic children with increased susceptibility to low levels of ambient summer air pollution.

METHODS

Cohort Identification

The National Cooperative Inner-City Asthma Study (NCICAS) was a multicenter study of risk factors for asthma morbidity among inner-city children. The design of the study and the methods used in it are reported in more detail elsewhere (8). Briefly, children and their parents were recruited from emergency departments and primary care clinics in the eight urban areas of the Bronx and East Harlem, NY; Baltimore, MD; Washington, DC; Detroit, MI; Cleveland, OH; Chicago, IL; and St. Louis, MO. At the time of recruitment, children were between the ages of 4 and 9 yr and resided in...
inner-city neighborhoods in which the income of at least 30% of residents was below the poverty level designated by the U.S. Government. Children in the study had either: (1) parentally reported physician-diagnosed asthma and symptoms in the 12 mo before the study; or (2) respiratory symptoms consistent with asthma, such as cough, wheezing, or shortness of breath, that had lasted more than 6 wk during the previous year, together with increased symptoms with exercise or cold air exposure or a family history of asthma. The protocol included an in-person baseline interview, a home survey, three brief telephone follow-up interviews at 3-mo intervals, and 2-wk peak flow and symptom diaries kept after the baseline interview and before each follow-up interview.

Exposure Measures

Concentrations of ozone, sulfur dioxide, nitrogen dioxide (NO₂), and particulate matter of < 10 µm size (PM₁₀) were obtained from the Aerometric Information Retrieval System database of the U.S. Environmental Protection Agency. Across the eight urban areas involved in the study, the most complete data were available for daily ozone levels, which serve as a marker for summer air pollution and which were therefore used to model subgroup differences in response to air pollution. The analysis was restricted to diaries completed between June 1 and August 31, 1993. A daily ozone metric was calculated from the 8-h average of hourly ozone concentrations from 10:00 A.M. to 6:00 P.M. The occurrence of rain in the preceding 24 h (yes/no) and the mean wet-bulb temperature (degrees Celsius) in the preceding 12 h were obtained from local airports and were included in all multivariate analyses. All children within an urban area were assigned common ozone and weather values.

Outcome Measures

At the baseline interview and during follow-up telephone calls, children and their parents were trained in the use of a mini-Wright peak flow meter (Clement Clarke, Columbus, OH). The analysis of susceptible subgroups included the following two outcome measures: (1) daily percent change from the diary-specific median of peak flow readings (%PEFR); and (2) incidence of symptoms, defined as the occurrence of wheezing, cough, or chest tightness among children who were symptom-free on the previous day. Morning and evening measures were modeled separately for several reasons. Morning values are better indicators of asthmatic individuals who are susceptible to airway narrowing, and focusing on morning measures may therefore identify children at greater risk for adverse health outcomes (10). Morning values also measure airway function at a time of most severe bronchoconstriction, when measurable differences between and within individuals may be greatest. In addition, we felt that evening measures might be influenced by daily medication use and time-activity patterns, and since data for these factors were unavailable and therefore could not be included in models of evening measures, the morning model could provide a less confounded estimate of the air pollution effect. For similar reasons, we chose to model the incidence rather than the prevalence of symptoms. Prevalence is a function of both the incidence and duration of symptoms. Because the duration of symptoms is likely to be influenced by changes in behavior that were not documented (such as increased medication use or decrease in activity), we felt that the incidence model would be less confounded by these factors.

Characterization of Susceptible Subgroups

The NCICAS protocol included extensive collection of demographic, psychosocial, home environment, and host characteristic data. A review of the epidemiologic literature suggested that several of these factors might modify the effect of air pollution on pulmonary function and symptoms. To identify susceptible subgroups, we defined characteristics from data collected during the baseline interview.

Birth characteristics of each subject were based on the mother’s report, and included low birthweight (LBW; < 5.5 lb.) and prematurity (≥ 3 wk). Given the overlap between these characteristics, we created a dichotomous composite measure based on the report of prematurity or LBW.

