### Addressing Community Concerns about Asthma and Air Toxics

#### Mary C. White,<sup>1</sup> Sherri A. Berger-Frank,<sup>1</sup> Dannie C. Middleton,<sup>1</sup> and Henry Falk<sup>2</sup>

<sup>1</sup>Health Investigations Branch, Division of Health Studies, <sup>2</sup>Office of the Assistant Administrator, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia, USA

People with asthma who live near or downwind from a source of toxic emissions commonly express concerns about the possible impact of hazardous air pollution on their health, especially when these emissions are visible or odorous. Citizens frequently turn to their local and state health departments for answers, but health departments face many challenges in addressing these concerns. These challenges include a lack of asthma statistics at the local level, limited exposure information, and a paucity of scientific knowledge about the contributions of hazardous air pollutants to asthma induction or exacerbation. Health agencies are creatively developing methods to address these challenges while working toward improving asthma surveillance data at the state and local levels. Recent community health investigations suggest that hazardous air pollutants that are occupational asthmagens or associated with odors may deserve more attention. In seeking to address community concerns about hazardous air pollution and asthma, community health investigations may also help to fill gaps in our scientific knowledge and identify areas for further research or environmental intervention. The solutions to community problems associated with environmental contamination and asthma, however, require sustained, coordinated efforts by public and private groups and citizens. Public health agencies can make a unique contribution to this effort, but additional resources and support will be required to develop information systems and epidemiologic capacity at the state and local levels. Key words: asthma; air pollutants, environmental; epidemiology; hazardous waste sites; odors; population surveillance. Environ Health Perspect 110(suppl 4):561-564 (2002).

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In 1998 the number of people with asthma in the United States was estimated at 17 million (1). Modeled estimates of concentrations of hazardous air pollutants in census tracts across the country suggest that it is fairly common for concentrations of many pollutants to exceed benchmark values for chronic disease risks (2). It follows that many people with asthma are potentially exposed to hazardous air pollution (also called air toxics). People with asthma living near or downwind from a source of toxic emissions commonly express concerns about the possible impact of hazardous air pollution on their health, especially when these emissions are visible or odorous. Citizens frequently turn to their government, not always with a great deal of trust, to address these concerns. Local and state health departments are on the front lines to respond to these concerns expressed by citizens and community groups.

Despite all the research on asthma and air pollution, little is directly applicable to the health agencies that must respond to community concerns about air toxics. As Loomis pointed out, the tendency in air pollution research has been "to look where the light is" ( $\mathcal{J}$ ). Thus, most of the research on air pollution has been conducted in large urban areas where air monitors routinely collect data on levels of ozone, particulate matter, and the other criteria pollutants. In most communities, air monitoring data for hazardous air pollutants is nonexistent or insufficient to evaluate health risks ( $\mathcal{A}$ ). In this article, we detail some of the challenges public health agencies encounter when responding to citizens' concerns about asthma and toxic air pollution at the local level.

### Investigating Community Reports of Increased Asthma Prevalence

Citizens or community groups might express concern over an unusually high prevalence of asthma in an area with known or suspected hazardous air pollution. A fundamental first step for public health agencies in responding to such concerns is to verify that more people have asthma (or that the asthma is more severe or more frequently aggravated) than would ordinarily be expected. However, data on asthma are limited at the local level. National data exist for specific asthma measures, including self-reported asthma prevalence, asthma office visits, asthma emergency department visits, asthma hospitalizations, and asthma deaths (5). With the exception of asthma deaths (a relatively rare outcome), asthma measures are not consistently available at the state or local level (6). As a result, most local and state health agencies cannot readily determine from existing data the prevalence of asthma in a particular community.

During the last several years, the federal government and other organizations have identified the need for better surveillance data for asthma and have initiated efforts to address this need (7–9). Several state and local health departments have initiated efforts to establish asthma surveillance activities with support from the Centers for Disease Control and Prevention (CDC) and other federal agencies (6). Improved asthma surveillance systems would enable public health agencies to identify unusual patterns or changes in the occurrence of asthma in specific communities. This may lead to the development of hypotheses regarding previously unrecognized risk factors, including hazardous air pollution.

