Geographic Clustering of Adult Asthma Hospitalization and Residential Exposure to Pollution at a United States—Canada Border Crossing

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Buffalo's West Side contains the Peace Bridge Complex (PBC), the busiest US-Canada crossing point for commercial traffic in the eastern United States. Over the past decade, there has been a steady increase in commercial traffic primarily owing to the increased trade resulting from the North American Free Trade Agreement (NAFTA). Increased asthma prevalence and health care use resulting from traffic-related pollution around the US-Canada border crossing point has been a major focus of previous studies.²⁻⁷ These studies have provided evidence supporting the hypothesis that there is a high respiratory burden among residents living in close proximity to the NAFTA corridor.

The PBC is located in a densely populated, urban, and predominantly minority community. Although the increased traffic through the PBC has brought economic prosperity to the United States, Canada, and Mexico, the health effects of NAFTA trade associated with traffic pollution in the communities along the US-Canada border have not been extensively studied. A recent report on health effects in NAFTA corridors⁸ concluded that commercial traffic contributes significantly to air pollution in all NAFTA corridors, particularly to nitrogen dioxide and particulate matter, but the report did not include the PBC. Diesel exhaust particles have been shown to worsen respiratory symptoms and to lead to deterioration in lung function, especially among individuals with preexisting chronic conditions such as asthma. $^{9-12}$

Previous studies, such as those by Schwartz and Dockery, ^{9,10} Dockery et al., ¹³ Schwartz, ^{14,15} Schwartz and Morris, ¹⁶ Styer et al., ¹⁷ Norris et al., ¹⁸ Wong et al., ¹⁹ Samet et al., ²⁰ and Peters et al., ²¹ have reported an association between cardiopulmonary morbidity and mortality and exposure to respirable particulate matter. In a groundbreaking study,

Objectives. We conducted a case—control study of adulthood asthma and point-source respirable particulate air pollution with asthma-diagnosed case patients (n=3717) and gastroenteritis-diagnosed control patients (n=4129) to determine effects of particulate air pollution on public health.

Methods. We used hospitalization data from Buffalo, NY, neighborhoods for a 5-year period (1996 through 2000), geographic information systems techniques, the Diggle method, and statistical analysis to compare the locations of case patients and control patients in terms of proximity to different known pollution sources in the study area.

Results. We found a clustering of asthma cases in close proximity to the Peace Bridge Complex and the freeways and a dose–response relationship indicating a decreased risk of asthma prevalence the farther an individual resides from the source of exposure.

Conclusions. These findings provide a basis for the development of new hypotheses relating to the spatial distribution of asthma prevalence and morbidity in this community. (Am J Public Health. 2004;94:1250–1257)

the United States Environmental Protection Agency (EPA) concluded that long-term inhalation exposure to diesel exhaust particles is likely to pose a lung damage threat, including a risk for cancer, to humans.²² The study further noted that short-term exposures can cause irritation and inflammatory symptoms of a transient nature.²²

In previous studies, we reported an increased risk of asthma among residents living along the US-Canada border crossing and the major roadways feeding it. An association between increases in commercial traffic across the PBC and increases in health care use for asthma was reported in Lwebuga-Mukasa et al.²³ A sharp decrease in traffic after the September 11, 2001, World Trade Center terrorist attacks was associated with a decline in health care use for respiratory illnesses, which rebounded when traffic recovered.⁷ A house-to-house survey of 214 homes in the area (from 1996-1997) and another of 1644 homes (in 2002) found households in close proximity to the PBC to have asthma prevalence rates that were double those of households located on Buffalo's East Side

(J.S. Lwebuga-Mukasa, MD, PhD, unpublished data). These observations indicate that increased traffic on Buffalo's West Side may be associated not only with asthma exacerbations but also with increased prevalence in the community.

Cumulative evidence is also emerging from studies reviewed in Peterson and Saxon,²⁴ Kane et al., 25 Lwebuga-Mukasa and Dunn-Georgiou, 2,3,26 Lwebuga-Mukasa and Pszonak,⁴ and Lin et al.⁵ showing a higher respiratory burden on the communities residing in Buffalo's West Side than on the surrounding communities. However, there is little focus on the spatial relationships between increased risk of asthma and environmental exposure. In this study, we investigate the hypothesis that proximity to the major commercial routes; the PBC; and EPA-designated toxic air release sites and multiple-emission sites are associated with increased asthma risk. Characterization of environmental and human characteristics of clusters would provide a basis for identification of factors contributing to an increased asthma burden in the community and to the development of mitigation measures.

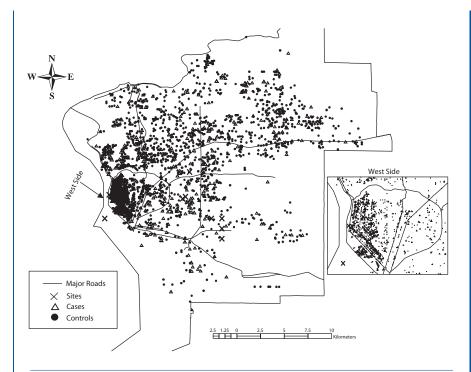


FIGURE 1—Resident locations of case patients and control patients, network of major roads, and focus sites in the study area.

