US ERA ARCHIVE DOCUMENT

<u>Proximity to Environmental Hazards:</u> Environmental Justice and Adverse Health Outcomes

By Juliana Maantay, Jayajit Chakraborty, and Jean Brender

Revised Final Draft – May 12, 2010

Prepared for the U.S. Environmental Protection Agency "Strengthening Environmental Justice Research and Decision Making: A Symposium on the Science of Disproportionate Environmental Health Impacts"

Juliana Maantay, Ph.D., M.U.P. [*]
Professor of Urban Environmental Geography
Acting Chair, Environmental, Geographic, and Geological Sciences Dept.
Director of the Geographic Information Science (GISc) Program, and Urban GISc Lab
Lehman College, City University of New York
250 Bedford Park Blvd. West
Bronx, NY 10468
718 960-8574 (tel); 718 960-8584 (fax)
Juliana.maantay@lehman.cuny.edu (e-mail)

Jayajit Chakraborty, Ph.D., M.S. Associate Professor Associate Chair Department of Geography University of South Florida 4202 East Fowler Avenue, NES 107 Tampa, FL 33620 813 974-8188 (tel); 813 974-4808 (fax) jchakrab@cas.usf.edu (email)

Jean D. Brender, Ph.D., R.N.
Professor, Dept. of Epidemiology & Biostatistics
Associate Dean for Research
School of Rural Public Health
219 SRPH Administration Building
TAMU 1266
Texas A&M Health Science Center
College Station, TX 77843-1266
979 862-1573 (tel); 979 458-1877 (fax)
jdbrender@srph.tamhsc.edu (email)

[*] = corresponding author

Table of Contents

I.	 Introduction
	 The Role of Geographic Information Science in Environmental Health Justice Research
	 Environmental Justice Research Studies
	 Summary of Section I and organization of the rest of the paper
II.	Methods and Models for Measuring Disproportionate Proximity and Exposure to Environmental Hazards
III.	Health outcomes and proximity to environmental hazards <u>pg. 55</u>
	 Adverse Pregnancy Outcomes and Childhood Cancers Cardiovascular, Respiratory, and other Chronic Diseases
	 Limitations of Spatial Epidemiology
IV.	Conclusions and recommendations
	Acknowledgementspg. 79
	References <u>pg. 80</u>
	<u>Appendices</u>
Appe	ndix A – Tables <u>pg. 100</u>
Table	
Table	Methodology for Spatial Definition of Proximity and Potential Exposure to Environmental Hazards
Table	3 Studies of Residential Proximity to Environmental Hazards and Adverse
	Pregnancy Outcomes with Reported Disparities by Race/Ethnicity or Socioeconomic Status
Table	
m 11	Outcomes, and Childhood Cancer
Table	5 Studies of Residential Proximity to Potential Environmental Hazards and Cardiovascular, Respiratory, and other Chronic Diseases

Appendix B – Figures <u>pg. 156</u>		
Figure 1	Spatial Coincidence Approach: Selection of Host Census Units	
Figure 2	Circular Buffers of Uniform Radius around Facilities of Concern	
Figure 3	Cumulative Distribution Functions for Hazard Proximity: Comparing Racial Characteristics of the Population	
Figure 4	A Typical Plume Footprint for a Hypothetical Chlorine Release Scenario using the ALOHA Model	
Figure 5	Selection of Census Units with a Circular Buffer using the Polygon Containment Method	
Figure 6	Selection of Census Units with a Circular Buffer using the Centroid Containment Method	
Figure 7	Selection of Census Units with a Circular Buffer using the Buffer Containment or Areal Apportionment Method	
Figure 8	Cadastral Dasymetric Mapping: Estimating Households within a Circular Buffer using Land Parcels	
Figure 9	Using Geographically Weighted Regression to Explore Relationships between Cancer Risk from Other (Minor) Point Sources of Air Toxics and Various Explanatory Variables in Florida: Distribution of Local t-statistic by Census Tract	

I. Introduction

Proximity to hazards, adverse health outcomes, and disproportionate impacts

The goal of this paper is to explore and answer the question: "Does proximity to environmental hazards result in adverse health outcomes and account for health disparities, and if so, *how* does proximity contribute to disproportionate environmental health impacts? In order to answer this question in a meaningful, comprehensive, and reliable manner, we have undertaken a substantive literature review and critique covering the salient research on these topics over the past two decades, including some earlier seminal works on the subject. One of the main objectives of this paper was to assemble the best information possible, to synthesize the body of knowledge on this topic, and to provide a state-of-the-science paper that would put forward the most cogent and scientifically defensible evidence that will assist the EPA and other regulatory agencies in making the best decisions and reforms required in order to minimize environmental injustices. This meta-analysis of the literature is the result of that effort.

Concerns about health and environmental hazards transcend the academic, scientific, and regulatory worlds: they are also of compelling interest to the public, who often recognize a relationship between environmental hazards and health. In a 1999 national telephone survey among U.S. voters (Hearne et al., 2000), 74% of respondents thought that environmental factors had an important impact on childhood cancer and 73% thought these factors had an impact on birth defects. Over one-half of the respondents expressed their opinion that air pollution, contaminated drinking water, and toxic waste had a "great deal" of impact on a person's health. These concerns often result in public perceptions of disease clusters near environmental entities such as hazardous waste sites, industrial facilities, and other potential sources of chemical releases. With the advent of geographic information systems (GIS), environmental scientists and public health researchers have been able to address these concerns more comprehensively and objectively with the use of various proximity analyses. In this report, we will discuss the various methods available to assess the relation between living near potential hazards and health outcomes. We will also systematically review studies that have examined residential proximity to environmental hazards in relation to environmental justice, adverse reproductive outcomes, childhood cancer, respiratory and cardiovascular conditions, and other adverse health outcomes.

Based on this review, implications for public health will be discussed, and recommendations for future research will be suggested based on gaps and weaknesses identified in the published studies.

Although the mainstream environmental movement of the 1950s and 1960s alerted the public to the dangers posed by pollution and environmental degradation, these impacts on people's health and the environment were not generally acknowledged (or thought) to be spatially or socially differentiated: everyone was presumed to be affected just about equally. The understanding that environmental problems may impact certain places and people more than others (and in a predictable pattern based on race and income) is a relatively new concept that gained nationwide attention in the late 1980s with the publication of the groundbreaking environmental justice study, "Toxic Wastes and Race in the United States: A National Report on the Racial and Socio-Economic Characteristics of Communities with Hazardous Waste Sites," published in 1987 under the auspices of the United Church of Christ's Commission for Racial Justice (United Church of Christ, 1987). This study found "race to be the most potent variable in predicting where commercial hazardous waste facilities were located in the U.S., more powerful than household income, the value of homes, and the estimated amount of hazardous waste generated by industry" (Bullard et al., 2007a).

Since the late 1960's, researchers have focused more specifically on the relationship between environmental health hazards and environmental health outcomes in nearby populations. Much of the subsequent research demonstrates the existence of an uneven geographic distribution of environmental health hazards, and potentially disproportionate environmental burdens and differential exposure risk in the United States, resulting in communities of color and low income neighborhoods bearing the highest burdens (Apelberg et al., 2005; Bryant, 1995; Bullard, 1994; Camacho, 1998; Chakraborty and Zandbergen, 2007; Geschwind et al., 1992; Goldman, 1993; Johnston, 1994; Linder et al., 2008; Mohai and Saha, 2007; Wilhelm and Ritz, 2003). Typically, proximity to pollution sources has been used as a proxy for exposure in many of these studies, rather than assessing actual health outcomes in relation to proximity, and usually a comparison is made between the socio-demographic characteristics of communities hosting such facilities and reference populations living where no such facilities are located. Facilities and land uses investigated are point sources of pollution, (such as Toxic Release Inventory facilities, power plants, and other stationary point sources of

air or water emissions) or non-point or area sources, (such as highways, landfills, hazardous waste sites, swine and poultry producing industries, and agricultural chemical applications), among others.

As will be shown in this report, much of the published literature has supported the hypothesis that proximity to environmental hazards translates to higher risks, including increased adverse health risks. Concern about proximity to industrial facilities and other noxious land uses stems from the fact that industrial areas generally carry a higher environmental burden than do purely residential neighborhoods in terms of pollution and risks (Maantay, 2001). Some of these burdens include poor air quality, noise and traffic safety; use and storage of hazardous materials; emissions of hazardous and toxic substances; illegal dumping; poor enforcement of environmental regulations; and inadequate response to environmental complaints (Maantay, 2001). In addition, there are many potentially negative consequences besides elevated exposures that are likely to be associated with living in close proximity to industrial emissions sources or hazardous waste sites, including odors, noise, traffic, contaminated soils, inferior housing, fewer amenities (e.g. parks, libraries), less safe neighborhoods and poorer environmental quality (Maantay, 2001; Perlin et al., 1995; Sexton, 1997).

That these health and quality-of-life impacts are visited disproportionately on the most vulnerable populations, those least likely to be able to combat them effectively, render these impacts even more detrimental to the public's health, and the need for remedy even more urgent. Some research has suggested that, not only are lower-income populations and communities of color more likely to live in close proximity to environmentally burdensome facilities and thus be more exposed to pollution, but that the health effects of exposure to these burdens are further modified by socio-economic status, and "due to material deprivation and psychosocial stress, [these populations] may be more susceptible to the health effects of air pollution," (O'Neill et al., 2003:1861).

Health disparities (adverse health outcomes disproportionately affecting minority and lower-income populations) are a well-documented phenomena in the United States. The National Center for Minority Health and Health Disparities (NCMHD) states that "African Americans, Hispanics, Native Americans, and Asian/Pacific Islanders, who represented 25 percent of the U.S. population, continued to experience striking health disparities, including

shorter life expectancy and higher rates of diabetes, cancer, heart disease, stroke, substance abuse, and infant mortality and low birth weight" (NCMHD, 2009).

The elimination of minority health disparities is also a goal of environmental justice and requires attention to both physical hazards and social conditions. Environmental conditions are believed to contribute to producing and maintaining minority health disparities, (Yen and Syme, 1999; Evans and Kantrowitz, 2002). The NIEHS Strategic Plan for eliminating such disparities notes, "both social and environmental exposures represent an important area of investigation for understanding and ameliorating the health disparities suffered by the disadvantaged of this nation" (NIEHS, 2004).

Environmental Health Justice

Environmental Justice, both as a term in our vocabularies and as a movement, came into being more than 20 years ago. Narrowly interpreted, Environmental Justice (EJ) is the attempt to document and address the disproportionate environmental and health burdens borne by the poor and people of color. In a broader context, EJ theory encompasses everything that is unsustainable about the world we have created, including rampant population growth, industrialization, pollution, consumption patterns, energy use, food production, and resource depletion. "The EJ movement has sought to redefine environmentalism as much more integrated with the social needs of human populations, and, in contrast with the more eco-centric environmental movement, its fundamental goals include challenging the capitalist growth economy, as well," (Pellow and Brulle, 2005:3).

The Environmental Protection Agency (EPA) currently defines environmental justice as:

Environmental Justice is the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies. EPA has this goal for all communities and persons across this Nation. It will be achieved when everyone enjoys the same degree of protection from environmental and health hazards and equal access to the decision-making process to have a healthy environment in which to live, learn, and work (U.S. EPA, 2009).

Another definition of environmental justice is "the provision of adequate protection from environmental toxicants for all people, regardless of age, ethnicity, gender, health status, social

class, or race" (Nordenstam, 1995:52), and the proximity of noxious land uses to populated areas is believed to jeopardize environmental health and justice. Although many researchers have focused on the disproportionate environmental burdens borne by the poor and communities of color, others have expanded the definition of environmental justice to include additional vulnerable populations, such as the very young, the elderly, the infirm and immunecompromised, pregnant women, immigrants, and future generations (Greenberg, 1993).

Environmental justice obtained its official integration into the governmental decision-making process in 1994, when President Bill Clinton signed Executive Order 12898, which read in part, "Each Federal agency shall make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority... and low-income populations." In 1998 President Clinton signed an Executive Order committing the nation to eliminate racial and ethnic minority health disparities, which was also reflected in one of the two overarching goals of *Healthy People 2010*. The National Institutes of Health (NIH) required each of its institutes to develop its own strategic plan for addressing disparity in the disease areas it studies, incorporated overall in "Addressing Health Disparities: The NIH Program of Action," (NIH, 2005).

In addition to Executive Order 12898, an important element of environmental justice activities is Title VI of the 1964 Civil Rights Act. Title VI prohibits recipients of federal financial assistance from discriminating on the basis of race, color, or national origin in their programs or activities. Thus, under Title VI, EPA has a responsibility to ensure that its funds are not being used to subsidize such discrimination. This statute has been used as the basis of several complaints in recent years alleging adverse impacts that disproportionately fall on people in protected classes, resulting from the issuance of pollution control permits by state and local governmental agencies that receive EPA funding. These complaints are addressed by EPA's Office of Civil Rights (OCR), which has developed draft guidance for evaluation. In the past year, the new EPA administration, headed by Lisa Jackson, has actively worked to expand the conversation on environmentalism and environmental justice, and is pursuing the integration of environmental justice and equity considerations into the agency's policy-making apparatus, including risk assessments, rule-making, and budget decisions.

In the wake of the devastation to Gulf Coast communities of color rendered by Hurricane Katrina and the inadequate governmental response, the United Church of Christ Justice and Witness Ministries commissioned a 20th anniversary report, *Toxic Waste and Race at Twenty*, 1987-2007: A Report Prepared for the United Church of Christ Justice & Witness Ministries," (Bullard et al., 2007a). Their study again found that race was the most significant variable in predicting where commercial hazardous waste facilities were located in the U.S., and that by applying new methodologies, it was found that disparities had worsened over the two decades. Using 2000 Census data, the authors found that African-Americans, Latinos, and Asian-Americans were 1.7, 2.3, and 1.8 times more likely than non-Hispanic whites to live within 3 kilometers of the nation's 413 commercial hazardous waste facilities across the entire country (p<0.001); the study found clustered and urban facilities to have similar or worse findings and statistically significant disparities in 9 of 10 EPA regions and 40 of 44 states with such facilities. The report was accompanied by an open letter to Congress, signed by more than 100 Environmental Justice Network leaders, calling for the federal government to "protect and enhance community and worker right-to-know" as one recommendation among ten in the authors' comprehensive plan, (Bullard et al., 2007b). The question remains whether disproportionate proximity/exposure of communities of color and low income populations to environmental health hazards translates into increased adverse health impacts for these populations.

The Role of Geographic Information Science in Environmental Health Justice Research

Since the late 1980's and beginning in earnest in the early 1990's, Geographic Information Systems have been used to examine the spatial realities of environmental injustice (Boer et al., 1997; Bowen et al., 1995; Burke, 1993; Chakraborty and Armstrong, 1997; Chakraborty et al., 1999; Maantay et al., 1997; Maantay, 2002; Morello-Frosch et al., 2001; Neumann et al., 1998; Perlin et al., 1995; Pollock and Vittas, 1995; Sheppard et al., 1999).

GIS methods have been used in environmental justice research primarily to analyze the spatial relationships between sources of pollution burdens and the socio-demographic characteristics of potentially affected populations. A GIS is "a powerful computer mapping and analysis technology that allows large quantities of information to be viewed and analyzed within

a geographic context," (Vine et al., 1997:598). GIS is more than just computer hardware and software. It is an integrated system of components, consisting of information about the real world that has been abstracted and simplified into a digital database of spatial and non-spatial features, which, in conjunction with specialized software and computer hardware, and coupled with the expert judgment of the GIS user or analyst, produces solutions to spatial problems or questions.

GIS technology is particularly well-suited for EJ research because it allows for the integration of multiple data sources (e.g., location of polluting facilities and population characteristics), representation of geographic data in map form, and the application of various spatial analytic techniques (e.g., buffering) for proximity analysis (Zandbergen and Chakraborty, 2006).

With GIS, it has become increasingly prevalent to try to map instances of environmental injustice, usually by geographically plotting facilities or land uses suspected of posing an environmental and human health hazard or risk, and then determining the racial, ethnic, and economic characteristics of the potentially affected populations compared with a reference population. This often results in dramatic maps showing toxic facilities concentrated in areas with high proportions of African Americans, Latinos, or Native Americans (United Church of Christ, 1987; Burke, 1993; Glickman and Hersh, 1995; Maantay et al., 1997; Clarke and Gerlak, 1998). Mapping became a favored method among researchers attempting to determine the existence of environmental injustice. Additionally, the wealth of environmental and demographic data now available on the Internet, as well as the proliferation of websites with interactive mapping applications available, have brought environmental justice mapping within reach of virtually anyone.

Although such maps can be unusually effective in visually demonstrating the disproportionate spatial distribution of noxious or hazardous facilities, these maps have also come under scrutiny and been criticized for being misleading and inaccurate, and their findings have often been contradicted by other spatial analyses. Mapping a phenomenon such as environmental injustice is not a straightforward exercise, and the difficulties encountered in producing such spatial analyses leave the maps open to a variety of interpretations and second-guessing. Just as no map can be viewed as an objective embodiment of the real world, maps

depicting environmental injustice are also social constructions, and therefore subjective and based on assumptions (Dorling and Fairbairn, 1997; Wood, 1992).

GIS approaches have thus proved to be quite controversial, and some researchers have questioned altogether the capabilities of GIS to adequately perform certain types of health research (Jacquez, 2000). Doubts also remain about the efficacy of GIS to pinpoint environmental injustices and the health impacts of pollution, and many researchers who use GIS have commented upon the challenges and limitations inherent in this method of spatial analysis (Clarke et al., 1996; Dunn et al., 2001; FitzGerald et al., 2004; Jarup, 2004; Kulldorff, 1999; Maantay, 2002; McMaster et al., 1997; Moore and Carpenter, 1999; Richards et al., 1999; Rushton et al., 2000; Sheppard et al., 1999; Vine et al., 1997; Wall and Devine, 2000; Yasnoff and Sondik, 1999).

Spatial and attribute data deficiencies, and methodological problems, especially those related to geographical considerations, are well-documented in these cited publications. Geographical considerations include the delineation of the optimal study area extent, determining the level of resolution and the unit of spatial data aggregation, and estimating the areal extent of exposure, as well as the various problems encountered in trying to statistically analyze and summarize spatial data. Due to the principle of spatial autocorrelation, which states that data from locations near one another in space are more likely to be similar than data from locations remote from one another, spatial data is by its very nature not randomly distributed, as traditional statistical approaches require (Tobler, 1979). Spatial autocorrelation, which is an inherent characteristic of geographically referenced data, thus becomes an impediment to the application of conventional statistical tests. These limitations are discussed in more detail in Sections II, III, and IV, below.

A fundamental concern with mapping environmental injustice is that it does not yield definitive findings about differential exposure levels or health outcomes for the population in proximity to the noxious facilities or land uses. This drawback makes these studies less useful in conclusively demonstrating (and measuring) the correspondence between the location of potential environmental burdens, exposures, and health effects. However, it is feasible to develop methods and tools for producing more meaningful spatial analyses, and recently health geographers and other researchers have been using GIS techniques effectively to show the

correspondence amongst factors such as proximity to hazardous facilities and land uses, adverse health outcomes, disproportionate exposure and risk, and health disparities.

Brulle and Pellow (2006:104) maintain that although there are many potential connections between environmental justice and health disparities research, the two remain, for all intents and purposes, separate disciplines. However, GIS and geospatial analysis can serve as a methodological framework to integrate or bridge these two areas of research. Although some of the papers that we reviewed for this report have succeeded in combining EJ and health disparities, (i.e., Grineski, 2007; Maantay, 2007; Chakraborty, 2009), one of the challenges we faced in assembling our literature review is that, generally speaking, papers fall into one or another of these categories, and only rarely merge all the topics we were interested in for the purposes of answering the research question posed at the beginning of the paper. Most research studies tend to look at health outcomes in relationship to environmental hazards, or at the correspondence of populations' socio-demographic factors to hazardous locations, or at health disparities based on disproportionate adverse health outcomes and socio-demographics, but studies do not usually examine more than one of these relationships at a time. Therefore our critique and evaluation of the salient literature for this report by necessity is composed of several completely separate bodies of literature. The following section discusses environmental justice studies and their findings.

Environmental Justice Research Studies

Rationale for study selection

The papers selected for review in this section reflect our efforts to provide a comprehensive overview and synopsis of relevant environmental justice studies and a longitudinal view of the research on EJ and proximity to environmental hazards over the past two decades, from the early 1990's to the present. In this way, we sought to provide background on the evolution of EJ research, as well as to evaluate the methods and data used, to report the findings, and to compare the results. The studies selected are significant because they are generally the most often-cited papers, and ones which have been consistently cited over time, an acknowledgement of their importance in the field. Several are considered the seminal papers on the topic. We attempted to find studies exhibiting as wide a range as possible in terms of

geographic extent studied, the variety of hazard types, and analytical methods employed. The researchers who conducted these studies also come from a diverse array of backgrounds and disciplines, and include geographers, sociologists, political scientists, atmospheric scientists, public health practitioners, urban planners, epidemiologists, environmental scientists, community-based planners and advocates, hazard analysts, environmental attorneys, and risk assessors.

The sub-sections below describe in general terms how most of the studies are constructed, regarding independent variables, dependent variables (hazard type), geographic scale or study extent, unit of analysis or resolution, and methods. The section concludes with a summary of the findings. See Table 1 for a detailed outline of the studies.

Independent Variables and definitions

Overview

The independent variables for most of the studies included both socioeconomic status (SES) and race/ethnicity, or focused specifically on either SES or race/ethnicity. The U.S. Census was usually the source for the socio-demographic information. The 2005 study by Apelberg et al. is typical of the usage of both race and SES as independent variables, using 2000 Census data. The study measured the percentage of white, African American and Hispanic people in each Baltimore census tract, and to measure SES, the study looked at the median household income, per capita income, percent owner-occupied homes, percent with public assistance income, percent below poverty level, and percent without a high school diploma.

Race as an Independent Variable

Definitions of race are not consistent, due to the subjective nature of "preconceived notions of a racialized social structure" (Omi and Winant 1994:59), where, particularly in the United States, racial divisions have been exploited as a strategy to maintain access to power and resources. However, the U.S. Census serves as a useful source of data related to race and ethnicity, although often recognized as being ambiguous, incomplete, and inconsistent itself. In 2000, the racial categories used by the U.S. Census include American Indian or Alaskan Native, Asian or Pacific Islander, Black, and White. In addition, the two ethnicity categories include Hispanic origin and Not of Hispanic origin, while persons of either ethnicity may be of any race.

Most studies included in this literature review treated people of Hispanic origin as its own ethnic/racial group similar to Census racial categories. Adding to the inconsistencies inherent in using census data, census categories have changed over time, reflecting what is perceived to be mainstream views of racial categories. Therefore, longitudinal analyses of racial/ethnic variables are fraught with difficulties, and may result in inaccuracies and misleading comparisons.

Chakraborty's 2009 paper provides an example of a typical use of race variables, using 2000 Census data at the census tract level in Tampa, Florida, to measure percentages of non-Hispanic whites, African Americans (non-Hispanic Black) and Hispanics or Latinos (of any race). As exemplified by this study, African American and Latino communities are often the primary focus of environmental justice studies, which is consistent with the attention paid in environmental justice and public health literature to health disparities faced by these populations in the United States, as well as these groups' significant proportions in the overall United States population. There are, however, a few environmental justice studies focusing on Native American communities, though these tend to be more anecdotal and descriptive than quantitative analyses (Bullard, 1994; The Akwesasne Task Force on the Environmental Research Advisory Committee, 1997; Clark and Gerlak, 1998; Berry, 1998; Camacho, 1998; Faber, 1998; Krakoff, 2002).

As demonstrated in the Findings sub-section below, race has a consistent spatial correspondence with the location of hazardous facilities and land uses, and a concomitant potential for disproportionate environmental exposures and disease burdens. Chakraborty and Zandbergen (2007) looked at the percentages of students who self-identify as black, Hispanic, white, and other at 153 public schools in Orange County, Florida. While the study did not explicitly measure SES variables, the authors point to the strong relationship between race, socioeconomic status, and educational attainment. The case of Orange County, Florida, is illustrative of a broader pattern in the United States, where SES factors such as employment status, education, and poverty are closely associated with race.

Residential segregation is a less-commonly explored factor, which poses environmental justice implications that could likely shed light on the connections between race and SES, as well as exposure to hazardous sites. Morello-Frosch and Jesdale (2006) measured racial segregation (as opposed to simply percentages of racial/ethnic populations within the population at large), asserting that systemic racial segregation poses important implications for community health and

individual well-being, and needed to be analyzed in order to better understand the origins and persistence of environmental health disparities. The authors measured segregation through the dissimilarity index (D), and calculated racial segregation for black, Hispanic, American Indian/Alaska Native, Asian/Pacific Islander, and white populations as an independent variable.

SES as an Independent Variable

Similar to race and ethnicity, the definition of SES is not concrete, and is somewhat fluid and inconsistent from study to study. There is no standard definition of "socio-economic status," but SES basically refers to a person's relative positioning and access to resources within a hierarchical social structure. Because the meaning(s) of SES are complex and dynamic, their quantitative (and qualitative) components also cannot be easily or consistently defined. This leads to the fact that each study we reviewed measured SES differently, although using similar methods and often based on U.S. Census data. Studies frequently looked at measures related to income, percent below poverty level, home ownership, education, and housing, and oftentimes expanded definitions of SES to incorporate education, employment, and family status. Recent EJ studies make a distinction between income and wealth, using the proportion of owner-occupied homes as an indicator of community wealth (Pastor et al., 2004; 2005; Gilbert and Chakraborty, 2008; Chakraborty, 2009). Linder, Marko, and Sexton (2008) regarded education as a correlate of health risk, and also looked at variables related to family status, such as lone-parent families and welfare or state reliance. Employment status or category were also frequently considered: for example, Linder, Marko, and Sexton (2008) examined percent of residents who worked in eight "blue-collar" occupations out of the 13 defined by the Census Bureau, and Sicotte and Swanson (2007) used the "population employed in manufacturing" as a proxy for SES. As another SES indicator, Mirabelli et al. (2006) measured student usage of free or reduced lunch.

While many studies looked at SES variables in conjunction with race/ethnicity, the two studies examining areas outside of the U.S., in Wales (Higgs 2009) and Hamilton, Canada (Buzzelli, 2003), did not take race into consideration and only used indicators of SES. This is likely attributable to these study regions having predominantly white populations. Higgs (2009) measured social deprivation, determined by reference to the Welsh Index of Multiple Deprivation of 2005, the official measure of deprivation used by the National Assembly for Wales. This is composed of seven deprivation indicators, namely: income, employment, health,

education, housing, access to services, and environment. There are no similar "official" measures of socioeconomic status or social deprivation used in the U.S.

Population Density

Green et al's study (2004), which measured proximity of schools to busy roads, looked at both race and SES data associated with social, economic, and housing status, and also looked at population density and total enrollment at schools as additional region-based variables. Additionally, Burke (1993), Bowen et al. (1995), Fisher (2005), Chakraborty (2009) and Maroko et al. (2009) all used population density as a variable, in addition to SES indicators and race. Population density serves as a useful independent variable to measure the degree to which an area is urbanized, which influences traffic volume as well as land use decisions. Several studies have also used metropolitan classification (urban/rural) as a categorical or dummy variable (Pastor et al., 2005; Baden et al., 2007; Gilbert and Chakraborty, 2008; Mohai et al., 2009). Rurality, also determined by population density, served as a variable in Norton et al's study (2007). In addition to measurements of race and the value of owner-occupied homes as a measure of wealth, they measured rurality by persons per square mile, because it serves as a possible determinant of landfill location, as these facilities require large parcels of unoccupied land for waste disposal. Rurality also often correlates with poverty, particularly in this paper's study region, eastern North Carolina.

Dependent variables/environmental hazard and proximity to hazard

Air pollution was the most common environmental burden examined in these studies, which typically measured proximity to sites that are pollution sources, including high-volume roads, power plants, and industrial facilities emitting air pollution. The dependent variable has been measured in various ways (as outlined more fully in Section II): presence of hazards, number or density of hazards, distance to hazards, or a measure of its magnitude, in terms of quantity of pollutants, toxicity, or health risk.

In order to measure health risk from air pollution, Morello-Frosch, Pastor, and Sadd (2001) used California's AB 2588 "Hot Spots" Guidelines and the U.S. Environmental Protection Agency's cancer risk guidelines. This study assessed cancer risk through inhalation unit risk (IUR) estimates, and used cancer potency data from the U.S. EPA and the California

EPA. Several similar studies on hazardous air pollutants used data from the EPA's National-Scale Air Toxics Assessment (NATA), which offers ongoing evaluations of air toxics and estimates of associated health risk. This data set has facilitated the making of connections between health risk and potential exposure, which has often proved difficult in environmental health studies. Studies using NATA data usually focus specifically on cancer risk estimates (Apelberg, 2005; Morello-Frosch and Jesdale, 2006; Linder et al., 2008), while recent studies have examined both cancer and respiratory disease risk (Gilbert and Chakraborty, 2008; Chakraborty, 2009).

Another source of data is the Toxic Release Inventory program of the Environmental Protection Agency, which includes annually collected data on releases and of certain toxic chemicals from industrial facilities. Utilization of TRI data was especially common before 2000, and Burke (1993), Bowen et al. (1995), Glickman and Hersh (1995), Perlin et al. (1995), Pollack and Vittas (1995), Chakraborty and Armstrong (1997), McMaster et al. (1997), and Sheppard et al. (1999) all used this data. Chakraborty and Zandbergen (2007) looked at potential exposure from TRI facilities as well as EPA's aerometric information retrieval system (AIRS) and major roads. Grineski (2007) used TRI data to collect a toxic air release score for each ZIP code.