Under cooperative agreements with the Agency for Toxic Substances and Disease Registry (ATSDR), several states are currently exploring creative ways to use existing data to examine asthma occurrence and its possible relationship with hazardous air pollution. For example, the Utah Department of Health is using geographic information systems (GIS) to examine the temporal and spatial variation in hospital visits for childhood asthma in four Utah urban counties (10). The four counties include 77% of the state's population, twothirds of the state's 270 hazardous waste sites, and 10 National Priority List (Superfund) sites. These analyses will examine if hospital visits for asthma are increased by residential proximity to hazardous waste sites or industrial emission sources. Similarly, the Massachusetts Department of Health will use school health records to identify students with asthma, and GIS to map the location of the pediatric asthma cases and potential sources of exposure in the Merrimack Valley. The area includes three municipal solid waste incinerators located within a 4-mile radius, as well as the largest medical waste incinerator in New England (11). The purpose of this study is to a) assess whether the pediatric asthma rate from each community in the Merrimack Valley is higher than the rate from its demographically similar comparison community, and b) examine whether pediatric asthma rates are higher in areas with greater opportunity for exposure from hazardous waste sites and industrial emission sources. The New York State Department of Health, in collaboration with researchers at Columbia University, is using data from emergency department visits and supplemental air monitoring information to evaluate the temporal

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Address correspondence to M.C. White, CDC, 4770 Buford Highway NE, Mailstop K55, Atlanta, GA 30341 USA. Telephone: (770) 488-3032. Fax: (770) 448-4639. E-mail: mcwhite@cdc.gov

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associations between asthma exacerbations and a panel of air contaminants among residents of the Bronx and Manhattan (12). In addition to the criteria air pollutants, other air contaminants measured include aldehydes and metals. Time-series analysis will be used to examine possible interaction between different air contaminants and acute asthma exacerbations.

# Assessing the Plausibility of Purported Associations

There is a paucity of scientific research that can be used to assess the likelihood and plausibility of an association between asthma and a specific toxic air pollutant or a combination of toxic air pollutants. In the 50 years since the London Fog episode, an impressive body of scientific research has been assembled on the adverse health effects on populations exposed to air pollution, but most of this research relates specifically to the six criteria pollutants (13,14). Exposure to outdoor air pollution is widely recognized as an important contributor to asthma exacerbations; this was well illustrated in the reports of "natural experiments" that occurred in Utah Valley, Utah, and in Atlanta, Georgia. In 1986 a steel mill in Utah Valley shut down during a labor dispute and ambient levels of fine particulate matter were cut in half. Compared with the number of hospital admissions recorded during the previous and subsequent years, the number of hospital admissions for asthma and bronchitis (especially among children) was 2-3 times lower during the winter when the steel mill was closed (15). During the summer Olympics in Atlanta in 1996, the city experienced a drop in vehicular traffic as well as motor vehicle emissions and ozone levels. Acute care visits and hospitalizations for childhood asthma in Atlanta also fell during this period (16).

A recent authoritative review on the subject of asthma and outdoor air pollution emphasized the distinction between asthma onset and asthma exacerbation and concluded that common outdoor air pollutants can aggravate asthma but are unlikely to be related to the development of asthma (17). At least two recent studies challenge this conventional wisdom by linking ozone exposure with adult-onset asthma (18) and with asthma induction among children playing sports (19). In addition, recent studies from Europe have linked traffic emissions with allergic disease in children (20,21). It is possible that some of the effects attributed to criteria pollutants may be due to constituents of the air pollution mix that would fit under the rubric of air toxics. The distinction between induction and exacerbation may be irrelevant to community residents who are more likely to perceive exposures that trigger symptomatic episodes as important causes of asthma.