METHODS

Study Area and Population

The study area covered 156 census tracts with an estimated population of 529846 people in Erie County, according to the 2000 population data obtained from the US Census Bureau.²⁷ About 40% of the study population was between 18 and 64 years of age. The study area includes inner-city areas characterized by low socioeconomic status, poor housing conditions, low education levels, high unemployment, and high percentages of minority population.²⁷ Figure 1 shows resident locations of case patients and control patients, the network of major roads, and focus sites in the study area.

Study Design

A cross-sectional study based on casecontrol data for adulthood asthma (case patients) and nonrespiratory disease (control patients) for a 5-year period (1996-2000) was conducted using hospitalization data. Case subjects (n=3717) and control subjects (n= 4129) consisted of asthma patients (Interna-

tional Classification of Diseases, 9th Revision [ICD-9] code 493)²⁸ and gastroenteritis patients (ICD-9 code 558), respectively, residing in Buffalo neighborhoods during the same period. The study was based on a database that was obtained from Kaleida Health Systems, a major provider of health care in western New York. The data were available at individual and zip code levels. Data with residential addresses was separated, processed, and geocoded for use in spatial analysis and a geographic information systems (GIS). At the census tract level, we analyzed cases patients per population and identified local asthma clusters. At the zip code level, a comparison of case patients and control patients was conducted to identify which zip codes had an elevated risk of asthma. We also conducted a field assessment of the previously identified focus sites.

Data Categories

The 3 data categories analyzed in this study are as follows: hospitalization and outpatient visits for asthma, 1996 through 2000; hospitalization and outpatient visits for gastroenteritis, 1996 through 2000; and focus sites. The data categories are described in detail in the following sections.

Hospitalization and outpatient visits for asthma, 1996 through 2000. The hospitalization and outpatient visits data were based on the patient records of kept by Millard Fillmore Health Hospitals, which are divisions of the Kaleida Health System, and covered admissions from January 1996 to August 2000. Some of these data have been published in Lwebuga-Mukasa et al.6 and in Oyana and Lwebuga-Mukasa.³⁰ The records contained the residential addresses of patients and their insurance status. The data were vital for the identification of spatial relationships between case address locations and polluting sources.

Hospitalization and outpatient visits for gastroenteritis, 1996 through 2000. Data on hospitalization and outpatient visits for gastroenteritis were obtained from the same source for the same time period. The database contained information on the case patients, including the patient's address and insurance status. The case patients were also categorized into 2 groups: clinical and emergency department cases. Gastroenteritis was used as the control disease. Only cases detected from 1996 through 2000 among residents of the study area were included in the case-control study.

Focus sites. Focus sites were previously obtained from the EPA Web site (http://www/ epa.gov/enviro/index.html) and mapped as point data, as shown in Figure 1. Three focus sites identified in Oyana and Lwebuga-Mukasa²⁹ were of primary interest, given their statistically significant association with increased risk for asthma, and 10 additional sites were also included. Six of these additional sites were test sites used in the validation of the model. The test sites were chosen randomly, outside of the statistically significant distance bands. However, the 13 focus sites used in this study do not constitute an exhaustive list of possible exposure sites in the study area.

We conducted a preliminary field assessment of the 3 sites (Birge Company, Ogrady Winnifred Silver, and Marnap Industries) to further our understanding of their surroundings in the study area. A working assumption was developed after finding out that 2 of the 3 focus

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sites had gone out of business, thereby attracting new business opportunities and developments in their original locations. We agreed that the focus sites represented air pollution in the preceding decade, when exposures might have occurred. This assumption seems reasonable for air pollution sources that have been in operation since the 1980s and whose dispersal is mediated by transport mechanisms (e.g., prevailing winds) that have not changed a great deal in the past 10 to 20 years.

Three additional sites (Miken Company, Nabisco Company, and Harrison Radiator) that are currently in operation and that have active licenses were obtained from the Department of Environmental Conservation, Buffalo. We also conducted a preliminary field assessment of these 3 sites that confirmed that the sites were functional.

Analytical Techniques

Rigorous analytical methods were applied to the case–control data to study whether there is an association between increased risk of asthma and pollution sources. The Diggle method was applied to test disease clustering of asthma around the focus sites. 30,31

The Diggle method is a focused cluster-detection approach appropriate for handling spatial data at the individual level. 31,32 The method compares the spatial pattern of case locations with the spatial pattern of control locations; for instance, using a more common control disease. The control acts as a null model of no clustering and normally reflects the spatial pattern of the population-at-risk. The test is based on maximizing the likelihood of the sample of case patients and control patients, which in turn is based on an exponential decline in risk as the squared distance from the source increases.

GIS techniques combined with statistical analysis were used to compare odds ratios (ORs) for the location of case patients and control patients in relation to proximity to different pollution sources in the study area. The choice of these analytical techniques was based on their wide applications in studying patterns of disease, prevalence, health care use, and incidence. These techniques have also been widely popularized by the development of the ClusterSeer³² (TerraSeer, Inc, Ann Arbor, Mich) and ARCGIS³³ (Environ-

mental Systems Research Institute [ESRI], Inc, Redlands, Calif) software packages, which handle large volumes of geographical data.

ClusterSeer Version 1.1.4 (TerraSeer);
ArcStreet USA, ARCGIS 8.1.2, and
ARCVIEW 8.1 (ESRI); and Microsoft Excel
(Microsoft, Inc, Redmond, Wash) software
packages were used in spatial analysis, GIS
mapping, and data analysis. All of the data
were compiled and analyzed at the Center
for Asthma and Environmental Exposure,
Kaleida Health Buffalo General Division,
University at Buffalo School of Medicine and
Biomedical Sciences.