Other studies which did not focus primarily on air pollution or releases of hazardous substances to the air measured proximity to industrial zones and the characteristics of populations living near intensification of major industrial zones (Maantay, 2001); proximity to solid waste facilities (Been and Gupta, 1996; Mohai and Saha, 2007; Norton, 2007; Higgs and Langford, 2009); access to parks and active recreational spaces (Maroko et al., 2009); and proximity to flood prone areas (Maantay and Maroko, 2009). Although natural hazards were not the focus of this review, we wanted to include at least one such research paper to point out the wide range of studies covering proximity to environmental burdens and environmental health justice.

Geographic scale (study extent), and unit of analysis (resolution)

Geographic Scale - Study extent

The studies included in this literature review ranged in geographic extent from individual neighborhoods to nation-wide studies, but the majority were small-extent studies focused on cities or metropolitan areas to allow for incorporation of location-specific knowledge. In

general, the cities and metropolitan areas selected for the studies reflected the geographic locations of the researchers, and perhaps over-represent major academic regions. The use of individual cities versus entire counties or metropolitan areas reflected both the research methodologies, selected data sets, and the location-specific geographies. For example, Sicotte and Swanson (2007) looked at the entire nine-county metropolitan statistical area including Philadelphia, PA, allowing them to disaggregate their results by county and neighborhood to compare results from suburban and urban areas in a manner that would not be possible using individual cities or a single county. On the opposite end of the extent spectrum were studies conducted on a national scale. Mohai and Saha (2007) conducted a study to replicate earlier nation-wide studies, using different spatial analysis methods, while Morello-Frosch and Jesdale (2006) focused on metropolitan areas within the continental U.S. to compare results using segregation as an explanatory factor to other national-scale studies that instead used racial/ethnic categories to help explain the same data sets (NATA) for environmental risk factors. Ash and Fetter (2004) examined urbanized areas nationwide to determine whether or not minority populations tend to experience higher levels of toxicity-adjusted exposure to air pollution from TRI facilities.

Unit of Analysis - Resolution

Because of their focus on race and socioeconomic status, most quantitative studies in the environmental justice literature--not just limited to those included in this paper--require census or similar population data, and therefore, enumeration units are the natural choice for the primary units of analysis for the independent variables. The majority of studies considered for this literature review used either census tracts or block groups as their unit of analysis depending on the extent of the study. The choice between census tracts and block groups varied with study extent, and either resulted from or was a reason for specific census categories used in the analysis--studies on individual cities tended to use census block groups, while most of the larger-extent studies used census tracts as their aggregation unit. Studies that looked at environmental effects on populations within schools used individual school sites as their unit of analysis (Green et al., 2004; Mirabelli et al., 2006), while Chakraborty et al. (2007) also included individual residences. Grineski (2007) was constrained to ZIP postal code zones as the unit of analysis on account of the available data resolution for asthma hospitalizations employed by the study, while

Hird (1993), Perlin et al. (1995), Bowen et al. (1995), and Daniels and Friedman (1999) used county-level data.

Several of the studies (McMaster et al., 1997; Dolinoy and Miranda, 2004; Baden et al., 2007; Mohai and Saha, 2007; Maantay and Maroko, 2009) were concerned with the effects of differing units of analysis on findings, and therefore used analysis units ranging from ZIP code postal zones down to census blocks. Mohai and Saha (2007) compared results from three levels of aggregation and concluded that areal apportionment techniques of distance-based spatial analysis produced very similar results regardless of the unit of analysis between ZIP code postal zones, census tracts, and census block groups. McMaster, Leitner, and Sheppard (1997) found that using smaller units of analysis allowed for detailed qualitative analysis that could be used to qualify and provide additional insight into the quantitative findings. Maantay and Maroko point out that data collected based on administrative boundaries created for political or other seemingly arbitrary purposes (e.g., ZIP code postal zones or census tracts) do not necessarily relate to the distribution of the underlying environmental phenomena being mapped, so using the smallest possible unit of analysis results in greater data resolution, minimizing potential misrepresentation of geographically heterogenous populations. They effectively disaggregated census block groups into individual tax parcels (cadastral units) using a GIS-based expert system (2009). Dolinoy and Miranda, in their 2004 study of Durham County, North Carolina, used data aggregated at various different scales and found that potential exposure disparities increase as the analytical unit becomes smaller (ZIP code to block), for both race and income. Baden et al. (2007) examined the sensitivity of EJ findings at three different geographic scales (nation, state, and county) using four different unit of analysis. Although the results depicted an inconsistent story, this study found strong evidence of environmental injustice for Blacks and Hispanics at the national and state levels when census tract and block group data were used.

Fisher et al. (2005) used a spatial analysis method, Ripley's *K* plots, borrowed from the epidemiology literature to identify statistically significant clusters of TRI facilities that could be used to establish proximity zones that served as the area for analysis, which, as discussed below, was also their method for determining proximity. However, they still relied on census tracts and block groups to provide population characteristics within the proximity zones. Downey (2006) used distance decay modeling to estimate hazard proximity to populations whose characteristics were aggregated by census tract.

Overview of proximity to environmental burdens

All of the studies included in this literature review attempted to establish the existence of inequitable distributional patterns of environmental factors relative to various axes of population characteristics. In general, these patterns can be revealed through either quantitative or qualitative methods; however for the epistemological purpose of public policy advocacy, which was at the core of the studies included in this literature review, quantitative methods of statistical and proximity analysis dominate. The utility of these methods is that they typically provide both a magnitude of disproportion and a probabilistic significance of the results. Quantitative methods are most useful for expediently and repeatably identifying larger-scale potential risk patterns that would be impractical using qualitative methods. All of the studies included in this literature review utilized a combination of quantitative proximity and/or statistical methods, while some also included qualitative components.

Qualitative methods, such as archival research or ethnographic studies, can reveal more detailed and holistic analyses of patterns of disproportionate environmental burdens, especially those not contained within the widely-available datasets used in most quantitative studies, such as TRI or NATA. An example of the use of qualitative analysis is the neighborhood-scale study of the Phillips community in Minneapolis, MN, conducted by McMaster, Leitner, and Sheppard (1997), which incorporated local knowledge of potentially vulnerable populations not captured by census data. In a study mixing quantitative and qualitative methods, Maantay (2001) used archival research on industrial zoning changes over a 40 year period in relationship to the socio-demographic characteristics of proximate populations to show a pattern of inequity in land use zoning practices and policies. More recently, Mohai et al. (2009) used individual-level survey data from a baseline sample of the Americans Changing Lives Study to examine racial and socioeconomic disparities in the distribution of polluting industrial facilities. Grineski (2009) also used qualitative methods in her study on asthmatic children (i.e., interviews with parents) to demonstrate how individuals interact with polluted urban environments and attempt to modify such environments.

The first methodological problem most of the studies addressed was how to establish the geographic extent of populations most vulnerable to environmental factors. The specific methods used to address this problem depended on how the environmental factors were

measured. Studies that used proximity to sources of environmental factors as a proxy for exposure effects employed techniques such as spatial coincidence, simple buffer intersection, and dasymetric mapping to associate the environmental factors with specific populations within the individual units of analysis (see Section II of this paper for definitions and explanations of these terms). Other studies used exposure- or health-risk estimates calculated for the population within each individual unit of analysis and therefore didn't require additional proximity analyses. Maroko et al. (2009) used a kernel density analysis to map density of park activity sites and acreage, and Geographically-Weighted Regression (GWR) of these variables in relationship to the socio-demographics of the census block group units of analysis.

The second methodological problem was how to determine if the environmental factors were "explained" by population variables, or put simply, whether or not the distribution of environmental factors was "equitable." As with the problem of defining the vulnerable populations, Fisher et al. point out that within the environmental justice literature, "no clear consensus on appropriate spatial statistics has emerged, because each statistic addresses different types of questions" (2005). Within the reviewed studies, statistical methods included generalized linear models, hypothesis testing (inferential statistics), and descriptive statistics.

While it is a fair assumption that all of the studies ask the question of disproportionate distributions of environmental goods and bads, the studies make different assumptions about how to measure "disproportion." Even the term "disproportionate impacts" is difficult to define to everyone's satisfaction. In this paper, we use the term to mean impacts that are not distributed evenly, uniformly, or equally across all populations and geographies, but rather tend to be more severe or of greater magnitude for certain groups in certain areas, as predicted by race/ethnicity and socioeconomic status, and in a way that is out of proportion with these groups' populations, both in terms of absolute numbers and as a percentage of the general population.

<u>Determination of Vulnerable Population or Exposure Risk</u>

Studies that relied on the U.S. EPA's NATA or Cumulative Exposure Project (CEP) data (Apelberg et al., 2005; Chakraborty, 2009; Linder et al., 2008; Morello-Frosch et al., 2001; Morello-Frosch and Jesdale, 2006) did not require separate geospatial methods since the data are mapped to census tracts as part of their underlying estimation methodology (Chakraborty, 2009). Similarly, Buzzelli et al. (2003) used spatial interpolation of Total Suspended Particulate (TSP)

concentrations to determine potential exposure levels at the centroid of each census tract within the study area.

The majority of studies that looked at proximity to point and mobile sources as an indicator of risk used the spatial coincidence method (Burke, 1993; Anderton et al., 1994; Goldman and Fitton, 1994; Bowen, 1995; Perlin et al., 1995; Been and Gupta, 1996; Cutter et al., 1996; Boer et al., 1997; Brooks and Sethi, 1997; McMaster et al., 1997; Daniels and Freidman, 1999; Fricker and Hengartner, 2001; Pastor et al., 2004; Mennis and Jordan, 2005; Baden et al., 2007; Norton, 2007). The spatial coincidence method is fully described in Section II of this paper, but briefly stated, it refers to an analytic approach that assumes potential exposure to environmental hazards is confined to boundaries of pre-defined geographic entities or census enumeration units (e.g., ZIP codes, census tracts, or block groups) that contain such hazards.

Many other studies used proximity analysis (or distance-based analysis) in the form of circular buffers (or line buffers in the case of mobile sources) with radii ranging from 100 yards to 4 miles to establish proximate populations, (Glickman and Hersh, 1995; U.S. GAO, 1995; Chakraborty and Armstrong, 1997; Neuman et al., 1998; Perlin et al., 1999; Boone, 2001; Bolin et al., 2002; Maantay, 2007; Chakraborty and Zandbergen, 2007; Mohai and Saha, 2007; Sicotte and Swanson, 2007; Higgs and Langford, 2009), and several of these studies compared the results of buffer analysis with spatial coincidence methods. Buffer analysis in EJ studies usually consists of the creation of circular or linear zones (buffers) around environmentally burdensome facilities/land uses, which represent areas of potential environmental impact from or exposure to pollutants, and then comparing the socio-demographic characteristics of the populations living within these zones (the potentially exposed populations) to those of the populations outside the buffer zones.

Instead of using circular buffers, Pollock and Vittas (1995) and Stretesky and Lynch (1999) used the distance between block group centroids and TRI facilities as a proxy for exposure, while Glickman and Hersh (1995), Chakraborty and Armstrong (1997), Dolinoy and Miranda (2004), and Fisher et al. (2007) employed air pollutant dispersion models to delineate plume buffers around facilities. Spatial coincidence methods were widely used as the exclusive method for spatially joining populations with environmental factors, although a handful of studies used multiple techniques to disaggregate their population data (Chakraborty and Armstrong, 1997; Higgs and Langford, 2009; Mohai and Saha, 2007; Sheppard et al., 1999).

Maantay and Maroko (2009) focused on flood risk, but employed disaggregation techniques like those used in the hazardous air pollutant studies to map flood zones onto census data. Fisher et al. (2005) used a spatial statistics technique called Ripley's K to locate statistically significant clusters of TRI facilities on a regional scale, then looked the entire neighborhood population within the area containing the most significant cluster of facilities. Chakraborty and Zandbergen (2007) used proximity analysis, but because their units of analysis were individually geocoded schools and residences, they were able to exactly map their population statistics onto the proximity ranges, which is not possible with census data due to anonymity requirements for data collection. Unique within the reviewed studies, Mirabelli et al. (2006) conducted voluntary surveys at schools to assess respondents' observations of swine odor at their school sites, using the degree of odor as a proxy for the presence of hazardous air pollutants resulting from concentrated swine feeding operations. Unlike those studies focused on hazardous air pollutants, Maroko et al. (2009) looked at access to parks, which prompted them to consider the physical infrastructure of the study area. Therefore, they used kernel density analysis, a form of spatial analysis, to determine proximate park density throughout the study area.

Evaluation of Disproportionate Risk or Burden

The studies included in this review used a range of statistical methods to describe the relationship between independent and dependent variables as evidence of disproportionate environmental risks or burdens. The majority of studies used generalized linear statistical methods to test for correlation between environmental factors and proximate population characteristics (linear or multivariate regression) or to test for significant differences between vulnerable and non-vulnerable populations (logistic regression, inferential statistics). These methods all calculate a measure of disproportion/correlation and an associated probabilistic significance level. Studies that used multivariate regression were able to test for independence between the explanatory variables to provide an additional detail about the findings, for example, that race was predictive of environmental risk even when controlling for household income. Similarly, several of the studies controlled for or considered the effects of spatial autocorrelation using appropriate methods such as Moran's *I* (Buzzelli et al., 2003; Sicotte and Swanson, 2007;

Chakraborty, 2009) or Monte Carlo randomization (Sheppard et al., 1999) to estimate the significance of their findings.

Nearly as prevalent as statistical methods in the reviewed studies were qualitative methods such as archival research and descriptive statistics to generally describe the extent and level of disproportion of environmental factors. For example, Maroko et al. (2009) augmented their indeterminate quantitative results with historiographic research and field surveys of physical access factors at parks within a case study area. In looking at differences between buffering methods in analysis of hazardous air pollutants, Chakraborty and Armstrong (1997) used descriptive statistics to compare results across methods, an acknowledgement that there is a great deal of uncertainty in aggregated population statistics and to provide statistical significance of differences in results across methods is meaningless since all of the results are themselves estimates with unquantified statistical significance.

Summary of Findings of Environmental Justice Research Studies

The majority of studies showed that both race and SES predicted a disproportionate spatial distribution of environmental burdens. When these two suites of variables were compared, SES variables pointed to more significant risks of exposure than race (McMaster et al., 1997; Apelberg, 2005; Mirabelli, et al., 2006; Grineski, 2007; Linder et al., 2008), however race tended to be predictive of disproportion even when controlling for SES (Morello-Frosch et al., 2001; Pastor et al., 2005; Morello-Frosch and Jesdale, 2006; Chakraborty, 2009). Linder, Marko, and Sexton (2008) found that, in addition to ethnicity and poverty being a significant indicator of cancer risk, the inverse relationship between a neighborhood population's overall level of education and cumulative cancer risk levels showed the strongest statistical associations across all social and economic variables. And studies pointed to the significant association between race and SES in their study areas, for example, McMaster et al. (1997:181) highlights "concentrated poverty" as an effective measure of potential exposure to hazardous air pollutants, emphasizing how it measures both income *and* race/ethnicity, due to the fact that people of color tend to be highly concentrated in areas with high poverty rates.

When studying other vulnerable populations, Chakraborty and Armstrong (2001) found that a significantly high proportion of the special needs population (a self-identified group of individuals with a physical or mental disability) in Linn County, Iowa, was residing in areas

potentially susceptible to worst-case extremely hazardous substances (EHS) releases. A similar study in Hillsborough County, Florida, demonstrated that a significantly large proportion of non-White and impoverished individuals resided in areas exposed to multiple worst-case EHS releases (Chakraborty, 2001).

Chakraborty (2009) had a number of unique outcomes. First, while measuring factors related to both race/ethnicity and SES, the study did not significantly prove inequity based on socioeconomic status. It did, however find a persistent pattern of racial and ethnic inequity in the distribution of estimated health risks from vehicular air pollution based on NATA data, even when controlling for relevant socioeconomic factors, and more so for the Hispanic population than African Americans. Additionally, this study found that residential population density indicates the strongest association with both cancer and respiratory risk at the tract level in Tampa Bay, pointing to the significant health risks associated with living in densely populated urban areas. Another unique finding was that households with no automobiles are disproportionately located in areas exposed to higher cancer and respiratory risks in comparison to the rest of the population, despite the fact that automobiles were the cause of health risks examined in these studies.

There were several notable studies that did not find a definitively positive correspondence between the location of environmental hazards and high percentages of minority or low socio-economic status populations. Mennis and Jordan (2005) reported mixed results in their study of TRI facilities in New Jersey, finding that the relationship between TRI facility density and explanatory factors vary significantly over space, and minority proportion has a significant and positive effect in most, but not all, urban and suburban areas in the state. Boone's 2001 study of Baltimore's TRI facilities found that tracts with White, working-class people are more likely to host TRI facilities than primarily Black tracts, but this pattern can be explained by a long history of residential and occupational segregation. The results were also inconclusive for some of the earlier environmental justice studies which primarily used the spatial coincidence method of ascertaining the binary presence or absence of hazardous facilities within a given geography (Anderton et al., 1994; Cutter et al., 1996). These results may be more attributable to the coarse unit of data aggregation used, as well as to the limitations of the spatial coincidence method used to approximate the potentially exposed population, rather than reflecting the realities of potential exposure.

Although TRI facilities have been used in many environmental health justice studies as the main locations from which to measure environmental exposures to vulnerable populations, and often act as a kind of a proxy for general environmental impacts areas, there are many limitations inherent with relying on TRI facilities to capture the entire range of environmental burdens that may affect a particular place or population group. For instance, only selected industrial sectors or polluting activities (limited to 23,000 facilities in the U.S.) and selected chemicals (approximately 650 at present) are included in TRI. Within the selected sectors and activities, facilities with fewer than 10 full-time employees are exempt from reporting, thus eliminating the vast majority of polluting facilities from consideration in the impact/exposure assessment. Additionally, facilities releasing toxics each year at levels under the reporting threshold set for an individual chemical (or in a form different than that designated for reporting - in dust or fibrous form, for example) are exempt from reporting. Limitations on regulation and data gathering obligations authorized under federal environmental statutes (e.g., grandfather clauses, toxic materials sent for recycling without intervening processing) will transfer to limits on TRI data (Nationally Consistent Environmental Justice Screening Approaches Work Group, 2010). Therefore, the TRI database captures only the tip of the iceberg with respect to adverse environmental burdens borne by communities in close proximity to these sites, because frequently these facilities are located where other industrial uses are permitted, and typically many of these uses fall below the reporting thresholds for TRI and are not tracked at a national level in any publicly-accessible database.

Studies focusing on air pollutants (TRI facilities, mobile sources, and so forth) dominated the literature and most consistently predicted disproportionate burdens. However, a number of studies examined proximity to other environmental burdens, such as Superfund sites (Baden et al., 2007); hazardous waste TSDFs (Anderton et al., 1994; Boer et al., 1997; Bolin, 2002; Cutter et al., 1996; Fricker and Hengartner, 2001; Goldman and Fitton, 1994;); solid waste landfills (Been and Gupta, 1996; Higgs and Langford, 2009; Mohai and Saha, 2007; Norton, 2007; and the U.S. GAO, 1995); and noise pollution from airports (Most et al., 2004). Most of these also found a positive spatial correspondence between minority/socio-economic status and proximity to hazards, with the exception of a few of the earlier studies (Anderton et al., 1994; Cutter et al., 1996; U.S. GAO, 1995).

Additionally, several studies looked at the disproportionate distribution of other environmental factors, including access to parks (Maroko et al., 2009), industrial land-use zoning (Maantay, 2001), and flood zones (Maantay and Maroko, 2009; Maantay et al, 2009a). Maantay (2001) showed zoning practices having a discriminatory impact, while Maantay and Maroko (2009) did not prove city-wide disparities in potential exposure to flood risk by race, although this varied amongst the different boroughs of New York City. However, the study showed that minority populations were dramatically undercounted as living in flood zones, when compared with non-Hispanic whites, as contrasted with the conventional methods of calculating population numbers. This points to the importance of taking into consideration factors related to historical and current patterns of residential settlement, industrial development, de-industrialization, and gentrification in certain areas of the city, and cultural changes over the years concerning the desirability of living along the waterfront and therefore the flood zones. Maroko et al. (2009) does not definitively demonstrate inequity in park access based on race or SES, but rather shows "unpatterned inequity," meaning that there is not an even distribution of parks and recreational areas in New York City, but the pattern cannot be predicted based on race or class. The study emphasizes the importance of further exploration of this topic using various quantitative and qualitative methods.

Studies that focused on methods for determining the proximity of disadvantaged populations to hazardous sites indicated that methods used in previous studies likely underestimated the disproportionate burdens faced by economically disadvantaged and non-white populations (Chakraborty, 1997; Morello-Frosch and Jesdale, 2006). Studies that included qualitative assessments generally found specific instances of disproportionate distributions of environmental factors that were not revealed through geostatistical analyses (Maantay, 2001; Maroko et al., 2009; McMaster et al., 1997). Maantay and Maroko (2009) recommend further use of geospatial methods such as dasymetric mapping and similar means of disaggregating population data, particularly in heterogenous urban areas. Dasymetric mapping refers to a process of disaggregating spatial data to a finer unit of analysis, using additional (or "ancillary") data to help refine locations of population or other phenomena (Mennis 2003). This disaggregation process will result in areas of homogeneity that take into account (and more closely resemble) the actual phenomena being modeled, rather than areal units based on administrative or other arbitrary boundaries (Maantay et al., 2007).

Norton's finding (2007) that the siting of public facilities was not disproportionate by race while the siting of privately owned facilities shows discriminatory impacts raises significant policy questions. The recent shift towards privately owned solid waste facilities, public-private partnerships for solid waste management, and increasing size and decreased number of solid waste facilities pose further risk for disproportionate siting decisions. For example, private companies may be subject to less stringent siting requirements than publicly-owned or -operated facilities.

A previous literature review by Maantay (2002) on studies conducted throughout the 1990s overwhelmingly found disproportionate burdens based on race (Perlin et al., 1993, Pollak and Vitas, 1995). Most of the studies also found disproportionate burdens based on income, however, several of the studies found inverse or non-linear relationships between income and proximity to noxious facilities, which are likely similar to the "halo" effect described by Higgs and Langford (2009), who use this term to illustrate their findings that the most "deprived" populations sometimes do not live in the closest proximity to the noxious facility, but rather at a moderate distance away, creating a nimbus-like aura of deprived population around the facility or land use, with an interstitial area of relatively more affluent population, or one having a lower percentage of minority residents, sandwiched between the more deprived populations and the facility. This was not, however, a prevalent finding.

Maantay (2002) points out that many of the earlier studies conducted prior to the mid1990s focused exclusively on distributions of noxious facilities (e.g., Toxic Substance Disposal
Facilities (TSDFs), Superfund sites, Resource Conservation and Recovery Act (RCRA) sites, and
TRI facilities) and suffered from "definitional, conceptual, methodological, and data problems,
which limited their usefulness and raised questions as to the ability of GIS to assess
environmental health or equity." Some of these problems were due to factors that are integral
limitations with the datasets themselves, such as discussed above with regards to the TRI data.
The shifts in the more recent studies included in this paper toward more complex data sets
providing modeled estimates of exposure concentrations and health risks such as the NATA, and
GIS techniques such as dasymetric mapping for data disaggregation, are demonstrative of
attempts to improve the reliability and utility of environmental justice studies.

As scientists continue to examine proximity to environmental hazards and health disparities by race and SES in order to better understand the persistence of inequity in exposure

to environmental hazards and associated health risks, Morello-Frosch, Pastor, and Sadd succinctly state their recommended objectives for future research. Scientists should aim to "elucidate how institutional discrimination, uneven regional development, and a spatialized political economy shape distributions of environmental hazards, which in turn determine variations in community exposures and susceptibility to environmental hazards" (Morello-Frosch et al., 2001:572). The refinement of GISc techniques and dasymetric mapping as well as increasing access to geographically detailed data sets such as NATA pose multiple opportunities for an increasing rigor in environmental justice research that can even more effectively serve to advance just and equitable policy.

Summary of Section I and organization of the rest of the paper

In Section I, we have presented an outline of environmental health justice as an advocacy movement, a legal and regulatory framework, and a focus of research inquiry. We review research studies spanning more than two decades that demonstrate the significant spatial correspondence between the locations of environmentally burdensome facilities/land uses and communities of color and lower-income communities, as well as the disproportionate distribution of these environmental burdens. Section II presents an historical overview of analytical methods used to examine environmental health justice, showing the evolution of the methods, the increasing sophistication of analyses, and offering a constructive critique of the analytical approaches, including a discussion of methodological and data limitations. Section III addresses the question of whether or not the proximity to environmentally burdensome facilities/land uses results in actual adverse health outcomes, and to what extent these are borne disproportionately by communities of color and lower-income populations. Health outcomes examined in these studies include adverse pregnancy outcomes, childhood cancers, and cardiovascular, respiratory and other chronic diseases. Section IV offers conclusions and recommendations for improving future research on these issues, and the necessity of including, in a more systematic and robust way, the realities of environmental health justice and disproportionate health impacts in policyand decision-making.

Thus, the organization of this paper mirrors the trajectory of environmental health justice research itself: from the earlier environmental justice studies - which show that certain populations, closely predicted based on race/ethnicity and income, are disproportionately located

in proximity to environmentally hazardous facilities, and that proximity may result in increased risk of exposure - to studies showing that this increased exposure risk often results in adverse health outcomes for populations proximate to these noxious facilities and land uses.

II. Methods and Models for Measuring Disproportionate Proximity and Exposure to Environmental Hazards

This section of the review explores how proximity to environmental hazards and potential exposure to their adverse health effects have been analyzed in previous empirical studies on environmental justice (EJ). More specifically, we examine how the assessment of differential proximity and exposure to environmental health hazards in quantitative EJ research has evolved from comparing the prevalence of minority or low-income populations in geographic entities hosting pollution sources and distance-based buffer zones to more refined methods that utilize GIS, pollutant fate and transport models, and various geostatistical techniques to estimate disproportionate environmental exposure and health risks. Methods used to estimate the number and socio-demographic characteristics of people residing in areas potentially exposed to hazards are also discussed and reviewed. Spatial analytic approaches and methods reviewed in this section apply to environmental health justice studies, as well as to studies relating health outcomes to proximity to environmental hazards.

Spatial Definition of Proximity and Potential Exposure to Hazards

A variety of spatial analytic approaches have been used in the EJ research literature to measure residential proximity to environmental hazard sources and estimate the geographic boundaries of areas potentially exposed to their adverse effects. The different methodologies that have been employed in prior quantitative studies to derive the spatial definition of potential exposure can be classified into three broad categories: (a) spatial coincidence analysis; (b) distance-based analysis; and (c) pollution plume modeling. These are described and discussed in detail below.

Spatial Coincidence Analysis

Spatial coincidence, in context of environmental justice research, can be used broadly to refer to an analytic approach that assumes potential exposure to environmental hazards is confined to the boundaries of pre-defined geographic entities or census enumeration units (e.g., ZIP codes, census tracts, or block groups) that contain such hazards. This basic methodology

that has been utilized in numerous EJ studies requires the researcher to first identify locations of relevant environmental hazards on a map. The next step is to estimate either the presence of the hazard being examined or a measure of its magnitude, for each unit of analysis within the study area. All individuals residing in an analytical unit containing an environmental hazard (referred to a host unit) are considered to be living in equal proximity to that hazard, and only people living in the host unit are considered to be living in proximity to that hazard. The demographic and socioeconomic characteristics of host units are statistically compared to all other analytical (non-host) units in the study area to determine disproportionate residential proximity or exposure.

The implementation of the spatial coincidence approach for EJ analysis has also evolved and changed over time. More specifically, several different methodologies have been used in previous studies to quantify potential exposure to environmental hazards within each host unit, as shown in Table 2. The most widely used and traditional method, referred to as *unit-hazard coincidence* (Mohai and Saha, 2006), utilizes the location of an environmental hazard source within each analytical unit as a surrogate for environmental exposure or health risk. Several influential and widely-cited EJ studies conducted at the national level have used the presence or absence of hazardous facilities within each ZIP code (e.g., United Church of Christ 1987; Goldman and Fitton 1994) or census tract (e.g., Anderton et al., 1994; Been, 1995) to determine disproportionate risk burdens. National and state level EJ studies have even used the county as a spatial unit for unit-hazard coincidence analysis (e.g., Hird, 1993; Daniels and Friedman, 1999; Tiefenbacher and Hagelman, 1999).

The choice of analytical unit (county, ZIP code, census tract, or block group) to represent the host area or impacted community has been subject of considerable debate in the EJ literature (McMaster et al., 1997; Williams, 1999; Mennis, 2002). Researchers have also examined how EJ results associated with the application of the unit-hazard coincidence method varies across multiple spatial scales, or the sensitivity of this technique to the size of the spatial unit selected (Glickman and Hersh, 1995; Cutter et al., 1996; Taquino et al., 2002; Baden et al., 2007). Although these studies are not comparable because of specific dissimilarities in study area and nature of the environmental hazard examined, their findings clearly suggest that different units of analysis potentially lead to different conclusions regarding the statistical effects of specific explanatory factors (e.g., proportions of minority or low-income populations) on the presence of

hazards. Researchers have concluded that an EJ study conducted at a single spatial scale or based on particular areal unit cannot produce reliable findings because one can never tell how the analytical results are affected by the nature of data aggregation (Sui, 1999; Mennis, 2002). Data aggregated at higher levels of governmental unit such as a county or metropolitan area (coarse spatial resolution), however, have been documented to be less reliable as indicators of disproportionate burdens and less accurate in identifying affected populations, compared to data aggregated to smaller units (finer spatial resolution) such as census block groups (McMaster et al., 1997; Sheppard et al., 1999; Maantay, 2002). It is generally acknowledged that using the smallest practicable unit of analysis yields the most accurate and realistic results in terms of environmental justice and health outcomes (Maantay, 2007), although other studies have shown that the use of larger areal units (coarser spatial resolution) often increases the strength and significance of the statistical relationship between environmental risk indicators and sociodemographic variables (Cutter et al., 1996; Taquino et al., 2002).