The possibility cannot be completely discounted that some air toxics contribute to the development of asthma. Asthma is generally accepted as a complex disease with a multifactorial etiology (22–24). Air pollutants may interact with various environmental, genetic, and personal factors in various ways at different points in the causal pathway. Few investigations have been published that examine the possible association between exposure to hazardous air pollutants and asthma prevalence. The Kanawha County Health Study in West Virginia reported positive associations between exposure to volatile organic compounds (VOCs) from chemical manufacturing plants and measures of asthma among elementary school children, including increased prevalence of physician's diagnosis of asthma and increased symptoms consistent with reactive airways (persistent wheezing and attacks of shortness of breath with wheezing) (25).

### Environmental Exposure to Occupational Asthmagens

Soybean dust was a recognized cause of occupational asthma long before it was linked to community outbreaks of asthma in Barcelona, Spain, and New Orleans, Louisiana (26, 27). Several of the compounds on the U.S. Environmental Protection Agency (U.S. EPA) list of hazardous air pollutants, including several metals, isocyanates, and some aldehydes, are known to cause asthma among occupationally exposed adults (28). Environmental exposure to these substances is not recognized as contributing to asthma prevalence. As Bates has pointed out, there is reason to be suspicious of air pollutants, but there is little epidemiologic research on this issue (29).

Recent investigations of environmental hazards from a foam-manufacturing facility in North Carolina suggest the need for further investigation of the possible impact of ambient exposures to agents known to cause asthma in the workplace. In this particular case, the manufacturer introduced a new process to produce foam that required the use of excess toluene diisocyanate, a substance that causes occupational asthma (*30*). After this change was introduced, residents near the plant complained about odors and health problems. They requested assistance from the state, local, and federal governments to investigate emissions from the plant.

The State of North Carolina made clinical evaluations available to self-selected adult residents who were concerned about nonspecific symptoms or illnesses and emissions from the plant. Physicians at Duke University in Durham, North Carolina, were contracted by the state to conduct a comprehensive examination, including pulmonary function testing and antibody testing for diisocyanates. They reported that 22 of 36 tested adults reacted during methacholine challenge testing, and 6 of 33 tested adults had antibodies to one or more diisocyanates (31). The report from this clinical evaluation concluded that the results were ". . . highly suggestive of environmental exposure from the plant," and that "... a plausible link exists between exposure . . . and symptoms experienced by community residents." Additional testing of 113 residents for antibodies to diisocyanates found 10 residents had specific antibodies to one or more diisocyanates, although 1 of the 10 may have been exposed at work (32). Environmental monitoring by ATSDR documented that diisocyanates were detected in the air. On the basis of both the environmental and biological monitoring, ATSDR concluded that there was evidence of a completed exposure pathway for diisocyanates in this community (33). The plant ceased foamproduction operations in September 1997.

ATSDR, in collaboration with the Randolph County Health Department and physicians at Duke University, subsequently undertook an investigation of respiratory symptoms among school-age children who lived within 1 mile of the plant during the period when the new curing process was used (34). The primary objectives of the investigation were to screen children who had potentially been exposed to emissions from the plant and provide diagnostic evaluations by asthma specialists for symptomatic children. A total of 259 children were identified from a list of students registered in the county schools system, and GIS plots of residences indicated that 225 of these students lived in the study area. Telephone interviews with the parents or guardians of these children identified an additional 24 siblings who were added to the study population. Interviews were completed with the parents or guardians of 231 (92.8%) of 249 children, but the interviews clarified that some children had not lived in the study area for at least 2 months during the period when the quick-curing process had been used (one of the study eligibility requirements). Of the 204 children who met the study eligibility criteria and for whom telephone screening interviews with their parents were completed, more than half (118) were found to have a history of asthma or respiratory symptoms consistent with asthma. Free medical evaluations were offered for these children, but only 55 of the 118 eligible children participated in these evaluations. Of these 55 participants, asthma was diagnosed in 28 children and considered possible in another 10 children. If we conservatively assume that none of the other 63 symptomatic children had asthma (unlikely), one lower-bound estimate of asthma prevalence among school-aged children in this community would be approximately 20% (38 of 204). This investigation was conducted to be responsive to local concerns and not as hypothesis-driven research, and thus no comparison population was used. On the basis of national statistics and studies in other communities, the proportion of children found to have respiratory symptoms or asthma in this predominantly white, semirural community appeared elevated. This apparent elevation in respiratory symptoms and asthma, although not explained, raises the possibility that poorly controlled releases from this facility contributed to these respiratory problems and suggests the need for more research on environmental exposure to occupational asthmagens.