The data were also loaded into ARCStreet USA to match the physical addresses with geographical latitudes and longitudes. ARC-Street USA contains the most up-to-date street addresses in the United States. Address matching was based on the Dynamap/ Zip+4 Centroids and Correspondences Files (Geographic Data Technology, Inc, Lebanon, NH). Geographic Data Technology provided this comprehensive street database to ESRI. Case patients' and control patients' addresses were mapped as point data. We had 2340 and 2571 case and control patients, respectively, with matched addresses. We obtained an accuracy level of over 90% during the address-matching exercise. The geocoded data were processed in ARCGIS 8.1.2 for further spatial analysis.

It was assumed that those who lived within 1 km of the emission sites and busily traveled roadways were exposed to vehicle exhaust fumes and pollutants from suspected sources of pollution, and that those living farther away (>2 km) were assumed to be unexposed. Rijnders et al.³⁴ recommended that variables such as degree of urbanization, traffic density, and distance to a nearby highway or any potential pollution source can be used to estimate exposure to traffic-related air pollution. Milligan et al.³⁵ also used a distance of more than 2 km in their study to estimate exposure resulting from traffic-related air pollution.

Epidemiological methods based on ORs and 95% confidence intervals were used to compute the spatial risk relationships between case patients and control patients (using a significance level of $P \le .05$). A 2 \times 2 table analysis was conducted to demonstrate the rela-

tionship between 2 dichotomous or binary variables (exposed and unexposed groups).

RESULTS

Case-Control Demographics

In the Kaleida database, there were 3717 patients hospitalized because of asthma. There were 6265 hospital discharges for asthma during the period between 1996 and 2000. The majority (80%) of the patients were adults (aged 17–64 years). Thirty-two percent were from the city of Buffalo, 3.2% were from the town of Amherst, 3.1% were from Williamsville, and 61.7% were from other places. There was a notable increase in hospital admission between 1996 and 2000, especially in zip codes 14201 and 14213.

In the Kaleida database, there were 4129 patients hospitalized because of a nonrespiratory disease—gastroenteritis. Inpatients constituted 3.9% of the patients, and the remaining 96.1% were outpatients. Emergency department and clinic patients constituted 36.4% and 40.3% of the total, respectively. The number of patients admitted with gastroenteritis remained constant over this period. Annual admissions ranged from 800 to 829, with an average of 826 patients admitted annually from 1996 to 2000. Zip codes 14201, 14213, and 14221 contributed 44% of the patients during this period.

Table 1 lists the odds ratios from the casecontrol study at the zip code level between 1996 and 2000. A comparison of odds ratios by zip codes shows certain zip codes with statistically significant increased odds of having asthma, relative to nonrespiratory disease. We observed a positive association between possible exposure and outcome at the 5% significance level in zip codes 14201, 14213, 14207, and 14204. All of these zip codes that were statistically significant with odds ratios greater than the value 1 are located on Buffalo's West Side. In zip codes 14221, 14214, 14217, 14150, and 14227, we observed a negative association between possible exposure and outcome at the 5% significance. Zip codes that were statistically significant with odds ratios less than the value 1 are located further away from the West Side of Buffalo. The remaining zip codes had statistically nonsignificant results.

TABLE 1—Exposure Based on Geographic Locations Identified at the Zip Code Level: Odds Ratios From a Case—Control Study, 1996–2000

	Case Patients (n = 3717)		Control Patients (n = 4129)			
Zip Code	% Diagnosed Asthma	Asthma Hospitalization Rates (per 10 000)	% Diagnosed Gastroenteritis	Gastroenteritis Hospitalization Rates (per 10 000)	Odds Ratio (95% Confidence Interval)	
14228	3.82	767	4.53	1010	0.84 (0.67, 1.05)	
14201	15.77	349	11.04	271	1.51 (1.32, 1.72°)	
14213	24.19	275	18.16	229	1.44 (1.29, 1.61 ^a)	
14203	0.43	129	0.34	113	1.27 (0.61, 2.61)	
14068	1.67	114	1.99	150	0.84 (0.60, 1.17)	
14026	0.08	109	0.15	218	0.53 (0.14, 2.08)	
14222	3.55	105	2.88	95	1.24 (0.96, 1.60)	
14207	6.54	101	5.01	86	1.33 (1.10, 1.61 ^a)	
14209	2.18	89	2.49	113	0.87 (0.65, 1.17)	
14216	4.20	63	4.94	82	0.84 (0.68, 1.04)	
14204	1.75	58	0.97	35	1.82 (1.22, 2.72 ^a)	
14226	4.95	57	5.55	71	0.89 (0.73, 1.08)	
14202	0.43	56	0.53	78	0.81 (0.43, 1.54)	
14221	8.18	55	14.48	109	0.53 (0.46, 0.61 ^a)	
14208	1.32	34	1.19	34	1.11 (0.75, 1.66)	
14212	1.53	29	1.11	23	1.38 (0.93, 2.05)	
14214	1.61	28	2.25	43	0.71 (0.51, 0.98b)	
14217	1.88	27	3.39	55	0.55 (0.41, 0.73 ^b)	
14215	3.44	27	3.92	35	0.87 (0.69, 1.11)	
14223	1.91	27	2.47	39	0.77 (0.57, 1.04)	
14211	2.64	25	2.16	23	1.23 (0.92, 1.64)	
14150	3.63	25	5.74	44	0.62 (0.50, 0.77 ^b)	
14210	0.78	16	0.75	17	1.04 (0.63, 1.73)	
14227	0.91	13	1.72	28	0.52 (0.35, 0.79 ^b)	
14206	0.89	13	1.07	17	0.83 (0.53, 1.30)	
14220	0.78	10	0.58	8	1.35 (0.78, 2.33)	
14218	0.59	10	0.44	8	1.34 (0.72, 2.52)	
14219	0.35	10	0.15	4	2.34 (0.88, 6.22)	

