

Pediatric patient asthma-related emergency
department visits and admissions in Washington, DC,
from 2001-2004, and associations with air quality,
socio-economic status and age group

Steven M Babin^{1§}, Howard S Burkom¹, Rekha S Holtry¹, Nathaniel R Tabernero¹,
Lynette D Stokes^{2,3}, John O Davies-Cole², Kerda DeHaan², Deitra Hazelwood Lee^{2,4}

¹Johns Hopkins University, Applied Physics Laboratory, 11100 Johns Hopkins Road,
Laurel, MD 20723, USA

²Environmental Public Health Tracking Program, Bureau of Epidemiology and Health
Risk Assessment, District of Columbia Department of Health, 825 North Capitol
Street NE, 3rd Floor, Washington, DC 20002, USA

³Currently employed by the Division of Energy Employees Occupational Illness
Compensation, US Department of Labor, Washington, DC 20210, USA

⁴Currently employed by the US Government Accountability Office, Washington, DC
20548, USA

§Corresponding author

Email addresses:

SMB: steven.babin@jhuapl.edu
HSB: howard.burkom@jhuapl.edu
RSH: rekha.holtry@jhuapl.edu
NRT: nathaniel.tabernero@jhuapl.edu
LDS: stokes.lynette@dol.gov
JODC: john.davies-cole@dc.gov
KD: kerda.dehaan@dc.gov
DHL: leedh@gao.gov

Abstract

Background

The District of Columbia (DC) Department of Health, under a grant from the US Centers for Disease Control and Prevention, established an Environmental Public Health Tracking Program to demonstrate possible relationships between ambient air quality and acute asthma exacerbations. The goal of this contextual pilot study is to quantify short-term associations between daily pediatric emergency department (ED) visits and admissions for asthma exacerbations with ozone and particulate concentrations, socio-economic status, and age group.

Methods

Data included daily counts of de-identified asthma-related pediatric ED visits for DC residents and daily ozone and particulate concentrations during 2001-2004. Daily temperature, mold, and pollen measurements were also obtained. After a cubic spline was applied to control for long-term seasonal trends in the ED data, a Poisson regression analysis was applied to the time series of daily counts for selected age groups.

Results

Associations between pediatric asthma ED visits and outdoor ozone concentrations were significant and strongest for the 5-12 year old age group, for which a 0.01-ppm increase in ozone concentration indicated a mean 3.2% increase in daily ED visits and a mean 8.3% increase in daily ED admissions. However, the age group with the highest rate of asthma-related ED visits were the 1-4 yr olds. For 1-17 yr olds, the rates of both asthma-related ED visits and admissions increased logarithmically with

the percentage of children living below the poverty threshold and levelled off when this percentage exceeded 30%.

Conclusions

Significant associations were found between ozone concentrations and asthma-related ED visits, especially for 5-12 year olds. The result that the most significant ozone associations were not seen in the age group (1-4 yrs) with the highest rate of asthma-related ED visits may be related to the clinical difficulty in accurately diagnosing asthma among this age group. There appears to be a real increase in relative risk of asthma ED visits for children living in higher poverty zip codes versus other zip codes. The results observed among age and poverty groups could suggest designs for future epidemiological studies that could include more information on individual exposures and other risk factors.

Background

Elevated concentrations of ground-level ozone and particulates have been shown to be associated with increased incidence of asthma exacerbations [1-3]. Aeroallergens, such as pollen and mold, may trigger allergy-induced asthma symptoms. Different locations have different aeroallergen seasons and, in Washington, DC, the following are the pollen seasons for trees, grasses, and weeds, respectively: February through June, May through August, and July through October [4]. Also, either very low or very high ambient temperature may be associated with asthma exacerbations. Higher temperatures may be associated with asthma exacerbations because they occur when there is more sunlight and sunlight is necessary for emissions to be converted to ground-level ozone. At the other temperature extreme, studies have suggested that very cold, dry air acts as an airway irritant, yet primarily results in exacerbations of exercise-induced asthma [5-7], which is not a part of this study.

The District of Columbia Department of Health (DC DOH), under a grant from the US Centers for Disease Control and Prevention (CDC), established an Environmental Public Health Tracking Program (EPHTP) to demonstrate possible relationships between ambient ozone and particulates and short-term asthma health outcomes, and to identify areas and populations most likely to be affected by pollution [8].

Therefore, the goals of this small pilot study were to determine the degree to which asthma exacerbations are associated with ozone and particulate concentrations in the short-term, on the order of days after presumed exposure, and to identify pediatric populations that may be at increased risk of these health effects. Asthma exacerbations were determined by daily counts of pediatric ED visits for asthma-

related problems. As will be described, the spatial variations among pollutant measurements on a given day were rather small. This is consistent with DC being an urban area with little industry and whose major source of ozone and PM_{2.5} is the considerable amount of vehicular traffic that permeates the 159 km² land area of DC. Therefore, it was decided to examine other spatial features that show more significant regional variation and these include residence zip code and socio-economic status of the pediatric population. Some interesting results were thereby obtained, especially with regard to socio-economic status.

Methods

The concentrations of ozone and particulates of aerodynamic diameter 2.5 µm or less (PM_{2.5}) were obtained from available measurements at five locations within DC from October 2001 through September 2004. Ozone measurements in units of parts per million (ppm) were made hourly at:

- Takoma Elementary School,
- River Terrace Elementary School, and
- Southeast (SE) end of McMillan Reservoir.

Daily PM_{2.5} measurements in units of µg m⁻³ were made at:

- River Terrace Elementary,
- National Park Service Office at Haines Point, and
- SE end of McMillan Reservoir.

Note that two of the locations above measured both ozone and PM_{2.5}. For the purposes of our analysis, the hourly ozone data were converted into 8-hour daily maximum ozone concentrations to conform to non-attainment criteria adopted by the

US EPA (see, for example, <http://www.epa.gov/ozonedesignations/> and <http://www.epa.gov/oar/oaqps/greenbk/oindex.html>). While PM_{2.5} and ozone were each measured at three different locations within DC, the spatial variation among measurements for each day during the three-year period was found to be negligible compared to differences expected to cause asthma problems. Therefore, we used the daily mean of the available measurements among these sites to represent each day's ozone or PM_{2.5} concentration.

Aeroallergen data were collected at the Walter Reed Army Medical Center by the US Army Centralized Allergen Extract Laboratory. While not collected every day, these data are collected about three days a week and more frequently from spring through early fall. These data covered the months of October 2001 through September 2004, except for December 2002 when no measurements were taken. These data consisted of daily counts of grass, weed, and tree pollen and mold spores in units of grains m⁻³. Daily temperature data were obtained from the National Weather Service measurements at Reagan National Airport in DC.