## Exposures Indoors and Outdoors

It is often difficult to obtain exposure information about hazardous air pollutants at the community level. When exposure data are unavailable within a community, proximity may be used as a crude surrogate measure of exposure. Some databases do exist, such as the U.S. EPA Toxic Release Inventory, that provide emission estimates for major exposure sources. This information can be used together with sophisticated modeling procedures to estimate chronic exposures in communities with large sources of air emissions (but exposures from multiple smaller sources will be underestimated) (35). In the absence of actual exposure data, modeled estimates and assumptions about exposures may be subject to considerable misclassification error.

Indoor sources of several VOCs can contribute more to a person's total exposure than outdoor exposures (36); however, this may not be true in a community located downwind of a petrochemical plant or other stationary source of air emissions (37). The protective value of staying indoors during periods of poor air quality may also be diminished when hazardous air pollutants are elevated. VOCs and fine particles (particulate matter with a mass median aerodynamic diameter less than 2.5  $\mu$ m) readily penetrate indoor environments, especially homes (37,38).

Large, stationary sources of air toxics are often located in, or adjacent to, areas of lower socioeconomic status, and issues of environmental justice cannot be ignored (*39*). In these communities, residents who feel they have been disproportionately affected by outdoor air toxics may resent efforts to shift attention to indoor allergens, many of which are also are associated with poverty (e.g., cockroaches and rodent urine). As one advocate described,

Maybe it is easier to blame our asthma rates on our poverty, on our race and ethnicity, than to

look seriously at what those in charge have been doing to our community for years. If you blame us, it also means that you don't have to do something about that polluting facility or about those diesel trucks (40).

In communities where environmental justice is a concern, local community and environmental justice groups often have been very effective at guiding decisions related to the support and implementation of research and other public health activities (41).

### Air Toxics Odors as Risk Factors for Asthma

Emissions of air toxics from a variety of stationary sources can be associated with noticeable and sometimes noxious odors. Odors can exacerbate symptoms among persons with asthma, but the evidence of this relationship is largely anecdotal (42-44). The magnitude of health risk posed by odorous air pollutants to people with asthma has not been quantified; most local environmental control agencies treat odors as a nuisance problem. However, two recent investigations (45,46) suggest that exposure to odorous compounds in the ambient air can lead to measurable adverse health effects among persons with asthma.

In response to community health concerns about landfill emissions on Staten Island, New York, ATSDR investigated the effect of odor and air pollutants from the landfill on persons with asthma (45). After considerable outreach efforts, a cohort was assembled of 148 people between 15 and 65 years of age who had been diagnosed with asthma and lived near the landfill. During the summer of 1997 cohort members completed daily diaries that recorded respiratory symptoms, medication use, twice-daily peak flow measurements, and self-reported odor perception for 6 weeks. The results indicated that the smell of rotten eggs or garbage was associated with a modest increase in respiratory symptoms (odds ratio [OR], 1.5; 95% confidence interval [CI], 1.3-1.7) and medication use (OR, 1.3; 95% CI, 1.1-1.4), as well as a marginal decline in lung function.

In Dakota City, Nebraska, the regional office of the U.S. EPA and ATSDR undertook continuous air monitoring of ambient hydrogen sulfide (H2S) levels to characterize its temporal and spatial distribution in the area for a 15-month period. H<sub>2</sub>S has an odor typically described as that of rotten eggs, and its odor threshold is fairly low (0.5 ppb) (47). Numerous sources of H<sub>2</sub>S exist in this community, but the community was most concerned about the uncovered waste lagoons of a large beef-processing plant. A recent timeseries analysis examined daily hospital visits and measures of H<sub>2</sub>S and total reduced sulfur. The study results suggested a modest association between both measures and hospital visits

for asthma and other respiratory diseases among children (46).