Note. Denominators derived from population data from the 1990 US Census; case patients and control patients derived from hospitalization and outpatient visits for asthma (ICD-9 code 493) and gastroenteritis (ICD-9 code 558) from Kaleida database, 1996-2000.

Spatial Analysis of Case-Control Study

The Diggle method, as used within ClusterSeer, was applied to test the null hypothesis of no clustering of case patients in comparison with a common control disease around a focal point, at α =0.05. GIS was used to determine whether spatial associations were between emission sites, major roadways, and residential locations of case patients and control patients. Sites were defined to include the PBC, air, toxic, and multiple re-

leases, as shown in Figure 1. The busily traveled roadways were defined according to the type and volume of traffic on the basis of data from the Department of Transportation, as well as on information obtained from the residences, to include Main Street, Bailey Avenue, Niagara Street, Seneca Street, Delaware Avenue, Interstate 198, Interstate 190, and Route 33 (Figure 1).

Figure 1 shows the increased odds of having asthma among the residences living in

close proximity to the sites or interstate roadways. Overall, there was a lower risk of asthma diagnosis for adults living farther away from sites and busily traveled roadways.

We analyzed exposed and unexposed case patients and control patients living within 1 km or farther away from sites and roadways. Table 2 shows increased odds of having asthma among the residences living in close proximity to the sites or roadways—there was a higher risk of asthma diagnosis for adults who lived in close proximity to those areas.

Case-control data showed that patients living along Main Street, Bailey Avenue, Niagara Street, Seneca Street, and Interstate 190 all had increased odds of having asthma. Most of the case patients at sites had statistically significant increased odds of asthma within 0.5 km versus more than 2 km away from the roadways, but the control patients did not. Apart from Interstate 190, which had statistically significant increased odds of asthma both at 0.5 versus 2.0 km and 1 versus 2 km, 4 roadways (Main Street, Bailey Avenue, Niagara Street, and Seneca Street) were also statistically significant at 1 versus 2 km. Three roadways (Delaware Avenue, Interstate 198, and Route 33) that carry mostly automobile traffic did not seem to have much effect when case patients and control patients were compared at 0.5 or 2 km farther away (within 500 m or more than 2 km).

The highest odds ratios were observed at air release sites (stationary sources of air pollution), with OR=15.77 at 0.5 versus 2 km. Residents living at 0.5 km from air release sites have 15 times the odds of having asthma among all emission sites compared with those living more than 2 km away. The PBC had OR=4.41 at 0.5 versus 2.0 km, toxic sites had OR=0.70 at 0.5 versus 2.0 km, and multiple release sites had OR=1.93 at 0.5 versus 2.0 km.

The evaluation of increased risk of diagnosed asthma near the focus sites that were identified in Oyana and Lwebuga-Mukasa²⁹ was carried out using the Diggle method and the individual-level data. Figure 2 both shows asthma clusters identified by 2 methods using group-level data^{36,37} and shows the distribution of asthma cases per 1000 people. For this analysis, we have increased the scope of the investigation to include test and alterna-

^aPositive association between exposure and outcome at the 5% significance level.

^bNegative association between exposure and outcome at the 5% significance level.

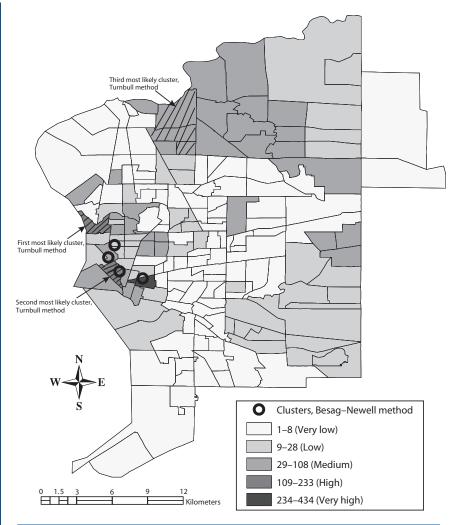


FIGURE 2—Asthma clusters identified by 2 methods and the distribution of asthma cases per 1000 people.