For health outcomes, our data consisted only of daily counts of asthma-related pediatric emergency department (ED) visits by DC residents between October 2001 and September 2004. Asthma-related visits were defined as those ED visit records in which one of the first three of nine possible diagnosis fields listed an asthma code number. These ED record fields contain numbers based on the International Classification of Diseases Ninth Revision (commonly called ICD-9) codes used by hospital and physicians' office personnel to report billing information to insurance companies [9]. ICD-9 codes may indicate either symptoms or specific diseases, and

those beginning with 493 indicate an asthma diagnosis. Based on discussions with medical personnel about ED coding practices among different DC hospitals, the criterion of an asthma code in one of the first three fields was chosen as an indication that an asthma exacerbation was a principal reason for the visit. The pediatric patients were those with an age between 1 and 17 years on the ED visit date. Patient identification was not included in the data except for residence zip code, age at time of visit, date of visit, and whether or not the patient was admitted. Data were grouped into these patient age groups: 1-4 years, 5-12 years, 13-17 years, and 1-17 years. Analysis was performed independently on each of these age groups. In addition to these data, we also obtained pediatric population by zip code and age group for DC according to the 2000 US Census. This US census data also provided for each zip code the percent of the child population living in households with income considered to be below the US poverty level.

We found that there were generally two annual peaks in the daily ED visits for pediatric asthma exacerbations in DC: the highest peak in September-November and a secondary peak in March-May. Because annual peaks in ozone and PM_{2.5} tend to occur in the summer months, this discrepancy results from strong seasonal effects on exacerbations that are associated with other factors [10]. For example, several studies [11,12,13] have noted increased asthma morbidity triggered by respiratory viral infections (e.g., colds) that occur during late summer and early autumn. While we had no data on respiratory infections, they represented one of the trends that were taken into account by the selection of knots for the spline to be described subsequently. We had no data on indoor and behavioral factors related to asthma exacerbations. No day-of-week effects could be identified in the asthma-related ED

daily count data. Because the focus of the study was only on short-term associations of air pollutants (i.e., days) and asthma ED visits, it was necessary to control for the above stronger long-term influences that were not part of the study. Many investigators have used a natural cubic spline function to represent the strong seasonal signals as a control for confounders in order to observe short-term asthma associations with pollutants [14,15]. Once the long-term trends are removed, the short-term associations become apparent. Our spline curve was derived from a best fit to the time series of daily asthma-related ED visits for DC children of ages 1-17 yrs over a three-year period. This spline had twelve knots to represent the frequency of variation of strong seasonal changes over the study period. Because there were 3 years of data and 4 seasons per year, 12 knots appeared to be a reasonable choice. These knots were not evenly spaced, but were selected to coincide with large long-term changes in the daily ED visits. Once these long-term trends were removed, the residual data (Fig 1) were examined for short-term (on the order of days) associations with ozone and PM2.5. Note in Fig. 1 that, while changes in variance remain, the long-term trends have effectively been removed.

While there were a number of choices for an analysis model, our goal was to quantify short-term associations in the time series of the ED daily counts with the environmental data. Our health data covered three years and only consisted of de-identified, zip-code level, daily counts of asthma-related ED visits in a pediatric population. A Poisson regression analysis assumes that counts of independent, rare events follow a Poisson distribution and models the logarithm of daily visit counts as a linear function of the explanatory variables. The Poisson regression model adjusts for data skewness by using a log transformation and is useful for distributions of non-

negative data, and data for which the variance increases as the mean. Poisson analysis therefore seemed appropriate for daily counts of asthma-related ED visits. While other regression models may also be appropriate, we chose the Poisson regression for the above reasons and the fact that it had successfully been used in other studies similar to ours (e.g., [16,17,18]).

Our database for analysis included date, daily ED visit and admission counts for each age group (1-4, 5-12, 13-17, and 1-17 yrs), daily 8-hr maximum ozone concentrations (based on the mean of the available measurement sites for that day), daily PM_{2.5} concentrations (based on the mean of the available measurement sites for that day), daily maximum temperature, daily tree, grass, and weed pollen concentrations, daily mold concentrations, month and season, and spline curve predictions. Because exercise-induced asthma diagnosis codes were virtually non-existent in our ED data and are those asthma exacerbations associated with cold temperatures, we examined only the associations with maximum daily temperature. Significant associations in this database were determined using the statistical analysis tools contained in the Stata [19] statistical software package developed by Stata Corporation of College Station, TX. Our null hypotheses were that there were no significant associations of ozone, particulates, aeroallergen, or socioeconomic status with pediatric asthma-related ED visits.

Results

Same-Day Effects

For ED visits, only a slight association was found with ozone effects for the 1-17 yrs age group. However, the 5-12 yrs age group showed a much stronger association (Table 1), with a 3.2% increase in visits indicated for an increase of 0.01 ppm in ozone concentration (95% Confidence Interval, or CI, = 1.4-5.0). Significant effects of tree pollen and ambient temperature were also seen for the 5-12 yrs age group. For hospital admissions from ED visits, the analysis was significant for the 1-17 yrs age group (Table 2), with a 0.01-ppm increase in ozone being associated with a 4.5% increase in asthma-related ED admissions (95% CI = 0.6-8.5). The 5-12 yrs age group showed even greater ozone impacts (Table 2), with a 0.01-ppm increase in ozone concentration being associated with an 8.3% increase in asthma-related ED admissions (95% CI = 2.6-14.4). Weed pollen and ambient temperature also showed significant associations with ED admissions for both the 5-12 yrs and 1-17 yrs age groups. Other age groups did not show significant impacts from ozone, and no significant associations of PM_{2.5} were seen over this three-year period.

Lagged Day Effects

We also looked for associations of ozone and PM_{2.5} concentrations with ED visits and admissions up to 4 days later. These lagged effects were examined in separate models and are shown in Table 3. For ED visits, the 1-17 yrs age group showed significant associations only with grass pollen. An increase in grass pollen of 10 grains m⁻³ was associated with a 2.6% increase in asthma-related ED visits (95% CI = 0.3-5). The 5-12 yrs age group showed significant lagged associations with ozone, tree pollen, and grass pollen. A 0.01 ppm increase in ozone was associated with a

1.9% increase (95% CI = 0.2-3.7) after 1 day, a 2.3% increase (95% CI = 0.6-4.1) after 2 days, a 2.8% increase (95% CI = 1.1-4.6) after 3 days, and a 3.3% increase (95% CI = 1.6-5.1) after 4 days, for asthma-related ED visits.