Regardless of the particular analytical unit selected, the unit-hazard coincidence method is problematic because of three reasons. First, most applications of this methodology do not usually draw a distinction between spatial units that host one environmental hazard source and those in which two or more sources are located. A dummy variable is typically used to indicate whether or not each unit of analysis contains a hazard, thus ignoring the number of hazards within host units, as well as the quantity and toxicity of emitted pollutants. Second, this approach does not account for boundary or edge effects. These effects are concerned with the possibility that a hazardous facility could be so close to the boundary of the host unit that a neighboring spatial unit could be equally exposed to pollution. A resident in a census tract containing a hazard (host unit), for example, may live farther away from the hazard than another person in an adjacent tract which does not contain any hazards (non-host unit). Unless the hazard is located near the geographic center of the spatial unit, the representativeness of the socio-demographic data used to analyze EJ becomes questionable. Third, the unit-hazard coincidence method assumes that the adverse impacts of environmental hazards are confined only to the boundaries of their host units. It fails to consider that pre-defined geographic entities such as census units, ZIP code areas, or counties are unlikely to represent the shape or size of the area potentially exposed to the entire range of environmental health hazards associated with a polluting facility or hazard source.

Figure 1 illustrates the problematic nature of the assumptions associated with the unit-hazard coincidence method. The map depicts the distribution of 12 environmentally hazardous facilities across a subset of census tracts in a hypothetical county. Most of these facilities are located near the boundaries of multiple tracts and closer to adjacent tracts than to the far end of their own host tract. A few non-host tracts are surrounded by multiple facilities located immediately outside their boundaries, potentially facing greater exposure to hazards than some of the host tracts. Given the spatial distribution of these hazardous facilities, it appears unlikely that their adverse impacts are confined solely to their host census units. This problem becomes more pronounced when the size of census units vary substantially within a study area. An additional limitation is that all tracts hosting hazardous facilities are treated equally, although the number of facilities within each host tract is not identical in Figure 1.

The inability to distinguish between host analytical units based on the number or magnitude of hazards can be addressed by summing the number of hazardous facilities located in each unit or the amount of pollutants released within each unit in a given study area. Instead of treating all host units equally, several EJ studies have extended the basic spatial coincidence approach by estimating either the total number of hazards, total volume of emitted pollutants, or toxicity-weighted volumes of all emitted pollutants associated with each host unit. The summation technique has been used to enumerate the frequency of toxic facilities within census tracts (Burke, 1993; Fricker and Hengarner, 2001) and ZIP codes (Ringquist, 1997), as well as the number of airborne toxic releases within counties (Cutter and Solecki, 1996). Since certain EPA databases such as the Toxic Release Inventory provide detailed data on annual quantities of toxic chemicals released at each facility, a more refined assessment of the magnitude of pollution associated with each host spatial unit is possible. While several EJ studies have relied on the total pounds of emitted pollutants from TRI facilities, others have used data on chemical toxicity indicators to weight annual release quantities and compute toxicity-adjusted emissions for each spatial unit in the study area, as shown in Table 2. Since the TRI database does not include toxicity data for released chemicals, researchers have use surrogate measures such as threshold limit values (TLVs) to weight the pounds of emissions for each pollutant. Although TLVs are available for many of the chemicals on the TRI list, it remains a problematic index for health risk and equity assessment because it was developed and intended to only assess occupational safety among a healthy worker population (Maantay, 2002).

The incorporation of data on the quality and quantity of pollution emitted from each hazard source have allowed researchers to distinguish between host spatial units on the basis of the magnitude of potential environmental risk and thus improve upon the basic unit-hazard coincidence method that examines the mere presence of hazards. Applications of spatial coincidence analysis that utilize emissions or toxicity data, however, are still limited by their inability to: (a) consider the exact geographic location of the hazard within the host spatial unit; and (b) determine the geographic extent of potential exposure to the hazard. Although the spatial coincidence approach facilitates statistical comparisons between host and non-host areas, it assumes that exposure to environmental hazards is distributed uniformly within and confined only to the boundary of the spatial unit containing a hazard.

Distance-Based Analysis

In order to address the limitations of the spatial coincidence approach, EJ studies have analyzed residential proximity on the basis of the distance from hazardous facilities to nearby spatial units. A variety of simple and advanced distance-based techniques have been suggested and implemented for measuring disproportionate proximity or exposure to environmental hazards in the EJ research literature. The most widely used method is commonly known as buffer analysis. Buffer generation is a spatial analytic technique provided by GIS software programs for creating new polygons around point, line, or area features on a map. Since the mid-1990s, a large number of EJ studies have used GIS-based circular buffers of various radii around point sources of hazards to identify areas and populations exposed to their adverse effects (Table 2). The demographic and socioeconomic characteristics of areas lying inside such buffer zones are statistically compared to the rest of the study area (outside the buffers) to determine disproportionate residential proximity or exposure to the hazards of concern. Figure 2 provides a typical example of buffer analysis, based on circles of radii one-half mile centered at each hazardous facility in our hypothetical county. The underlying census tracts can be used to estimate the characteristics of the population residing within these buffer zones, as described later in this section.

The radius of circular buffers in EJ studies have ranged from 100 yards (Sheppard et al., 1999) to 3 miles (U.S. GAO, 1995; Mohai and Saha, 2006). Distances of 0.5 and 1.0 mile from facilities of concern, however, have been used most frequently (Glickman, 1994; Zimmerman,

1994; Chakraborty and Armstrong, 1997; Neumann et al., 1998; Bolin et al., 2000; Baden and Coursey, 2002; Boone, 2002; Harner et al., 2002; Maantay, 2007; Kearney and Kiros, 1999; Mohai et al., 2009). Instead of using a single radius or buffer, several studies have also constructed three or more circular rings at increasing distances from environmental hazard sources (e.g., Neumann et al., 1998; Perlin et al., 1999; Sheppard et al., 1999; Atlas et al., 2002; Perlin et al., 2002; Pastor et al., 2004; Walker et al., 2006). To represent relative risk and differentiate between buffers, some EJ studies have estimated pollutant concentrations, release volumes, or toxicity-weighted emissions within a fixed radius of each TRI facility in the study area (e.g., Bolin et al., 2002; Neumann et al., 1998; Harner et al., 2002).

Circular buffer analysis provides a more accurate or realistic geographic representation of potential exposure to hazards than the spatial coincidence because it does not assume that the adverse effects are restricted solely to the boundaries of pre-defined analytical units hosting the hazard. There are, however, specific limitations associated with its application in EJ analysis. First, the facility or hazard representing the center of the circle is assumed to be small enough to be treated as a point. For undesirable land uses such as Superfund sites that are large in size, a generated circular buffer may not accurately depict the area surrounding the site if the radius is too small. Some hazardous sites, therefore, need to be delineated as a polygon instead of a point and the buffer should be constructed around the polygon (Liu, 2001). Although applications of the buffer method for EJ analysis have not considered this issue, the shape and size of the hazard source needs to be first examined before deciding which type of buffer is appropriate. A second limitation is the radius of the circular buffer in most EJ studies is chosen arbitrarily and buffers around all hazards in a study area usually have the exact same radius. The properties and quantities of hazardous substances stored or released at each individual facility have been rarely incorporated in the determination of buffer radii to reflect the spatial extent of environmental exposure. The operational parameters of emission releases (e.g., release height, exit velocity, exit temperature) are also not considered in the determination of the buffer size. An additional limitation of the circular buffer and other distance-based approaches is the implicit assumption that the emissions from a facility are uniformly dispersed in all directions around the facility without respect to prevailing wind patterns.

The circular buffer method remains a widely-used approach because it can be easily implemented using GIS software, provides a simple visual representation (circles centered at

point sources), and makes statistical comparisons between potentially exposed (inside circle) and non-exposed (outside circle) areas and populations convenient. An important assumption of this method, however, is that the adverse effects of a hazard are limited only to the specified circular area or distance. EJ studies using this technique thus consider only areas within a fixed distance of hazards to be potentially exposed to pollution and areas outside to remain unaffected. While this binary or dichotomous assumption makes comparisons convenient, the results are highly sensitive to the choice of buffer radius, as demonstrated in previously mentioned studies utilizing more than one circle around facilities of concern. In addition, a discrete measurement such as 'within 1 mile of a facility' is unlikely to reflect a more continuous and gradual reduction in environmental exposure with increasing distance from the hazard (Waller et al., 1997). Using multiple circular buffers can overcome this limitation to a certain degree, but the determination of the number of buffers to use and choice of radii for each buffer remain ambiguous and do not necessarily result in a more accurate representation of potential exposure (Zandbergen and Chakraborty, 2006). In any event, this characterization still results in a binary analysis (exposed vs. not exposed) and even with additional buffers provides an ordinal comparison (e.g., high, medium, or low levels of exposure, and no exposure) based on tenuous assumptions.

Continuous distances, based on the calculation of the exact distance between each hazard and the locations of the potentially exposed population, represent an alternative to the use of discrete distances. Several EJ studies have utilized the distance from the centroid of each census tract or block group to their nearest hazard source as an indicator of residential proximity (Pollock and Vittas, 1995; Gragg et al., 1995; Stretesky and Lynch, 1999; Margai, 2001; Mennis, 2002). The description and analysis of continuous distances can be enhanced through the use of a cumulative frequency distribution (CDF). A CDF is essentially a graph that depicts the number or percentage of observations falling below every threshold value. Applied to any set of hazardous facilities, a CDF can be plotted as distance versus a potentially exposed population and would indicate how the size of this population (as a percentage of the total in the study area) increases with proximity. The vertical axis of a CDF curve typically represents the percentage of the population (from 0 to 100), and the horizontal axis represents potential exposure, typically measured as distance from the hazard. If the exact locations of each member of the population are unavailable and the analysis is based on data aggregated to spatial units (e.g., census tract or

block group), the point-to-point distance from each census unit centroid to the nearest hazard source can be used to generated a CDF curve.

Figure 3 provides an illustration of a pair of CDF curves that compare the location patterns of two racial subgroups with respect to a set of hazardous facilities in a hypothetical county. The CDFs representing the cumulative proportions of the non-White and White population in the county are depicted as a function of distance to the nearest facility, estimated on the basis of block group data. The vertical axis shows the cumulative number of non-White and White residents as a percentage of their respective county totals. The collective percentage of the population in each racial subgroup increases from 0 to 100 as distance from the facilities increase. If the CDF curve for non-Whites is higher than the curve for Whites at any specific distance, the percentage of the county's non-White population residing within that distance of their nearest facility exceeds the corresponding percentage of the White population. According to Figure 3, approximately 13.0 percent of non-White residents in this county reside in block groups located within a mile of the polluting facilities, compared to about 12.4 percent of the White population. Almost 50 percent of non-Whites and only 40 percent of Whites reside within a distance of two miles from these facilities. While the gap between the two curves increases substantially with distance from the facilities, the CDF for non-Whites always remains higher than the CDF for Whites and suggests a racially inequitable location pattern in this county.

The limitations of conventional buffer analysis based on arbitrary and discrete distance values can also be assessed from Figure 3. A circular buffer of radius smaller than 0.75 mile would indicate almost similar percentages of non-White and White residents, or the lack of evidence for disproportionate proximity. A buffer analysis based on a radius of 1 mile or 2 miles, in contrast, would yield a significantly higher non-White proportion and a different statistical result. Since discrete buffer distances are typically chosen without any knowledge of the actual empirical CDF, our results from Figure 3 indicate that this approach is likely to result in an incomplete characterization of environmental exposure and potentially lead to biased results.

Several EJ studies have demonstrated that the CDFs are particularly well-suited for assessing disproportionate proximity because they overcome the limitations of choosing arbitrary and discrete buffer distances (Waller et al., 1997; 1999; Zandbergen and Chakraborty, 2006; Chakraborty and Zandbergen, 2007). These studies also indicate the usefulness of CDFs in

comparing various subgroups of the residential population, to evaluate proximity patterns on the basis of race (e.g., White vs. Black), ethnicity (e.g., Hispanic vs. non-Hispanic), or economic status (e.g., below poverty vs. above poverty level).

While most proximity-based analyses of EJ assume that the adverse effects of an environmental hazard decline with increasing distance in a linear fashion, a few studies have utilized curvilinear distance decay functions to model residential proximity. Pollock and Vittas (1995) hypothesized three functional forms of exposure (linear, square root, and natural logarithm) with respect to distance from TRI facilities in Florida. They suggested that the square root function represents a gentle decline in slope, but the logarithmic function provides a steeper slope. The natural logarithm of the distance to the nearest facility was selected as a proxy for exposure. A GIS-based distance decay modeling technique was recently developed by Downey (2006) and applied to estimate residential proximity to TRI facilities in Detroit. This technique also weights each hazard's estimated adverse effect by distance such that the adverse effect declines continuously with distance from the hazard, thus providing more accurate estimates of proximity-based exposure. While this technique was found to be flexible enough to incorporate any appropriate distance decay function, several different curvilinear and reverse curvilinear functions were used to estimate neighborhood proximity to TRI activity.

An inherent limitation of this distance decay approach is that researchers are unaware of the actual and precise rate at which the adverse impacts of an environmental hazard decline with increasing distance (distance decay rate). The mathematical functions used to calculate distance decay are typically based on assumptions about the distance decay process rather than on precise knowledge of the process (Downey, 2006). Regardless of the distance decay rate or function utilized, it is important to consider that distance serves only as a surrogate for exposure and the actual extent of human exposure may not be a simple function of distance. In reality, distance decay functions are likely to vary substantially based on circumstances of release, types and quantities of substances released, and local factors (e.g., wind, temperature, topography).

Although distance-based approaches for EJ analysis have evolved from the use of discrete buffers to continuous functions, there are still limited by the fact that mere proximity many not always provide a valid proxy for exposure to environmental pollution. Additionally, the buffer method and the continuous distance approach fails to consider directional biases in the distribution of environmental hazards by assuming that their adverse effects are equal and

uniform in all directions. A resident who lives one mile upwind from a hazardous facility, however, is unlikely to face the same level of toxic exposure compared to someone living a mile downwind from the same facility. Although physical processes do not operate in a perfectly symmetrical (isotropic) manner, distance-based analyses of EJ assume that exposure is invariant to prevailing wind direction and other factors that influence the movement and dispersal of emitted pollutants.

Pollution Plume Modeling

To provide a more realistic and accurate representation of areas potentially exposed to the adverse effects of a hazard, several EJ studies have utilized detailed information on toxic chemical emissions and local meteorological conditions to model the environmental fate and dispersal of pollutants released from each hazard source. *Geographic plume analysis* is a methodology that integrates air dispersion modeling with GIS to estimate areas and populations exposed to airborne releases of toxic substances (Chakraborty and Armstrong,1996; 1997). Dispersion models typically combine data on the quantity and physical properties of a released chemical with site-specific information and atmospheric conditions to estimate pollutant concentrations downwind from the emission source. This information is then used to identify the geographic extent or boundary of the area potentially exposed to the chemical's spreading plume--the plume footprint. The footprint represents the area where ground-level concentrations of the pollutant are predicted to exceed a user-specified limit or threshold level (Figure 4).

Most applications of geographic plume analysis for EJ research have relied on ALOHA (Areal Locations of Hazardous Atmospheres), an air dispersion model designed to support emergency responses to hazardous chemical accidents. ALOHA is a computer modeling package distributed by the National Safety Council in cooperation with the National Oceanic and Atmospheric Administration (NOAA) and the EPA that is designed for estimating plume extent and concentration for short-duration chemical releases. Previous studies of disproportionate exposure to environmental hazards have used the ALOHA model to generate, at each facility of concern, a single plume footprint (Chakraborty and Armstrong, 1996), a composite footprint reflecting historical weather patterns (Chakraborty and Armstrong, 1997; 2004), or plume-based circular buffers whose radii are based on worst-case chemical release scenarios (Chakraborty, 2001; Margai, 2001; Chakraborty and Armstrong, 2001). In order to delineate the boundaries of

adversely impacted areas, other EJ studies have used the Industrial Source Complex Short Term (ISCST) air dispersion model (Dolinoy and Miranda, 2004; Fisher et al., 2006; Maantay et al., 2009), ash deposition models (Bevc et al., 2007), and various noise pollution models (Chakraborty et al., 1999; Most et al., 2004).

The application of pollutant fate and transport modeling allows the concentration of toxic pollutants released from a hazard source and their estimated health risks to: (a) decline continuously with increasing distance from the emitting source; and (b) vary according to compass direction. Plume-based buffers are thus capable of addressing the problems of previous analytic approaches which assume that residing in either a census unit containing a hazard (spatial coincidence) or within a specific distance from a hazard (distance-based) results in environmental exposure and adverse health risks. These are, however, certain limitations associated with plume modeling. First, air dispersion models typically required large volumes of site-specific and facility-specific information, such as the facility's stack height and diameter, gas exit velocity and exit temperature, accurate emissions data on each chemical released (e.g., average hourly quantities and rates), as well as detailed meteorological information (e.g., average monthly or hourly wind speed and direction). The input data necessary for plume modeling is rarely available for all environmental hazard sources in a given study area (Maantay, 2007). Simplifying assumptions are frequently made when such information is unavailable, leading to inaccurate estimates of the potentially exposed area or population. Second, some dispersion models such as ALOHA assume that topography is always flat and are unable to provide accurate concentration estimates when the atmosphere is stable or wind speeds are very low. Third, the creation of plume modeling data sets that encompass all toxic facilities and chemical emissions in a large geographic area is a time-consuming and expensive process (Downey, 2006). As a consequence, a limited number of large scale plume model data sets have been constructed and those that exist are limited to specific types of hazards.

Data sets derived from pollutant fate and transport modeling that cover the entire U.S. include the Risk-Screening Environmental Indicators (RSEI) and National-Scale Air Toxic Assessment (NATA). These national scale databases developed by the EPA are particularly appropriate for EJ research, not only because they allow researchers to estimate the potential health risks associated with specific environmental hazards and specific analysis units, but also because the plume modeling and risk assessment techniques used to derive these data take into

account factors such as wind speed, wind direction, air turbulence, smokestack height and the rate of chemical decay and deposition.

The Risk-Screening Environmental Indicators (RSEI) model can be used to estimate potential human health risks from air pollutants based on toxicity and atmospheric dispersion of chemicals emitted by facilities in the TRI database. For each individual TRI site and pollutant, the RSEI integrates information on the facility location, the quantity and toxicity of the chemical, fate and transport through the environment, the route and extent of human exposure, and the number of people affected for up to 44 miles (101 km) from the source of release. The ambient concentrations of each TRI pollutant is determined for each square kilometer of the 101-km by 101-km grid in which the facility is centered. The model uses standard assumptions about human exposure to derive a surrogate dose which is an estimate of the amount of chemical contacted by an individual per kilogram of body weight per day. The RSEI combines chemical-specific toxicity weights with the surrogate dose delivered by each release to obtain a partial score for each square kilometer cell that represents the relative toxicity-adjusted potential human health effects from chronic exposure. These partial scores from chemical releases at different facilities are summed to obtain a cumulative score for each 1 km by 1km grid cell that can be used evaluate the potential for chronic health risk within a study area.

EJ studies have merged risk scores from the RSEI grids with census demographic data to analyze disproportionate exposure to TRI pollutants in the entire U.S. (Bowes et al., 2001; Ash and Fetter, 2004) and in Philadelphia (Sicotte and Swanson, 2007). Since the toxic pollution plumes used to obtain the risk estimates can extend in any direction for up to 44 miles from a TRI facility, the RSEI modeling technique had the advantage of allowing hazards and emissions in one unit of analysis to affect people living in other units. While this represents a distinct improvement over the spatial coincidence or distance-based approaches, there are several problematic assumptions associated with the RSEI. Each facility in the RSEI database, for example, is given a single smokestack height estimate. Many industrial facilities, however, have multiple smokestacks of varying height. Smokestack height estimates are often based on the median smokestack height for an entire industry (based on the facility's SIC code) and in the RSEI model stack height is assumed to have has the greatest impact on predicted concentrations of air pollutants. Additionally, the RSEI model assumes constant emissions rates and uses chemical decay estimates that are not necessarily accurate (Downey, 2006).

The National-Scale Air Toxic Assessment (NATA) was designed to guide air pollution reduction and related prioritization efforts, has also emerged as a valuable data set for estimating exposure concentrations and public health risks associated with inhalation of air toxics from different types of sources. While criteria air pollutants include common contaminants such as particulate matter, sulfur dioxide, nitrogen oxides, ozone, carbon monoxide, and lead, air toxics (also known as hazardous air pollutants) include 188 specific substances identified by the U.S. Congress in the Clean Air Act Amendments of 1990. Air toxics are known to or suspected of causing cancer and other serious health problems, including respiratory, neurological, immune, or reproductive effects (U.S. EPA, 2008). The methodology used to generate estimates of health risk for the NATA comprises several steps. The National Emissions Inventory (NEI) serves as the data source on air toxics emissions from various stationary and mobile outdoor sources. The NEI data is used as input to a Gaussian dispersion model that accounts for atmospheric decay to provide an estimate of the annual ambient concentration of air toxics. Estimates of ambient concentrations from the ASPEN are then included as input in an inhalation exposure model that incorporates activity patterns that may influence personal exposure to ambient pollutants. From these exposure concentrations, the NATA estimates potential public health risks (cancer, respiratory, and neurological) from inhalation of air toxics following the EPA's standard risk characterization guidelines, which assume a lifelong exposure to current levels of air emissions. The census tract is the smallest spatial unit for which estimates of exposure and health risk are provided in the NATA.

Tract level estimates of lifetime cancer risk from the 1996 NATA have been utilized for EJ analysis in Maryland (Apelberg et al., 2005), California (Pastor et al., 2005), and 309 metropolitan areas of the U.S. (Morello-Frosch and Jesdale, 2006). Recent studies have used the 1999 NATA to examine the disproportionate distribution of cancer and respiratory risks in Florida (Gilbert and Chakraborty, 2008), and the metropolitan areas of Houston (Linder et al., 2008) and Tampa Bay (Chakraborty, 2009). An important advantage of the NATA is spatial compatibility with census demographic data—the modeled risk estimates are available at the level of spatial units (tracts) that provide population and housing characteristics. In addition, this database provides cumulative health risk estimates for ambient exposure to multiple types of toxic emission sources. The NATA thus allows EJ analysis to extend beyond major stationary sources such as TRI facilities and include smaller emitters, as well as various off-road and on-

road mobile sources. However, the NATA is somewhat limited by its specific focus on only one category of air pollutants (HAPs) and one avenue of human exposure (inhalation) to air toxics. The adverse health risks of exposure to criteria air pollutants and through other pathways such as ingestion and skin contact are not taken into account.

Although plume modeling techniques and data sets represent a significant improvement over the spatial coincidence and distance-based approaches, these are often based on necessary and problematic assumptions and may not be as accurate as many researchers think. More importantly, their use is limited to particular research questions (those having to do with specific public health risks) and hazards (those covered by the plume models). It is important to consider that health risks associated with exposure to pollution may not be the only set of risks imposed by environmental hazards. The presence of a hazard can also adversely affect nearby property values, perceptions of local health risks, psychological stress, local employment opportunities, sense of community and local economic activity (Downey, 2006). These potential negative impacts cannot be analyzed on the basis of plume modeling methods and data.

Estimating Characteristics of Proximate Populations

After delineating the geographic boundaries of areas potentially exposed to the adverse effects of environmental hazards, EJ studies have employed a variety of spatial analytic techniques to estimate the demographic and socioeconomic characteristics of the population residing in such areas. These techniques can be classified into two basic categories, depending on the level of spatial aggregation of the residential population data: *point interpolation* and *areal interpolation*. When the addresses of all individuals or households relevant to the study are available and can be located on a map, **point interpolation** is the appropriate method. Street address information is typically used in conjunction with a detailed street network to determine an accurate location of each individual or household in the study area, utilizing the geocoding capabilities of commercial GIS software. The total number and the socio-demographic characteristics of individuals or households potentially exposed to an environmental hazard can be estimated on the basis of points that fall inside a distance-based or plume-based buffer zone (point-in-polygon overlay). The earliest application of this method can be found in a study conducted by Mohai and Bryant (1992) on hazardous waste facilities in Detroit. Data from a metropolitan-wide probability sample survey were utilized to determine if the racial and

economic status of respondents living within circular buffers of radii 1 mile and 1.5 miles were different from respondents who reside outside the buffers. Subsequent EJ studies using distance-based and plume-based approaches have relied on point interpolation to estimate the number of self-identified individuals with special needs in Cedar Rapids, Iowa (Chakraborty and Armstrong, 2001), various characteristics of survey respondents in Fort Lauderdale, Florida (Bevc et al., 2007), and the racial/ethnic status of school children in Orange County, Florida (Chakraborty and Zandbergen, 2007). These studies utilized the address matching functionalities of GIS software to geocode the locations of relevant individuals to the street network of their respective study areas. A more recent study (Mohai et al., 2009) utilized addresses of survey respondents in the American Changing Lives Study to examine the racial/ethnic and socioeconomic characteristics of residents living within one mile of TRI facilities.

Although point interpolation can be easily implemented to estimate potentially exposed populations effectively and accurately, a major limitation is data availability on the street addresses of all individuals and households relevant to the analysis. Individual or household level data on demographic and socioeconomic characteristics of residents are not publicly available and can only be obtained through an extensive social survey that encompasses the entire study area. Consequently, EJ studies have relied mainly on socio-demographic information collected by the U.S. Census Bureau and other agencies that are commonly aggregated at the level of pre-defined administrative boundaries or census enumeration units. If the area potentially exposed to a hazard is represented by a distance-based or plume-based buffer, the shape and size of the buffer area is unlikely to match the underlying administrative or census units that contain aggregated population data (see Figure 2 or Figure 4). A method of areal interpolation (polygon-on-polygon overlay) is necessary to transfer data from census units (source zone) to the boundaries of areas potentially exposed to the adverse effects of a hazard (target zone). Several different areal interpolation techniques have been utilized in previous EJ studies to accomplish this objective. These are described below and illustrated in Figures 5 to 7, using a circular buffer around a single facility of concern.

The simplest method is *polygon containment*, where all spatial units or census polygons that either intersected or entirely enclosed by a distance-based or plume-based buffer are selected (Chakraborty and Armstrong, 1997; Liu, 2001). Also referred to as *adjacency analysis* (Most et al. 2004) or the *boundary intersection method* (Mohai and Saha, 2006), population

characteristics of any given buffer zone are derived through a simple aggregation of all census units that are within or in contact with the buffer (Figure 5). The effective buffer zone obtained through this method, however, will not resemble the original buffer (i.e., circle or plume footprint) because it is based on the boundaries of the selected census polygons. The polygon containment method also does not make any distinction between census polygons that are completely enclosed and those that are partially enclosed or barely touched by the original buffer. This could lead to a severe overestimation of the potentially exposed population if people residing in a partially enclosed census unit are actually concentrated in a particular section of the unit that falls outside the buffer boundary. A variation of this method is to use a cutoff criteria to limit the inclusion of census polygons that are partially contained within the given distancebased or plume-based buffer. The area of the census enumeration unit intersected by the buffer and the proportion of its area located inside the buffer are first calculated. A decision rule is then implemented to include only those census polygons that have a significant overlap with the designated buffer zone. The most common practice is to include census units that have more than half of their area within the circle or plume used to represent the buffer. This variation of the polygon containment method is often referred to as 50% area containment method (Mohai and Saha, 2006; 2007).

The second method for estimating population characteristics within a buffer zone is known as *centroid containment* (Chakraborty and Armstrong, 1997; Maantay and Maroko, 2009). This technique selects only those census polygons whose geographic centers or centroids are located within the buffer, thus limiting the number of census units that can be included (Figure 6). It assumes that a point (centroid) represents the entire polygon in terms of its population characteristics. Like the polygon containment method, the shape and size of the effective buffer zone will not resemble the original circle or plume. Additionally, this method is likely to provide inaccurate estimates of the potentially exposed population if the actual residences of people in census units intersected by the buffer are not concentrated near the centroid of the unit. For plume-based buffers that are irregular in shape and become narrower at the source, this method could lead to an underestimation of the population exposed to a hazard. A census enumeration unit that contains the hazard, for example, can be excluded if its centroid is located outside the plume footprint used to represent the buffer.