Skin testing for aeroallergens was done with the skin prick-puncture method on the volar surface of the forearm, using the Multi-test device (Lincoln Diagnostics, Inc., Decatur, IL) (11). The panel of 14 test allergens included Penicillium, dog, cat, mouse, rat, two types of dust mite, cockroach, grass mix, Alternaria, orchard, oak, maple, and ragweed. Three categories of atopy were created.

Daily medication use was not collected in the NCICAS diaries. At the time of the baseline interview, however, parents reported the names of all medications used or prescribed in the 3 mo before the interview. On the basis of these reports, children were classified into one of four medication groups: (1) no medications; (2) only β₂-agonists or methylxanthines; (3) cromolyn (without steroids); or (4) steroids.

Parentally reported current maternal smoking was used to characterize exposure to environmental tobacco smoke (ETS). Data on daily use of air conditioners was unavailable; however, homes with air conditioning were identified on the basis of the reports of either a room or central air conditioner. The presence of a gas or electric stove was also reported. An index of crowding was created by dividing the number of people living in the home by the number of rooms (excluding bathrooms), with an index above 1.0 defining crowdedness.

Dust samples were collected according to standard methods (12, 13) and were assayed for dust mite (Dermatophagoides farinae [der f] and Dermatophagoides pteronyssinus [der p]), cat (Felis domesticus 1 [fel d 1]), and cockroach (Blatella germanica 1 [bla g 1]). Antigen levels in the bedroom were used to define exposure. On the basis of proposed threshold levels for the induction of disease, high exposures were defined by > 2 µg of allergen per gram of dust for a combined measure of der p 1 and der f 1, > 2 µg/g dust for fel d 1, and > 8 U/g dust for bla g 1 (14). Because of budget restrictions, antigen levels in the dust were measured only once (within a month of the baseline interview) and data on this were collected for approximately one-third of the homes in the study. Diaries were repeated throughout the year, and dust samples were therefore collected on an average of 12 wk before the start of the diaries included in this part of the study (range: 28 wk before to 6 wk after starting the diary). Because the dust sample data might therefore not have corresponded to a child’s actual exposures at the time the child’s diary was completed, we also evaluated self-reported antigen exposure, including the presence of carpeting in the child’s bedroom (for dust mite), the

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presence of a cat in the home, and reports of cockroaches. Exposure to molds in the home was limited to reports of moisture or ceiling leaks.

We examined whether peak flow responses to ozone were different for children who were and were not experiencing asthma symptoms. Symptom status was defined in the following two ways: (1) the current day's report of symptoms (yes/no); and (2) groupings based on the median of the percent of diary days with symptom reports (< 25% versus ≥ 25% of diary days).

Statistical Methods

Observations recorded in diaries and those for specific individuals are not independent of one another, and we therefore used longitudinal regression analyses that accounted for repeated measures in analyzing these data. We used linear mixed models and generalized estimating equations to model repeated measures for normally distributed and binary outcomes, respectively. Each method takes into account the correlation structure of observations recorded in diaries and those for specific individuals. Because the estimates provided by the equations are based on outcome and risk-factor data at the individual level, they are useful for making direct inferences about the role of risk factors at the individual level.

The percent change in morning peak flow (%PEFR) was modeled with the SAS Proc Mixed program (SAS Institute, Cary, NC) (15). The incidence of symptoms was modeled with the SAS Proc Genmod Program (16) for binary distributions. Indicator variables for urban area, visit number at which the diary was obtained (i.e., baseline and 3-, 6-, or 9-mo assessment), and day of study (since June 1, 1993) were entered simultaneously as fixed effects in all models. A linear term for 12-h average wet-bulb temperature and a binary indicator for the occurrence of rain in the preceding 24 h were included as fixed effects. In a model of the entire cohort, the interaction term between urban area and ozone was not significant for either of the two study outcomes, and was therefore, not included in the models of subgroup effects. An independence error structure provided the best fit relative to other covariance options on the basis of Akaike's Information Criteria (AIC), and was used in models for both outcome measures.