A 100 grains m^{-3} increase in tree pollen was associated with a 1.2% increase (95% CI = 0.4-2.1) after 1 day, a 1.5% increase (95% CI = 0.7-2.4) after 2 days, a 1.1% increase (95% CI = 0.3-2) after 3 days, and a 1.1% increase (95% CI = 0.2-2) after 4 days, for asthma-related ED visits for 5-12 yr olds. Grass pollen also showed a progressively increasing effect when lagged from 1 to 3 days, and the association diminished for greater lags. Specifically, a 10 grains m^{-3} increase in grass pollen was associated with a 3.5% increase (95% CI = 0.1-7.1) after 1 day, a 4.3% increase (0.9-7.9) after 2 days, a 5.5% increase (95% CI = 2.2-9) after 3 days, and a 3.1 % increase (95% CI = -0.5-6.8) after 4 days, for asthma-related ED visits.

Covariate Interactions

We analyzed the data for interactions among ozone, PM_{2.5}, and different types of pollen and mold spore counts. However, no significant interactions were identified, so these results are not shown. This result may be due to the small number of high pollen days in the 36 months of data.

Socioeconomic Status

As mentioned earlier, one of the goals of the EPHTP is to identify areas and populations that may be at greater risk of health effects of pollution [8]. . Because the ozone and PM_{2.5} concentrations did not vary appreciably by location within DC, we compared the geospatial information of residence in zip codes of low socioeconomic status with other zip codes. The asthma-related ED visit rate for each

zip code was determined by dividing the number of asthma-related ED visits from a zip code by the pediatric population of that zip code. When this ED visit rate was plotted against the percentage of children living in households with income below poverty level, a logarithmic relationship was observed (Fig. 2). Similar results were obtained when ED admission rate was used instead of visit rate. Note that there is a strong, nearly linear relationship as the percent below poverty rises to nearly 5%. Above 5%, the relationship becomes curvilinear. Above about 30%, the relationship becomes almost linear again but with a very slow rise. We believe this is the first time such a logarithmic relationship has been observed in asthma-related ED visit and admission rates. These logarithmic correlations are very high (R^2 close to unity), indicating that this relationship is highly significant. Fig. 2 shows both asthma-related ED visit and admission rates increased with the percentage of the pediatric population below poverty level, suggesting that over-utilization of hospital ED for routine primary care of asthma exacerbation was not totally to blame for the positive slope of these curves. Together, the curves in Fig. 2 suggest a real increased risk of acute asthma exacerbations as the percent of children below poverty level increases.

Based on the curves in Fig. 2, we defined “high child poverty” zip codes as those for which the US census data indicated that at least 30% of children under 18 years old lived in households below the poverty level. Based on the 2000 US census, ten DC zip codes met this criterion. The map in Fig. 3 indicates these ten zip codes with cross-hatching. The colors correspond to the asthma-related ED visit rates in the legend. Note that these higher poverty zip codes tend to have higher ED visit rates than other zip codes.

It is important to emphasize the contextual and ecologic nature of this study. Given the patient's residence zip code, we knew neither which patients within a zip code were in households classified as below the poverty level, nor which patients had sufficient exposure to outdoor air pollutants. With these stipulations, we estimated zip code-level relative risks of acute asthma exacerbation for pediatric residents of high child-poverty zip codes versus those from all DC zip codes. For each relative risk estimate, the numerator was the count of daily ED visits from each residence zip code, and the denominator was a US Census population estimate for the year 2000 for that zip code. Both numerator and denominator were age-group specific (e.g., counts of only 5-12 yr olds used in both numerator and denominator). Table 4 presents these relative risk estimates with upper and lower 95 % confidence limits showing that all estimates are statistically significant. These confidence intervals were calculated using the method of Katz et al [20]. These aggregated calculations support the hypothesis that children of each age group have increased relative risk of requiring an ED visit for asthma exacerbation if their residence is in a high-poverty zip code. The second part of Table 4 presents relative risk estimates in which the numerators were daily counts of only those ED visits that resulted in hospital admission. These tables suggest that, once a child visits an ED for an asthma exacerbation, there is an increased risk of that child needing admission if that child's residence is in a high child poverty zip code. These results are consistent with the curves seen in Fig. 2. Therefore, our results appear to corroborate the hypothesis that, relative to DC children in general, children from high poverty zip codes visit the hospital ED with more severe asthma symptoms, making admission more likely.

For 5-12 yr olds living in these high poverty zip codes, a 0.01 ppm increase in ozone concentration was associated with a 6.8% increase in daily ED admissions (95% CI = 0.5-13.5, $p < 0.033$). However, for this same age group from all zip codes, the same increase in ozone concentration resulted in an 8.3% increase (Table 2). For all the different age groups, our data did not reveal significantly greater associations between ozone and asthma-related visits or admissions for those children in high poverty zip codes versus all zip codes.

Discussion

We performed Poisson regression analyses of time series of daily counts of pediatric asthma-related hospital ED visits and admissions to seek associations with environmental risk factors. The dataset covered the period from October 1, 2001, through September 30, 2004, and included only DC residents. Separate analyses were done to analyze environmental effects on more serious cases by limiting the subset of counts to those for which the visits resulted in hospital admissions. The risk factors included in the analysis were daily concentrations of ozone, PM_{2.5}, and other environmental factors such as pollen.

During the study period, we found no statistically significant effect of PM_{2.5} on pediatric asthma-related ED visits or admissions. This is likely related to the relatively low levels of PM_{2.5} during our study period (daily PM_{2.5} never reached Code Red levels). While other studies have shown significant associations between particulates and pediatric ED asthma admissions [21], Fusco et al [22] also found no significant relationship between pediatric asthma and particulates. These different results may be related to different particulate size distributions and concentrations

among different locations, different types of sources at those locations (e.g., smokestack industries, diesel vs. gasoline vehicular traffic), and different periods of time.

The 8-hour daily maximum ozone concentrations reached Code Red levels on only five days during the study period. We found some significant associations of ozone and tree pollen on counts of ED visits and admissions for the 1-17 yrs age group, and the largest associations were found on the 5-12 yrs age group. We also calculated significant but smaller associations of ED visits with grass pollen and temperature. Significant ozone associations were seen in the 5-12 yrs age group even when the daily ED visit data were lagged by up to 4 days from the ozone data. Interestingly, regression using grass pollen data did not show a significant same-day effect, but for daily ED visits, this effect became significant for a one-day lag and became stronger as the lag increased to three days before dropping to non-significant levels after three days. Tobias et al [23] noted similar lags of 2 to 3 days, depending on the type of pollen.