TABLE 2-Spatial Analysis of Case-Control Study Showing Odds Ratios

	Odds Ratios		95% Confidence Interval	
Sites	0.5 vs 2 km	1 vs 2 km	0.5 vs 2 km	1 vs 2 km
Peace Bridge Complex	4.41 ^a	0.52	3.26, 5.97	0.39, 0.70
Air release	15.77 ^a	0.91	9.93, 25.04	0.67, 1.22
Toxic release	0.70	0.42 ^b	0.40, 1.22	0.29, 0.60
Multiple release	1.93 ^a	0.56 ^b	1.60, 2.32	0.46, 0.69
Interstate 190	1.30 ^a	3.26ª	1.02, 1.66	2.67, 3.97
Interstate 198 and Route 33	0.67 ^b	0.49 ^b	0.51, 0.87	0.39, 0.63
Main St, Bailey Ave, Niagara St, and Seneca St Delaware Ave	1.11 0.65 ^a	1.36 ^b 0.96	0.95, 1.30 0.51, 0.83	1.15, 1.61 0.76, 1.21

Note. Case patients and control patients derived from hospitalization and outpatient visits for asthma (ICD-9 code 493) and gastroenteritis (ICD-9 code 558) from Kaleida database, 1996-2000.

tive sites (geographic coordinates) around the focus sites to evaluate how the P value changed. Our null hypothesis was that the case and control occurrences have the same underlying spatial distribution. The alternative hypothesis was based on case subject locations having different spatial patterns in comparison with the control locations and on the fact that the density of the case locations was higher than that of the control near the focus sites.

Model results for the 13 focus sites showed that certain geographical areas were significantly more affected by asthma than others. For instance, our study shows that the chance of achieving more extreme outcomes (if the null hypothesis was true) for the model parameters for 4 focus sites (Birge Company, PBC, Ogrady Winnifred Silver, and Miken Company) located in Buffalo's West Side had $P \le .0001$. We further observed that focus sites located in Buffalo's West Side have associations that are very highly significant, and the effects appear to extend over a large area. However, there were also modest associations for 2 focus sites (Marnap Industries, $P \le .053$; and Nabisco Company, P≤.045) located on Buffalo's East Side, and the effects appear to extend over a small area. There was no evidence of association for Harrison Radiator. also located on Buffalo's East Side, as well as the other 6 test sites. Overall, the analysis of case-control data establishes further evidence of an association between diagnosed asthma and 4 focus sites located on Buffalo's West Side. Our study also finds modest associations for locations within Buffalo's East Side, especially the Nabisco Company, which manufactures Milk-Bone bakery products and releases respirable air-borne dust particles. These associations warrant further investigation.

Comparability of Case Patients and Control Patients

Insurance status as a possible confounder was evaluated during the study period. A comparison of insurance status for case patients and control patients was conducted to evaluate whether a significant difference existed between asthma and gastroenteritis patients in 34 subcategories of insurance status reported in the databases, which were identified following these 5 tallies: total number of

Positive association between exposure and outcome at the 5% significance level.

^bNegative association between exposure and outcome at the 5% significance level.

case patients, inpatients, outpatients, emergency room use, and clinical case patients. Overall, based on a t test, there was no statistical difference in insurance status between asthma and gastroenteritis patients in the subcategories described above. However, there were some slight differences that were observed in 3 subcategories: outpatients $(t_{\rm observed}=1.669, df=66, t_{\rm critical}=1.668, P \le .049)$, clinical $(t_{\rm observed}=2.562, P \le .006)$, and case patients diagnosed in 2000 $(t_{\rm observed}=1.694, P \le .047)$.

Additional analysis was conducted on the basis of the 4 categories of benefits (private benefit programs, public-sponsored benefit programs, self-sponsored benefits, and others) that were identified with insurance status data. In this particular analysis, it was assumed that those patients who were most likely to have access to private preventive care belonged to the private benefit programs category. Insurance status data for patients seeking emergency and urgent care in the hospital showed that 34.85% and 30.28% of the patients with asthma and gastroenteritis diagnoses, respectively, were most likely to have private preventive care as well. The remaining subcategories showed that publicsponsored benefits were used by 26.99% and 28.41% of the asthma and gastroenteritis patients, self-sponsored benefits constituted 20.62% and 22.15% of the asthma and gastroenteritis patients, and others constituted 17.5% and 18.9% of patients with asthma and gastroenteritis diagnoses, respectively. Overall, the comparison of insurance status for patients seeking care in the emergency room during the study period shows a comparable ratio of 1:1 in all 4 subcategories. The insurance status of case patients with asthma diagnosis and diagnosed gastroenteritis was therefore comparable, lending support for gastroenteritis as an appropriate candidate for the case-control study.

DISCUSSION

There are 4 major findings from this study: first, the distribution of asthma, after accounting for spatial variation in the population at risk, is nonhomogenous; second, areas in which case patients have high rates of asthma appear clustered in proximity to air-polluting

sites, including the PBC and the busily traveled roadways supplying it, in addition to EPA-designated toxic air release sites; third, the decrease in asthma prevalence as a function of distance from sites indicates that pollutants from the sites are not only associated with the worsening of asthma symptoms but may also play a role in the etiology of asthma; and fourth, further analysis of common features of pollutants may help elucidate mechanisms relating exposures to the genesis of asthma. To date, studies have focused on highlevel exposures and have paid relatively little attention to local multiple exposures. Identification of clusters associated with different sources may provide insights into how mixtures of pollutants may interact and lead to development of asthma in susceptible individuals. The study findings support and expand our earlier observations in Lwebuga-Mukasa and Dunn-Georgiou,^{2,3} Lwebuga-Mukasa and Pszonak, 4 Oyana and Lwebuga-Mukasa, 29 and Lwebuga-Mukasa et al.⁶ These findings are also consistent with previous findings that have been reported in Peterson and Saxon,²⁴ Kane et al., 25 Briggs et al., 38 Donaldson et al.,12 Dockery,39 Loh et al.,40 Lin et al.,5 and Lin et al.41 The findings confirm earlier reports that indicated that increases in NAFTA traffic across the PBC were related to increases in prevalence and health care use for asthma.²³ This study, together with the previous studies, provides a basis for systematic investigation of environmental exposures in communities and their effect on residents.