### The Need for More Community-Based Investigations

The Pew Environmental Health Commission reported that most of the research money spent by the federal government on asthma in 1999 was used to support research on asthma treatment or basic research into cellular processes and mechanisms (7). Only a small percentage of federal funds was used to support public health activities and research into asthma etiology. Although many have recognized the lack of data on the potential contribution of air toxics to the burden of asthma, ongoing asthma surveillance at the community level and more formal epidemiologic studies are needed for this possible relationship to be examined. At the community level, decisions continue to be made about plant operating permits, ambient air emission controls, land use, zoning, etc.; many of these decisions may be controversial or questioned by community residents concerned about asthma. Better asthma surveillance data and information on exposure at the local level would enable exploratory analyses to better define the objectives and design of an epidemiologic study, and such research could be used to evaluate the adequacy of existing environmental controls for protecting public health.

Any epidemiologic study of asthma and air toxics will, by definition, involve community residents as the study population. Although the political controversies and legal actions in some communities may prove formidable to the conduct of a well-grounded, scientifically objective study, this is certainly not always the case. To be successful, the involvement of residents in the planning and implementation of any study is essential. The National Institute of Environmental Health Sciences and the U.S. EPA currently support 12 Centers for Children's Environmental and Disease Prevention Research, which facilitate the combination of multidisciplinary basic and applied research with community-based prevention research projects (48). Several of these centers have established academic-community partnerships to creatively conduct asthma research, and similar efforts could be expanded to other communities affected by air toxics.

The experiences of specific communities may offer unique opportunities to better understand the potential contribution of air toxics and asthma. Recent advances in study methodologies for environmental lung diseases can be applied to community investigations (49). By applying the best scientific methods, investigators will use the results not only to address local questions but also to advance their broader scientific knowledge of the problem.

### Summary

Public health agencies face a number of challenges when responding to local concerns about asthma and air toxics. As states continue to explore innovative ways to use existing asthma data at local levels, more information will be available for investigation of the prevalence of asthma in limited geographic areas. To address the need for better asthma data at the state and local levels, CDC is working with several state and local health departments to establish asthma surveillance activities. The systematic collection and tracking of health outcome data, as recommended by the Pew Environmental Health Commission, can lead to the development of hypotheses regarding previously unrecognized risk factors, including hazardous air pollution. In addition to improved surveillance data, the conduct of investigations of asthma and hazardous air pollutants at the community level might help fill data gaps and suggest priorities for further research or environmental interventions. Recent community-based investigations indicate that more attention should be given to hazardous air pollutants that are occupational asthmagens or associated with odors.

Community health investigations can only begin to address community concerns about hazardous air pollution and asthma. Epidemiologic investigations of air toxics and asthma are likely to have the greatest impact if conducted as part of long-term, coordinated efforts by public and private groups and citizens to address health concerns about environmental contamination. The enhancement of data information systems and epidemiologic capacity at the state and local levels would strengthen the ability of public health agencies to contribute to such efforts.

#### **REFERENCES AND NOTES**

- Rappaport S, Boodram B, Centers for Disease Control and Prevention. Forecasted state-specific estimates of selfreported asthma prevalence—United States, 1998. Morb Mortal Wkly Rep 47:1022–1025 (1998).
- Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106:245–251 (1998).
- Loomis D. Sizing up air pollution research. Epidemiology 11:2–4 (2000).
- Kyle A, Wright C, Caldwell J, Buffler P, Woodruff T. Evaluating the health significance of hazardous air pollutants using monitoring data. Public Health Rep 116:32–44 (2001).
- Mannino DM, Homa DM, Pertowski CA, Ashizawa A, Nixon LL, Johnson CA, Ball LB, Jack E, Kang DS. Surveillance for asthma—United States, 1960-1995. Morb Mortal Surveill Summ 47:1–27 (1998).
- Boss LP, Kreutzer RA, Luttinger D, Leighton J, Wilcox K, Redd SC. The public health surveillance of asthma. J Asthma 38:83–89 (2001).