In separate models, the test for difference across subgroups was done by including main-effect terms for ozone and for each subgroup of interest, along with an interaction term for subgroup by ozone exposure. The use of interaction terms instead of stratification of the data allowed the model to be fitted for all observations, and increased the stability of the estimates. We report any findings that were statistically significant (p < 0.05), or for which there was at least a twofold difference across subgroup characteristics regardless of statistical significance. Estimates were rescaled to reflect an increase in ozone across the interquartile range (15 ppb).

RESULTS

A total of 1,528 children completed the NCICAS baseline interview, of whom 846 children completed at least one peak-flow diary between June 1 and August 31, 1993. A total of 910 diaries were returned during this interval, which provided 11,622 child-days of observation. Children with more severe asthma, as defined by reports of the use of multiple classes of medication, were more likely to complete diaries. Pollution levels were unrelated to the completeness of the diaries. The characteristics of the population described in this report are presented in Table 1.

Across the eight urban areas involved in the study, the mean 8-h average ozone concentration was 48 ppb. Fewer than 5% of days exceeded the current U.S. Environmental Protection Agency standard of 80 ppb (157 μg/m³) as an 8-h mean ozone concentration. The 12-h mean wet-bulb temperature (8:00 A.M. to 8:00 P.M.) was 19° C. Pearson's correlation coefficient for current-day ozone and wet-bulb temperature was 0.32. The percent of days with rain was 39%.

The single-day lag models suggested a minimal immediate impact of ozone on each study outcome measure for both the morning and evening. The ozone effect at longer lag times was seen only in the morning outcome measures. No significant associations were seen between single- or multiple-day ozone metrics and any evening outcome measure. For morning outcomes the ozone effect increased over several days, and the strongest association was seen for multiple-day moving averages. A 15 ppb increase in the 5-d moving average ozone concentration (with lags of 1 to 5 d) was associated with a 0.59% decline in morning %PEFR (95% confidence interval [CI]: 0.13% to 1.05%). The incidence of morning symptoms was most strongly associated with a 4-d moving average (lags of 1 to 4 d) (odds ratio [OR] = 1.16, 95% CI: 1.02 to 1.30).

The cumulative effects obtained from unrestricted lag and second-degree polynomial lag models for morning %PEFR were nearly identical to those obtained from the 5-d moving average (cumulative declines of 0.54%, 0.51%, and 0.59%, respectively). These unrestricted lag models suggested that ozone exposures for 3 to 5 d earlier have a greater impact on morning %PEFR than do more immediate exposures. The unrestricted and polynomial distributed lag models for morning symptoms also yielded cumulative estimates similar to those obtained with moving averages (summary OR = 1.13, 1.14, and 1.16, respectively).

Subgroup differences in response to ozone are reported in Tables 2 and 3. The greatest differences in %PEFR in response to ozone were found to accord birthweight or prematurity status, which was available for 814 of the 846 children for whom data were evaluated. The 170 children of LBW or who had had premature birth had a 1.8% decline in morning %PEFR (95% CI: 1.0% to 2.7%) per 15 ppb increase in 5-d average ozone, as...
compared with only a 0.3% decline (95% CI: −0.2% to 0.8%) for the 644 full-term or normal birthweight children. These estimates were unaffected by adding a main-effects term for median peak flow into the model. When restricted to the children for whom acceptable spirometry data were available, inclusion of a main-effects term (FEV\textsubscript{1} % predicted or FVC % predicted) did not alter the conclusion that children of LBW or premature birth had significantly greater responses to ozone. The effect was strongest among but was not limited to the 5% of children of very LBW. A 15 ppb increase in ozone was associated with declines of 3.5%, 1.5%, and 0.3% for children with birthweights < 3.3 lb., 3.4 to < 5.5 lb., and ≥ 5.5 lb., respectively, which suggests an exposure-response relationship.

Other characteristics associated with greater effects of ozone on morning %PEFR included male sex, reported cromolyn use (without steroids), household crowding, presence of an electric rather than a gas stove in the home, and self-reported exposure to indoor allergens. Nonatopic children showed greater declines in morning %PEFR in response to ozone than did atopic children.