The third and most widely-used method is buffer containment (Chakraborty and Armstrong, 1997; Liu, 2001). Instead of either completely including or excluding partially enclosed polygons, this method includes all census units lying within the buffer and a fraction of the population from units that are interested by the buffer (Figure 7). Recent studies have referred to this approach as the *areal apportionment* method (Mohai and Saha, 2006; Kearney and Kiros, 2009). Unlike the other containment techniques, the buffer containment method has the advantage of retaining the area and shape of the original circle or plume used to delineate the buffer zone. An areal weighting technique is typically utilized to determine the population characteristics of the buffer zone (Maantay and Maroko, 2009). Specifically, the population of each census unit is weighted by the proportion of its area that falls inside the circular or plumebased buffer. An important limitation of this technique is the assumption that the population of a census unit and all its characteristics are distributed uniformly within its boundary. The application of the buffer containment method could thus lead to inaccurate estimates of the potentially exposed population if the actual residences of people within a census unit are concentrated in specific areas instead of being dispersed throughout the unit. The assumption of uniform distribution becomes particularly problematic for areal weighting when underlying census units are larger in size. As an alternative to areal interpolation, one EJ study has utilized a hybrid method known as cross-area transformation (Most et al., 2004). The population characteristics of census units that are intersected or partially contained within a buffer are estimated by borrowing data from the completely contained census units, based on the remaining area of the buffer zone that falls outside the boundaries of all fully contained units. In addition to completely excluding census units intersected by the buffer, this method assumes that any census unit completely enclosed by a given buffer will adequately reflect the characteristics of people living in the larger area.

All of these methods have been widely employed in the analysis of disproportionate exposure to hazards and no single best technique has emerged. The application of *dysametric mapping* in combination with areal interpolation has been suggested as a promising approach (Holt et al., 2004; Zandbergen and Chakraborty, 2006; Maantay et al., 2007). Dysametric mapping is a technique that uses ancillary information to redistribute spatial data in a more accurate and logical manner (Mennis, 2002). This technique can be used to develop a more refined distribution of the population residing within a census enumeration unit using land cover

information. However, dysametric mapping is somewhat cumbersome to carry out reliably and is very sensitive to the assumptions about the population density difference among land cover categories. Recent studies suggest that cadastral dysametric mapping represents a meaningful improvement on the use of the aggregated demographic data when geocoded locations of individuals or households are unavailable (e.g., Maantay and Maroko, 2009; Maantay et al., 2008). Figure 8 provides an example of how a map showing the boundaries of land parcels can be used to estimate households residing within one-half mile of a hazardous facility. Additional details such as housing tenure, ownership, and values can also be utilized to assess socioeconomic status of proximate households.

Geostatistical Techniques for Assessing Disproportionate Proximity and Exposure

A variety of statistical methodologies have been employed in EJ research to determine if race/ethnicity and economic status is statistically related to indicators of proximity or exposure to environmental hazards. To compare the demographic and socioeconomic characteristics of proximate areas (census units or buffer zones) that contain hazards to areas that do not contain hazards, several researchers have used two-sample inferential tests of means and proportions. Most studies, however, have relied on linear correlation or multivariate regression analysis to establish the statistical significance of the association between environmental risk and relevant characteristics of the residential population. The dependent variable in regression models for EJ analysis has been represented by the presence of hazards (Burke, 1993; Sadd et al., 1999; Pastor et al., 2004; Mohai and Saha, 2006; Baden et al., 2007), distance to hazards (Pollock and Vittas, 1995; Stretesky and Lynch, 1999; Mennis, 2002), quantity of pollutants released (Bowen et al., 1995, Ringquist, 1997; Daniels and Friedman, 1999), toxicity-weighted emissions (Brooks and Sethi, 1997; Ash and Fetter, 2004; Sicotte and Swanson, 2007), and estimated health risks of exposure to air toxics (Morello-Frosch et al., 2001; Pastor et al., 2005; Gilbert and Chakraborty, 2008). While least squares regression is an effective and widely used technique for measuring the strength and significance of relationships between the dependent and multiple explanatory factors, it is based on two assumptions (independence and homogeneity) that are rarely met by spatially distributed data and variables.

Spatial Dependence and Spatial Autoregressive (SAR) Models

The independence assumption of linear regression ignores the notion that locational proximity often results in value similarity when most demographic or socioeconomic variables are mapped. This basic concept was articulated by Tobler (1970, 236) as "everything is related to everything else, but near things are more related than distant things" and is known as Tobler's first law (TFL) of geography. The practical implication of TFL is that observations from nearby locations are often more similar than what can be expected on a random basis. This phenomenon is known as spatial dependence, and more formally as positive spatial autocorrelation. The presence of such autocorrelation can be problematic for standard statistical tests such as correlation and regression that assume independently distributed observations and errors. Regression analysis of spatially distributed variables can thus lead to incorrect statistical inference regarding model coefficients when spatial autocorrelation is present and when model specifications fail to include proper corrections for spatial dependence. Although EJ analysis is based on spatial data, most previous studies have assumed observations and error terms to be independent, thus violating one of the classical regression assumptions and ignoring spatial effects that could lead to incorrect inferences about the significance of key explanatory variables such as the proportion of racial/ethnic minorities or people in poverty.

A large body of literature in geographic analysis has focused on the development of methods that can be used to detect violations of the independence assumption and models that account for spatial autocorrelation in the data (e.g., Cliff and Ord, 1981; Anselin and Bera, 1988; Anselin, 2005). Spatial regression models, such as simultaneous autoregressive (SAR) models, are statistical models that consider spatial dependence as an additional variable in the regression equation and estimate its effect simultaneously with effects of other explanatory variables. The use of spatial regression has increased in recent years, in part, due to the availability of user-friendly spatial analysis software programs such as *GeoDa* (Anselin, 2005) that are that capable of implementing the underlying spatial econometric techniques. With regards to EJ research, recent studies have begun to utilize SAR models that explicitly consider the effects of spatial dependence in the data (Pastor et al., 2005; Grineski and Collins, 2008; Chakraborty, 2009). Although SAR model performance and results depend on several analytical choices related to model specification (lag or error) and the approach used to select spatial neighbors (contiguity or distance), these EJ studies demonstrate the effectiveness of spatial regression models in reducing

spatial dependence of residuals and satisfying the assumption of independently distributed errors in regression.

Spatial Heterogeneity and Geographically Weighted Regression (GWR) Models

In addition to independence, the classical linear regression model assumes a generating process that is considered to be stationary or homogeneous. When applied to a regression model, this means that a single set of global parameters is adequate to describe the process and there are no spatial variations in the relationships between the dependent and independent variables. It has become increasingly evident, however, that local variations in statistical relationships can play a significant role in the analysis of spatial data. The use of a single or 'global' regression model for an entire study area assumes model parameters do not vary spatially and thus ignores local differences in statistical associations between the dependent and independent variables. Since conventional multivariate regression models that are used in EJ research do not account for this spatial variability and only provide global results for the whole study region, they have the potential to mask important geographic differences in relationships and ignore local processes.

Geographically weighted regression (GWR) is a local spatial statistical technique used to analyze spatial nonstationarity, defined as when the measurement of relationships among variables differs from location to location (Fotheringham et al., 2002). There are two ways in which GWR differs from conventional linear regression. First, a separate regression is carried out at each location or observation using only the other observations that fall within a user-specified local area surrounding that location. Second, the technique includes a statistical device that weighs the attributes of nearby observations within the local area more highly compared to the attributes of distant observations. Instead of generating a single global regression equation or one set of regression parameters for an entire study area, GWR produces a separate regression equation or a unique set of parameters for each observation or spatial unit. Maps generated from GWR analysis can be used to explain and interpret how various regression diagnostics and the strength of statistical relationships differ from place to place within a study area.

Figure 9 illustrates, for example, how the nature and significance of the statistical association between lifetime cancer risk from minor point sources of air toxics (1999 NATA) and four specific explanatory variables vary across census tracts in the state of Florida. For each variable or GWR model coefficient, red shading is used to depict tracts showing a positively

significant t-statistic, blue shading is used to show tracts with a negatively significant t-statistic, and the grey shading is used to display tracts with a non-significant t-statistic. These maps clearly indicate that the relation between environmental health risk and the presence of racial/ethnic minority or impoverished residents could be significantly positive in some areas, significantly negative in other areas, and not significant at other locations, within the same state or study region. Traditional multivariate regression, however, is incapable of uncovering these spatial variations in statistical relationships and could potentially lead to incorrect conclusions regarding disproportionate proximity or exposure to environmental hazards.

With regards to EJ research, only one published article has used GWR to examine disproportionate proximity to hazards (Mennis and Jordan, 2005). This study focused on modeling the statistical association between the density of TRI facilities and race/ethnicity and other socioeconomic factors in New Jersey. Maps based on the output from GWR analysis were used to illustrate how relationships among race, class, employment, urban concentration, and land use with facility density vary significantly over space within the state. This study and other recent studies mentioned in this section demonstrate and emphasize the necessity to advance statistical methodologies for measuring the spatial association between exposure to environmental hazards and the racial/ethnic or socioeconomic characteristics of the residential population. Instead of relying only on conventional statistical methods, there is growing need for future research on disproportionate proximity or exposure to incorporate geostatistical techniques that are particularly appropriate for analyzing spatial data and relationships.

Limitations and Data Needs

This review has explored how the assessment of proximity and potential exposure to environmental hazards in quantitative EJ research has emerged from simple coincidence or distance-based methods to more sophisticated techniques that utilize pollutant fate and transport models or provide modeled estimates of health risks from cumulative exposure to multiple pollutants and emission sources. Methodological debates have also evolved from the choice of ZIP code or census tract for coincidence analysis to the selection of appropriate distance decay functions or geostatistical techniques that are more suitable for analyzing spatial data, variables, and relationships. In spite of the methodological improvements in measuring disproportionate proximity and exposure to hazards, this research still remains constrained by several limitations.

First, a majority of EJ studies have focused exclusively on night-time exposure to environmental hazards by utilizing demographic and socioeconomic data from the U.S. Census. Since census variables represent residential or night-time populations, they cannot be used to assess day-time risk. Most studies implicitly assume that people are non-mobile and are not exposed to pollution at non-residential locations. However, daily mobility typically results in residents moving to and from various locations, such as to work or to school. The journey-towork commute between the suburbs and central cities could have important implications for EJ research and policy. A large number of affluent and White suburban residents, for example, could be spending a considerable amount of time during the day in census units near downtown locations that are exposed to adverse health risks. At the same time, minority residents who commute daily to suburban job locations may face lower levels of exposure for most of the day, thus reversing the inequity patterns reported in empirical studies (Chakraborty, 2009). In addition to geographic mobility, other factors that are not accounted for by the use of environmental exposure at residential locations as a surrogate for personal exposure include differences between ambient concentrations and those indoors or in vehicles. Although these factors are now considered by the NATA in its estimation of exposure concentrations and health risks, the analysis of EJ and disproportionate impacts is still based on census socio-demographic data which represents night-time residences of people. Future EJ research, however, should explore the use of additional data sources to construct temporally sensitive models that examine the day-time distribution of urban populations. Available data that can be used for this purpose include those on employment and people in day-time institutions such as schools and daycare centers (McMaster et al., 1997). Such information can be used to develop an independent model of the population distribution for the hours of 7 am to 5 pm to complement census residential data. The EPA's new Consolidated Human Activity Database (CHAD) is another useful resource that can be utilized for this purpose.

A second limitation in assessing disproportionate proximity or health effects is the difficulty in obtaining data at a spatial resolution that is sufficiently detailed to reliably demonstrate the connection between environmental conditions and potential exposure or adverse health outcomes. As mentioned previously, the lack of address-specific and individual level information forces most EJ researchers to use socio-demographic data that are tied to analytical units such as ZIP codes, census tracts, or block groups. This is problematic because boundaries

used to create these areal units of analysis are arbitrary with respect to the distribution of environmental hazards and exposure to environmental risk. Since the boundaries of areas potentially exposed to hazards do not coincide spatially with the boundaries of census units, simplistic assumptions about the residential population distribution are necessary to estimate the number and characteristics of people at risk on the basis of areal interpolation. Although interpolation inaccuracies can be reduced by using spatial units that are smaller in size (i.e., census blocks), socioeconomic variables are not published by the U.S. Census at the block level of aggregation. Survey data serves as a useful alternative to examine individual or household level racial/ethnic and socioeconomic disparities in residential proximity to environmental hazards and provides several advantages (Mohai et al., 2009). First, addresses of survey respondents can be geocoded and represented as points on a map, leaving little ambiguity in determining their locations with respect to pollution sources or hazardous facilities. Second, the use of individual or household level data avoids the ecological fallacy of incorrectly assuming that relationships observed for areal units translate to relationships at the micro or individual level. Third, survey data typically provides more comprehensive and detailed information about the life circumstances of people living near hazardous sites than are available from the short and long forms of the decennial census. This allows better adjustments for confounding factors and offers additional insights on the characteristics of residents living near environmental hazards.

Another data problem relates to the utilization of outdated census information for EJ analysis. A temporal mismatch occurs, for example, when researchers evaluate the current distribution of environmental hazards (2009) with respect to demographic and socioeconomic data from the 2000 Census. While values of certain socioeconomic variables (e.g., median household income) may not have changed significantly since the latest census, data on age and race/ethnicity reach their usable limit in a few years (McMaster et al., 1997). Although adjusted estimates are often published by American Community Survey and private vendors, these are available only at coarser spatial resolutions such as counties and metropolitan statistical areas. To obtain more detailed and finer resolution (census tract or block group level) estimates necessary for assessing disproportionate proximity to hazards, researchers need to wait until the next census information is published (2011). The problem repeats itself though every cycle of the decennial census, as key variables gradually become dated and of questionable accuracy.

Finally, it is important to consider that most studies that examine disproportionate proximity or exposure to environmental hazards have relied on cross-sectional analysis to assess spatial disparities in the current patterns of adverse health risks (outcome equity). Regardless of the method and model utilized, the findings of such studies cannot be used to evaluate the processes that led to the observed risk disparities (process equity). Without detailed historical data on environmental conditions and socio-demographic characteristics, it is difficult to establish if hazardous facilities in a given study area were deliberately placed in locations more proximate to minority or low-income residents, or if subsequent residential choices and other factors caused the inequitable outcomes. While the dispersion of toxic pollutants is an explicitly spatial process, geographic patterns of explanatory variables such as race/ethnicity and related disparities are shaped by various underlying social, political, economic, and historical processes. Although cross-sectional research based on statistical data from the latest census cannot explain the processes leading to current racial/ethnic disparities, the findings represent a starting point for longitudinal investigation on the historical production of environmental injustice. Studies conducted in metropolitan areas such as Los Angeles (Pulido, 2000) or Phoenix (Bolin et al., 2005) demonstrate that a combination of qualitative and quantitative methods are necessary to improve our understanding of the causes and consequences of disproportionate exposure to hazards.

While this section of our paper has examined methodologies for assessing disproportionate proximity or exposure to environmental health hazards and their sources, Section III addresses the question of whether proximity to such hazards and locally undesirable facilities/land uses results in actual adverse health outcomes, and to what extent these adverse health outcomes are borne disproportionately by people of color and lower socioeconomic status.

III. Health outcomes and proximity to environmental hazards

Introduction

As already reviewed in Sections I and II, numerous studies provide evidence that ethnic/racial minorities and persons of lower socioeconomic status are more likely than non-Hispanic Whites and persons of higher socioeconomic status to live near potential environmental hazards. Public health and environmental health agencies are often confronted, however, by concerns that perceived excess adverse health outcomes are due to residential proximity to such hazards. The question also remains whether living in close proximity to various environmental entities such as hazardous waste sites, industrial facilities, and busy roadways is an actual risk factor for adverse pregnancy outcomes and childhood and adult health disorders.

A comprehensive review of the literature was conducted to determine how proximity to environmental hazards impacts the health of residential populations, including adverse pregnancy outcomes, childhood cancer, cardiovascular and respiratory illnesses, end-stage renal disease, and diabetes. With respect to cancer, we primarily focused our review on childhood cancers. Given the relatively long induction and latent period of solid tumors in adults, residential histories would need to be determined up to 15 to 30 years prior to diagnosis of cancer to capture pertinent environmental exposures. Although several recently published studies have included extensive residential histories (Bonner et al., 2005; Nie et al., 2007), there remains a dearth of such studies in the literature. According to a forthcoming chapter containing a comprehensive literature review of environmental health studies that use residential histories, only 26 such studies could be found, out of myriad health studies using only current residential addresses (Boscoe, forthcoming). Other health outcomes such as adverse pregnancy outcomes and childhood cancer have shorter latent periods between induction of disease from putative exposures and diagnosis; therefore, these types of outcomes are more easily studied in relation to proximity to environmental hazards. Although the pathogenesis of various respiratory and cardiovascular conditions may take many years before manifestation of overt disease, environmental exposures to pollutants could have acute effects, i.e., precipitating asthma attacks or myocardial infarction in susceptible individuals.

The studies reviewed utilized a wide variety of methods to examine the relation between proximity to potential environmental hazards and adverse health outcomes including spatial coincidence analyses (e.g., residence in a zip code with one or more hazardous waste sites), distance-based analyses (e.g., residence within one mile of industrial facilities as defined by a one-mile buffer), and pollution plume modeling, with distance-based analyses used most frequently. Tables 3, 4, and 5 provide details on the approaches used to assign exposure in each cited study in this Section.

Adverse Pregnancy Outcomes and Childhood Cancers

Adverse Pregnancy Outcomes

The relation between a maternal residence near potential environmental hazards and pregnancy outcomes has been investigated for a variety of outcomes including spontaneous abortion, fetal death, congenital malformations, low birth weight, preterm birth, and neonatal and infant deaths. For the purposes of this review, we mainly focused on studies that have been published within the past 15 years. We reviewed a total of 54 studies that examined the relation between residential proximity to one or more potential environmental hazards and adverse pregnancy outcomes. In this review, studies were also examined for evidence of racial/socioeconomic disparities between residential proximity to these potential hazards and adverse pregnancy outcomes, and Table 3 lists the studies that specifically examined racial/ethnic and/or income disparities in pregnancy outcomes in relation to residential proximity. Table 4a provides the characteristics of all studies reviewed by year published, types of study designs used, study populations, pregnancy outcomes included, methods of exposure assessment, major findings, and limitations. Investigators most often focused exposure assessment on maternal proximity to hazardous waste sites or landfills, but a few studies examined residential proximity to industries, municipal waste incinerators, cropland and areas with pesticide applications, and roadways and traffic density/pollution.

Study findings

Few studies examined health impacts specifically in relation to race or socioeconomic status (Table 3). Of those that did, conclusions were mixed as to the impact on specific racial or income groups. In a study of maternal residential proximity to hazardous waste sites and

chromosomal anomalies, increased risk for Klinefelter variants was confined to births to Hispanic women (Brender et al., 2008a). In the same study population, however, neural tube defects were associated with residential proximity within one mile of an industrial facility only among non-Hispanic White women (Suarez et al., 2007). Among various ethnic/racial groups studied, Orr et al., (2002) noted the strongest associations between a maternal residence in a census tract with one or more NPL hazardous waste sites and birth defects among American Indians/Alaska Natives. In Israel, Bedouin populations showed increased risk for major congenital malformations and perinatal mortality with residential proximity to an industrial park but no increased risk was noted for the Jewish populations with this residential characteristic (Bentov et al., 2006; Sarov et al., 2008). In a Canadian population, risks for preterm and low birth weight births in relation to maternal residential proximity to highways were strongest for births among highly educated women and women who lived in wealthy neighborhoods (Genereux et al., 2007).

Of all studies reviewed (Table 4a), several studies found positive associations between a maternal residence near waste sites and central nervous system defects (Geschwind et al., 1992; Croen et al., 1997 [neural tube defects]; Dolk et al., 1998 [neural tube defects]; Dodds & Sevior, 2001 [neural tube defects]; Orr et al., 2002 [neural tube defects]), heart defects (Shaw et al., 1992; Croen et al., 1997; Dolk et al., 1998; Malik et al., 2004; Yauck et al., 2004 [among offspring of women 38 years or older]; Langlois et al., 2009 [truncus arteriosus], Elliott et al., 2009), surgical correction of gastroschisis and exomphalos (Elliott et al., 2001), hypospadias and epispadias (Elliott et al., 2009), and chromosomal anomalies in offspring (Geschwind et al., 1992 [sites with plastics]; Orr et al., 2002; Vrijheid et al., 2002; Brender et al., 2008a [Klinefelter variants among Hispanic births]).

Fewer studies explored the relation between maternal residential proximity to waste sites and adverse pregnancy outcomes other than congenital malformations. Most of these studies reported minimal or no association except with maternal residential proximity to pesticide-contaminated waste sites and fetal deaths (Mueller et al., 2007), PCB-contaminated sites and low birth weight among male births (Baibergenova et al., 2003), municipal solid waste landfills and low birth weight or small for gestational age births (Goldberg et al., 1995), and any hazardous waste site and low and very low birth weight (Elliott et al., 2001).

In Europe and Japan, pregnancy outcomes were examined in relation to maternal residential proximity to incinerators or crematoriums. Associations were noted between these residential characteristics and risk for neural tube defects (Dummer et al., 2003), heart defects (Dummer et al., 2003), oral clefts (Cordier et al., 2004), renal dysplasia (Cordier et al., 2004), stillbirths (Dummer et al, 2003), and infant deaths (Tango, 2004). However, Tango et al. (2004) found no excess of deaths due to congenital malformations among births to mothers who lived near municipal solid waste incinerators in Japan.

In several populations, maternal residential proximity to industrial complexes was associated with increased risk of adverse pregnancy outcomes including central nervous system defects (Marshall et al., 1997; Bentov et al., 2006; Suarez et al., 2007 [neural tube defects]), oral clefts (Brender et al., 2006a), chromosomal anomalies (Brender et al., 2008a), undescended testis (Czeizel et al., 1999), perinatal mortality (Sarov et al., 2008), and low birth weight (Bhopal et al., 1999). Vinceti et al. (2001) noted that births with cardiovascular, musculoskeletal, and oral cleft defects were more likely to occur among women living in an industrial area contaminated with lead relative to women living away from this area.

Increased risks for low birth weight and preterm births were noted among offspring of mothers who resided near highways (Genereux et al., 2007) and in areas with high traffic density (Wilhem and Ritz, 2003), although no statistically increased risk was noted for fetal and early neonatal deaths with this maternal residential characteristic (de Medeiros et al., 2009).

Mothers living near cornfields at delivery were more likely to give birth to babies with limb malformations (Ochoa-Acuna and Carbajo, 2009), an association that might be attributed to exposure to pesticides used on this crop. Rull et al. (2006) noted elevated risks for neural tube defects among offspring of women who lived within 1000 meters of applications of pesticides classified as amides, benzimidazole, methyl carbamate, organophosphates, benomyl, or methomyl. Bell et al., (2001a) found risk for fetal deaths due to congenital malformations elevated if mothers lived near areas of pesticide applications.

Limitations

Without exception, the studies included in this review had one or more limitations that need to be considered in the interpretation of results and implications for environmental and public health. All studies used residential proximity to some degree to assign exposure which

might have led to exposure misclassification. Residential exposure to site contaminants, industrial emissions, traffic emission, and pesticide applications will also vary by climatic and topographic characteristics of geographic areas. These conditions were rarely considered in the studies discussed with the exception of those by Bentov et al., (2006), Cordier et al., (2004), Goldberg et al. (1995), Vinceti et al. (2008, 2009), and Wilhem and Ritz (2004). Furthermore, an ecologic study design was used in several instances in which rates of pregnancy outcomes were compared between geographic areas of potential exposure and non-exposure without assignment of specific exposures to each maternal address. Such a design can lead to an ecologic bias in which associations at an aggregate level do not represent exposures at the individual level among mothers with and without adverse pregnancy outcomes (Morgenstern, 2008).

Many studies used maternal address at delivery rather than address around conception and during the first trimester to assign exposure. While the addresses at delivery might be relevant for some adverse pregnancy outcomes, it can be problematic for assigning exposure in studies of chromosomal and non-chromosomal congenital malformations in which the time around conception and even earlier or the first trimester of pregnancy are respectively the most relevant. The estimated percent of women who change addresses between the time of conception and delivery has ranged between 12 and 33 percent depending on maternal populations examined (Canfield et al., 2006; Fell et al., 2004; Khoury et al, 1988; Shaw & Malcoe, 1992).

Residual confounding can also be an issue in which information regarding factors that are related both to exposure and outcome are measured imprecisely or are not available at all. Maternal addresses are not randomly distributed, but depend on several factors such as socioeconomic status, parental occupation, and race/ethnicity. These same factors may also increase or decrease the likelihood of living near potential environmental hazards. Race/ethnicity (Perlin et al., 2001; Woodruff et al., 2003; Brender et al., 2006b), maternal education (Brender et al., 2006b) and maternal occupation (Brender et al., 2008b) have been associated with maternal residential proximity to sources of pollution. These factors are also risk factors for several adverse pregnancy outcomes.

Data sources for pregnancy outcomes varied by the type of outcome studied. Ideally, birth defect registries provide the most accurate and complete source of data for congenital malformations, a source that was used by most studies reviewed that examined the relation between maternal residential proximity to environmental hazards and birth defects. By

September 2007, nearly every state in the U.S. had a birth defects registry in place (National Birth Defects Prevention Network, 2007), but these registries vary in scope and whether active or passive surveillance is used. While birth certificates are a poor source data source for congenital malformations (DiGiuseppe et al., 2002; Gore et al., 2002), several studies have found these vital records excellent data sources for birth weight (Roohan et al., 2003), gestational age, and preterm birth (DiGiuseppe et al., 2002). In the studies reviewed of fetal deaths, fetal death certificates usually served as the data source for this pregnancy outcome. However, fetal deaths tend to be underreported, especially those occurring before 28 weeks gestation (Harter et al., 1986; Goldhaber, 1989).

Summary of Adverse Pregnancy Outcomes Research

Risk estimates for adverse pregnancy outcomes in relation to maternal residential proximity to sources of pollution have varied across studies. Increased risks for central nervous system defects (including neural tube defects), congenital heart defects, chromosomal anomalies, low birth weight, and small for gestational age were noted in several U.S. and European study populations that lived close to hazardous waste sites. Odds ratios and relative risks for other types of birth defects in relation to this residential characteristic tended to be close to 1.0, indicating no association. Several studies also noted a maternal residence near active sites with chemical emissions (industries, incinerators, crematoriums) to be associated with fetal deaths, infant deaths, low birth weight, central nervous system defects, oral clefts, heart defects, renal dysplasia, and chromosomal anomalies. Residential proximity to pesticide applications or waste sites containing these chemicals was associated with fetal deaths, limb malformations, and neural tube defects. In several studies, women who lived near highways were more likely to have preterm births and low birth weight offspring.

Childhood Cancer

With respect to the relation between childhood cancer and residential proximity to potential environmental hazards, most published studies focused on all childhood cancers combined, leukemia, and/or brain cancer. Table 4b lists the 25 studies reviewed by characteristics and findings. As with adverse pregnancy outcomes, review of published research primarily focused on studies published within the past 15 years.

Study findings

Residential proximity to roadways and other indices of increased exposure to traffic-related pollution were associated with increased risk of childhood leukemia in a number of European studies (Crosignani et al., 2004; Harrison et al., 1999), but not noted in several U.S. study populations (Langholz et al., 2002; Reynolds et al., 2002b, 2004; Von Behren et al., 2008) or in a Danish population (Raaschou-Nielsen et al., 2001). Crosignani et al. (2004) estimated traffic-related benzene emissions through a Gaussian diffusion model and observed an OR of 3.91 (95% CI 1.36, 11.27) for childhood leukemia with benzene concentrations greater than 10 ug/m³. Residential proximity to roadways/traffic emissions was also associated with Hodgkin lymphoma in Danish children (Raaschou-Nielsen et al., 2001).

Risk for childhood cancer was examined in relation to residential proximity to cropland and pesticide applications in U.S. populations in California (Reynolds et al., 2002a, 2005; Rull et al., 2009) and Texas (Carozza et al., 2009). A birth address within 1000 meters of cropland showed some association with germ-cell tumors, non-Hodgkin lymphoma, and Burkitt lymphoma although the elevated ORs were based on small numbers of cases (Carozza et al., 2009). In an exposure metric that consisted of residential proximity within one-half mile of pesticide applications (pounds per square mile), Reynolds et al. (2005) noted slightly elevated ORs for leukemia with birth addresses near applications of pesticides that were probable or possible carcinogens and near applications of organochlorine or organophosphate pesticides. Risk of acute lymphoblastic leukemia (ALL) was elevated in children who lived within one-half mile (lifetime residences) of applications of organophosphates, chlorinated phenols, and triazines and pesticides classified as insecticides or fumigants (Rull et al., 2009); ALL risk was associated with moderate but not high exposures.

Risk of childhood cancer risk was examined in relation to residential proximity to other sources of contaminants including industries reporting under the U.S. Toxic Release Inventory, petrochemical plants, petrol stations, repair garages, nuclear power plants, and landfill sites and hazardous waste sites. Increased risk for childhood leukemia was found with a residential address near petrol stations (Harrison et al., 1999; Steffen et al., 2004, Weng et al., 2009), repair garages (Steffen et al., 2004), and nuclear power plants (Kaatsch et al., 2008; Spix et al., 2008). Children whose mothers lived during pregnancy near industries covered under the TRI were more likely to have brain cancer especially if the mother lived within one mile of a facility with

emissions of carcinogens (Choi et al., 2006). In a series of studies in which Knox (2000, 2006) and Knox and Gilman (1997) examined migration asymmetries via birth and death addresses of children who died from cancer in Great Britain, significant migration asymmetries were noted (close residential proximity more likely at birth than at death) for addresses near bus stations, railway stations, ferries, railways, roads, municipal and hospital incinerators, a variety of industries, airfields, and harbors.