The most significant effects of ozone on asthma-related ED visits and admissions were found in the 5-12 yrs age group. Compared to adults, children have a higher alveolar ventilation relative to body mass, as well as a higher peripheral airways resistance, resulting in children having a greater risk of adverse ventilatory effects. As children grow, respiratory rate decreases from about 22 breaths per minute at age 4 to around 14 breaths per minute by age 16 years [24], thereby approaching normal values for adults. Because the 5-12 yrs age group represents school-age children, it is possible that this includes a significant number of children who spend a larger amount

of time outside than the 1-4 yrs age group. If so, then the 5-12 year old children would be at greater risk for effects of outdoor pollutants. Significant associations found in 5-12 yrs age group for tree and weed pollen concentrations also suggest that this age group may be at greater risk for outdoor exposures triggering reactive airway conditions due to greater time spent outdoors. Ziska et al. [25] observed that ragweed plants in urban locations, where carbon dioxide levels are high, produced more pollen than ragweed plants in rural areas where amounts of carbon dioxide were low. Our result that weed pollen was significantly associated with ED admissions may be related to the fact that ragweed is the primary component of weed pollen and is known to produce huge amounts of pollen that are considered among the most highly allergenic of all pollen.

When averaged over the 36 months of this study, the annual rates of asthma-related ED visits (visits divided by the year 2000 DC census for that age group) were 0.070, 0.033, 0.022, and 0.029 for the age groups 1-4 yrs, 5-12 yrs, 13-17 yrs, and 1-17 yrs, respectively. It is noteworthy that the most significant associations of asthma exacerbations with outdoor environmental factors were not seen in the age group with the highest rate of asthma-related ED visits, the 1-4 yr olds. This may be related to less time spent outdoors and to the clinical difficulty in accurately diagnosing asthma in this age group. Because of the developmental factors mentioned above, children of ages 1-4 yrs may experience significant wheezing and shortness of breath due to allergens or other factors, but these effects often wane as the child develops. Therefore, there is some debate whether these children should have been diagnosed with asthma as opposed to a more temporary reactive airway condition. It is thus

possible that diagnoses of asthma in this age group may not represent its true incidence.

An even more interesting finding resulted when we compared pediatric populations from zip codes in which more than 30% of the children lived below the poverty level (defined as “high child poverty zip codes”) with all other zip codes within DC. Fig. 2 reveals a significant logarithmic relationship between the percentage of children in a zip code living below the poverty level and both asthma-related ED visits and admissions. This figure also shows that the ratio of ED admission to visit rates tends to increase as the percent zip code population below poverty increases. At and above about 30%, the rate of increase in both asthma-related ED visit and admission rates levels off. Increased rates for both asthma-related ED visits and admissions were seen in residents of high child poverty zip codes compared with other zip codes. Table 4 suggests a real increased relative risk of acute asthma exacerbations that require hospital admission among the pediatric residents of high-poverty zip codes. Possible explanations for this observation include postponing primary care until symptoms become severe, discrepancies in outdoor air exposure (e.g., lack of air conditioning), discrepancies in indoor air quality [26], and discrepancies in health care access and availability [27,28,29]. For example, Levy et al [27] found that only 36% of asthmatic children in three public housing developments in Boston had been prescribed any daily controller medication.

It is necessary to emphasize that this study has its limitations. Because of the data available, this investigation is limited to a contextual study involving measured outdoor risk factors for a population with a spatial resolution no finer than the zip

code level. Krieger et al [30] have shown that inhomogeneities in socio-economic status within a zip code may have statistical impacts that are significant for some health outcomes, such as cancer incidence, and not for others, such as cancer mortality. Our asthma data did not include census tract information. Depending on the homogeneity of DC zip codes for socio-economic status, analysis at the census tract level may be more informative than at the zip code level to which this study was constrained. Another limitation was that the period of our study was only 36 months, while datasets in some similar studies cover much longer time intervals. The spatial analysis of environmental effects was limited in resolution because ozone and PM2.5 data were available from only three stations within DC during the study period. The fact that there were only five Code Red ozone days and no Code Red PM2.5 days during this study suggests limited opportunities to observe relationships between these pollutants and asthma-related ED visits, although asthma-related ED visits may also result from conditions below Code Red levels. Despite these limitations, the results of this study, corroborative of similar studies noted above, may be used for hypotheses and designs of more comprehensive, individual-based epidemiological studies.

Conclusions

We conclude that asthmatic exacerbations, as measured by hospital ED visits and admissions, have significant short-term associations with ozone, especially among 5-12 year olds. The finding that the most significant associations were not seen in the age group with the highest number of asthma-related ED visits, the 1-4 yr olds, may be a result of the clinical difficulty in accurately diagnosing asthma in this age group.

We also found an interesting logarithmic relationship between asthma-related ED visit and admission rates and the percentage of children living below the poverty level within a zip code (Fig. 2). The curves in Fig. 2 suggest that asthmatic children in high-poverty zip codes may not be over-utilizing ED visits and that there appears to be a real increased relative risk of acute asthma exacerbations that require hospital admission among these children. Therefore, the results observed among age and poverty groups above may suggest designs for future studies on efforts to offer health education and services such as asthma maintenance therapy to families in high poverty areas.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

SMB participated in the study design and analyses, drafted the manuscript, and provided support as a meteorologist and physician. HSB designed and developed the statistical analyses. RSH coordinated implementation of the study and contributed her expertise in respiratory therapy and public health issues such as socioeconomic status. NRT participated in the spatial analysis and provided geographic information systems analyses. LDS acquired federal funding and collaborated in the study conception, design, and implementation, as well as provided support for understanding the ED data. JODC collaborated in the study conception and implementation. KD provided

support for study design and relevant public health issues including socioeconomic status. DHL collaborated in the study conception and design. All authors read and approved the final manuscript.

Acknowledgements

The authors would like to express their sincere appreciation to Susan Kosisky and her staff at the United States Army Centralized Allergen Extract Laboratory, Department of Allergy-Immunology, Walter Reed Medical Center, for providing aeroallergen data. This research is sponsored by the District of Columbia Department of Health under contract POHC-2006-C-0100. The views and conclusions contained in this document are those of the authors and should not be interpreted as necessarily representing the official policies, either expressed or implied, of the United States Government.

References

1. Gent JF, Triche EW, Holford TR, Belanger K, Bracken MB, Beckett WS, Leaderer BP: Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA* 2003, 290:1859-1867.
2. Jaffe DH, Singer ME, Rimm AA: Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. *Environ Res* 2003, 91:21-28.

3. Peden DB: Pollutants and asthma: role of air toxics. *Environ Health Persp* 2002, 110(Suppl 4):565-568.

4. National Allergy Bureau, AAAAI: US Pollen Seasons 2003, [[http://www.aaaai.org/nab/index/cfm?p=uspollen seasons](http://www.aaaai.org/nab/index/cfm?p=uspollen%20seasons)].