Within the study cohort of 846 children, the OR for the incidence of morning symptoms was 1.15 per 15 ppb increase in 4-d average ozone (95% CI: 1.02 to 1.30). Children with LBW or prematurity had an OR for the incidence of morning symptoms of 1.42 per 15 ppb increase in 4-d average ozone (95% CI: 1.10 to 1.82), as compared with the remaining children, for whom the OR was 1.09 (95% CI: 0.95 to 1.24). Children in the cromolyn group had greater ozone effects that children in the groups reporting use of β-agonists or xanthines only, of steroids, or of no medications. Nonatopic children had a greater incident of morning symptoms in response to increases in 4-d average ozone. Other characteristics associated with greater effects of ozone on the incidence of morning asthma symptoms included male sex, the presence of cats in the home, and carpeting in the child's bedroom.

We examined whether the effect of ozone was stronger among children who were both exposed to and sensitized to indoor allergens (roach, cat, dust mite). Within the study cohort of 846 children, no clear patterns were seen for exposure and sensitization to roach or mite, or from an indicator for any of the three indoor allergens. Children who lived in homes with cats and were sensitized to them, however, had a substantially greater incidence of symptoms in response to increases in ozone (OR = 1.80 per 15 ppb increase) than did children who were unexposed or not sensitized (OR = 1.11). This enhanced effect of ozone was also seen for %PEFR, and was additionally seen when exposure was based on allergen levels from dust samples rather than on self-reported housing characteristics.

There was no evidence of subgroup differences based on age, maternal smoking status, household income, symptom status, reports of moisture or leaks in the home, or asthma severity, and these data are therefore not presented.

Models were repeated, with stratification by the indicator variable, for LBW or premature birth (Table 4). The birth-characteristic subgroups did not differ significantly from one another with respect to the characteristics found in Table 1, except that children who had been full-term infants or of normal birthweight were more likely to be male. The sample sizes for several of the subgroups, such as that for antigen levels in dust, were too small to permit additional stratification, and these analyses are therefore not presented.

Among the 644 children who had been full-term infants or of normal birthweight, greater effects on morning %PEFR were seen for children who were male, nonatopic, reported cromolyn use, or lived in crowded homes or homes with electric stoves. Increased symptoms were associated with nonatopic status, reports of cromolyn use, and presence of an electric stove in the home.

Although estimates for subgroup differences within the small cohort of LBW or premature children (n = 170) are rather unstable, the findings with regard to this may identify areas for future study. Within this subset of children, greater declines (at least twofold) in morning %PEFR in response to ozone were seen among children who reported no use of medications, who reported using steroids, who lived in crowded homes, or who were exposed to indoor allergens as based on self-reports. A higher incidence of symptoms associated with ozone was found among boys and among children reporting no medications. There were no clear patterns according to atopy, type of stove in the home, presence of air conditioning, or allergen exposure.
DISCUSSION

This present study suggests that some subgroups of asthmatic, inner-city children may be more susceptible than others to the adverse effects of summer ozone. LBW and premature birth were associated with substantially greater responses to ozone, even after adjustment for median PEFR or baseline measures of pulmonary function. Although these classifications were based on maternal reports and not on medical records, the percent of children classified as having LBW (17%) or a premature birth (13%) was very comparable to published data based on medical records, suggesting that the self-reporting used in the study was quite accurate (17).

Several recent studies have examined the impact of air pollution on preterm birth (18), LBW (19), and infant (20, 21), and intrauterine (22) mortality. Our findings suggest that among asthmatic children, birth characteristics continue to be associated with increased susceptibility to air pollution later in life. There is extensive evidence that LBW and prematurity are associated with many adverse outcomes, including heart disease and hypertension (23). Moreover, these factors are also associated with reduced lung function, higher levels of airways reactivity, and increased susceptibility to lung damage (24, 25). Inadequate weight gain of the fetus or premature birth may result in alteration of the structure and function of airways at birth. These effects have been found to remain for many years beyond birth (24, 25), and may explain why LBW and prematurity were found to increase children's susceptibility to the respiratory insults of air pollution.