- Pew Environmental Health Commission. Attack Asthma: Why America Needs a Public Health Defense System to Battle Environmental Threats. Baltimore, MD:Johns Hopkins School of Public Health, 2000.
- DHHS. Asthma Work Group. Action against asthma. Washington, DC:U.S. Department of Health and Human Services, 2000.
- The American Lung Association. Urban air pollution and health inequities: a workshop report. Environ Health Perspect 109(suppl 3):357–374 (2001).
- Ball W, Gillette M, Williams G. An analysis of childhood asthma and environmental exposure in Utah. In: Program of American Public Health Association 2001 Annual Meeting, 21-25 October 2001, Atlanta, Georgia. Available: http://apha.confex.com/apha/129am/techprogram/paper\_ 25652.htm [accessed 23 January 2002].
- 11. Knorr R. Personal communication.
- 12. Luttinger D. Personal communication.
- American Thoracic Society, Committee of the Environmental and Occupational Health Assembly. Health effects of outdoor air pollution. Am J Respir Crit Care Med 153:3–50 (1996).
- American Thoracic Society, Committee of the Environmental and Occupational Health Assembly. Health effects of outdoor air pollution. Part 2. Am J Respir Crit Care Med 153:477–498 (1996).
- Pope CA, 3rd. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. Am J Public Health 79:623–628 (1989).
- Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague WG. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. JAMA 285:897–905 (2001).
- 17. Koenig JQ. Air pollution and asthma. J Allergy Clin Immunol 104:717–722 (1999).
- McDonnell WF, Abbey DE, Nishino N, Lebowitz MD. Longterm ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG Study. Environ Res 80:110–121 (1999).
- McConnell R, Berhane K, Gilliard F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. Asthma in exercising children exposed to ozone: a cohort study. Lancet 359:386–391 (2002).
- Wyler C, Braun-Fahrlander C, Kunzli N, Schindler C, Ackermann-Liebrich U, Perruchoud AP, Leuenberger P, Wuthrich B. Exposure to motor vehicle traffic and allergic sensitization. The Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Epidemiology 11:450–456 (2000).
- Kramer U, Koch T, Ranft U, Ring J, Behrendt H. Trafficrelated air pollution is associated with atopy in children living in urban areas. Epidemiology 11:64–70 (2000).
- Plopper CG, Fanucchi MV. Do urban environmental pollutants exacerbate childhood lung diseases? Environ Health Perspect 108:A252–A253 (2000).
- Gilliland FD, McConnell R, Peters J, Gong H Jr. A theoretical basis for investigating ambient air pollution and children's respiratory health. Environ Health Perspect 107(suppl 3):403–437 (1999).
- 24. Beggs PJ, Curson PH. An integrated environmental asthma model. Arch Environ Health 50:87–94 (1995).
- Ware JH, Spengler JD, Neas LM, Samet JM, Wagner GR, Coultas D, Ozkaynak H, Schwab M. Respiratory and irritant health effects of ambient volatile organic compounds. The Kanawha County Health Study [see comments]. Am J Epidemiol 137:1287–1301 (1993).
- Anto JM, Sunyer J, Rodriguez-Roisin R, Suarez-Cervera M, Vazquez L. Community outbreaks of asthma associated with inhalation of soybean dust. Toxicoepidemiological Committee. N Engl J Med 320:1097–1102 (1989).
- White MC, Etzel RA, Olson DR, Goldstein IF. Reexamination of epidemic asthma in New Orleans, Louisiana, in relation to the presence of soy at the harbor. Am J Epidemiol 145:432–438 (1997).
- 28. Leikauf GD, Kline S, Albert RE, Baxter CS, Bernstein DI, Buncher CR. Evaluation of a possible association of urban

air toxics and asthma. Environ Health Perspect 103 (suppl 6):253–271 (1995).