Limitations

Studies of proximity to environmental hazards and childhood cancer shared similar limitations as those with proximity and adverse pregnancy outcomes. Exposure assignment was frequently based on one address such as address at birth, diagnosis, or death which potentially introduced exposure misclassification by not accounting for residential mobility. In a California study of childhood leukemia (Urayama et al., 2009), 65.8% of the study children moved between birth and date of diagnosis with over one-third of those who moved having at least one residence outside of the county of birth residence. During the year before the child's birth, 25% of the children's mothers changed residences at least once. Use of address at diagnosis or death may also be inappropriate with respect to temporality in that insufficient time might have been spent in the particular residential area to link nearby sources of pollution to cancer incidence.

Potential residual confounding was also a limitation in several studies, especially lack of adjustment for parental occupation. Maternal/paternal occupations have been associated with risk for childhood cancers (Savitz and Chen, 1990; Colt and Blair, 1998), and maternal occupations and residential proximity to sources of pollution appear related (Brender et al., 2008b).

Summary of Childhood Cancer Research

The results of several studies suggested an association between risk of childhood cancer and residential proximity to TRI facilities, highly trafficked roads, nuclear power plants, pesticide applications, and petrol stations or repair shops. These positive associations were not consistently found, however, and several well-designed studies found no association between childhood cancer and proximity to roadways, pesticide applications, nuclear plants, and hazardous waste sites. None of the reviewed studies compared the impact of this residential

characteristic across race or income although many adjusted risk estimates for these demographic factors to reduce confounding. In these studies, too few children were available with the residential proximity characteristics of interest for stratified analyses by race and income. Assignment of potential residential exposures appeared challenging in such studies, and most often a single address either during the prenatal period or during childhood was used to define proximity.

Cardiovascular, Respiratory and other Chronic Diseases

A comprehensive review of the literature was conducted to examine the relation between residential proximity to environmental hazards and cardiovascular, respiratory, and other chronic diseases. Studies employing a range of geospatial techniques, such as proximity analysis and air dispersion modeling, and using Geographical Information Systems Science (GISc) as an organizing framework, were selected to examine the public health effects of living near environmental burdens. A total of 20 studies were identified, representing a wide range of hazards and health outcomes. See Table 5 for a detailed outline of the research studies reviewed in this section. This section is organized into the following outcome/exposure categories:

- Cardiovascular and respiratory illness air pollution;
- PCB toxicity, end-stage renal disease, diabetes hazardous waste sites; and
- Cancer industrial & nuclear plants, air pollution.

Cardiovascular and Respiratory Disease – Air Pollution

Research studies using GISc to investigate the effects of mobile and stationary sources of air pollution on cardiovascular and respiratory illness are the most common exposure studies reviewed for this section. Despite a substantial body of literature on the topic, evidence of the connection between air pollution and various health effects remains mixed. A search of the literature revealed 14 articles that investigated the impact of air pollution on various health outcomes. Two studies assessed cardiovascular illness such as stroke, coronary heart disease, or chronic obstructive pulmonary disease, 12 articles examined asthma or respiratory symptoms, and one article pertained to both cardiovascular and respiratory diseases.

Study Findings

Two ecological studies found significant associations between stroke mortality and air pollution (Hu et al., 2008; Maheswaran and Elliott, 2003). Hu et al. (2008) determined the observed and expected stroke mortality at the census tract level for Escambia and Santa Rosa counties in Northwest Florida. The standardized mortality rates (SMR) were calculated for each census tract by dividing the observed count by the expected count. Air pollution was characterized by the presence of Toxic Release Inventory sites and other point sources (i.e., dry cleaners, sewer treatment plants, solid waste disposal, and superfund sites), and roads with high average vehicle traffic counts. Using location, air pollution density surfaces for point sources and traffic were calculated and hierarchical logistic regression was employed. The mean ageadjusted stroke rate in the study areas was over eight times the expected rate and there was a significantly elevated risk of stroke mortality in census tracts with high levels of air pollution. Maheswaran and Elliott (2003) also looked at relationship between stroke mortality and residential proximity to main roads at the census enumeration district (CED) in England and Wales. Mobile sources of air pollution were evaluated using road network data that characterized exposure as distance categories from the centroid of each CED to the nearest main road. Logistic Regression controlling for age, sex, socioeconomic (SES) deprivation, and urbanization determined the associations between stroke mortality and distance categories. CEDs with distances of less than 200 meters to main roads had significantly higher stroke mortality rates than those with distances of over 1,000 meters. This association held when stratified by sex and a significant dose-response relationship was determined for distance categories.

Aylin et al. (2001) examined how residential proximity to operational coke works plants in England and Wales impacts the cardiorespiratory health of vulnerable populations including older adults and children. The nine plants in operation are known local sources of air pollution including sulfur dioxide, oxides of nitrogen, and particulates such as black smoke and soot. Hospitalization data for coronary heart disease, stroke, respiratory disease, chronic obstructive pulmonary disease, and asthma was collected for children under 5 and adults age 65 and over living within a CED with a centroid distance of 7.5 km or less to a coke works plant. For each CED, observed and expected disease risk ratios were calculated and compared based on distance categories to the plants. Despite significant elevated risk ratios with proximity to the Teeside plant for coronary heart disease in older adults, and respiratory disease and asthma among

children, no other results demonstrated a significant association. The impact of air pollution around the Teeside coke works plant should be further investigated, especially for vulnerable populations. Although there was no elevated risk of cardiorespiratory hospitalizations with proximity to coke works (except for Teeside), the authors only took into account a single measure of air pollution, which most likely underestimated the exposure potential. Taken together, these studies suggest that there is a significant association between residential exposure to combined sources of air pollution and stroke mortality.

A case-crossover study by Smargiassi et al. (2009) examined the effects of residential proximity to point source air pollution on asthma among children. Asthma hospitalization data for children two-four years of age was collected and the risk of asthma episodes was calculated for residential postal codes for the East End of Montreal Island. Exposure was estimated using the American Meteorological Society/Environmental Protection Agency Regulatory Model (AERMOD), an air dispersion model, and sulfur dioxide (SO₂) emissions data from two petroleum refineries and other point sources measured via two fixed air-monitoring sites. AERMOD computed estimations of daily SO₂ exposure at the centroid of each postal code as well as average hourly predictions and daily peaks. Logistic regression was used to evaluate SO₂ exposure in relation to asthma hospitalization days versus control days using a time-stratified approach. Results revealed that short-term increases in SO₂ are significantly associated with a higher number of asthma related emergency department visits and hospital admissions in children residing near refineries.

Another study by Maantay et al. (2009c) used AERMOD to model air pollution from stationary point sources in the Bronx, New York City. The article expanded on a prior study by the author that assessed the impact of mobile and point source air pollution on asthma hospitalization in adults and children residing in the Bronx (Maantay, 2007). In the earlier study, doctor-diagnosed asthma hospitalization data were obtained from the New York Statewide Planning and Research Cooperative System (SPARCS) and stratified by age and sex. Exposure was classified by creating buffers (ranging from 150 meters to 0.5 miles) around TRI, National Emissions Inventory (NEI), and HAPs sites as well as limited access highways, which average 50,000 vehicles per day. Proximity analysis determined whether the asthma cases fell within the buffers and revealed that residing within exposure buffers was significantly associated with asthma hospitalization for both children and adults. In addition, standardized incidence ratios

showed that observed asthma rates were significantly higher than expected values at the census block group. In an effort to validate the results from the proximity analysis, the author modeled five criteria air pollutants (PM₁₀, PM_{2.5}, NO_x, CO, SO₂) from stationary point sources on the NEI list to create plume buffers (Maantay et al., 2009c). Asthma hospitalizations were geo-coded to actual individual patient addresses, and then rates were calculated inside and outside of the plume buffers using tax-lot data for population estimates. Regression analysis revealed that the odds of being hospitalized for asthma was significantly higher inside the buffers versus outside. These studies demonstrate that residential proximity to point source air pollution is associated with asthma hospitalization regardless of which exposure measurement methodology was used.

A prior literature review by Maantay and Porter-Morgan (2004) found additional evidence of a connection between residential proximity to mobile sources air pollution and asthma or respiratory symptoms in children and adults. Edwards et al. (1994) examined the association between living near heavily trafficked roads and asthma hospitalization rates using a case-control proximity analysis in Birmingham, England. Study subjects included cases consisting of children under 5 years of age with an asthma hospitalization and controls from both the hospital and the community. To determine residential exposure to heavily trafficked roads, participant addresses were linked via the user postcode and fixed-distance buffers of 200 and 500 meters were established around major roads. Statistical analysis using chi-square tests were performed to determine the odds of asthma hospitalization among children living within road buffers versus those residing outside. The odds of asthma hospitalization among children living within 200 meters of a major road was significantly higher among cases than community controls. In addition, the odds of living near roads with high traffic flows (i.e., > 24,000 vehicles per hour) was significantly higher for cases as compared with both hospital and community controls.

Three other studies assessed the same association and failed to find a significant relationship between proximity to roads and asthma hospitalizations among children (English et al., 1999; Livingstone et al., 1996; Wilkinson et al., 1999). However, the odds of residing in high traffic flow areas were significantly higher for children experiencing more than one asthma hospitalization per year than for children having only one incident (English et al. 1999). Several other studies found an increase of self-reported chronic respiratory symptoms such as wheeze, pulmonary function, attacks, and the use of respiratory medicine, with residential proximity to

major roadways (Oosterlee et al., 1996; van Vliet et al., 1997; Venn et al., 2001; Wjst et al., 1993).

Summary of Cardiovascular and Respiratory Disease Research

The results from these studies suggest that residential proximity to both stationary sources (TRIs, NEIs, HAPs, petroleum refineries, etc.) and, with a few exceptions, heavily trafficked roads is significantly associated with asthma hospitalizations (Edwards et al., 1994; Maantay and Porter-Morgan, 2004; Maantay et al., 2009c; Smargiassi et al., 2009). In addition, exposure to mobile sources air pollution increases the occurrence of chronic respiratory symptoms by exacerbating asthma (English et al., 1999; Oosterlee et al., 1996; Vliet et al., 1997; Venn et al., 2001; Wjst et al., 1993). The studies reviewed (Hu et al., 2008; Maheswaran and Elliott, 2003; Aylin et al., 2001) also suggest that there is a significant association between residential exposure to combined sources of air pollution and stroke mortality.

PCB Toxicity, End Stage Renal Disease, Diabetes – Hazardous Waste Sites

Three studies we reviewed examined the impact of residing near hazardous wastes sites using GISc although the health outcomes of interest were different: cord blood Polychlorinated biphenlys (PCB) level, end-stage renal disease (ESRD), and diabetes.

Study Findings

Choi et al. (2006) obtained data from a cohort of mother-infant pairs participating in a study on PCB exposure and child development to assess whether living near a PCB-contaminated superfund site, New Bedford Harbor in Massachusetts, is related to high cord serum PCB levels among infants. Cord blood PCB levels were taken at birth from participants residing in New Bedford, Acushnet, Fairhaven, and Dartmouth, Massachusetts. PCB concentrations were reported as the sum of 51 congeners and grouped into light and heavy PCB categories based on relative volatility. Residential history and individual risk factors (i.e., diet, birthplace, etc.) were collected via a questionnaire and distances to the superfund hotspot were calculated. Inverse distance weighting was used to create a surface of PCB cord serum levels within a 5-mile radius of the hotspot and linear regression controlling for neighborhood SES was employed. The authors found no association between cord serum PCB toxicity and distance to the superfund site; individual characteristics (maternal age and birthplace) remained the most

significant predictors. However, children born before or during dredging of the Harbor had significantly higher PCB levels than those born after dredging, suggesting that the impact of remediation should be further reviewed.

In an effort to determine the risk of ESRD with residential proximity to hazardous waste sites, Hall et al. (1996) conducted a case-control study of 20 New York State counties. Cases and controls were selected from the Health Care Financing Administration and matched by age, sex, and race. A questionnaire assessed residential history, occupation, and individual risk factors such as hobbies, health, and lifestyle behaviors. Hazardous waste sites were compiled from the New York Inactive Hazardous Waste Site Registry and 1-mile buffers around each site were created. Based on site history, sampling data, and other reports, exposure categories were assigned to 25 sectors within each buffer. Logistic regression controlling for SES and other confounders was used to assess the relationship between ESRD and residence within an exposure sector. Although risk of ESRD was elevated for subjects residing within buffers and in medium and high exposure sectors, the results were not significant.

Kouznetsova et al. (2007) conducted an ecological study to determine whether exposure to persistent organic pollutants (POPs) found near hazardous waste sites was associated with diabetes risk in New York. SPARCS data was used to calculate diabetes hospitalization rates in patients 25-74 years of age by ZIP code, controlling for age, sex, race, income, and urban/rural population density. Hazardous waste sites were grouped into exposure categories by ZIP code: POP sites (dioxins/furans, PCBs, persistent pesticides), non-POP sites (volatile organics and metals, etc.), and clean sites. Significantly higher diabetes hospitalization rates were found in POP ZIP codes versus both clean and non-POP sites. Upon stratification, the rate ratios were highest for blacks and older age groups.

Summary of Research on PCB Toxicity, Renal Disease, and Diabetes – Hazardous Waste Sites

Although there is some evidence linking residential proximity to hazardous waste sites and adverse health impacts (Choi et al., 2006; Hall et al., 1996; Kouznetsova et al., 2007), the dearth of literature makes cross-study comparisons difficult. Although there may be an association between exposure to hazardous waste sites and outcomes such as PCB toxicity, ESRD, and diabetes, more research is needed.

Cancer - Industrial and Nuclear Plants and Air Pollution

Three studies exploring the relationship between environmental burdens and cancer using GISc were reviewed. The environmental exposures of interest in these studies range from industrial plants, a nuclear facility, and air pollution. The evidence is mixed regarding proximity to industrial plants and nuclear facilities.

Study Findings

Morris and Knorr (1996) conducted a case-control proximity analysis to examine the relationship between radioactive emissions from the Pilgrim nuclear power plant in Plymouth, Massachusetts and adult leukemia. Cases were selected from the Massachusetts Cancer Registry and hospital data. Eligibility requirements included residence within 1 of 22 towns that were located in a 22-mile radius of the Pilgrim Plant and an age of 12 or older at time of diagnosis. Two controls were randomly matched to each case by age, sex, vital status, and year of death. A telephone survey with the subject (or surrogate in case of death) determined residential history, occupation, health status, and sociodemographic information. Exposure potential was calculated for both residential proximity to Pilgrim during years of high radioactive release (1974-1977) and as an individual exposure summary score derived from time spent at work, home, downwind from Pilgrim, and reported emissions. Conditional logistic regression was used to calculate the odds of leukemia with distance to the plant controlling for cigarette smoking, SES, and occupation in a high-risk environment. No significant associations were found between proximity to Pilgrim and adult leukemia. However, using the exposure score, there was evidence of a dose response relationship where the odds of leukemia was significantly higher for subjects within higher exposure score categories than the lowest. However, when stratified by sex, only women in the highest exposure score group had significantly higher odds of leukemia suggesting that risk may be different for subpopulations.

Two studies examined the relationship between cancer and industrial plants with mixed results (Johnson et al., 2003; Wilkinson et al., 1997). In a case-control study, Johnson et al. (2003) investigated the connection between Non-Hodgkin's Lymphoma (NHL) and residential proximity to industrial plants in Canada, including copper, lead, and nickel smelters, steel, petroleum refineries, and kraft and sulfite pulp mills. Cases were selected from the National

Enhanced Cancer Surveillance System based on a NHL diagnosis between 1994-1998 and residence within the study area comprised of eight Canadian Provinces. Controls were obtained through health insurance plans and finance property assessment databases. A questionnaire assessed individual risk factors (i.e., smoking, drinking), work and residential history, and SES. Individuals were grouped by distance categories to plants and years spent within each category. Odds ratios were obtained using unconditional logistic regression controlling for province, age, sex, smoking status, alcohol use, SES, residential history, chemical exposure (self-reported), and high-risk occupational history. The authors found no significant association between distance to the combined measure of industrial plants and NHL. However, the odds of NHL were significantly higher for subjects residing within two miles of a copper smelter and one-half mile of a sulfite pulp mill. In addition, women residing within two miles of any industrial plant were significantly more likely to have follicular NHL.

A small area study in Waltham Abbey, England by Wilkinson et al. (1997) explored cancer incidence and mortality around the pesticide and fertilizer producing Pan Britannica Industries Plant. Observed and expected cancer incidence and death rates were calculated and compared for electoral wards using a distance decline model around Pan Britannica. Although the authors found significant differences between observed and expected rates of cancer incidence and mortality for distance from 0-1 km and 0-7.5 km around the plant, there was mixed evidence of a dose-response relationship and the rate ratios were within the range for the region. Also, the observed incidence for skin melanoma, and cancers of the lung, pancreas, and stomach were significantly higher than expected values.

Summary of Research on Cancer – Industrial and Nuclear Plants and Air Pollution

Similar to the hazardous waste site studies, studies assessing cancer risk from exposure to various environmental hazards suffer from comparability problems. Residential proximity to certain industrial plants appears to be linked to cancer risk although the significance of the results depends upon the specific type of plant, population subgroup, and cancer types. Leukemia was significantly associated with proximity to Pilgrim nuclear power plant among women (Morris and Knorr 1996). Although one study failed to find a relationship between cancer and exposure to the Pan Britannica Plant (Wilkinson et al., 1997), another found significant associations between other industrial plants and some cancers: for instance, NHL was

significantly associated with proximity to a copper smelter and sulfite pulp mill (Johnson et al., 2003).

Limitations of Spatial Epidemiology

Before the results from the literature review can be interpreted, it is important to understand how the limitations of this review will impact the ability to draw conclusions. This literature review encompasses a broad range of environmental hazards and health outcomes but few studies evaluate the same associations between specific diseases. In addition, the geographical location of interest as well as the target population (i.e., children or adults) varies by study. Therefore, the results of these studies should be interpreted with caution due to inconsistency from methodological and data limitations inherent in the design of geospatial studies. The following paragraphs summarize these challenges, which include measurement misclassification, comparisons across appropriate spatial units, and controlling for confounders. Each of these limitations will be discussed followed by a synthesis of the literature results.

Despite the increase in interest of understanding the health impacts of residing near environmental hazards, it remains difficult to measure consistency across studies due to variation in both outcome and exposure measurement. This means that similar research questions are addressed using different definitions, variables, and methodological techniques (Diez Roux, 2001). For example, exposure is most often defined using distance to the specific environmental hazards (i.e., major roads, hazardous wastes sites, etc.). However, this can only serve as a crude proxy for exposure and does not accurately represent individual exposure levels to ambient conditions, or body or target organ dose (Aylin et al., 2001; Choi et al., 2006; Edwards et al., 1994; English et al., 1999; Hall et al., 1996; Johnson et al., 2003; Kouznetsova et al., 2007; Livingstone et al., 1996; Maheswaran and Elliott, 2003; Morris and Knorr, 1996; Oosterlee et al., 1996; van Vliet et al., 1997; Venn et al., 2001; Wilkinson et al., 1997; Wilkinson et al., 1999; Wjst et al., 1993). In addition, the studies differ with regards to how they classify the exposure variables: some include a range of exposures (general air pollution) whereas others assess a specific source (mobile sources air pollution), and still others look at a specific pollutant (i.e., PM_{2.5}).

Individual-based exposure measurement is preferable although not always available, which may introduce bias from exposure misclassification (Elliott and Savitz, 2008). The same

issues are also apparent in the measurement of the health outcome of interest especially when self-reported data on a particular disease or symptoms is used rather than an official medical diagnosis or health record (Oosterlee et al., 1996; van Vliet et al., 1997; Venn et al., 2001; Wjst et al., 1993). Measurement misclassification has lead to inconsistencies in the evidence linking environmental hazards to specific health outcomes and renders comparability across studies difficult.

Another challenge is defining the geographical area to ensure that it is meaningful and representative of the exposure being measured, the target population, and the outcome of interest. However, the spatial delineation or classification of the unit of analysis is rarely considered systematically due to a dependence on existing datasets. Many studies have relied on censusdefined or administrative boundaries (i.e., census tract, ZIP code) as a proxy for residential location when individual address was unknown. This method, known as the "container approach," (or "spatial coincidence analysis" – see Section II of this paper) determines exposure based on whether a specific environment hazard is present within a particular geographic unit of aggregation (Maroko et al., 2009). This implies that all residents within a particular boundary are all impacted equally by the hazard of interest, without an accurate assessment of individual exposure. For example, a case may live next to a particular hazard of interest but if the hazard is not located within their unit of analysis (i.e., ZIP code) then that case would not be defined as exposed. In general, the larger the unit of aggregation, the more likely it is that bias will be introduced due to heterogeneity across and within these units (Maroko et al., 2009), and ecological fallacy may result. However, these types of small scale studies (using larger units of aggregation) may serve as indicators of where further and more detailed investigation should take place (Elliott and Savitz, 2008).

It is essential to ensure that certain factors present in the underlying study population, known as confounders, are not influencing the relationship between environmental hazards and health outcomes. However, despite the importance of controlling for confounders, many of the studies reviewed did not include potential confounders such as race/ethnicity, SES, behavioral risk factors, and workplace or school exposure. These underlying disease determinants tend to vary across the unit of analysis and if they are coincident with the exposure measures, then these spatial confounders will bias the results of the study. SES status and certain lifestyle behaviors (i.e., smoking) may be particularly powerful confounders. Again, choosing a smaller unit of

analysis can provide some protection against bias because confounding may be less of a threat and more easily controlled in the analysis (Elliott and Savitz, 2008).

In conclusion, research using GISc and other geospatial techniques to explore the public health burdens of residential proximity to environmental hazards is in its infancy. This literature review investigated the associations between only a few environmental exposures and health outcomes and is not by any means to be considered exhaustive. In addition, consistency across the studies is vulnerable due to the differences in study designs and methodology. However, despite the limitations mentioned above, a few general trends have emerged and are worth discussing.

Air pollution and its impact on respiratory and cardiovascular disease are of particular importance given the extent of the exposed population. Although there are a few exceptions, the evidence linking stroke and asthma to residential exposure of both point and mobile sources of air pollution is strong (Edwards et al., 1994; Maantay and Porter-Morgan, 2004; Maantay, 2007; Smargiassi et al., 2009). In addition, air pollution, particularly from roads with a high traffic volume, can exacerbate self-reported chronic respiratory symptoms thus exacerbating existing asthma (English et al., 1999; Maantay et al., 2009c; Oosterlee et al., 1996; van Vliet et al., 1997; Venn et al., 2001; Wjst et al., 1993).

Hazardous waste sites are an oft-mentioned source of concern for environmental justice advocates yet the lack of research investigating their relationship to adverse health impacts is problematic. This review found only three articles focusing on these sites and each study specified a different outcome of interest, which makes comparison difficult. In short, proximity to hazardous waste sites was associated with diabetes but not ESRD nor PCB toxicity (except before and during dredging) (Choi et al., 2006; Hall et al., 1996; Kouznetsova et al., 2007). However, more research assessing these relationships is clearly needed.

Lastly, residential proximity to certain industrial plants appears to be associated to cancer risk although the significance of the results depends upon the industry and population of interest. Adult leukemia was significantly associated with residential proximity to Pilgrim nuclear power plant among women (Morris and Knorr, 1996), all-type NHL was significantly associated with distance to a copper smelter and sulfite pulp mill, and women residing within two miles of any industrial plant were more likely to have follicular NHL (Johnson et al., 2003).

IV. <u>Conclusions/Recommendations</u>

Conclusions

Based on our review and evaluation of existing research on the relationship amongst proximity to environmental hazards, environmental justice, and adverse health outcomes, we have reached the following conclusions:

- A higher proportion of minorities and lower-income populations reside near environmental hazards, such as Toxic Release Inventory facilities; National Emissions Inventory facilities; other sources of hazardous air pollutants; hazardous waste treatment, disposal, and storage facilities; landfills; sewage treatment plants; power plants; major roadways; solid waste facilities; industrial zones in general; and air craft noise from airports.
- 2. Although the results are mixed, several studies have found significant relationships between residential proximity to environmental hazards and adverse health outcomes, such as adverse pregnancy outcomes, (including increased risks for central nervous system defects, congenital heart defects, oral clefts, renal dysplasia, limb malformations, chromosomal anomalies, preterm births, low birth weight, small-forgestational-age, fetal deaths, and infant deaths); childhood cancers (including leukemia, brain cancer, germ-cell tumors, non-Hodgkin lymphoma, and Burkitt lymphoma); asthma hospitalizations and chronic respiratory symptoms; stroke mortality; PCB toxicity, end-stage renal disease, and diabetes. Although populations living close to environmental hazards appear more likely to have adverse health outcomes, proximity does not necessarily equate to individual level exposure.
- 3. Given that racial/ethnic minorities and/or lower-income populations are more likely to live near such environmental hazards and research has indicated that this residential characteristic might be associated with adverse health outcomes, it is highly likely that there is a disproportionate impact of this exposure on the health of minorities and lower-income populations.
- 4. However, few studies have examined whether such exposures are more or less likely to increase risk for adverse health outcomes among minority and lower-income populations. This dearth of studies is possibly due to a limitation of the available

health data, which often does not accurately or completely report race and ethnicity of the health outcome cases.

- 5. Methods for assessing spatial proximity and potential exposure to hazards have evolved from comparing the prevalence of minority or low-income residents in predefined geographic units hosting hazardous facilities to more rigorous techniques that are based on precise distances between hazards and people, quantity and quality of emitted pollutants, chemical fate and transport modeling, and data sets which provide modeled estimates of adverse health risks from cumulative exposure to multiple pollutants and emission sources.
- 6. The lack of address-specific, individual/household data and information on day-time locations of people are major impediments in measuring disparities in proximity or exposure to environmental health hazards accurately and comprehensively.
- 7. While conventional statistical methods such as correlation or regression have been used extensively in previous studies to evaluate racial/ethnic or socioeconomic disparities, these techniques violate several classical statistical assumptions (i.e. independence and homogeneity) and may not be appropriate for analyzing spatial data and relationships.

Recommendations

Given the conclusions above, which are based on the evidence of disparities by race and income in relation to proximity to environmental hazards, the adverse health outcomes for populations in close proximity to environmental hazards, and acknowledgement of the health disparities experienced in general by communities of color and lower-income communities, we suggest that these factors be given serious consideration in the decision-making process by governmental environmental and health agencies regarding the siting of environmentally-burdensome facilities and land uses, in regulatory and enforcement efforts concerning pollution, and in the active promotion of environmental health justice and environmental health protection.

We believe that there is sufficient evidence right now to justify the application of the Precautionary Principle to protect people from the deleterious effects of living near environmental hazards. This means that, even in the absence of complete scientific proof, we have enough evidence of potential harm being done to take steps to rectify the problem, and that

there is a social responsibility to protect the public from exposure to harm when all available evidence points to plausible risk. Nevertheless, economic and political forces will likely require stringent proof that specific recommendations, like the establishment of protective buffer zones around noxious land uses, will be effective. And, indeed, the question remains whether it is advisable to create "no-man's-lands" of environmentally hazardous areas under the probably erroneous assumption that if no one lives near these facilities and land uses, they are no longer dangerous or causing any problems. That might only lead to a false sense of security, which is dangerously close to creating an "out of sight, out of mind" situation. Perhaps actively confronting whether there is a need for these facilities and land uses to exist in the first place would get more to the root of the problem.

A major drawback to re-crafting national environmental regulations with an eye to addressing the proximity issue is that the land use and zoning decision-making process is one of the largest drivers of the problem of proximity to environmental hazards and environmental health justice. It may also be one of the largest drivers of the solutions to the problem. But land use and zoning, by and large, are regulated at the most local levels of government, so any protective solution involving land use regulation would likely not be applied consistently on a national basis, potentially leading to more intensive "ghetto-ization" of environmentally hazardous uses, and the expansion of, or additional, "sacrifice zones."

An in-depth discussion of the policy implications of our review is beyond the scope of this paper, and would require a different set of skills and expertise than the authors possess, in order to analyze existing regulations and determine what would need to be changed and how to do it in such a way as to better protect people from environmental hazards. However, some of the more obvious practical applications of our review are perhaps easier to state. They fall into the category of "common-sense" guidelines, and constitute approaches that would be difficult to argue against. These might include things like prohibiting the siting of schools near highways, and being cognizant of pesticide drift when planning residential locations or other sensitive land uses. Our technical recommendations are informed primarily by the limitations of current research, as described in detail in the previous sections. We recommend that the following deficiencies in available data, research methods, and research emphasis be addressed:

1. Research gaps - there are significant gaps in current research, especially regarding the assessment of overall health outcomes in relation to proximity to environmental

- hazards, and regarding the relationships between these issues and minority, low-income, and other populations considered to be more vulnerable.
- 2. Data needs the data necessary for more definitive research on these relationships require increased accuracy and higher spatial resolution. Data on health outcomes need to be made available at the individual patient level, which is possible now since issues of maintaining patient information confidentiality can successfully be handled through geo-coding masking and randomization techniques in graphic display. Aggregated health data is not sufficiently fine-grained enough for most research on the relationship between proximity to environmental hazards, health outcomes, and characterization of affected populations. Data on environmental quality factors, meteorological conditions, and physical environmental infrastructure parameters are generally not complete or exact enough to serve as inputs to complex models, and these need to be augmented by better data as well.
- 3. Methodological approaches conventional statistical methods, which have been used for many health studies, are not the most appropriate or effective methods for finegrained spatial analysis, but more location-based geostatistical methods have not been adopted as frequently as would be desirable, due to the fact that many health and environmental researchers who conduct this type of research lack awareness of these methods and knowledge of their utilization. Increased education and training in geostatistical analytic techniques would be useful to encourage new research incorporating these methods, and to assist researchers in developing additional new geographically-based methods. Furthermore, although environmental modeling is often held out as the gold-standard of environmental impact assessment, it is still relatively cumbersome, labor-intensive, computer-intensive, and necessitates a high level of computational skills, as well as requiring extensive data inputs that are usually quite difficult to obtain. Better and more generalized, easy-to-use models should be developed, preferably models that are well-integrated or closely-coupled with GISc software, rather than stand-alone models. Multidisciplinary teams, such as those with expertise in GIS, epidemiology, environmental science, and statistical modeling, as well as community scientists, are in the best position to investigate the

- relation between proximity to environmental hazards and adverse health outcomes (Maantay et al., 2009b).
- 4. Paucity of environmental impacts investigated many studies investigate the same type of hazard, (for example, TRI facilities) usually because of data limitations and the default use of hazard databases available at the national scale. Most studies look at only one or two environmental hazards at a time. Cumulative and synergistic impacts have rarely been examined, yet these types of impacts may have a larger than acknowledged connection to adverse health outcomes.
- 5. Residential focus vs. daytime location studies in this review that used census data to assess disproportionate impacts examined proximity to hazards from the perspective of residential location of the potentially exposed population, although, except for small children and perhaps the elderly, most people do not spend the majority of their time at home. The true environmental impact on various populations can only be ascertained by achieving a better understanding of where people actually are located, other than simply their residential addresses.
- 6. Exposure assessment most studies of proximity to environmental hazards and health outcomes based exposure assessment on a single residential address. This approach does not take into account residential mobility and is potentially a significant source of exposure misclassification. Furthermore, the appropriate temporal sequence was a problem in some studies in which data on current environmental conditions were linked to past residential locations.