In several analyses done in the present study, boys appeared to have greater responses to increases in ozone than did girls, although the differences were not consistent across outcome measures. It has been reported elsewhere that boys were more susceptible than girls to the effects of environmental tobacco smoke, dampness, and unvented kitchen stoves (26, 27). It follows that boys may also be more susceptible to the effects of ambient air pollution. The greater effect in boys may be due to greater time spent outdoors and more physical activity, both of which are factors that would increase exposure to and the respiratory dose of ambient air pollution (28). Others have shown that correction for these factors increases effect estimates (29). Time spent outdoors and physical activity data were not included in the NCICAS protocol and therefore could not be included in present analyses.

Medication classifications based on a one-time assessment during a baseline interview may be inadequate for fully controlling for recent medication use. Despite this limitation, we found that within the normal birthweight and full-term subgroup, children who reported using cromolyn without steroids experienced greater effects of ozone. Children who did not report using any medications and those who reported using steroids, β-agonists, or methylxanthines had very similar responses to ozone. Among the subgroup with LBW or premature birth, children who did not report using any medications showed greater susceptibility to ozone. This may have been a subgroup that was inadequately medicated, given their higher risk for respiratory problems. Children who reported using steroids also showed greater declines in %PEFR. Report use of steroids is likely to be a marker for asthma severity rather than a true reflection of a harmful effect of steroids on responses to ozone. In fact, within both the entire cohort and when it was stratified by birth characteristics, children who reported using steroids also reported from four to six times more hospitalizations and emergency room visits for asthma during the follow-up year than did children who reported using no medications (data not shown).

The findings for air conditioner use are inconsistent. Air conditioning appeared to be protective only for symptoms within the subgroup with LBW or premature birth. However, air conditioners are effective in reducing indoor ozone levels (30), and children who lived in air-conditioned homes may therefore have had a substantially lower exposure and reduced risk from outdoor ambient levels of ozone. It is not clear, however, why this would not be supported in the group of children of normal birthweight or full-term birth.

An unexpected finding was that electric rather than gas stoves were associated with an increased risk of effects of ozone exposure. Gas stoves are an important indoor source of NO₂, a well-established respiratory irritant. However, indoor NO₂ will quench ozone that penetrates indoors, reducing ozone exposures. Children who live in homes with gas stoves may therefore have lower and more variable exposures to ozone than indicated by outdoor monitors. Because only 35 children in the LBW group in our study had electric stoves in their homes, the estimates for the effect of stove type on response to ozone appear to be highly unstable.

Interactions between ozone and antigens have been reported, and may be an important mechanism in pollutant-induced exacerbations of asthma (31). There is speculation that air pollutants increase the permeability of the bronchial mucosa to antigens, thereby decreasing the threshold for the antigen exposure needed to produce sensitization, and increasing the incidence of allergic asthma and asthma symptoms (32). We found that children who were both exposed and sensitized to cat allergen had greater responses to ozone. This increased susceptibility was not found for the other allergens, but the sample sizes were small and estimates for such susceptibility were unstable. We therefore also examined the effect of allergen exposure regardless of sensitization status. Among children of normal birthweight or full-term birth, exposure to household antigens (by self-reported carpeting, cockroaches, or a cat in the home) was associated with greater responses to ozone.

The results were less consistent across antigens when based on dust-sample levels. Only cat antigen (fel d 1) was associated with an increased response for both study outcomes; cockroach exposure (bla g 1) was associated with an increased effect on %PEFR. Analysis of dust samples is often considered to be the "gold standard" in exposure assessment (33). However, the interval between the dust sampling and collection of diary information in the present study may have compromised the representativeness of exposures during the diary period.

When we disregarded allergen exposure status, there was no clear pattern of responses across atopy categories among the children of LBW or...
premature birth. In the remaining 79% of the cohort, children with positive skin tests had smaller responses to ozone. Two recent studies of children of similar ages also found that the effect of outdoor pollutants was limited to nonatopic children (34, 35), and nonatopic children have been shown to be more responsive to the effect of ETS (36). Given the multifactorial etiology of asthma, it may be that for nonatopic children, risk factors such as irritant effects of air pollution play a more dominant role in asthma exacerbations, whereas among atopic children, respiratory events are triggered by antigen exposure.