- Bates DV. Observations on asthma. Environ Health Perspect 103(suppl 6):243–247(1995).
- Banks DE, Rando RJ, Barkman HW Jr. Persistence of toluene diisocyanate-induced asthma despite negligible workplace exposures. Chest 97:121–125 (1990).
- Darcey D, Lipscom H. Report of Clinical Evalutions, Archdale/Glenola Area Residents, Randolph County, NC. Durham, NC:Duke University Medical Center, 1998.
- Orloff KG, Batts-Osborne D, Kilgus T, Metcalf S, Cooper M. Antibodies to toluene diisocyanate in an environmentally exposed population. Environ Health Perspect 106:665–666 (1998).
- ATSDR. Health Consultation for the Trinity American Corporation. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1997.
- ATSDR. Diagnostic Evaluation of Children with Respiratory Symptoms and Potential Exposure to Diisocyanates Released from the Trinity American Corporation, Glenola, North Carolina. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 2001.
- ATSDR. Petitioned Public Health Assessment Newton Community, Gainesville, Georgia, Exposure to Air Pollution. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 2002.
- 36. Wallace LA, Pellizzari ED, Hartwell TD, Sparacino C, Whitmore R, Sheldon L, Zelon H, Perritt R. The TEAM (Total Exposure Assessment Methodology) Study: personal exposures to toxic substances in air, drinking water, and breath of 400 residents of New Jersey, North Carolina, and North Dakota. Environ Res 43:290–307 (1987).
- Lewis CW. Sources of air pollutants indoors: VOC and fine particulate species. J Expos Anal Environ Epidemiol 1:31–44 (1991).
- Suh HH, Bahadori T, Vallarino J, Spengler JD. Criteria air pollutants and toxic air pollutants. Environ Health Perspect 108(suppl 4):625–633 (2000).
- American Lung Association. Urban air pollution and health inequities: a workshop report. Environ Health Perspect 109(suppl 3):357–374 (2001).
- 40. Warren B. Asthma and community advocacy for clean air. In: Proceedings of the Conference on Working Together to Combat Urban Asthma, hosted by the Center for Urban Epidemiologic Studies at the New York Academy of Medicine, 4-5 May 1998, New York, New York. Available: http://www.nyam.org/publications/online/asthmaconference/WARREN-F.htm [accessed 10 January 2002].
- Claudio L, Torres T, Sanjurjo E, Sherman LR, Landrigan PJ. Environmental health sciences education—a tool for achieving environmental equity and protecting children. Environ Health Perspect 106(suppl 3):849–855 (1998).
- Shim C, Williams MH Jr. Effect of odors in asthma. Am J Med 80:18–22 (1986).
- Shusterman D. Critical review: the health significance of environmental odor pollution. Arch Environ Health 47:76–87 (1992).
- Shusterman D. Odor-associated health complaints: competing explanatory models. Chem Senses 26:339–343 (2001).
- ATSDR. A Panel Study of Acute Respiratory Outcomes, Staten Island, New York. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 2000.
- 46. Campagna D, Kathman S, Pierson R, Inserra S, Phifer B, Middleton D, Zarus G, White MC. Impact of ambient hydrogen sulfide and total reduced sulfur levels on hospital visits for respiratory diseases among children and adults in Dakota City and South Sioux City, Nebraska [Abstract]. Am J Respir Crit Care Med 163:A562 (2001).
- ATSDR. Toxicological Profile for Hydrogen Sulfide. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1999.
- Dearry AD, Collman GW, Saint C, Fields N, Redd S. Building a network of research in children's environmental health. Environ Health Perspect 107(suppl 3):391 (1999).
- Tager IB. Current view of epidemiologic study designs for occupational and environmental lung diseases. Environ Health Perspect 108(suppl 4):615–623 (2000).