These deficiencies in research focus, methodological techniques, exposure assessment, and data availability and access may be mitigated by providing more targeted funding to help correct some of these problems, and ensure that future research does not suffer from these drawbacks. This would lead to increased reliability of results, stronger evidence, increased understanding of the complex interactions of environment-human factors, and better hope for finding real solutions to environmental health injustices and environmentally-related diseases and conditions.

Acknowledgements

Dr. Maantay would like to acknowledge the invaluable assistance of three doctoral student researchers in the Urban GISc Lab at Lehman College, City University of New York (CUNY). Thanks to their unstinting hard work, fresh insights, and enthusiasm, the research effort for this paper was made so much more interesting (and more fun!), in addition to enhancing the resulting document.

- Rachael Weiss, Research Fellow, DPH program, CUNY Graduate Center;
- Keith Miyake, Research Assistant, Earth and Environmental Sciences Ph.D. Program,
 CUNY Graduate Center;
- Laurel Mei Turbin, Research Assistant, Earth and Environmental Sciences Ph.D.
 Program, CUNY Graduate Center.

Dr. Maantay would also like to take this opportunity to thank the following organizations for supporting her research in environmental health justice over the past years, which in many ways enabled her contributions to the writing of this paper:

National Institute for Environmental Health Science; National Center for Minority Health and Health Disparities; NOAA-CREST, the National Oceanic and Atmospheric Administration's Cooperative Remote Sensing Science and Technology Center; U.S. Environmental Protection Agency; South Bronx Environmental Justice Partnership; Bronx CREED (Center to Reduce and Eliminate Racial and Ethnic Health Disparities); Montefiore Medical Center and Albert Einstein College of Medicine; PSC-CUNY Faculty Research Awards; George N. Shuster Fellowship.

References

Akwesasne Task Force on the Environmental Research Advisory Committee. Superfund Cleanup at Akwesasne: A Case Study in Environmental Justice. *International Journal of Contemporary Sociology* 1997;34(2):267-290.

Anderton D, Anderson J, Oakes J, Fraser M. Environmental equity: the demographics of dumping. *Demography*. 1994;31:229-248.

Anselin L. *Exploring Spatial Data with GeoDa: A Workbook*. Urbana-Champaign, IL: Spatial Analysis Laboratory, Department of Geography, University of Illinois; 2005.

Anselin L, Bera, A. Spatial dependence in linear regression models with an introduction to spatial econometrics. In: Ullah A, Giles D, eds. *Handbook of Applied Economic Statistics*. New York: Marcel Dekker; 1998:237-289.

Apelberg BJ, Buckley TJ, White RH. Socioeconomic and racial disparities in cancer risk from air toxics in Maryland. *Environmental Health Perspectives*. 2005;113(6):693-699.

Ash M, Fetter TR. Who lives on the wrong side of the environmental tracks? Evidence from the EPA's Risk-Screening Environmental Indicators model. *Social Science Quarterly*. 2004;78:793-810.

Atlas M. Few and far between? An environmental equity analysis of the geographic distribution of hazardous waste generation. *Social Science Quarterly*. 2002;83(1):365-378.

Aylin P, Bottle A, Wakefield J, Jarup L, Elliott P. Proximity to coke works and hospital admissions for respiratory and cardiovascular disease in England and Wales. *Thorax.* 2001 03;56(3):228-233.

Baden BM, Coursey D. The locality of waste within the city of Chicago: a demographic, social, and economic analysis. *Resource and Energy Economics*. 2002;24: 53-93.

Baden BM, Noonan DS, and Turaga RM. Scales of justice: is there a geographic bias in environmental equity analysis? *Journal of Environmental Planning and Management*. 2007; 50:163-185.

Baibergenova A, Kudyakov R, Zdeb M, Carpenter DO. Low birth weight and residential proximity to PCB-contaminated waste sites. *Environ Health Perspect*. 2003;111(10):1352-7.

Been V. Analyzing evidence of environmental justice. *Journal of Land Use and Environmental Law.* 1995;11 (1): 1-36.

Been V, Gupta F. Coming to the nuisance or going to the barrios? A longitudinal analysis of environmental justice claims. *Ecology Law Q* 1996;XXIV(1):1–35.

Bell EM, Hertz-Picciotto I, Beaumont JJ. A case-control study of pesticides and fetal death due to congenital malformations. *Epidemiology*. 2001a;12(2):148-156.

Bell EM, Hertz-Picciotto I, Beaumont JJ. Case-cohort analysis of agricultural pesticide applications near maternal residence and selected causes of fetal death. *Am J Epidemiol*. 2001b;154(8):702-710.

Bentov Y, Kordysh E, Hershkovitz R, et al. Major congenital malformations and residential proximity to a regional industrial park including a national toxic waste site: An ecological study. *Environ Health.* 2006;5:8(Mar 29).

Berry, KA. Race for Water? Native Americans, Eurocentrism, and Western Water Policy. In: Camacho, D.E., ed., 1998, *Environmental Injustices, Political Struggles: Race, Class and the Environment*, Duke University Press.

Bevc CE, Marshall BK, Picou JS. Environmental justice and toxic exposure: toward a spatial model of physical health and psychological well-being. *Social Science Research*. 2007;36: 48-67.

Bhopal RS, Tate JA, Foy C, Moffatt S, Phillimore PR. Residential proximity to industry and adverse birth outcomes. *Lancet*. 1999;354(9182):920-921.

Boer JT, Pastor M, Sadd JL, Snyder LD. Is there environmental racism? the demographics of hazardous waste in Los Angeles. *Social Science Quarterly*. 1997;78:793–810.

Bolin B, Matranga E, Hackett EJ, Sadalla EK, Pijawka KD, Brewer D, Sicotte D. Environmental equity in a sunbelt city: The spatial distribution of toxic hazards in Phoenix, Arizona. *Environmental Hazards*. 2000;2: 11–24.

Bolin B, Nelson A, Hackett E, Pijawka D, Smith S, Sadalla E, Sicotte D, Matranga E, O'Donnell, M. The ecology of technological risk in a sunbelt city. *Environment and Planning A*. 2002;34: 317-339.

Bonner MR, Han D, Nie J, Rogerson P, Vena JE, Muti P, et al. Breast cancer risk and exposure in early life to polycyclic aromatic hydrocarbons using total suspended particulates as a proxy measure. *Cancer Epidemiol Biomarkers Prev.* 2005;14(1):53-60.

Boone CG. An assessment and explanation of environmental inequity in Baltimore. *Urban Geography*. 2002;23(6):581-595.

Boscoe F. The use of residential history in environmental health studies. In: Maantay JA and McLafferty S, eds. *Geospatial Analysis of Environmental Health*, forthcoming, Springer Verlag, Dordrecht, NL.

Bouwes N, Hassur SM, and Shapiro MD. Empowerment through risk-related information: EPA's Risk-Screening Environmental Indicators project. *Working Paper DPE-01-06*. Amherst, MA: Political Economy Research Institute; 2001.

Bowen WM, Salling MJ, Haynes KE, Cyran EJ. Towards environmental justice: spatial equity in Ohio and Cleveland. *Annals of the Association of American Geographers* 1995;85(4):641-663.

Bowman JD. GIS model of power lines used to study EMF and childhood leukemia. *Public Health GIS News and Information*. 2000;23:7-10.

Boyle E, Johnson H, Kelly A, McDonnell R. Congenital anomalies and proximity to landfill sites. *Ir Med J.* 2004;97(1):16-18.

Brender JD, Zhan FB, Suarez L, Langlois PH, Moody K. Maternal residential proximity to waste sites and industrial facilities and oral clefts in offspring. *J Occup Environ Med.* 2006a; 48(6):565-572.

Brender JD, Zhan FB, Suarez L, et al. Linking environmental hazards and birth defects data. *Int J Occup Environ Health*. 2006b;12(2):126-133.

Brender JD, Zhan FB, Langlois PH, Suarez L, Scheuerle A. Residential proximity to waste sites and industrial facilities and chromosomal anomalies in offspring. *Int J Hyg Environ Health*. 2008a;211(1-2):50-58.

Brender JD, Suarez L, Langlois PH, Steck M, Zhan FB, Moody K. Are maternal occupation and residential proximity to industrial sources of pollution related? *J Occup Environ Med*. 2008b;50(7):834-839.

Brooks N, Sethi R. The Distribution of Pollution: Community Characteristics and Exposure to Air Toxics. *Journal of Environmental Economics and Management*. 1997;32(2):233-250.

Brulle RJ, Pellow DN. Environmental Justice: Human Health and Environmental Inequalities. *Annual Review of Public Health*, 2006;27(1):103-124.

Bryant B, ed. *Environmental Justice: Issues, Policies, and Solutions*. Washington, DC: Island Press; 1995.

Bullard RD (Ed). *Unequal Protection: Environmental Justice and Communities of Color*. San Francisco: Sierra Club Books; 1994.

Bullard RD, Mohai P, Saha R, Wright B. *Toxic Waste and Race at Twenty, 1987-2007: A Report Prepared for the United Church of Christ Justice and Witness Ministries.* Cleveland, Ohio: Justice and Witness Ministries, United Church of Christ; 2007a.

Bullard RD, Mohai P, Wright B, Saha R et al. An Open Letter to the Members of Congress: Toxic Wastes and Race at Twenty, 1987-2007: Grassroots Struggles to Dismantle

Environmental Racism in the United States. July 20, 2007; 2007b. (http://www.ejrc.cau.edu/TWART_Letter_Congress.pdf Accessed March 26, 2009.)

Burke LM. Environmental Equity In Los Angeles. *National Center for Geographic Information and Analysis (NCGIA): Technical Report 93-6.* Santa Barbara, CA: NCGIA; 1993.

Buzzelli M, Jerrett M. Burnett R, Finklestein N. Spatiotemporal Perspectives on Air Pollution and Environmental Justice in Hamilton, Canada, 1985–1996. *Annals of the Association of American Geographers*. 2003;93(3):557-573.

Camacho DE, ed. *Environmental Injustices, Political Struggles: Race Class, and the Environment.* Durham, NC: Duke University Press; 1998.

Canfield MA, Ramadhani TA, Langlois PH, Waller DK. Residential mobility patterns and exposure misclassification in epidemiologic studies of birth defects. *J Expo Sci Environ Epidemiol*. 2006;16(6):538-543.

Carozza SE, Li B, Elgethun K, Whitworth R. Risk of childhood cancers associated with residence in agriculturally intense areas in the United States. *Environ Health Perspect*. 2008;116(4):559-565.

Carozza SE, Li B, Wang Q, Horel S, Cooper S. Agricultural pesticides and risk of childhood cancers. *Int J Hyg Environ Health*. 2009;212(2):186-195.

Chakraborty J. Acute exposure to extremely hazardous substances: an analysis of environmental equity. *Risk Analysis*. 2001;21:883-894.

Chakraborty J. Evaluating the environmental justice impacts of transportation improvement projects in the US. *Transportation Research Part D.* 2006;11:315–323.

Chakraborty J. Automobiles, air toxics, and adverse health risks: environmental inequities in Tampa Bay, Florida. *Annals of the Association of American Geographers*. 2009;99(4):647-697.

Chakraborty J, Armstrong MP. Exploring the use of buffer analysis for the identification of impacted areas in environmental equity assessment. *Cartography and Geo Info Systems*. 1997;24:145-157.

Chakraborty J, Armstrong MP. 2001. Assessing the impact of airborne toxic releases on populations with special needs. *The Professional Geographer*. 2001;53:119-131.

Chakraborty J,Armstrong MP. Using geographic plume analysis to assess community vulnerability to hazardous accidents. *Computers, Environment, and Urban Systems* 1995;19(5.6):1.17. 49, 223-227.

Chakraborty J, Schweitzer L, Forkenbrock, D. Using GIS to assess the environmental justice consequences of transportation system changes. *Transactions in GIS*. 1999;3(3):239-258

Chakraborty J, Zandbergen P. Children at risk: Measuring racial/ethnic disparities in potential exposure to air pollution at school and home. *Journal of Epidemiology and Community Health*; 2007;61:1074-1079.

Choi HS, Shim YK, Kaye WE, Ryan PB. Potential residential exposure to Toxic Release Inventory chemicals during pregnancy and childhood brain cancer. *Environ Health Perspect*. 2006;114(7):1113-1118.

Choi AL, Levy JI, Dockery DW, Ryan LM, Tolbert PE, Altshul LM, et al. Does living near a Superfund site contribute to higher polychlorinated biphenyl (PCB) exposure? *Environ.Health Perspect*. 2006 07;114(7):1092-1098.

Clarke J, Gerlak A. Environmental racism in southern Arizona. In: *Environmental Injustices, Political Struggles: Race, Class, and the Environment* (Camacho DE, ed). Durham, NC: Duke University Press; 1998.

Clarke KC, McLafferty S, Templaski B. On epidemiology and geographic information systems: a review and discussion of future direction. *Emerging Infectious Diseases* 1996;2 (2)85–92.

Cliff AD, Ord JK. Spatial processes: Models and Applications. London: Pion Limited; 1981.

Colt JS, Blair A. Parental occupational exposures and risk of childhood cancer. *Environ Health Perspect*. 1998;106(Suppl 3):909-925.

Cordier S, Chevrier C, Robert-Gnansia E, Lorente C, Brula P, Hours M. Risk of congenital anomalies in the vicinity of municipal solid waste incinerators. *Occup Environ Med*. 2004;61(1):8-15.

Cresswell PA, Scott JE, Pattenden S, Vrijheid M. Risk of congenital anomalies near the Byker waste combustion plant. *J Public Health Med.* 2003;25(3):237-242.

Croen LA, Shaw GM, Sanbonmatsu L, Selvin S, Buffler PA. Maternal residential proximity to hazardous waste sites and risk for selected congenital malformations. *Epidemiology*. 1997;8(4):347-354.

Crosignani P, Tittarelli A, Borgini A, et al. Childhood leukemia and road traffic: A population-based case-control study. *Int J Cancer*. 2004;108(4):596-599.

Cutter SL, Holm D, Clark L. The role of geographic scale in monitoring environmental justice. *Risk Analysis*. 1996;16:517-526.

Czeizel AE, Hegedus S, Timar L. Congenital abnormalities and indicators of germinal mutations in the vicinity of an acrylonitrile producing factory. *Mutat Res.* 1999;427(2):105-123.

Daniels G, Friedman S. Spatial inequality and the distribution of industrial toxic releases: evidence from the 1990 TRI. *Social Science Quarterly*. 1999;80:244-62.

De Medeiros AP, Gouveia N, Machado RP, de Souza MR, Alencar GP, Novaes HM, de Almelda MF. Traffic-related air pollution and perinatal mortality: a case-control study. *Environ Health Perspect*. 2009;117(1):127-32.

Diez Roux AV. Investigating neighborhood and area effects on health. *Am J Public Health*. 2001; 91(11):1783-1789.

DiGiuseppe DL, Aron DC, Ranbom L, Harper DL, Rosenthal GE. Reliability of birth certificate data: a multi-hospital comparison to medical records information. *Matern Child Health J.* 2002;6(3):169-79.

Dodds, L, Seviour R. Congenital anomalies and other birth outcomes among infants born to women living near a hazardous waste site in Sydney, Nova Scotia. *Can J Public Health*. 2001;92(5):331-334.

Dolinoy DC, Miranda ML. GIS modeling of air toxics releases from TRI-reporting and non-TRI reporting facilities: impacts for environmental justice. *Environmental Health Perspectives*. 2004;112 (17):1717-1724.

Dolk H, Vrijheid M, Armstrong B, Abramsky L, et al. Risk of congenital anomalies near hazardous-waste landfill sites in Europe: the EUROHAZCON study. *Lancet*. 1998;352(9126):423-427.

Dorling D, Fairbairn D. *Mapping: Ways of Representing the World*. Harlow, UK: Addison, Wesley, Longman, Ltd;1997.

Downey L. Environmental racial inequality in Detroit. Social Forces. 2006;85(2): 772-796.

Dummer TJ, Dickinson HO, Parker L. Adverse pregnancy outcomes around incinerators and crematoriums in Cumbria, north west England, 1956-93. *J Epidemiol Community Health*. 2003;57(6):456-461.

Dunn EC, Kingham PS, Rowlingson B, Bhopal SR, Cocking S, Foy JWC, Acquilla DC, Halpin J, Diggle P, Walker D. Analysing spatially referenced public health data: a comparison of three methodological approaches. *Health and Place* 2001;7:1–12.

Edwards J, Walters S, Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. *Arch.Environ.Health* 1994 1994;49(4):223-227.

Eizaguirre-Garcia D, Rodriguez-Andres C, Watt GC. Congenital anomalies in Glasgow between 1982 and 1989 and chromium waste. *J Public Health Med.* 2000;22(1):54-58.

Elliott P, Briggs D, Morris S, et al. Risk of adverse birth outcomes in populations living near landfill sites. *BMJ*. 2001;323(7309):363-368.

Elliott P, Richardson S, Abellan JJ, et al. Geographic density of landfill sites and risk of congenital anomalies in England. *Occup Environ Med.* 2009;66(2):81-89.

Elliott P, Savitz DA. Design issues in small-area studies of environment and health. *Environ.Health Perspect.* 2008 08;116(8):1098-1104.

English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L. Examining associations between childhood asthma and traffic flow using a geographic information system. *Environ.Health Perspect.* 1999;107(9):761-767.

Evans GW, Kantrowitz E. Socioeconomic Status And Health: The Potential Role of Environmental Risk Exposure. *Annual Review of Public Health*. 2002;23(1):303.

Faber DR. The Struggle for Ecological Democracy: Environmental Justice Movements in the United States, The Guilford Press: New York, NY; 1998.

Fell DB, Dodds L, King WD.. Residential mobility during pregnancy. *Paediatr Perinat Epidemiol*. 2004;18(6):408-414.

Fielder HM, Poon-King CM, Palmer SR, Moss N, Coleman G. Assessment of impact on health of residents living near the Nant-y-Gwyddon landfill site: retrospective analysis. *BMJ*. 2000;320(7226):19-22.

Fisher JB, Kelly M, Romm J. Scales of environmental justice: combining GIS and spatial analysis for air toxics in West Oakland, California. *Health & Place*. 2006;12: 701–714.

Fitzgerald M, Schuurman N, Dragicevic S. The utility of exploratory spatial data analysis in the study of tuberculosis incidences in an urban Canadian population. *Cartographica* 2004;39(2):29–39.

Fotheringham SA, Brunsdon C, Charlton ME. *Geographically Weighted Regression: The Analysis of Spatially Varying Relationships*. Chichester, U.K.: Wiley; 2002.

Fricker RD, Hengartner NW. Environmental equity and the distribution of toxic release inventory and other environmentally undesirable sites in metropolitan NYC. *Environmental and Ecological Statistics*. 2001;8:33-52.

Genereux M, Auger N, Goneau M, Daniel M. Neighbourhood socioeconomic status, maternal education, and adverse birth outcomes among mothers living near highways. *J Epidemiol Community Health*. 2008;62(8):695-700.

Geschwind SA, Stolwijk JA, Bracken M, Fitzgerald E, Stark A, Olsen C, Melius J. Risk of congenital malformations associated with proximity to hazardous waste sites. *Am J Epidemiol*. 1992;135:1197-207.

Gilbert A. Air Toxics and Equity: A Geographic Analysis of Environmental Health Risks in Florida. Master's Thesis. Tampa, FL: University of South Florida; 2009.

Gilbert A, Chakraborty J. Health risks from hazardous air pollutants: analyzing environmental injustice in Florida. *The Florida Geographer*. 2008;39:71-94.

Gilbreath S, Kass PH. Fetal and neonatal deaths and congenital anomalies associated with open dumpsites in Alaska native villages. *Int J Circumpolar Health.* 2006;65(2):133-147.

Glickman TS. Measuring environmental equity with Geographical Information Systems. *Renewable Resources J.* 1994;12:17-21.

Glickman TS, Hersh R. Evaluating environmental equity: the impacts of industrial hazards on selected social groups in Allegheny County, Pennsylvania. *Discussion Paper 95-13*, Washington, DC: Resources for the Future; 1995.

Goldberg MS, Goulet L, Riberdy H, Bonvalot Y. Low birth weight and preterm births among infants born to women living near a municipal solid waste landfill site in Montreal, Quebec. *Environ Res.* 1995;69(1):37-50.

Goldhaber MK. Fetal death ratios in a prospective study compared to state fetal death certificate reporting. *Am J Public Health*. 1989;79(9):1268-1270.

Goldman BA, Fitton L. *Toxic Wastes and Race Revisited: An Update of the 1987 Report on the Racial and Socioeconomic Characteristics of Communities with Hazardous Waste Sites.*Washington, DC: Center for Policy Alternatives; 1994.

Goldman BA. *Not Just Prosperity: Achieving Sustainability with Environmental Justice*. Washington, DC: National Wildlife Foundation; 1993.

Gore DC, Chez RA, Remmel RJ, Harahan M, Mock M, Yelverton R. Unreliable medical information on birth certificates. *J Reprod Med*. 2002;47(4):297-302.

Gragg RD, Christaldi RA, Leong S, Cooper M. The location and community demographics of targeted environmental hazardous sites in Florida. *Journal of Land Use & Environmental Law*. 1996;12:1-24.

Green RS, Smorodinsky S, Kim JJ, McLaughlin R, Ostro B. Proximity of California Public Schools to Busy Roads. *Environmental Health Perspectives*. 2004;112(1):61-66.

Greenberg M. Proving environmental inequity in siting locally unwanted land uses. *J Risk - Iss Health Saf* 1993;4(3):235–252.

Grineski SE. Incorporating health outcomes into environmental justice research: The case of children's asthma and air pollution in Phoenix, Arizona. *Environmental Hazards*. 2007;7:360-371.

Grineski SE. Human-environment interactions and environmental justice: How do diverse parents of asthmatic children minimize hazards? *Society and Natural Resources*. 2009;22 (8):727-743.

Grineski SE, Collins T. Exploring environmental injustice in the Global South: Maquiladoras in Ciudad Juárez. *Population and Environment*. 2008;29: 247–270.

Gunier RB, Hertz A, Von Behren J, Reynolds P. Traffic density in California, socioeconomic and ethnic differences among potentially exposed children. *Journal of Exposure Analysis and Environmental Epidemiology*. 2003;13: 240–246.

Hall HI, Kaye WE, Gensburg LS. Residential proximity to hazardous waste sites and risk of end-stage renal disease. *J. Environ. Health* 1996;59:17-22.

Harrison RM, Leung PL, Somervaille L, Smith R, Gilman E. Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations. *Occup Environ Med.* 1999;56(11):774-780.

Harter L, Starzyk P, Frost F. A comparative study of hospital fetal death records and Washington State fetal death certificates. *Am J Public Health*. 1986;76(11):1333-1334.

Hearne SA, Locke PA, Mellman M, Loeb P, Dropkin L, Bolger G, Fink N, Byrnes M. 2000. Public Opinion About Public Health — United States, 1999. *Morbidity and Mortality Weekly Report Centers for Disease Control.* 2000;49(12):258-260.

Higgs G, Langford M. GIScience, environmental justice, & estimating populations at risk: The case of landfills in Wales. *Applied Geography*, 2009;29(1):63-76.

Hird JA. Environmental policy and equity: the case of Superfund. *Journal of Policy Analysis and Management*. 1993;1(2):323-343.

Holt JB, Lo CP, Hodler RW. Dasymetric estimation of population density and areal interpolation of census data. *Cartography and Geographic Information Science*. 2004;31(2):103-121.

Hu Z, Liebens J, Rao KR. Linking stroke mortality with air pollution, income, and greenness in northwest Florida: an ecological geographical study. *Int J Health Geogr* 2008 05/01;7:20-20.

Jacquez GM. Spatial Analysis in Epidemiology: Nascent Science or a Failure of GIS? *Journal of Geographical Systems* 2000;2:91–97.

Jarup, L., 2004. Health and Environment Information Systems for Exposure and Disease Mapping, and Risk Assessment. Environmental Health Perspectives v. 112 no. 9 p. 995-7.

Jarup L, Briggs D, de Hoogh C, et al. Cancer risks in populations living near landfill sites in Great Britain. *Br J Cancer*. 2002;86(11):1732-6.

Jarup L, Morris S, Richardson S, et al. Down syndrome in births near landfill sites. *Prenat Diagn*. 2007;27(13):1191-1196.

Johnston BR, ed. *Who Pays the Price? The Sociocultural Context of Environmental Crisis.* Washington, DC: Island Press; 1994.

Johnson KC, Pan S, Fry R, Mao Y. Residential proximity to industrial plants and non-Hodgkin lymphoma. *Epidemiology*. 2003;14(6):687-693.

Kaatsch P, Spix C, Schulze-Rath R, Schmiedel S, Blettner M. Leukaemia in young children living in the vicinity of German nuclear power plants. *Int J Cancer*. 2008;(4):122:721-726.

Kearney G, Kiros GE. A spatial evaluation of socio demographics surrounding National Priorities List sites in Florida using a distance-based approach. *International Journal of Health Geographics*. 2009;8:33.

Khoury MJ, Stewart W, Weinstein A, Panny S, Linday P, Eisenberg M. Residential mobility during pregnancy: implications for environmental teratogenesis. *J Clin Epidemiol*. 1988;41(1):15-20.

Kloppenborg SCh, Brandt UK, Gulis G, Ejstrud B. Risk of congenital anomalies in the vicinity of waste landfills in Denmark; an epidemiological study using GIS. *Cent Eur J Public Health*. 2005;13(3):137-143.

Knox EG. Childhood cancers, birthplaces, incinerators, and landfill sites. *Int J Epidemiol*. 2000;29(3):391-7.

Knox EG. Roadways, railways, and childhood cancers. *J Epidemiol Community Health*. 2006;60(2):136-141.

Knox EG, Gilman EA. Hazard proximities of childhood cancers in Great Britain from 1953-80. *J Epidemiol Community Health.* 1997;51(2):151-159.

Kouznetsova M, Huang X, Ma J, Lessner L, Carpenter DO. Increased rate of hospitalization for diabetes and residential proximity of hazardous waste sites. *Environ.Health Perspect.* 2007 01;115(1):75-79.

Krakoff S. "Tribal Sovereignty and Environmental Justice." Pp. 161-183 in Justice and Natural Resources: Concepts, Strategies, and Applications, edited by Kathryn M. Mutz, Gary C. Bryner, and Douglas S. Kenney. Washington: Island Press; 2002.

Kriesel W, Centner TJ, Keeler AG. Neighborhood exposure to toxic releases: are there racial inequities? *Growth and Change*. 1996; 27:479–99.

Kuehn CM, Mueller BA, Checkoway H, Williams M. Risk of malformations associated with residential proximity to hazardous waste sites in Washington State. *Environ Res.* 2007;103(3):405-412.

Kulldorff M. Geographical information systems (GIS) and community health: some statistical issues. *Journal of Public Health Management Practice*. 1999;5(2):100–106.

Langholz B, Ebi KL, Thomas DC, Peters JM, London SJ. Traffic density and the risk of childhood leukemia in a Los Angeles case-control study. *Ann Epidemiol*. 2002;12(7):482-487.

Langlois PH, Brender JD, Suarez L, Zhan FB, Mistry JH, Scheuerle A, Moody K. Maternal residential proximity to waste sites and industrial facilities and conotruncal heart defects in offspring. *Paediatr Perinat Epidemiol.* 2009;23(4):321-331.

Linder SH, Marko D, Sexton K. 2008. Cumulative Cancer Risk from Air Pollution in Houston: Disparities in Risk Burden and Social Disadvantage. *Environmental Science & Technology*. 2008; 42(12):4312-4322.

Liu CC, Chen CC, Wu TN, Yang CY. Association of brain cancer with residential exposure to petrochemical air pollution in Taiwan. *J Toxicol Environ Health A*. 2008;71(5):310-314.

Liu F. Environmental Justice Analysis: Theories, Methods, and Practice. New York: CRC Press; 2001.