Asthma prevalence and hospitalization rates are substantially higher among Puerto Ricans than among Mexicans (37, 38). Hispanic persons from the two New York sites involved in our study were predominately Puerto Rican, whereas the Hispanic persons from the Chicago site were predominately Mexican. We therefore examined ethnic differences in ozone effect separately, for each of the two cities. Stratification by birth characteristics resulted in groups that were too small for meaningful comparisons. The data suggest, however, that in either birth-characteristic group, responses were similar for Puerto Ricans and black persons in New York, but that in Chicago the Mexican individuals in the study showed greater responses to ozone than did black persons.

There are several limitations to our findings. Our study focused on a single pollutant, and does not address the impact of copollutants. In the communities in which the study was conducted, ozone was present with copollutants, and was therefore used as a marker for summer pollution. It is therefore not surprising that the acute effects of ozone reported in chamber-exposure studies are not replicated in free-living populations, and that more delayed effects were seen in our study. Analyses of other pollutants suggested that within the study cohort, these pollutant did not strongly confound the overall association with ozone (data not shown), and are therefore unlikely to influence the ozone-related subgroup differences identified in this report.

Medication use was reported only at the baseline interview, and data on daily medication use was not collected. It is possible that in the interval between the baseline interview and the completion of the diary, medications were added or deleted from a child's treatment plan, and that on a day-to-day basis, compliance with the treatment plan varied. The possibly resulting misclassification is unlikely to have been related to ozone levels, and it is likely that those subjects who reported using a particular medication at baseline were more likely to have used it during the interval over which the diaries were collected. Future studies of the effect of ambient pollutants should consider the collection of daily information about medication use, as well as about daily air conditioner use, time spent outdoors, and physical activity.

The majority of the subgroups in the study were defined by characteristics determined at the time of the baseline interview, and may have changed with time. However, only 20% of the diaries used in the study data analysis were completed more than 3 mo after the baseline interview. Except for medications and antigen levels, the subgroup characteristics are unlikely to have changed substantially within a few months, and it is therefore reasonable to assume that the baseline characteristics of subjects were applicable to exposures at the time the diaries were collected. It is also reasonable to assume that changes in these characteristics were unrelated to daily exposures to ozone.

The study did not include outdoor monitoring of antigen levels. Others have shown that outdoor antigens are independent risk factors for respiratory outcomes and do not confound the association with ozone (39, 40). Also, if increases in ozone were highly correlated with outdoor antigen levels, one would expect to see a greater effect among children with positive skin tests to outdoor allergens. This was not found to be true among the population in our study (data not shown).

This study did not address exposures away from home, such as at school. However, because the study period was limited to June through August, exposures at school were likely to have been minimal and would not have influenced the study findings. Given the sociodemographics of the study population, it is unlikely that many children were at summer camp while completing the study diaries. Exposures in day-care and other environments outside the home could not be addressed in the study, but should be considered in future studies.

Because our primary interest was in the effect of ozone, only diaries completed during the summer were included. During the winter months, the role of indoor allergens and pollutants may be stronger, since children spend more time indoors, with closed windows, and ventilation is decreased. Future studies of the interaction of indoor characteristics with winter pollutants are warranted.

Conclusion

Among the cohort of inner-city children with asthma examined in the present study, ozone was found to be significantly associated with measurable declines in morning %PEFR and increases in morning asthma symptoms. These data suggest that several asthmatic subgroups susceptible to effects of ozone may exist. Children of LBW or of premature birth are at greater risk for respiratory problems, and appear to be substantially more susceptible to the effects of summer air pollution than children of normal birthweight or full-term gestation. The study underscores the need to consider effects of prenatal and possible early postnatal development on pulmonary function and ongoing susceptibility to a myriad of adverse exposures. Further investigation is needed to replicate these findings in other asthmatic populations, and to evaluate whether LBW and prematurity are associated with greater effects of air pollution in nonasthmatic cohorts.

Footnotes

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