5. Berk JL, Lenner KA, Jr EM: Cold-induced bronchoconstriction: role of cutaneous reflexes vs. direct air way effects. *J Appl Physiol* 1987, 63:659-664.

6. Jamason PF, Kalkstein LS, Gergen PJ: A synoptic evaluation of asthma hospital admissions in New York City. *Am J Respir Crit Care Med* 1997, 156:1781-1788.

7. Davis MS, Malayer JR, L V, M RC, C ME, Williamson KK: Cold weather exercise and air way cytokine expression. *J Appl Physiol* 2005, 98:2132-2136.

8. McGeehin MA, Qualters JR, Niskar AS: National Environmental Public Health Tracking Program: bridging the information gap. *Environ Health Perspect* 2004, 112:1409-1413.

9. Hart AC, Hopkins CA (Eds): *2003 ICD9CM Expert for Hospitals, Sixth Edition*. Salt Lake City, UT: St Anthony Publishing 2003.

10. Kimes D, Levine E, Timmins S, Weiss SR, Bollinger ME, Blaisdell C: Temporal dynamics of emergency department and hospital admissions of pediatric asthmatics. *Environ Res* 2004, 94(1):7-17.

11. Gern JE, Busse WW: Association of rhinovirus infections with asthma. *Clin Microbiol Reviews* 1999, 12(1):9-18.
12. Johnston NW, Johnston SL, Duncan JM, Greene JM, Keadze T, Keith PK, Roy M, Wasserman S, Sears MR: The September epidemic of asthma exacerbations in children: a search for etiology. *J Allergy Clin Immunol* 2005, 115(1):132-138.
13. Dales RE, Schweitzer I, Toogood JH, Drouin M, Yang M, Dolovich J, Boulet J: Respiratory infections and the autumn increase in asthma morbidity. *Eur Respir J* 1996, 9:72-77.
14. Norris G, YoungPong SN, Koenig JO, Larson TV, Sheppard L, Stout JW: An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Persp* 1999, 107(6):489-493.
15. Galan I, Tobias A, Banegas JR, Aranguiz E: Short-term effects of air pollution on daily asthma emergency room admissions. *Eur Respir J* 2003, 22:802-808.
16. White MC, Cody RP, Lioy PJ: Exacerbations of childhood asthma and ozone pollution in Atlanta. *Environ Res* 1994, 65:56-68.
17. Sunyer J, Spix C, Quenel P, Ponce-de Leon A, Ponka A, Barumandzadeh T, Touloumi G, Bacharova L, Wojtyniak B, Vonk J, Bisanti L, Shwartz J, Katsouyanni

K: Urban air pollution and emergency admissions for asthma in four European cities: the APHEA Project. *Thorax* 1997, 52:760-765.

18. Norris G, Larson T, Koenig J, Claiborn C, Sheppard L, Finn D: Asthma aggravation, combustion, and stagnant air. *Thorax* 2000, 55:466-470.

19. Stata, Inc: *Stata Release 8: User's Guide*. College Station, TX: Stata Press 2003.

20. Katz D, Baptista J, Azen SP, Pike MC: Obtaining confidence intervals for the risk ratio in cohort studies. *Biometrics* 1978, 34(3):469-474.

21. Atkinson RW, Anderson HR, Sunyer J, Ayres J, Baccini M, Vonk JM, Boumghar A, Forastiere F, Forsberg B, Touloumi G, Schwartz J, Katsouyanni K: Acute effects of particulate air pollution on respiratory admissions: Results from the APHEA 2 Project. *Am J Respir Crit Care Med* 2001, 164:1860-1866.

22. Fusco D, Forastiere F, Michelozzi P, Spadea T, Ostro B, Arca M, Perucci C: Air pollution and hospital admissions for respiratory conditions in Rome, Italy. *Eur Respir J* 2001, 17:1143-1150.

23. Tobias A, Galan I, Banegas JR, Aranguiz E: Short term effects of airborne pollen concentrations on asthma epidemic. *Thorax* 2003, 58:708-710.

24. Wallis LA, Healy M, Undy MB, Maconochie I: Age related reference ranges for respiration rate and heart rate from 4 to 16 years. *Arch Dis Child* 2005, 90:1117-1121.
25. Ziska LH, Gebhard DE, Frenz DA, Faulkner S, Singer BD, Straka, JG: Cities as harbingers of climate change: Common ragweed, urbanization, and public health. *Journal of Allergy and Clinical Immunology*. 2003, 111:290-295.
26. Walker B, Stokes LD, Warren R: Environmental factors associated with asthma. *J Natl Med Assoc* 2003. 95: 152-166.
27. Levy JI, Welker-Hood LK, Clougherty JE, Dodson RE, Steinbach S, Hynes HP: Lung function, asthma symptoms, and quality of life for children in public housing in Boston: a case-series analysis. *Environ Health: A Global Access Science Source* 2004. 3:13, doi:10.1186/1476-069X-3-13.
28. Miller JE: The effects of race/ethnicity and income on early childhood asthma prevalence and health care use. *Am J Public Health* 2000. 90(3):428-430.
29. Pollack HA, Dombkowski KJ, Zimmerman JB, Davis MM, Cowan AE, Wheeler JR, Hillemeier AC, Freed GL: Emergency department use among Michigan children with special health care needs: an introductory study. *Health Services Research* 2004. 39(3): 665-692.

30. Krieger N, Chen J, Waterman P, Soobader M-J, Subramanian S, Carson R:
Geocoding and monitoring of US socioeconomic inequalities in mortality and
cancer incidence: Does the choice of area-based measure and geographic level
matter? *Am J Epidemiol* 2002. 156: 471-482.

Figures Legends

Figure 1 – Daily ED Visit Residuals

Daily time series plot of the residuals of daily pediatric asthma ED visits for 1-17 yr olds (observed minus the spline predicted).

Figure 2 - ED Visit and Admission Rates for Ages 1-17 Yrs vs Percent below Poverty Level in Zip Code

Upper plot shows the asthma-related ED admit rate and the lower plot shows the asthma-related ED visit rate for children ages 1-17 years versus the percent of children living below poverty levels for zip codes within DC.

Figure 3 - Asthma-related ED Visit Rates by Zip Code for FY2001-2003

Asthma-related ED visit rates averaged over fiscal years 2001-2003 (October 2001-September 2004) for ages 1-17 years for zip codes within DC. The hatched zip codes are those for which the 2000 US census determined that 30% or more of the children lived below poverty levels. The grey zips are those for which there is little or no pediatric population showing the asthma-related ED admissions rate for children ages 1-17 years versus the percent of children living below poverty levels for zip codes within DC.