Levels of Geographic Resoloution

This study has benefited from the use of more than 1 level of geographic resolution. Our spatial analysis has been conducted here at 2 different levels. At the individual level, we analyzed and compared spatial locations of case patients and control patients at varying distances from sites. At the zip code level, we computed odds ratios to evaluate which zip codes were heavily burdened by respiratory illnesses. In a previous study, 29 we analyzed census tract (group)-level data to obtain case patients per population size and to identify local asthma clusters. These different levels of geographic resolution enabled us to gain more insight into the problem of respiratory illnesses faced by communities living in

close proximity to the PBC, major roadways, and pollution sites.

Proximity to Sites

This study pinpoints proximity to air release sites including the PBC as a significant contributor to increased asthma exacerbations in the study area. For the residents living within 0.5 km from air release sites, the odds of having asthma were 15 times greater compared with those of patients living more than 2 km away. We also observed a 4-fold increase in the odds of having asthma among residents living in close proximity to the PBC compared with those living in nonexposed areas farther away. It is probable that these sites significantly affect the quality of inhalable air, which in turn could trigger episodes of airway inflammation among individuals with asthma. It is therefore reasonable to suspect that communities living in close proximity to these sites are exposed to high levels of particulate emissions 42 that contribute to increased asthma exacerbations. In addition, this particular finding might explain the high rates of hospitalization and emergency room use already reported in previous studies.^{4,23} Overall, the extreme increase in asthma risk associated with toxic air release sites points to the need to minimize toxic releases because of their potential health risk to residents nearby.

Significant Associations

Six statistically significant associations of diagnosed asthma near the focus sites, in comparison with the geographical distribution of gastroenteritis, were found using the Diggle method. Although 1 focus site located on Buffalo's East Side did not reach statistical significance, there was modest evidence of increased diagnosed asthma in 2 focus sites located in this area. Of the 13 sites for which we fitted models, we observed a higher increased density of case locations in comparison with control locations on Buffalo's West Side than in other areas. These associations are consistent with those identified using K-means and nearest-neighborhood hierarchical clustering techniques, the score test of Lawson and Waller, the Bithell score risk test, and Besag and Newell's methods. The PBC site was not statistically significant by 1

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method—the score test of Lawson and Waller, ²⁹ but all the other methods, including the Diggle model, found that particular site very highly significant.

The analysis of case-control data establishes further evidence of associations between diagnoses of asthma and the 4 focus sites located on Buffalo's West Side. However, we cannot necessarily attribute all of the effects to these 4 focus sites, because statistically significant P values extend over a large area. Other possible explanations are explored below. This study also found modest associations for 2 focus sites located on Buffalo's East Side, especially for the Nabisco Company, which produces grain and flour, and these associations warrant further investigation. Current data do not permit determination of whether the increase in diagnosed asthma is related to residents working at the factory or whether exposure was secondary to air pollution.

Our study had 3 limitations. First, because the hospital data we used were from a single hospital system, we could not examine other hospital admissions in the same area. Second, it was not possible to determine why patients were admitted to the hospitals (i.e., the patient's original complaint). Third, the data set did not contain information pertaining to the living conditions of the patients; this information could explain other exposures to asthma risk factors.

Although there is mounting cumulative evidence associating increased risk of asthma to traffic-related pollution, it is reasonable to suspect the existence of a number of other prime risk factors, such as possible mixtures of pollutants and the interaction of local ecological factors that might better explain spatial variations of asthma in the study area. We suspect that these risk factors are key contributors of asthma on Buffalo's West Side, given that previous studies⁶ have absolved possible confounders such as exposure to environmental tobacco smoke, race, and income.

Interpretations

These interpretations were arrived at on the basis of the following factors. First, there is a significant release of diesel exhaust particles⁴² from a busily traveled state highway in which the bulk of commercial traffic serving the NAFTA trade corridor flows through this community. In the summer of 2002, a preliminary chemical analysis of respirable particles collected from the PBC by Baier, one of the research scientists studying traffic-related pollution, revealed high ammonium carbonates, nitrates, and sulfates in the study area (R. E. Baier, oral communication, August 23, 2002). His analysis further revealed a net addition of silicates and iron-bearing particles to the respirable fraction, which was independent of wind direction. Baier suggested that the net increase of respirable silicate-rich and iron-rich matter was attributable to truck emissions, based on further comparisons to spectra of impacted aerosol samples from a Cheektowaga truck stop, where numerous idling trucks were present. These unusually high levels of silicate-rich and iron-rich matter are residues of burning diesel that may combine with local factors and contribute to chronic airway and lung inflammation 44-49 and set the stage for airway hyperresponsiveness, a characteristic of asthma, and worsening of asthma symptoms among individuals who already have the disease. Second, there is a significant number of manufacturing industries located on Buffalo's West Side that might be emitting respirable particulates. The evidence of clusters of asthma about the focus sites we studied illustrates this point. Third, local ecological factors may be important, including the urban heat island phenomenon, 49,50 residential overcrowding, and most important, meteorological conditions influenced by the presence of Lake Erie and the Niagara River, which have an effect on the dispersion of particulate pollutants. There is a low density of vegetation on Buffalo's West Side compared with other areas in the study area, which causes the area to be warmer than normal. Finally, the age of the housing units is a potential confounder that warrants further investigation.