Livingstone AE, Shaddick G, Grundy C, Elliott P. Do people living near inner city main roads have more asthma needing treatment? Case control study. *BMJ* 1996 03/16;312(7032):676-677.

London SJ, Thomas DC, Bowman JD, Sobel E, Cheng TC, Peters JM. Exposure to residential electric and magnetic fields and risk of childhood leukemia. *Am.J.Epidemiol.* 1991 11/01:134(9):923-937.

Maantay JA. Zoning, Equity, and Public Health. *American Journal of Public Health*. 2001;91 (7):1033–1041.

Maantay JA. Mapping Environmental Injustices: Pitfalls and Potential of Geographic Information Systems (GIS) in Assessing Environmental Health and Equity. *Environmental Health Perspectives*. 2002;110(Supp 2):161-171.

Maantay JA. Asthma and air pollution in the Bronx: methodological and data considerations in using GIS for environmental justice and health research. *Health and Place*. 2007 03/28;13(1):32-56.

Maantay JA, Maroko A. Mapping urban risk: Flood hazards, race, & environmental justice in New York. *Applied Geography*. 2009;29(1):111–124.

Maantay JA, Maroko A, Porter-Morgan H. A New Method for Population Mapping and Understanding the Spatial Dynamics of Disease in Urban Areas, *Urban Geography*. 2008;29(7):724-738.

Maantay JA, Maroko A, Herrmann C. Mapping Population Distribution in the Urban Environment: The Cadastral-based Expert Dasymetric System (CEDS), *Cartography and Geographic Information Science*, 2007;34(2): 77-102.

Maantay JA, Maroko AR, and Culp G. Using Geographic Information Science to Estimate Vulnerable Urban Populations for Flood Hazard and Risk Assessment in New York City, in Showalter, P., and Lu, Y. eds., *Geotechnical Contributions to Urban Hazard and Disaster Analysis*, 2009a, Springer-Verlag.

Maantay J A, Maroko AR, Alicea C, and Strelnick A H. Geographic Information Systems, Environmental Justice, and Health Disparities: The Need for An Interdisciplinary Approach to Study Asthma and Air Pollution in the Bronx, New York, in: Freudenberg, N., Saegert, S., and Klitzman, S., editors, *Urban Health and Society: Interdisciplinary Approaches to Research and Practice*, Chapter 5, 2009b; 93-126, Jossey Bass.

Maantay JA, Tu J, Maroko AR. Loose-coupling an air dispersion model and a geographic information system (GIS) for studying air pollution and asthma in the Bronx, New York City. *Int.J.Environ.Health Res.* 2009c;19(1):59-79.

Maantay JA, Porter-Morgan H. *Asthma Literature Review*. Unpublished Study. City University of New York, Lehman College, Urban GISc Lab; 2004.

Maantay JA, Timander L, Graziosi G, Meyers L. *The Bronx Toxic Release Inventory Report*. New York: Center for a Sustainable Urban Environment/U.S. Environmental Protection Agency, Region 2; 1997.

Maheswaran R, Elliott P. Stroke mortality associated with living near main roads in England and wales: a geographical study. *Stroke*. 2003;12/13;34(12):2776-2780.

Malik S, Schecter A, Caughy M, Fixler DE. Effect of proximity to hazardous waste sites on the development of congenital heart disease. *Arch Environ Health*. 2004;59(4):177-181.

Margai FL. Health risks and environmental inequity: a geographical analysis of accidental releases of hazardous materials. *The Professional Geographer*. 2001;53(1):422-434.

Maroko A, Maantay J, Sohler N, Grady K, Arno P. The complexities of measuring access to parks and physical activity sites in New York City: a quantitative and qualitative approach. *Int.J.Health Geogr.* 2009;8(1):34.

Marshall EG, Gensburg LJ, Deres DA, Geary NS, Cayo MR. Maternal residential exposure to hazardous wastes and risk of central nervous system and musculoskeletal birth defects. *Arch Environ Health*. 1997;52(6):416-425.

McMaster RB, Leitner H, Sheppard E. GIS-based environmental equity and risk assessment: methodological problems and prospects. *Cartogr Geogr Inform Sys* 1997;24(3):172–189.

Mennis J. Using Geographic Information Systems to Create and analyze statistical surfaces of population and risk for environmental justice analysis. *Social Science Quarterly*. 2002;83:281–97.

Mennis J. Generating surface models of population using dasymetric mapping. *The Professional Geographer* 2003; 55(1): 31-42.

Mennis J, Jordan L. The distribution of environmental equity: exploring spatial non-stationarity in multivariate models of air toxic releases. *Annals of the Association of American Geographers*. 2005;95(2):249-268.

Mirabelli MC, Wing S, Marshall SW, Wilcosky TC. Race, Poverty, and Potential Exposure of Middle-School Students to Air Emissions from Confined Swine Feeding Operations. *Environmental Health Perspectives*, 2006;114(4):591-596.

Mohai P, Bryant B. Environmental racism: reviewing the evidence. In: Bryant B, Mohai B, eds. *Race and the Incidence of Environmental Hazards: A Time for Discourse*. Boulder, CO: Westview Press; 1992:163–176.

Mohai P, Lantz PM, Morenoff J, House JS, Mero RP. Racial and socioeconomic disparities in residential proximity to polluting industrial facilities: evidence from the Americans' Changing Lives Study. *American Journal of Public Health*; 2009;99(S3):649-655.

Mohai P, Saha R. Reassessing racial and socio-economic disparities in environmental justice research. *Demography*. 2006;43(2): 383–399.

Mohai P, Saha R. 2007. Racial inequality in the distribution of hazardous waste: a national-level reassessment. *Social Problems*. 2007;54:343–70.

Moore D, Carpenter TE. Spatial analytical methods and geographic information systems: use in health research and epidemiology. *Epidemiologic Reviews* 1999;21(2):143–161.

Morello-Frosch R, Jesdale W. Separate and Unequal: Residential Segregation and Estimated Cancer Risks Associated with Ambient Air Toxics in U.S. Metropolitan Areas. *Environ Health Perspect.* 2006;114:1-8.

Morello-Frosch R, Pastor M, Sadd J. Environmental justice and Southern California's "riskscape"—the distribution of air toxics exposures and health risks among diverse communities. *Urban Affairs Review*. 2001;36:551–578.

Morgan OW, Vrijheid M, Dolk H. Risk of low birth weight near EUROHAZCON hazardous waste landfill sites in England. *Arch Environ Health*. 2004;59(3):149-151.

Morgenstern H. Ecologic studies. In: Rothman KJ, Greenland S, Lash TL, eds. *Modern Epidemiology*. Philadelphia, PA: Wolters Kluwer/Lippincott Williams and Wilkins; 2008:511-531.

Morris MS, Knorr RS. Adult leukemia and proximity-based surrogates for exposure to Pilgrim plant's nuclear emissions. *Arch. Environ. Health* 1996 1996;51(4):266-274.

Morris SE, Thomson AO, Jarup L, de Hoogh C, Briggs DJ, Elliott P. No excess risk of adverse birth outcomes in populations living near special waste landfill sites in Scotland. *Scott Med J.* 2003;48(4):105-107.

Most MT, Sengupta R, Burgener MA. Spatial scale and population assignment choices in environmental justice analyses. *The Professional Geographer*. 2004;56(4): 574–586.

Mueller BA, Kuehn CM, Shapiro-Mendoza CK, Thomashek KM. Fetal deaths and proximity to hazardous waste sites in Washington State. *Environ Health Perspect*. 2007;115(5):776-80.

National Birth Defects Prevention Network (NBDPN). State Birth Defects Surveillance Program Directory. *Birth Defects Res A Clin Mol Teratol*. 2007;79(12):815-873.

National Center for Minority Health and Health Disparities, 2009 website http://ncmhd.nih.gov/hdFactSheet_gap.asp

National Institutes of Environmental Health Sciences, Strategic Plan for the Elimination of Health Disparities, www.niehs.nih.gov/translat/hd/hd-strat.pdf (Accessed December 19, 2004.)

National Institutes of Health, 2005. Addressing Health Disparities: The NIH Program of Action http://healthdisparities.nih.gov/whatare.html (accessed January 2, 2005)

Nationally Consistent Environmental Justice Screening Approaches Work Group, *Draft Report* of the Nationally Consistent Environmental Justice Screening Approaches Work Group to the National Environmental Justice Advisory Council (NEJAC), January 11, 2010

Neumann CM, Forman DL, Rothlein JE. Hazard screening of chemical releases and environmental equity analysis of populations proximate to toxic release inventory facilities in Oregon. *Environmental Health Perspectives*. 1998;106(4): 217-226.

Nie J, Beyea J, Bonner MR, Han D, Vena JE, Rogerson P, et al. Exposure to traffic emissions throughout life and risk of breast cancer: the Western New York Exposures and Breast Cancer (WEB) study. *Cancer Causes Control*. 2007;18(9):947-955.

Nordenstam BJ. Transformation of grassroots environmental justice into federal agency environmental policy. In *Environmental Science and Engineering Fellow Program*, 1995

Reports (pp. 51-65). Washington, DC: American Association for the Advancement of Science; 1995.

Norton JM, Wing S, Lipscomb HJ, Kaufman JS, Marshall SW, Cravey AJ. Race, Wealth, and Solid Waste Facilities in North Carolina. *Environmental Health Perspectives*. 2007;115(9):1344-1350.

Ochoa-Acuna H, Carbajo C.. Risk of limb birth defects and mother's home proximity to cornfields. *Sci Total Environ*. 2009;407(15):4447-4451.

Omi M, Winant H. Racial Formation in the United States. New York and London: Routledge; 1994.

O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, Wilkinson P, Fletcher T, Cifuentes L, Schwarz J. Health, wealth, and air pollution: advancing theory and methods. *Environmental Health Perspectives*. 2003;111(16):1861-1870.

Oosterlee A, Drijver M, Lebret E, Brunekreef B. Chronic respiratory symptoms in children and adults living along streets with high traffic density. *Occup.Environ.Med.* 1996 04;53(4):241-247.

Orr M, Bove F, Kaye W, Stone M. Elevated birth defects in racial or ethnic minority children of women living near hazardous waste sites. *Int J Hyg Environ Health*. 2002;205(1-2):19-27.

Palmer SR, Dunstan FD, Fielder H, Fone DL, Higgs G, Senior ML. Risk of congenital anomalies after the opening of landfill sites. *Environ Health Perspect*. 2005;113(10):1362-1365.

Pastor M, Morello-Frosch R, Sadd J. The air is always cleaner on the other side: race, space, and ambient air toxics exposures in California. *Journal of Urban Affairs*. 2005;27(2):127-148.

Pastor M, Sadd JL, Morello-Frosch R. 2004. Waiting to inhale: the demographics of toxic air releases in 21st century California. *Social Science Quarterly*. 2004;85(2):420-440.

Pellow DN, Brulle RJ, eds. *Power, Justice, and the Environment: A Critical Appraisal of the Environmental Justice Movement.* The MIT Press, Cambridge, MA; 2005.

Perlin SA, Setzer RW, Creason J, Sexton K. Distribution of industrial air emissions by income and race in the United States: an approach using the toxic release inventory. *Environmental Science Technology*. 1995;29(1):69-80.

Perlin SA, Wong D, Sexton K. Residential proximity to industrial sources of air pollution: interrelationships among race, poverty, and age. *Journal of the Air and Waste Management Association*. 2001;51:406–421.

Perlin SA, Sexton K, Wong D. An examination of race and poverty for populations living near industrial sources of air pollution. *J Exp Anal Env Epdemiol*. 1999;9:29-48.

Perlin SA, Wong D, Sexton K. Residential proximity to industrial sources of air pollution: interrelationships among race, poverty, and age. *J Air Waste Manag Assoc*. 2001; 51(3):406-421.

Pollock PH, Vittas ME. 1995. Who bears the burden of environmental pollution? race, ethnicity, and environmental equity in Florida. *Social Science Quarterly*. 76(2):294-310.

Raaschou-Nielsen O, Hertel O, Thomsen BL, Olsen JH. Air pollution from traffic at the residence of children with cancer. *Am J Epidemiol*. 2001;153(5):433-443.

Reynolds P, Von Behren J, Gunier RB, Goldberg DE, Hertz A, Harnly ME. Childhood cancer and agricultural pesticide use: an ecologic study in California. *Environ Health Perspect*. 2002a;110(3):319-324.

Reynolds P, Von Behren J, Gunier RB, Goldberg DE, Hertz A, Smith D. Traffic patterns and childhood cancer incidence rates in California, United States. *Cancer Causes Control.* 2002b; 13(7):665-673.

Reynolds P, Von Behren J, Gunier RB, Goldberg DE, Hertz A. Residential exposure to traffic in California and childhood cancer. *Epidemiology*. 2004;15(1):6-12.

Reynolds P, Von Behren J, Gunier RB, Goldberg DE, Harnly M, Hertz A. Agricultural pesticide use and childhood cancer in California. *Epidemiology*. 2005;16(1):93-100.

Richards TB, Croner CM, Rushton G, Brown CK, Folwer L. Geographic information systems and public health: mapping the future. *Public Health Reports*. 1999:114, 359–373.

Ringquist EJ. Equity and the distribution of environmental risk: the case of TRI facilities. *Social Science Quarterly*.1997;78:811–829.

Roohan PJ, Josberger RE, Acar J, Dabir P, Feder HM, Gagliano PJ. Validation of birth certificate data in New York State. *J Community Health*. 2003;28(5):335-346.

Rull RP, Gunier R, Von Behren J, et al. Residential proximity to agricultural pesticide applications and childhood acute lymphoblastic leukemia. *Environ Res.* 2009;109(7):891-899.

Rull RP, Ritz B, Shaw GM. Neural tube defects and maternal residential proximity to agricultural pesticide applications. *Am J Epidemiol*. 2006;163(8):743-753.

Rushton G, Elmes G, McMaster R. Considerations for improving geographic information system research in public health. *URISA Journal*. 2000;12(2):31–49.

Sadd JL, Pastor Jr. M, Boer JT, Snyder LD. 1999. "Every breath you take...": the demographics of toxic air releases in Southern California. *Economic Development Quarterly*. 1999;13:107-23.

Sarov B, Bentov Y, Kordysh E, et al. Perinatal mortality and residential proximity to an industrial park. *Arch Environ Occup Health*. 2008;63(1):17-25.

Savitz DA, Chen JH. Parental occupation and childhood cancer: review of epidemiologic studies. *Environ Health Perspect.* 1990;88(Aug):325-337.

Sexton K. Sociodemographic aspects of human susceptibility to toxic chemicals:Do class and race matter for realistic risk assessment? *Environmental Toxicology and Pharmacology*. 1997;4 261-269.

Sharp L, Black RJ, Harkness EF, McKinney PA. Incidence of childhood leukaemia and non-Hodgkin's lymphoma in the vicinity of nuclear sites in Scotland, 1968-93. *Occup Environ Med.* 1996;53(5):823-831.

Shaw GM, Malcoe LH. Residential mobility during pregnancy for mothers of infants with or without congenital cardiac anomalies: a reprint. *Arch Environ Health*. 1992;47(3):236-238.

Shaw GM, Schulman J, Frisch JD, Cummins SK, Harris JA. Congenital malformations and birthweight in areas with potential environmental contamination. *Arch Environ Health*. 1992;47(2):147-154.

Sheppard E, Leitner H, McMaster RB, Hongguo T. GIS based measures of environmental equity: exploring their sensitivity and significance. *J Expos Anal Environ Epidemiol* 1999;9:18–28.

Sicotte D, Swanson S. 2007. Whose risk in Philadelphia? Proximity to unequally hazardous industrial facilities. *Social Science Quarterly*. 2007;88:515-534.

Smargiassi A, Kosatsky T, Hicks J, Plante C, Armstrong B, Villeneuve PJ, et al. Risk of asthmatic episodes in children exposed to sulfur dioxide stack emissions from a refinery point source in Montreal, Canada. *Environ.Health Perspect*. 2009 04/21;117(4):653-659.

Sosniak WA, Kaye WE, Gomez TM. Data linkage to explore the risk of low birthweight associated with maternal proximity to hazardous waste sites from the National Priority List. *Arch Environ Health*. 1994;49(4):251-255.

Spix C, Schmiedel S, Kaatsch P, Schulze-Rath R, Blettner M. Case-control study on childhood cancer in the vicinity of nuclear power plants in Germany 1980-2003. *Eur J Cancer*. 2008;44(2):275-284.

Steffen C, Auclerc MF, Auvrignon A, et al. Acute childhood leukaemia and environmental exposure to potential sources of benzene and other hydrocarbons: a case-control study. *Occup Environ Med.* 2004;61(9):773-778.

Stretesky P, Lynch MJ. Environmental justice and the predictions of distance to accidental chemical releases in Hillsborough County, Florida. *Social Sci Q.* 1999;80(4):830–846.

Suarez L, Brender JD, Langlois PH, Zhan FB, Moody K. Maternal exposures to hazardous waste sites and industrial facilities and risk of neural tube defects in offspring. *Ann Epidemiol*. 2007;17(10):772-777.

Sui D. GIS, environmental equity analysis, and the modifiable areal unit problem (MAUP). In: Craglia M, Onsrud H, eds. *Geographic Information Research: Transatlantic Perspective*. London: Taylor and Francis; 1999:41-54.

Tango T, Fujita T, Tanihata T, et al. Risk of adverse reproductive outcomes associated with proximity to municipal solid waste incinerators with high dioxin emission levels in Japan. *J Epidemiol.* 2004;14(3):83-93.

Taquino M, Parisi D, Gill DA. Units of analysis and the environmental justice hypothesis: the case of industrial hog farms. *Social Science Quarterly*. 2002;83(1):298–316.

Tiefenbacher JP, Hagelman RR. 1999. Environmental equity in urban Texas: race, income, and patterns of acute and chronic toxic air releases in metropolitan counties. *Urban Geography*. 1999;19:516-533.

Tobler WR. A computer movie simulating urban growth in the Detroit region. *Economic Geography*. 1970;46:234-240.

Tobler W. Cellular Geography. In: Steven, G., Gunnar, O. (Eds.), *Philosophy in Geography*. Reidel, Dordrecht, NL; 1979.

Tsai J, Kaye WE, Bove FJ. Wilms' tumor and exposures to residential and occupational hazardous chemicals. *Int J Hyg Environ Health* 2006;209(1):57-64.

United Church of Christ Commission for Racial Justice. *Toxic Wastes and Race in the United States: A National Report on the Racial and Socio-economic Characteristics of Communities with Hazardous Waste Sites.* New York: United Church of Christ; 1987.

Urayama KY, Von Behren J, Reynolds P, Hertz A, Does M, Buffler PA. Factors associated with residential mobility in children with leukemia: implications for assigning exposures. *Ann Epidemiol.* 2009;19(11):834-840.

U.S. Environmental Protection Agency. *Environmental Justice*. (http://www.epa.gov/compliance/environmentaljustice/index.html Accessed March 13, 2009.)

U.S. GAO (Government Accountability Office). *Demographics of People Living Near Waste Facilities*. Washington DC: Government Printing Office; 1995.

van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ.Res*. 1997;74(2):122-132.

Venn AJ, Lewis SA, Cooper M, Hubbard R, Britton J. Living near a main road and the risk of wheezing illness in children. *Am.J.Respir.Crit.Care Med.* 2001 12/15;164(12):2177-2180.

Vinceti M, Rovesti S, Bergomi M, et al. Risk of birth defects in a population exposed to environmental lead pollution. *Sci Total Environ*. 2001;278(1-3):23-30.

Vinceti M, Malagoli C, Teggi S, et al. Adverse pregnancy outcomes in a population exposed to the emissions of a municipal waste incinerator. *Sci Total Environ*. 2008;407(1):116-121.

Vinceti M, Malagoli C, Fabbi S, et al. Risk of congenital anomalies around a municipal solid waste incinerator: a GIS-based case-control study. *Int J Health Geogr.* 2009;8(Feb 10):8.

Vine MF, Degnan D, Hanchette C. Geographic information systems: their use in environmental epidemiologic research. *Environ Health Perspect*. 1997;105(6):598–605.

Von Behren J, Reynolds P, Gunier RB, et al. Residential traffic density and childhood leukemia risk. *Cancer Epidemiol Biomarkers Prev.* 2008;17(9):2298-2301.

Vrijheid M, Dolk H, Armstrong B, et al. Chromosomal congenital anomalies and residence near hazardous waste landfill sites. *Lancet*. 2002;359(9303):320-322.

Walker G, Mitchell G, Fairburn J, Smith G. 2005. Industrial pollution and social deprivation: evidence and complexity in evaluating and responding to environmental inequality. *Local Environment*. 2005;10(4):361–377.

Wall P, Devine O. Interactive analysis of the spatial distribution of disease using a geographic information system. *Journal of Geographical Systems*. 2000;2:243–256.

Waller LA, Louis TA, Carlin BP. Environmental justice and statistical summaries of differences in exposure distributions. *J Exp Anal Env Epdemiol*. 1999;9:56-65.

Weng HH, Tsai SS, Chiu HF, Wu TN, Yang CY. Childhood leukemia and traffic air pollution in Taiwan: petro station density as an indicator. *J Toxicol Environ Health A*. 2009;72(2):83-87.

Wilhelm M, Ritz B. Residential proximity to traffic and adverse birth outcomes in Los Angeles county, California, 1994-1996. *Environ Health Perspect.* 2003;111(2):207-216.

Wilkinson P, Elliott P, Grundy C, Shaddick G, Thakrar B, Walls P, et al. Case-control study of hospital admission with asthma in children aged 5-14 years: relation with road traffic in north west London. *Thorax* 1999 12;54(12):1070-1074.

Wilkinson P, Thakrar B, Shaddick G, Stevenson S, Pattenden S, Landon M, et al. Cancer incidence and mortality around the Pan Britannica Industries pesticide factory, Waltham Abbey. *Occup.Environ.Med.* 1997 02;54(2):101-107.

Wjst M, Reitmeir P, Dold S, Wulff A, Nicolai T, von Loeffelholz-Colberg ,E.F., et al. Road traffic and adverse effects on respiratory health in children. *BMJ* 1993 09/04;307(6904):596-600.

Wood D. The Power of Maps. New York:Guilford Press; 1992.

Woodruff TJ, Parker JD, Kyle AD, Schoendorf KC. Disparities in exposure to air pollution during pregnancy. *Environ Health Perspect*. 2003;111(2):942-946.

Wulff M, Hogberg U, Sandstrom-Holmgren A. 1996. Congenital malformations in the vicinity of a smelter in Northern Sweden, 1973-1990. *Paediatr Perinat Epidemiol*. 1996;10(1):22-31.

Yasnoff WA, Sondik EJ. Geographic Information Systems (GIS) in public health practice in the New Millennium. *Journal of Public Health Management Practice* 1999;5(4):ix–xii.

Yauck JS, Malloy ME, Blair K, Simpson PM, McCarver DG. Proximity of residence to trichloroethylene-emitting sites and increased risk of offspring congenital heart defects among older women. *Birth Defects Res A Clin Mol Teratol.* 2004;70(10):808-814.

Yen IH, Syme SL. The Social Environment and Health: A Discussion of the Epidemiologic Literature. *Ann Rev Public Health*. 1999;20:287-306.

Yu CL, Wang SF, Pan PC, et al. Residential exposure to petrochemicals and the risk of leukemia: using geographic information tools to estimate individual-level residential exposure. *Am J Epidemiol*. 2006;164(3):200-207.

Zandbergen PA, Chakraborty J. Improving environmental exposure analysis using cumulative distribution functions and individual geocoding. *Int Journal of Health Geographics*. 2006;5:23.

Zimmerman R. Issues of classification in environmental equity: how we manage is how we measure. *Fordham Urban Law Journal*. 1994;29(3):633-669.

Appendix A

Table 1	Environmental Justice Research
Table 2	Methodology for Spatial Definition of Proximity and Potential Exposure to Environmental Hazards
Table 3	Studies of Residential Proximity to Environmental Hazards and Adverse Pregnancy Outcomes with Reported Disparities by Race/Ethnicity or Socioeconomic Status
Table 4	Studies of Residential Proximity to Potential Environmental Hazards and Adverse Pregnancy Outcomes, and Childhood Cancers
Table 5	Studies of Residential Proximity to Potential Environmental Hazards and Cardiovascular Respiratory, and other Chronic Diseases

Table 1. Environmental Justice Research

Study	Study Parameters	Environmental Indicators	Methods	Findings
Reference	(Study Extent /	(Category of Environmental	(Determination of	8
	Unit of Analysis /	Indicator /	vulnerable population or	
	Independent Variables)	What's Being Measured)	exposure risk/Evaluation of	
		_	disproportion)	
Anderton et	Extent: MSAs in the	Indicator: Commercial	Population: Spatial	No consistent
al., 1994	United States	hazardous waste treatment,	coincidence by tract	association between
		storage, and disposal (TSD)		location of TSD
	Unit: Census tract	facilities in MSAs	1 1	facilities and the
			of means test to compare	percentage of either
		Measuring: Characteristics of		minority or low-income
		the population outside and		groups.
	employment	inside host areas (based on	presence of facilities as	
		multiple spatial definitions), in	dependent variable	
		all MSAs and the 25 largest		
	T	MSAs	2 1 1 21 21 21	5:1.1:
Apelberg et	Extent: Maryland	Indicator: Hazardous air		Risk disparities for on-
al., 2005		pollutants	mapped to census tracts	road, area and non-road
	Unit: Census Tracts	M : I inlance of consequent	D:	sources exist by SES,
	L. J J V	Measuring: Linkage of cancer risk estimates from NATA to		and on-road and area by
	1 *	racial and socioeconomic	regression; statistical	race. On-road sources contribute most to
		characteristics of census tracts	significance (Pearson's chi-	
	assistance, poverty, and	characteristics of census tracts	squared and relative risk) of	
	education		differences in proportion of	
	Education		high risk census tracts	
			across quartiles of the	
			independent variables	
			independent variables	
Ash and	Extent: Census	Indicator: Toxic Release	Daniel ations. Tract lavel	African Americans tend
Fetter, 2004		Inventory (TRI) facilities and	Population: Tract level chronic risk estimates based	
retter, 2004	areas in the U.S.	emissions, based on EPA's	on pollution plume and	polluted cities and in
		RSEI model		more polluted areas
	Unit: Block group	RSEI model		within cities. Hispanics
		Measuring: Inequities in	Disproportion: Multivariate	
		cumulative risk from TRI		cities on average, but in
		emissions, based on toxicity and		more polluted
		atmospheric dispersion		areas within cities.
	education, housing			
Baden et al.,		Indicator: Superfund sites on	Population: Spatial	Different results for
2007		the National Priorities List	coincidence using four	different scales and
	(California), county (Los	(NPL)	different units of analysis.	units, but strong
	Angeles).			evidence of injustice for
		Measuring: Disparities	Disproportion: Multivariate	Blacks and Hispanics at
		associated with NPL site	logistic regression using	national and state level
		location at county, ZIP code,	μ.	with tract and block
		tract, and block group levels.	dependent variable.	group data.
	Independent Variables:			
	Race, ethnicity, income,			
	percent urban, MSA			
Been and	Extent: United States	<i>Indicator</i> : Solid-waste facilities		Black and Hispanic
Gupta, 1996			coincidence	populations were

Unit: Census Tracts Measuring: Longitudinal comparison of host and non-Independent Variables: Measuring: Longitudinal Disproportion: statistical significance of differences	in 1990; Black
Independent Variables: host census tracts prior to and significance of differences	
	populations did not,
Race, income, after facility siting in proportions of host and	while Hispanic
population density, non-host tracts; logit	populations were
unemployment, function to control for	predictive facility siting
occupation, housing correlation between	within tracts; working
value variables; linear regression	class and not poor
for longitudinal	neighborhoods
comparison; comparative	predictive of facility
static exercises	siting
Boer et al., Extent: Los Angeles Indicator: Hazardous waste Population: Spatial	Both race and ethnicity
1997 County, California treatment, storage, disposal coincidence to select tracts	
facilities(TSDFs) hosting any TSDF, large-	with TSDF location.
Unit: Census tracts capacity TSDFs, and those	
Measuring: Inequities in the within a mile of large-	communities located
Independent Variables: distribution of all TSDFs and capacity TSDFs.	near industrial areas
Race, ethnicity, SES, large-capacity TSDFs	most affected.
	most affected.
residential land, (processing more than 50 tons industrial land, annually) Disproportion: Univariate comparison of host and	
population density, non-host tracts; multvariat	2
registered voters. logit regression.	d
Bolin et al., Extent: Phoenix Indicator: Four types of Population: Combination	
2002 metropolitan area, hazardous industrial and toxic spatial coincidence and	environmental
Arizona waste sites circular buffer analysis to	injustice by class and
measure hazard density	race across a range of
Unit: Census tract Measuring: Inequities based on index for each tract and	hazards in the Phoenix
the number of hazards and type of hazard	metropolitan region
Independent Variables: hazard density indices for each	
Race, ethnicity, income tract Disproportion: Bivariate	
correlation with hazard	
counts and hazard density	
indices	
Boone, 2001 Extent: City of Indicator: TRI facilities. Population: Spatial	Tracts with White,
Baltimore, Maryland coincidence and circular	working-class people
Measuring: Historical analysis buffer.	are more likely to host
Unit: Census tract of the city's residential and	TRI facilities than
industrial geography to explain Disproportion: Compariso	
Independent Variables: current location pattern of TRI of host and non-host areas	r ·
Race, income, level of facilities. based on tract, circular	long history of
education. buffers, and locally define	
neighborhoods	occupational
li cignooi noods	segregation.
Bowen, 1995 Extent: Ohio Indicator: Hazardous air Population: Spatial	Disproportionate
pollutants coincidence examining	burdens by race found
Unit: County, with geographic unit with,	statewide and by
Cuyahoga County at <i>Measuring:</i> The degree of without, or adjacent to	county, but not in
tract level environmental hazard borne by hazardous facility; toxicity	
various population groups by index based on Threshold	tracts. Disproportionate
Independent Variables: measuring spatial distribution of Limit Values and total	burdens additionally
Population density, facilities in relation to pounds of emissions	found by SES variables.
race/ethnicity, value of demographic groups	
owner-occupied homes, Disproportion: Zero-order	
income, rent correlations, partial	are strongly related to
	franulation dansity
correlations, and analysis	n population density.