Tables

Table 1 - Risk Factor Associations with ED Visits for Ages 5-12 yrs

The numbers in parenthesis represent the lower and upper 95% confidence limits for the percent change in visits.

Risk Factor Increase	Risk Factor	Mean % Change in Daily Visits per Risk Factor Increase	T-test p-value
0.01 ppm	Ozone	3.2 (1.4, 5)	0.000
1 $\mu\text{g m}^{-3}$	PM2.5	-0.2 (-0.6, 0.2)	0.331
1000 spores m^{-3}	Mold Count	-0.2 (-1.1, 0.7)	0.707
10 grains m^{-3}	Weed Pollen	-2.9 (-6.8, 1.1)	0.148
100 grains m^{-3}	Tree Pollen	1.8 (0.9, 2.6)	0.000
10 grains m^{-3}	Grass Pollen	1.6 (-2, 5.4)	0.378
1 $^{\circ}\text{F}$	Temperature	0.2 (0.1, 0.4)	0.007

Table 2 - Risk Factor Associations with ED Admissions for Ages 1-17 yrs and for Ages 5-12 yrs

The numbers in parenthesis represent the lower and upper 95 % confidence limits for the percent change in admissions. The upper table is for ages 1-17 yrs and the lower table is for ages 5-12 yrs.

Risk Factor Unit Increase	Risk Factor	Mean % Change in Daily Visits per Risk Factor Unit Increase	T-test p-value
0.01 ppm	Ozone	4.5 (0.6, 8.5)	0.023
1 $\mu\text{g m}^{-3}$	PM2.5	0.2 (-0.6, 1.1)	0.548
1000 spores m^{-3}	Mold Count	1.1 (-0.8, 3)	0.250
10 grains m^{-3}	Weed Pollen	7.7 (0.7, 15.2)	0.029
100 grains m^{-3}	Tree Pollen	1.5 (-0.3, 3.3)	0.105
10 grains m^{-3}	Grass Pollen	0.7 (-7.4, 9.5)	0.874
1 °F	Temperature	0.5 (0.1, 0.8)	0.018

Risk Factor Unit Increase	Risk Factor	Mean % Change in Daily Visits per Risk Factor Unit Increase	T-test p-value
0.01 ppm	Ozone	8.3 (2.6, 14.4)	0.004
1 $\mu\text{g m}^{-3}$	PM2.5	-0.4 (-1.6, 0.8)	0.479
1000 spores m^{-3}	Mold Count	2 (-0.6, 4.6)	0.140
10 grains m^{-3}	Weed Pollen	10 (0.5, 20.5)	0.039
100 grains m^{-3}	Tree Pollen	1.8 (-0.8, 4.4)	0.173
10 grains m^{-3}	Grass Pollen	1.8 (-9.4, 14.5)	0.764
1 °F	Temperature	0.9 (0.4, 1.5)	0.001

Table 3 – Lagged Day Associations of ED Visits with Ozone and Pollen.

The top and bottom tables are for the 1-17 yr old and 5-12 yr old age groups, respectively. The mean % change in daily visits in these tables is per unit 0.01 ppm increase in ozone, 100 grains m⁻³ increase in tree pollen, and 10 grains m⁻³ increase in grass pollen.

Visits 1-17 yrs

Lag (days)	Ozone		Tree Pollen		Grass Pollen	
	Mean % Change in Daily Visits per Unit	T-test p-value	Mean % Change in Daily Visits per Unit	T-test p-value	Mean % Change in Daily Visits per Unit	T-test p-value
0	1 (-0.1, 2.1)	0.078	0.5 (0, 1.1)	0.068	0.7 (-1.8, 3.1)	0.602
1	0.1 (-1, 1.2)	0.879	0.4 (-0.2, 1)	0.202	1.9 (-0.4, 4.3)	0.112
2	-0.2 (-1.3, 0.9)	0.724	0.5 (-0.1, 1.1)	0.105	2.3 (-0.1, 4.7)	0.056
3	0.3 (-0.8, 1.5)	0.572	0.2 (-0.4, 0.8)	0.492	2.6 (0.3, 5)	0.030
4	0.2 (-0.9, 1.3)	0.754	0.2 (-0.4, 0.9)	0.441	-0.4 (-2.9, 2.3)	0.787

Visits 5-12 yrs

Lag (days)	Ozone		Tree Pollen		Grass Pollen	
	Mean % Change in Daily Visits per Unit	T-test p-value	Mean % Change in Daily Visits per Unit	T-test p-value	Mean % Change in Daily Visits per Unit	T-test p-value
0	3.2 (1.4, 5)	0.000	1.8 (0.9, 2.6)	0.0001	1.6 (-2, 5.4)	0.378
1	1.9 (0.2, 3.7)	0.030	1.2 (0.4, 2.1)	0.005	3.5 (0.1, 7.1)	0.046
2	2.3 (0.6, 4.1)	0.009	1.5 (0.7, 2.4)	0.0002	4.3 (0.9, 7.9)	0.013
3	2.8 (1.1, 4.6)	0.002	1.1 (0.3, 2)	0.01	5.5 (2.2, 9)	0.001
4	3.3 (1.6, 5.1)	0.000	1.1 (0.2, 2)	0.016	3.1 (-0.5, 6.8)	0.088

Table 4 - Relative Risk and Conditional Relative Risk for Asthma-related ED Visits and Admissions for High Poverty vs Other Zip Codes

The numbers in parenthesis represent the lower and upper 95 % confidence limits for the percent change in visits and admissions.

Age Groups	Outcome	Relative Risk for asthma-related ED visits/admissions for high poverty vs. others
1-17	visits	1.45 (1.39 - 1.51)
	admissions	1.75 (1.51 - 2.03)
1-4	visits	1.4 (1.32 - 1.48)
	admissions	1.73 (1.35 - 2.21)
5-12	visits	1.38 (1.3 - 1.47)
	admissions	1.77 (1.42 - 2.2)
13-17	visits	1.71 (1.54 - 1.9)
	admissions	1.73 (1.19 - 2.52)

Age Group	Given an asthma-related ED visit, the Relative Risk of being admitted: high poverty vs. others
1-17	1.2 (1.05 - 1.37)
1-4	1.23 (1 - 1.52)
5-12	1.18 (0.98 - 1.43)
13-17	1.17 (0.85 - 1.62)