CONCLUSIONS

There are 2 implications of the study findings: first, current traffic levels not only contribute to asthma and other respiratory disease exacerbations but may also contribute to high asthma prevalence on Buffalo's West Side in comparison with other Buffalo com-

munities; and second, identification of asthma clusters along busily traveled roadways and the PBC, in addition to other sites, indicates an etiological link between pollutants and high asthma prevalence rates. These implications are important for the development of new hypotheses relating to the spatial distribution of asthma prevalence and morbidity in this community. Although our study does not provide specific information pertaining to the chemical compositions of the focus sites, it provides evidence about the locations at which exposures may affect susceptible individuals.

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This article was accepted February 10, 2003.

Contributors

Tonny J. Oyana participated in data processing, geocoding, data analysis, and geographic information systems (GIS) modeling and wrote the article. Peter Rogerson advised on data analysis and also contributed to editing the article. Jamson S. Lwebuga-Mukasa provided the data sets for disease analysis and guidance on medical issues and also participated in editing the article. The spatial and GIS approach used in this study was developed by Tonny J. Oyana in consultation with Peter Rogerson and Jameson S. Lwebuga-Mukasa.

Acknowledgments

This research was supported in part by the Centers for Disease Control and Prevention and the Troup Fund, Kaleida Health Foundations (grant R01-CCR220259–01).

Human Participant Protection

All research reported in this article was approved by the University of Buffalo human sciences investigation review board.

References

- 1. Niagara Peninsula Transportation Needs Assessment Study: Executive Summary Report. Available at: http://www.mto.gov.on.ca/english/pubs/research/niagara.pdf. Accessed March 23, 2004.
- 2. Lwebuga-Mukasa JS, Dunn-Georgiou E. Health

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- implications of Peace Bridge Plaza Complex: traffic related pollution effects on an American community at a US-Canada Gateway. Symposium Proceedings: Engineering Solutions to Indoor Air Quality Problems. J Air Waste Manage Assoc. 2000;VIP-96:477-503.
- Lwebuga-Mukasa J, Dunn-Georgiou E. The prevalence of asthma in western New York elementary school-aged children. J Urban Health. 2000;77: 745-761
- 4. Lwebuga-Mukasa JS, Pszonak R. Patterns of asthma hospitalization in western New York. J Asthma. 2001; 38:1-6.
- Lin S, Munsie JP, Hwanga S, Fitzgerald E, Cayo MR. Childhood asthma hospitalization and residential exposure to state route traffic. Environ Res. 2002;88: 73 - 81.
- 6. Lwebuga-Mukasa JS, Wojcik R, Dunn-Georgiou E, Johnson C. Home environmental factors associated with increased respiratory symptoms in patients with asthma. J Health Care Poor Underserved. 2002;13: 214-228.
- 7. Lwebuga-Mukasa JS, Ayirookuzhi SJ, Hyland A. Traffic volumes and respiratory health care utilization among residents in close proximity to the Peace Bridge before and after September 11, 2001. J Asthma. 2004; 40:855-864
- 8. ICF Consulting. North American trade and transport corridors: environmental impacts and mitigation strategies. Prepared for the North American Commission for Environmental Cooperation. February 2001. Available at: http://www.cec.org/files/pdf/pollutants/ trade_corridors_final-el_en.pdf. Accessed December 15,
- Schwartz J, Dockery DW. Increased mortality in Philadelphia associated with daily air pollution concentrations. Am Rev Respir Dis. 1992;145:600-604.
- 10. Schwartz J, Dockery DW. Particulate air pollution and daily mortality in Steubenville, Ohio. Am J Epidemiol. 1992;135:12-19.
- 11. Schwartz J. Air pollution and hospital admissions for respiratory disease. Epidemiology. 1996;7:20-28.
- 12. Donaldson K, Gilmour IM, MacNee W. Asthma and PM₁₀. Respir Res. 2000;1:12-15.
- 13. Dockery W, Pope CA III, Xu X, et al. An association between air pollution and mortality in six US cities. N Engl J Med. 1993;329:1753-1759.
- 14. Schwartz J. Particulate air pollution and chronic respiratory disease. Environ Res. 1993;62:7-13.
- 15. Schwartz J. Air pollution and daily mortality: a review and meta-analysis. Environ Res. 1994;64:36-52.
- 16. Schwartz J, Morris R. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. Am J Epidemiol. 1995;142:23-35.
- 17. Styer P, McMillan N, Gao F, Davis J, Sacks J. The effect of airborne particulate matter on daily death counts. Environ Health Perspect. 1995;103:490-497.
- 18. Norris G, YoungPong SN, Koenig JQ, Larson TV, Sheppard L, Stout JW. An association between fine particles and asthma emergency department visits for children in Seattle. Environ Health Perspect. 1999;107: 489-493
- 19. Wong TW, Lau TS, Yu TS, et al. Air pollution and hospital admissions for respiratory and cardiovas-