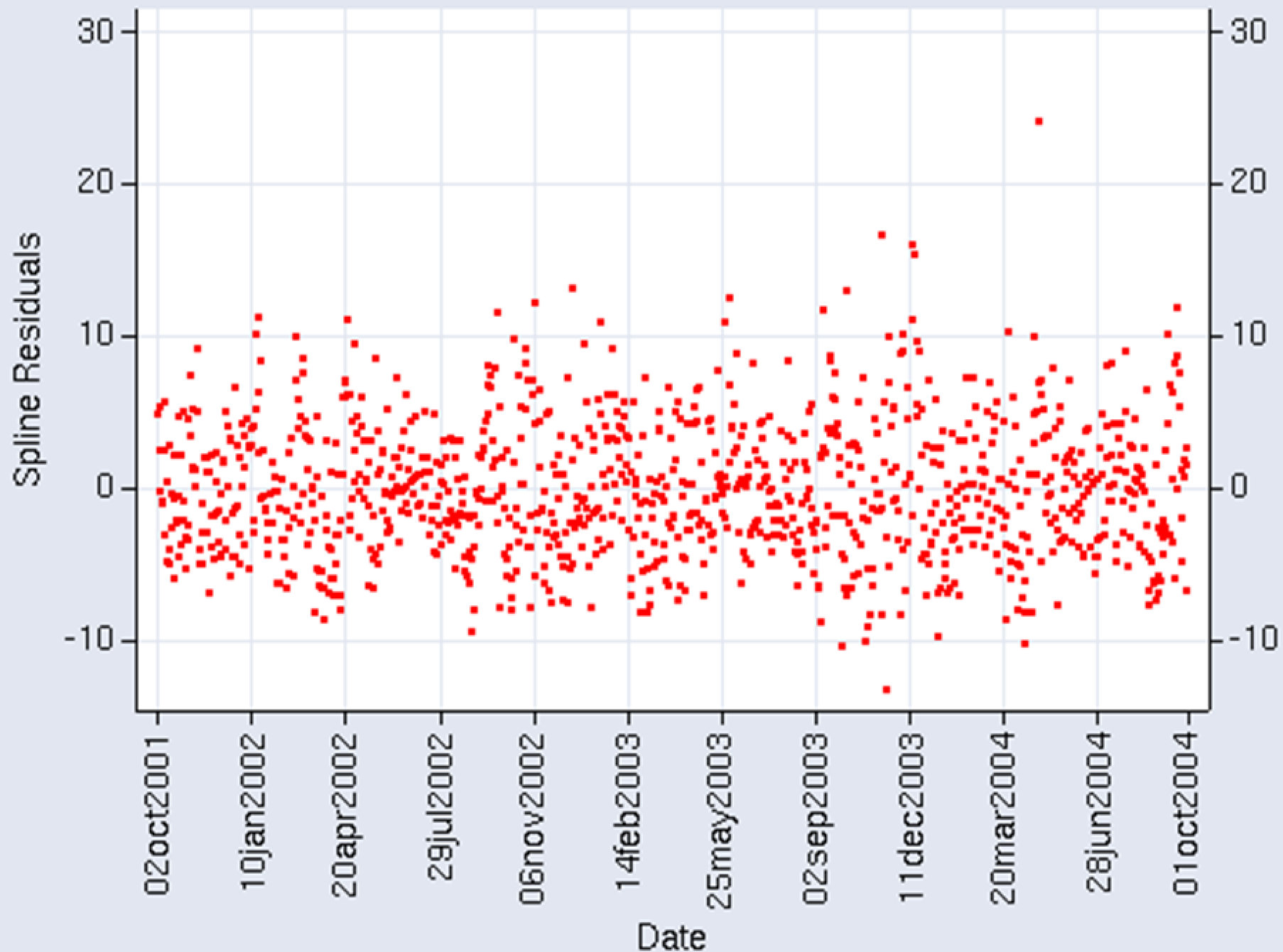
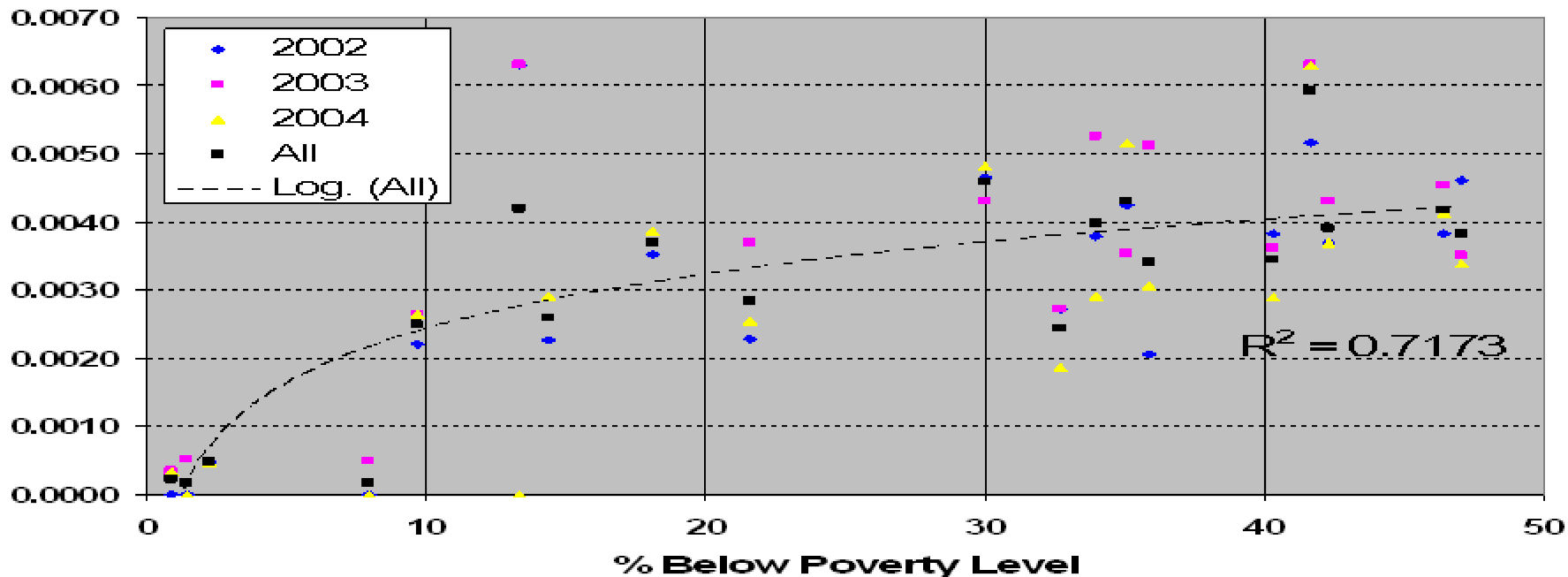