- cular diseases in Hong Kong. Occup Environ Med. 1999;56:679-683.
- 20. Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 US cities, 1987-1994. N Engl J Med. 2000;343: 1742-1749.
- 21. Peters A, Skrorkovsky J, Kotesovec F, et al. Association between mortality and air pollution in Central Europe. Environ Health Perspect. 2000;108:283-287.
- 22. US Environmental Protection Agency. Health Assessment Document for Diesel Exhaust. Washington, DC: US Environmental Protection Agency; May 1, 2002. USEPA EPA/600/8-90/057F.
- 23. Lwebuga-Mukasa JS, Oyana TJ, Thenappan A, Ayirookuzhi SJ. Association between volume and healthcare utilization for asthma among residents at a US-Canada border crossing point. J Asthma. 2004: 41(3)289-304.
- 24. Peterson B, Saxon A. Global increases in allergic respiratory disease: the possible role of diesel exhaust particles. Ann Allergy Asthma Immunol. 1996;77:
- 25. Kane M, Jaen CR, Tumiel L, Gonzalo MS, Bearman ML, O'Shea R. Unlimited opportunities for environmental interventions with inner-city asthmatics. I Asthma. 1999;36:371-379.
- 26. Lwebuga-Mukasa JS, Dunn-Georgiou E. A schoolbased asthma intervention program in Buffalo, New York, schools. J School Health. 2002;72:27-32.
- 27. US Census Bureau. 2000 US Population Census, US Dept of Commerce. Available at http://www.census. gov. Accessed September 15, 2002.
- 28. International Classification of Diseases. 9th Revision. Geneva, Switzerland: World Health Organization;
- 29. Oyana TJ, Lwebuga-Mukasa JS. Spatial relationships among asthma prevalence, healthcare utilization, and pollution sources in Buffalo neighborhoods, New York State. J Environ Health. 66(8):25-38.
- 30. Diggle PJ. A point process modeling approach to raised incidence of a rare phenomenon in the vicinity of a prespecified point. JR Stat Soc. 1990;153:
- 31. Diggle PJ, Rowlingson BS. A conditional approach to point process modeling of elevated risk. J R Stat Soc. 1994;157:433-440.
- 32. ClusterSeer version 1.1.4. Ann Arbor, Mich.: TerraSeer Environmental Insight. Available at: http://www. terraseer.com.
- 33. Environmental Systems Research Institute, AR-CGIS Software. Redlands, Calif.
- 34. Rijnders E, Janssen NAH, van Vliet PHN, Brunekreef B. Personal and outdoor nitrogen dioxide concentration in relation to degree of urbanization and traffic density. Environ Health Perspect. 2001;109(suppl 3): 411-417.
- 35. Milligan PJM, Brabin BJ, Kelly YJ, et al. Association of spatial distribution of childhood respiratory morbidity with environmental dust pollution. J Toxicol Environ Health Part A. 1998;55:169-184.
- 36. Besag J, Newell J. The detection of clusters in rare diseases. J R Stat Soc Ser A. 1991;154:143-155.
- 37. Waller LA, Turnbull BW, Clark LC, Nasca P.

- Chronic disease surveillance and testing of clustering of disease and exposure: application to leukemia incidence and TCE-contaminated dumpsites in upstate New York. Environmetrics. 1992;3:281-300.
- 38. Briggs DJ, Collins S, Elliott P, et al. Mapping urban air pollution using GIS: a regression-based approach. Int J Geogr Info Sci. 1997;11:699-718.
- 39. Dockery DW. Epidemiologic evidence of cardiovascular effects of particulate air pollution. Environ Health Perspect. 2001;109:483-486.
- 40. Loh P, Sugerman-Brozan J, Wiggins S, Noiles D, Archibald C. From asthma to airbeat: communitydriven monitoring of fine particles and black carbon in Roxbury, Massachusetts. Environ Health Perspect. 2002; 110(suppl 2):297-301.
- 41. Lin M, Chen Y, Burnett RT, Villeneuve PJ, Krewski D. The influence of ambient coarse particulate matter on asthma hospitalization in children: casecrossover and time-series analyses. Environ Health Perspect. 2002;110:575-581.
- 42. Widmer T, Vermette S. Impact of the Peace Bridge traffic on airborne particulates. Poster presented at: Fourth Annual Student Research and Creativity Celebration; Buffalo State College, Buffalo, NY; April 27,
- 43. Weitzman M, Gortmaker SL, Sobol AM, Perrin JM. Recent trends in the prevalence and severity of childhood asthma. JAMA. 1992;268:2673-2677.
- 44. Weiss KB, Gergen PJ, Crain EF. Inner-city asthma: the epidemiology of an emerging US public health concern. Chest. 1992;101(suppl 6):362S-367S.
- 45. Wijst M, Reitmeir P, Dold S, et al. Road traffic and adverse effects on respiratory health in children. BMJ. 1993:307:596-600.
- 46. Seaton A, Godden DJ, Brown K. Increase in asthma: a more toxic environment or a more susceptible population? Thorax. 1994;49:171-174.
- 47. Weiland SK, Mundt KA, Ruckmann A, Keil U. Self-reported wheezing and allergic rhinitis in children and traffic density on street of residence. Ann Epidemiol. 1994;4:243-247.
- 48. Takenaka H, Zhang K, Diaz-Sanchez D, Tsien A, Saxon A. Enhanced human IgE production results from exposure to the aromatic hydrocarbons from diesel exhaust: direct effects on B-cell IgE production. J Allergy Clin Immunol. 1995;95:103-115.
- 49. Woolum CA. Notes from a study of the microclimatology of the Washington, DC area for the winter and spring seasons. Weatherwise. 1964;17:263-271.
- 50. Myer WB. Urban heat island and urban health: early American perspective. Prof Geogr. 1991;43: 38 - 48