Figure 1

ED Admissions: Age Group 1-17



ED Visits: Age Group 1-17

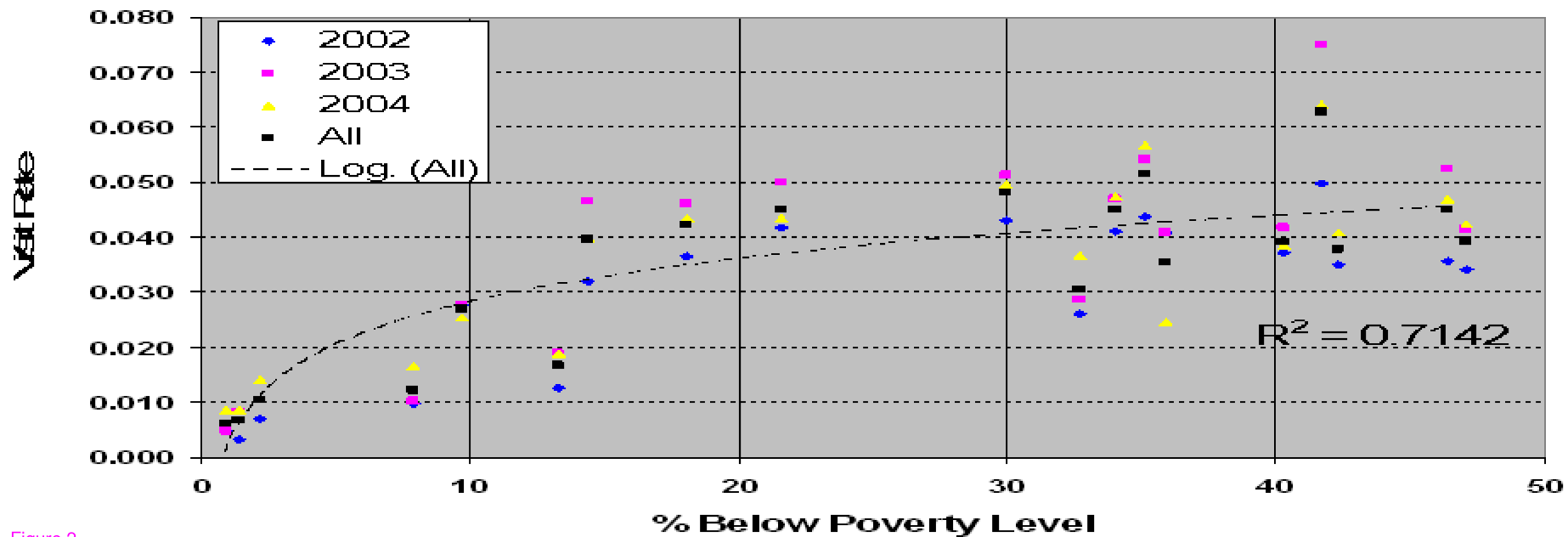


Figure 2

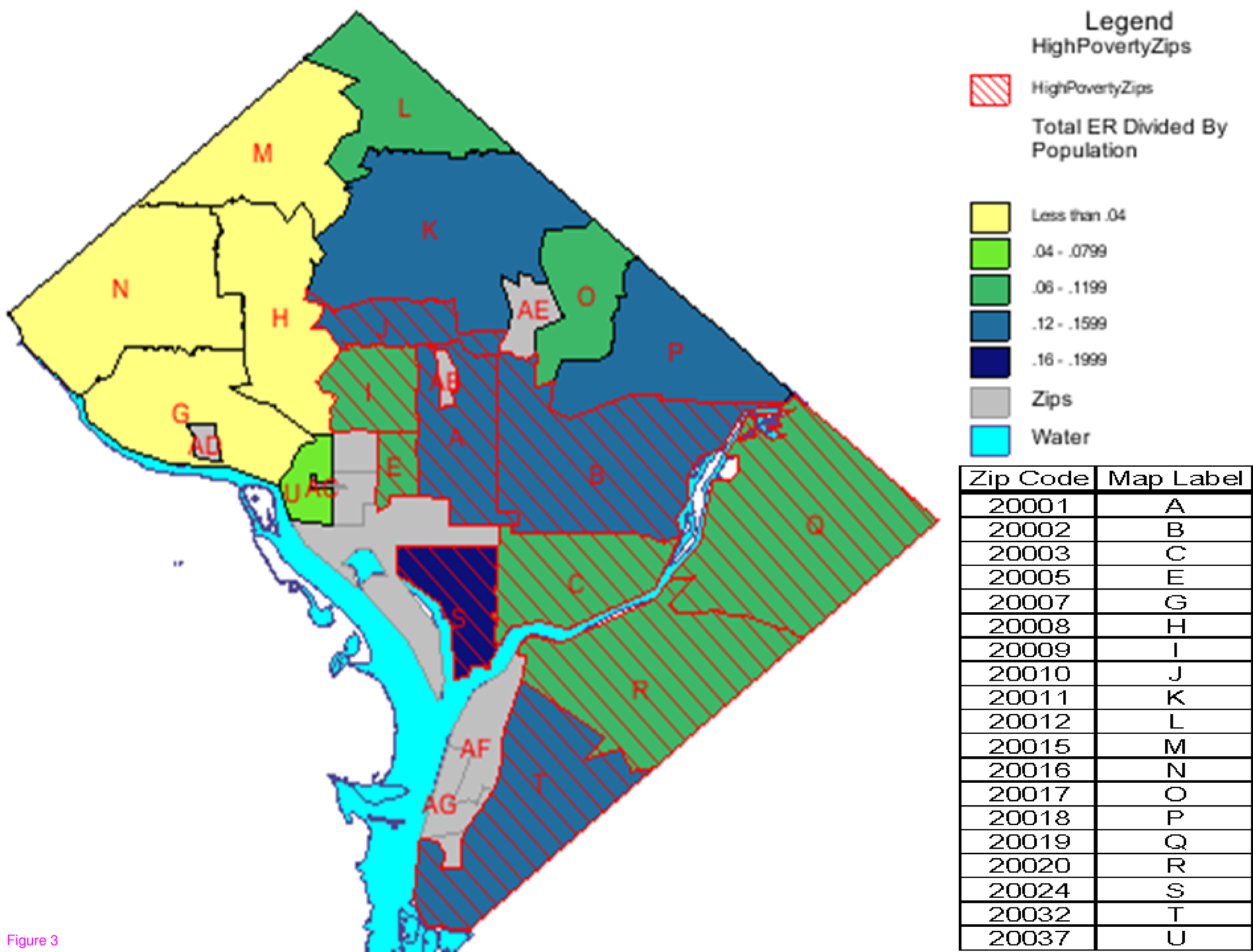


Figure 3