			assigned to one of three categories based on number	
			of TRI facilities	
Brooks and Sethi, 1997	Extent: United States	Indicator: TRI facilities and emissions	Population: Spatial	ZIP codes with higher Black proportions and
		Measuring: A ZIP code level	Disproportion: Multivariate	
	Race, income, level of	•	exposure index and logistic	to TRI releases.
	value, population	differences and the distance from the emission source used to analyze determinants of disparities.	regression using increase in exposure level as dependent variables.	
Burke, 1993	Extent: Los Angeles County, California	<i>Indicator</i> : Hazardous air pollutants	coincidence	Disproportionate burdens found by race and income, and
		Measuring: The significance of race in the siting of industrial	Disproportion: Bivariate analysis and bivariate	Hispanics are disproportionately
	Income, population	facilities when income and population density is controlled		exposed to TRI facilities regardless of
	density, race/ethnicity			concluded that it is not
D 11: 1	T II . 'I.	X // XX	D. I. J. MOD.	occurrence
Buzelli et al, 2003	Extent: Hamilton, Canada	<i>Indicator</i> : Hazardous air pollutants	Population: TSP concentrations estimated for census tract centroids using	
		Measuring: The relationship between changing TSP	point-kriging interpolation	value. However, disparities decrease
	1 *	characteristics of census tracts	Disproportion: Linear regression using OLS, SAR, and GAM methods	over time
	unemployment, type of employment, lone- parent families, government assistance		or ire, and or ire incurous	
Chakraborty, 2001	Extent: Hillsborough County, Florida	extremely hazardous substances	modeling to determine radii	
	Unit: Block groups	(EHS) Measuring: Inequities in the	buffers for each chemical at	White and impoverished individuals residing in
	Independent Variables: Racial and poverty status.	spatial distribution of exposure to worst-case releases of toxic and flammable chemicals from	Disproportion: Cumulative	areas exposed to

Chakraborty, 2009	Florida Metropolitan statistical area Unit: Census Tracts Independent Variables: Race, ethnicity, income, home ownership, and transportation disadvantaged individuals.	cancer and respiratory risks based on NATA data from inhalation exposure to vehicular emissions of air pollutants	statistics and multivariate regression using OLS; spatial regression using spatial thresholds for neighbor determination to control for spatial autocorrelation	Disproportionate burdens based on race, even when controlling for SES. Also, tracts characterized by high population density and low rates of home/car ownership face significant disparities
Chakraborty and Armstrong, 1997	Iowa Unit: Census block groups Independent Variables:	Indicator: TRI facilities and emissions Measuring: Differences in disparities for proximity to TRI facilities based on modeling method	weighting methods	
	income and race		Disproportion: Descriptive statistics to compare proximate/non-proximate populations	
Chakraborty and Armstrong, 2001	Iowa Unit: Gecoded residential locations	extremely hazardous substances (EHS) Measuring: Inequities in the spatial distribution of exposure to worst-case releases from EHS facilities	Population: Plume	special needs population residing in areas potentially susceptible to worst- case EHS releases
Chakraborty and Zandbergen, 2007	County, Florida Unit: Geocoded school and residence locations; Independent Variables: race (self-reported white, black, Hispanic, or other)	Measuring: Racial disparities in potential exposure to air pollution from TRI facilities, small facilities from EPA's aerometric information retrieval system (AIRS), and major	(concentric) buffer radii around facilities and roads; cumulative distribution function for schools and residences	Black and Hispanic children face a higher burden at any given distance from each type of pollution source
Cutter et al., 1996	Unit: Counties, census tracts, block groups Independent Variables:	Measuring: Inequities in the	Population: Spatial coincidence by county, tract, and block group Disproportion: Bivariate correlation to measure association between facility	A disproportionate burden on White and more affluent counties in urban areas. No relationship between facility location and race or income at the

	income, age, education,	facility at the county, census	presence and characteristics	tract and block group
				levels.
			means tests between host	
			and non-host units	
Friedman,	Extent: United States. Unit: County	Indicator: TRI facilities with air releases	coincidence by county.	Positive relationship between toxic air releases and Black
	Independent Variables:	Measuring: Inequities in the distribution of TRI sites	Disproportion: Multivariate logistic and OLS regression	explained by
	urban proportion, manufacturing	emission amounts (total pounds		urbanization and industrial location. Curvilinear association with economic status.
		Indicator: TRI facilities and non-TRI facilities (smaller emitters)	modeling to estimate	Exposure potential disparities increase as the analytical unit
		Measuring: Inequities in the	spatial units, at each scale	becomes smaller (ZIP code to block), for bot
	Independent Variables:	exposure to toxic air releases	Disproportion: Multivariate regression and cumulative distribution functions to compare exposure potential	race and income.
			differences, at each scale	
•	metropolitan area,	Indicator: TRI facilities Measuring: Disproportionate proximity to TRI facilities,		
	Unit: Census tract	based on several distance decay functions	Disproportion: Bivariate	composition of tracts had a strong
	Independent Variables: Race, ethnicity, income, level of education, housing value, employment.		distribution functions	independent effect on proximity to TRI facilities.
2005	Bay Area, California; West Oakland neighborhood, Oakland, California Unit: Census tracts, block groups, and blocks; neighborhood, city, county, region Independent Variables: race and population	clustering; disparities in point and mobile air pollutant sources; disparities in hazardous air pollutant dispersion for a single facility	Spatial statistical pattern analysis (Ripley's <i>K</i>) to locate statistically significant clusters of point sources of air toxics; population generalized by neighborhood; air dispersion modeling and buffer intersection for a single facility Disproportion: Descriptive	Disproportionate clust of point sources identified at the neighborhood scale, which corresponded to a community with high percentage of non-white and lower incorresidents than surrounding areas; mobile sources disproportionately located near dense populations of non-
	density		neighborhood analysis	populations of non- white and lower incon residents; Ripley's K i useful for identifying statistically significan point source clusters f regulatory prioritization

Fricker and	Extent: New York City	Indicator: Various types of	Population: Spatial	Hispanics are more
Hengartner,		environmentally undesirable	coincidence by tract	proximate to the
2001	New York.	facilities, including TRI sites,	confedence by tract	undesirable sites than
2001	New Tork.	TSDF facilities, landfills,	Disproportion: Log- linear	other groups. Both
	Unit: Census tracts	incinerators, bus garages, and	and logistic generalized	Hispanics and Black
		sewage treatment plants	linear models to determine	more proximate to sites
	Independent Variables:	be wage treatment plants	association of race and	in the Bronx and
	_	Measuring: The relationship	ethnicity with number of	Queens, and less
	population density,	between the total number of	sites, after accounting for	proximate to sites in
	indicators of water	undesirable sites in a tract and	SES variables	Manhattan.
		independent variables.		
	train track.	1		
Gilbert and	Extent: Florida	Indicator: Hazardous air	Population: NATA data	For both cancer and
Chakraborty,		pollutants	mapped to census tracts	respiratory risks,
2008	Unit: Census Tracts			evidence of inequity for
		<i>Measuring</i> : The distribution of	Disproportion: Bivariate	race, ethnicity, and
	Independent Variables:	cumulative cancer and	correlation, multivariate	population density.
		respiratory risks based on	regression analysis	Respiratory risk, but
	age, home ownership,	NATA data on inhalation		not cancer risk, was
		exposure to ambient air toxics		negatively associated
	urban designation			with urban areas.
		Indicator: Hazardous air	Population: Spatial	Disproprtionate burdens
Hersh, 1995	County, Pennsylvania	pollutants, potential chemical	coincidence by tract, block	found for income using
	77 1. 3.6 11.	releases, power plants	group and municipality;	all methods.
	Unit: Municipality,	M : D:	proximity analysis by half	Disproportionate
	census tract, block	Measuring: Disproportionate	mile and one mile buffers	burdens by race in all
	group, and block	risk of fatality based on race,	and plume buffers;	buffers, and not in all
		income and age due to exposure from TRI facilities	weights using RfD and	spatial coincidence models
	Independent Variables: race, income, age	If off TRI facilities	potency carcinogens;	inodeis
	race, meome, age		dispersion modeling using	
			ALOHA, ISCLT2,	
			COMPLEX1 models	
			COM ELM Models	
			Disproportion: GIS in	
			conjunction with census	
			data and impact models	
			estimated the individual	
			fatality rates for each social	
			group. These were	
			compared to the individual	
			fatality rates for the rest of	
			the county's population	
		Indicator: Commercial	Population: Spatial	Minority populations
Fitton, 1994		hazardous waste management	coincidence by ZIP code	were more likely to live
	Unit: ZIP code	facilities	n	in areas where facilities
	7 1 1 277 11		Disproportion: Difference	are located than they
	*	Measuring: Characteristics of	of proportions tests to	were in 1980.
		the population in host and non-		Race/ethnicity was a
	per capita income		ZIP codes in each group,	stronger indicator than
		ZIP codes into five groups based on their level of	and examine changes from 1980	income.
		hazardous activity	1700	
Green et al.,	Extent: California; high-	·	Population: Spatial	As proximity of schools
2004		pollutants (traffic counts on	coincidence for schools and	
	•	busy roads)	census tracts; identification	
l		_ /	, , , , , , , , , , , , , , , , , , , ,	7

	Unit: Census tract and geocoded school locations Independent Variables: race/ethnicity, income, housing data, born outside the U.S., population density, school-related data including number of students eligible for food and work assistance, English-learners, and total school enrollment	Measuring: Determine disparities between schools within 150m of busy roads by race and SES indicators	of road with most traffic within 150 meters of schools as hazard source Disproportion: Logistic regression to compare odds ratios across independent variables based on school groupings by categorical traffic levels	percentage of both black and Hispanic students increased substantially. Potential exposure to traffic also increased in relation to socioeconomic indicators, including English language- learners
Grineski, 2007	Extent: Phoenix, Arizona Unit: ZIP code Independent Variables: neighborhood social class measured by median income and value of homes, ozone, air toxics, race (African- American), ethnicity (Latino), indoor hazards based on proportions of rented households and age of housing	Indicator: Asthma hospitalizations Measuring: Socio-demographic, indoor hazard, and air quality factors that contribute to disparities in asthma hospitalizations	Multivariate regression	Asthma is negatively associated with neighborhood social class and positively associated with ozone, toxic air releases, the proportion of racial minorities, and indoor hazards. Ozone most strongly predicted asthma hospitalization
Grineski and Collins, 2008	Mexico Unit: Neighborhoods defined by Areas Geoestadisticas Basicas	Indicator: Industrial assembly plants or maquiladoras Measuring: Inequities in spatial relationships between residential socio-demographics and density of maquiladoras	density for each	more likely to locate in neighborhoods characterized by lower social class, and higher proportions of children and formal housing.
Higgs and Langford, 2009	Extent: Wales, United Kingdom Unit: Lower Super Output Areas (roughly equivalent to U.S. Census block groups) Independent Variables: Welsh Index of Multiple Deprivation 2005, comprised of:	population estimation to		Increasing buffer size diminished differences in deprivation profiles; deprived populations do not live in very close proximity, but at moderate (~1-4km) distances from landfill sites ("halo" effect). HRP is the preferable method in the UK

	· •		Г	1000
	income, employment, health, education, housing, access to services, and environment			context, and DBC may be preferable outside the UK because it's less dependent on a specific data source.
Marko, and Sexton, 2008 Maantay, 2001	(Houston, Texas) Unit: Census Tracts Independent Variables: SES, employment status, education, health risk, access to health care, crowding, household assets, race/ethnicity Extent: New York, New York	measuring: Cumulative cancer risk from airborne toxics based on the sum of all five emissions categories from 91 carcinogenic HAPs listed in NATA-1999 HAPEM5 Indicator: Industrial land-use zoning Measuring: Rezoning in industrial areas over time		Disproportionate industrial zoning based on race and SES. Industrial zoning increased in areas with higher than average minority populations,
Mantaay, Maroko, 2008/2009	Extent: New York, New York	Indicator: Flood zones Measuring: Vulnerability to	Population: buffer centroid containment and dasymetric disaggregation techniques	No disproportionate city-wide risk; higher risk at the borough
	<i>Unit</i> : Cadastral units, block groups	100-year floodsDifferences in disparities based on modeling	(CEDS, areal weighting)	level for Black residents in Manhattan, the Bronx, and Queens,

	x 1 1 .xx + 11	I ·	I	1.0 33711
	Independent Variables: race/ethnicity	data	statistics comparing relative likelihood versus city- and borough-wide expectations	and for White residents in Manhattan and Brooklyn; people of color are undercounted using areal weighting and centroid-containment versus CEDS
	and Suffolk County, New York. Unit: Census tract Independent Variables: Race, ethnicity, income, education, age, housing, employment.	Indicator: Accidental releases of hazardous materials Measuring: Whether these hazardous material accidents disproportionately affected disadvantaged neighborhoods.	Population: Plume modeling to generate circular buffers or impact zones for worst-case accidents Disproportion: Comparison of areas inside and outside impact zones based on difference of means tests and stepwise discriminant analysis.	
2009	York <i>Unit</i> : Census block	Indicator: Access to Parks Measuring: Access to parks as park acreage density and physical activity site density	Population: Adaptive kernel density estimation Disproportion: linear and spatial regression (OLS and GWR); verification using Monte Carlo simulation to test for variability	Indeterminate findings; statistical results showed no discernible consistent associations, but unpatterned inequality may exist
Leitner, and Sheppard 1997	Extent: Twin Cities	Indicator: Hazardous air pollutants Measuring: Potential risk based on proximity to and exposure quantities from TRI sites at different scales	Population: Spatial coincidence Disproportion: Descriptive statistics comparing aggregation units in terms of independent variables; qualitative inventory of environmental hazards incorporating "local knowledge"	Disproportionate risk based on race, poverty, and concentrated poverty; relative toxicity highest in areas with highest concentrated poverty; assessment at neighborhood scale reveals specific issues not captured by larger- scale studies
Mennis and Jordan, 2005	Extent: New Jersey Unit: Census tract Independent Variables: Race, class, employment, urban	Indicator: All TRI facilities and a subset of facilities releasing persistent bioaccumulative toxins (PBT) Measuring: Inequities in the distribution of TRI and PBT	Population: Spatial coincidence by census tract Disproportion: Multivariate OLS regression, followed by Geographically Weighted Regression	explanatory factors

		facility density; spatial variation		
	use	in statistical associations	spatial distribution of model	
		between facility density and	parameters and fit	areas in the state
Minobolli at	Extent: 226 Public	explanatory variables Indicator: Hazardous air	Duranianita Cabaalal	Diamanantianata
Mirabelli et al., 2006	schools in North Carolina	pollutants Measuring: Risk for exposure to	Proximity: Schools' reported odor levels as indicator of impact	Disproportionate exposure due to race and SES; odor better predicted by SES than
	sites Independent Variables: SES status as free or reduced lunch; race	swine-related airborne toxins	Disproportion: Prevalence ratios [logistic regression?]	race; high SES least likely to be within 3 mi of swine CAFO or to have reported odor; low-white/low-SES most likely to be within 3mi of swine CAFO; mean odor rating declined across tertiles of percent white and SES
Mohai et al., 2009	Unit: Individual level survey data from Americans Changing Lives Study (ACLS) Independent Variables: race, ethnicity, income, education, age, gender, metropolitan status, region of residence	address geocoding, with respect to TRI facility locations.	(1 mi radius) around TRI facilities Disproportion: Multivariate logistic regression, using presence of a TRI site within a mile as a dependent variable	Racial disparities more pronounced in urban areas of the Midwest and West, and in suburban areas of the South.
Mohai and Saha, 2007	Unit: ZIP codes, census tracts, census block groups Independent Variables: race/ethnicity, SES, employment status,	Measuring: Whether distance-	coincidence, buffer containment, and areal apportionment Disproportion: Inferential statistics (Student's t-test) between host and non-host	Disproportionate burdens very significant for all variables using 50% areal containment and areal apportionment; unit-based coincidence produced little differences for most variables between host and non-host tracts, however still statistically significant (except percent African American and SES); better consistency between methods and across unit sizes using areal containment and particularly with apportionment method.
Morello- Frosch and Jesdale, 2006	Extent: 309 metropolitan areas in the continental United States	pollutants	Population: NATA data mapped to census tracts Disproportion: Population	Disproportionate risk based on race. Increased racial segregation predicts

	Unit: Census Tracts	risk based on NATA outdoor air	risk index; calculation of	increased cancer risks
		toxics estimates	7	for all racial groups
	Independent Variables:		on Poisson regression,	combined, strongest for
	racial segregation		controlled for region,	Hispanics, somewhat
			population density, and	weaker for whites,
			tract-level SES	African Americans, and
				Asians; strong gradient
				observed for mobile
				and area emission
				sources and
				nonsignificant effects
				for point sources
Morello-	Extent: Southern	Indicator: Hazardous air		Disproportionate risk
Frosch et al.,	California (South Coast	pollutants		based on race, SES, and
2001	Air Basin)			land use. Race/ethnicity
		Measuring: Differences in		had positive and highly
	Unit: Census Tracts	lifetime cancer risk associated		significant association
		with air toxics exposures using	7	with cancer risk;
		CEP data, based on racial and	regression analysis	lifetime cancer risk
	Population density,	economic differences,		negatively associated
		controlling for other variables		with homeownership
	use/zoning	that may also explain exposure		and positively with
	use/zonnig	mat may also explain exposure		housing value; income
				has curvilinear
				relationship to cancer
				risk (risk decreases for
				`
				lowest income levels);
				land use is highly
3.6	T G. T			predictive of cancer risk
Most et al.,	Extent: St. Louis	Indicator: Aircraft noise		Higher percentage of
2004	County, St. Charles	impacts around St. Louis-		protected populations
	•	Lambert Field Airport	used to determine	residing within areas
	City, Missouri.			exposed to the highest
		U 1	noise level contours	levels of airport noise.
	Unit: Block group	spatial distribution of	estimated by FAA's	
		excessively high levels of	Integrated Noise Model	
		airport noise in two time-		
	` •	periods	Disproportion: Descriptive	
	low-income)		statistics to compare	
	populations.		percentages for protected	
			and non-protected groups	
N.T.	Г С	The state of the s	D 1 4 E' 1100	TDI C '11'-1
Newmann et	Extent: Oregon	Indicator: TRI facilities and	_	TRI facilities
al., 1998		emissions	circular buffers (equal areas	
	Unit: Census block		between circles)	located in lower income
		Measuring: Inequities based on		and minority
		a media-specific chronic		neighborhoods. No
	Race, ethnicity, and	toxicity index developed to rank	statistical analysis	relationship between
	household income	TRI chemical releases		hazard ranking of TRI
				facilities and the
				socioeconomic
				characteristics of host
				neighborhoods
Norton, 2007	Extent: North Carolina	<i>Indicator</i> : Solid-waste facilities	Population: spatial	Disproportionate
				burdens based on race and SES; prevalence of

	Groups	(1990 - 2003) of the locations of		new facilities where
		existing and newly permitted	Disproportion:	there previously had
	Independent Variables:	solid waste facilities	Prevalence odds ratios	been none was
	SES, race, population		using logistic regression	disproportionate based
	density and rurality,		analysis with multiple	on race for private but
	region within state		indicator variables	not public facilities; no
				disproportionate
				prevalence of newly
				permitted facilities in
				blocks containing
				existing facilities based
				on race
Pastor et al.,	Extent: California	Indicator: TRI facilities with air	Population: Spatial	A pattern of
2004		releases	coincidence by tract	disproportionate
	Unit: Census tract			exposure based on race,
		Measuring: Locational	Disproportion: Multivariate	with the highest
	Independent Variables:	inequities in the distribution of	regression analysis,	disparity for Hispanics,
	Race, ethnicity, home	all TRI sites with air releases	ordered and multinomial	after adjusting for
	ownership, population	and TRI sites reporting releases	logit regressions	varying levels of
	density, income,	of specific categories of toxic		pollution risk
	employment	chemicals		
Pastor et al.,	Extent: California	Indicator: Hazardous air	Population: NATA data	A pattern of
2005		pollutants	mapped to census tracts	disproportionate
	Unit: Census tract			exposure by race that
		Č .		persists even after
	Independent Variables:	cumulative cancer risk based on	statistics, multivariate OLS	controlling for other
	Race, ethnicity, income,	NATA data from inhalation	regression, and spatial error	
	home ownership, land	exposure to air pollutants from	regression to account for	as well as spatial
	use, population density,	mobile and stationary sources	spatial autocorrelation in	factors
	employment		the data	
Perlin et al.,	Extent: United States	Indicator: Hazardous air	Population: Spatial	Disproportionate
1995	** • •	pollutants	coincidence	burdens based on race.
	Unit: County	. 5:00		On average, annual
			Disproportion: Exposure	household income is
	Independent Variables:	exposure to airborne chemical	index using Population	higher in counties with
	race/ethnicity and	releases from industrial	Emission Index (PEI) based	higher TRI air releases
	socioeconomic status	operations in relation to socio-	on total pounds emissions	
		economic status and	divided by population in	
		race/ethnicity	county. PEI for particular	
			demographic group is	
			compared to PEI for	
			reference group of total white population in U.S.	
Perlin et al.,	Extent: Kanawha	Indicator: TRI facilities		Results from all study
1999	Valley, West Virginia;		circular buffers around TRI	
	Baton Rouge–New		facilities	African Americans and
	Orleans corridor,	characteristics of the population		those below the poverty
	Louisiana; and the	residing near TRI facilities in	Disproportion: Comparison	
	greater Baltimore	the three study areas	of proportions for relevant	live closer to the
	branci Daniiiore	and the study areas	sub-groups; cumulative	nearest TRI facility and
	metropolitan area		Day Eroups, Cumulant 10	promote rite invitity alla
	metropolitan area, Marvland			
1	metropolitan area, Maryland			also within 2 miles of
	Maryland		distribution function based	
			distribution function based	also within 2 miles of
	Maryland		distribution function based	also within 2 miles of

	status			
Pollak and Vittas, 1995	Extent: Florida Unit: Census block group Independent Variables: Race/ethnicity, income	Indicator: Hazardous air pollutants Measuring: Evenness in distribution of potential exposure to TRI pollutants	Population: Proximity analysis using natural log of distance to hazardous facility Disproportion: Regression analysis controlling for urbanization, population density, manufacturing employment and housing values	strongly found for African-American households. Although occupational and housing patterns account for much variation in proximity to TRI sites, both low- income and white groups exhibit average proximity in comparison to the rest of the population
Sheppard et al., 1999	Extent: Minneapolis, MN Unit: Census Block Groups Independent Variables: race, SES	Indicator: Hazardous air pollutants Measuring: Differences in proximity to TRI sites using different methods; validity of significance testing using randomization	Population: Spatial coincidence and areal apportionment using multiple buffer sizes Disproportion: proximity ratios (demographic % proximate to % not proximate) to measure relative differences; Monte Carlo simulation to test for significance	Indeterminate burdens based on race, disproportionate burdens based on SES. Strong association found for white and total populations below the poverty level, lower association for non-white below the poverty level; relationships were similar for spatial coincidence and small buffers, but larger buffers had stronger association; Monte Carlo simulation supports findings and adds a level of "significance" to the results
Sicotte and Swanson, 2007	Extent: Nine-county Philadelphia, PA MSA Unit: Census Block Groups Independent Variables: race, "most disadvantaged" (SES, education, unemployment), working class, people employed in manufacturing	Indicator: Hazardous air pollutants Measuring: Discrepancies in residential proximity to the most hazardous RESI facilities based on inhalation cumulative chronic health risk	Population: Buffer intersection with 1-km buffers around each facility Disproportion: Stepwise multiple regression using spatially weighted independent variables for each county individually and MSA as a whole; controlled for autocorrelation using rook contingency	Disparate impact based on race in entire MSA

				correlated with those employed in manufacturing
U.S. General Accounting Office, 1995	Unit: Block groups Independent Variables: Race and income.	Indicator: Nonhazardous municipal landfills. Measuring: Overrepresentation of minority and low-income populations in areas proximate to municipal landfills.	Population: Circular buffers of various radii and areal apportionment Disproportion: Difference of means test to compare minority and low-income proportions within buffer to the remainder of the host county	Minorities or low- income people not overrepresented near a majority of landfills in U.S. The proportion of minorities or low- income people living within 1 mile buffer was higher than the rest of the county in less than 50% of all landfills.
United Church of Christ, 1987	Unit: ZIP codes Independent Variables: Race, income, housing value	Indicator: Commercial hazardous waste management facilities. Measuring: Characteristics of population in four mutually exclusive groups of ZIP codes, (with/without facilities, with/without one of the five largest waste facilities).		Race was the most significant factor for facility location. Minority percentage in ZIP codes with facilities, on average, was twice as high as other ZIP codes.
of Christ,	Unit: Census tracts. Independent Variables: Race, ethnicity, income, education, housing	Indicator: Commercial hazardous waste management facilities. Measuring: Characteristics of the population with 3 km. of each facility, for comparison with areas lying beyond 3 km.	Population: Circular buffer (3 km. radius), with areal apportionment method Disproportion: Bivariate statistical comparsion tests and multivariate logistic regression	Percentages of Blacks, Hispanics and Asians in host areas are 1.7, 2.3 and 1.8 times greater than non-host areas. Race continues to be the most significant predictor of waste facility location, after accounting for other factors.

<u>Table 2. Methodology for Spatial Definition of Proximity and Potential Exposure to Environmental Hazards</u>

Approach	Risk Indicator	Examples: Author and Year of Study
Spatial Coincidence Analysis	Presence of a hazard (unit-hazard coincidence)	United Church of Christ 1987; Burke 1993; Hird 1993; Anderton et al. 1994; Goldman and Fitton 1994; Been 1995; Been and Gupta 1996; Cutter et al. 1996; Boer et al. 1997; Daniels and Friedman 1999; Fricker and Hengartner 2001; Boone 2002; Taquino et al. 2002; Walker et al. 2006; Baden et al. 2007.
	Total number or density of hazards	Burke 1993; Cutter and Solecki 1996; Ringquist 1997; Tiefenbacher and Hagelman 1999; Fricker and Hengartner 2001; Mennis and Jordan 2005.
	Total quantity of emitted pollutants	Bowen et al. 1995; Krisel et al. 1996; Boer et al. 1997; Tiefenbacher and Hagelman 1999; Daniels and Friedman 1999; Bolin et al. 2000.
	Toxicity-weighted quantity of pollutants	Bowen et al. 1995; Perlin et al. 1995; McMaster et al. 1997; Brooks and Sethi 1997; Bolin et al. 2000.
Distance- Based Analysis	Discrete distance from hazards (fixed buffer)	Glickman 1994; Zimmerman 1994; U.S. GAO 1995; Glickman and Hersh 1995; Chakraborty and Armstrong 1997; Neumann et al. 1998; Perlin et al. 1999; Sheppard et al. 1999; Bolin et al. 2000, 2002; Altas 2002; Baden and Coursey 2002; Boone 2002; Pastor et al. 2004; Mohai and Saha 2006, 2007; Walker et al. 2006; United Church of Christ 2007; Kearney and Kiros 2009; Mohai et al. 2009.
	Continuous distance from hazards	Pollock and Vittas 1995; Gragg et al. 1995; Stretesky and Lynch 1999; Cutter et al. 2001; Margai 2001; Mennis 2002; Waller et al. 1997; 1999; Zandbergen and Chakraborty 2006; Downey 2006; Chakraborty and Zandbergen 2007.
Pollution Plume Modeling	Geographic plume analysis	Glickman 1994; Glickman and Hersh 1995; Chakraborty and Armstrong 1997, 2001; Chakraborty et al. 1999; Chakraborty 2001; Margai 2001; Dolinoy and Miranda 2004; Most et al. 2004; Fisher et al. 2006; Bevc et al. 2007; Maantay 2007.
	Plume-based health risk estimate	Morello-Frosch et al. 2001; Bouwes et al. 2001; Asch and Fetter 2004; Apelberg et al. 2005; Pastor et al. 2005; Morello-Frosch and Jesdale 2006; Sicotte and Swanson 2007; Gilbert and Chakraborty 2008; Linder et al. 2008; Chakraborty 2009.

<u>Table 3. Studies of Residential Proximity to Environmental Hazards and Adverse</u>
<u>Pregnancy Outcomes with Reported Disparities by Race/Ethnicity or Socioeconomic Status</u>

Reference &	Population	Pregnancy	Disparities	Environmental
Year		Outcomes	Examined	Hazard & Disparities
Bentov et al.,	Beer-Sheva	Major	Jewish	Residential proximity
2006	subdistrict in	congenital	populations	to a regional
	Israel, 1995-2000	malformations	(urban, urban	industrial park was
		combined and	satellite, and	associated with
		subcategorized	agricultural	increased rates of
		into major	localities);	major congenital
		congenital	Bedouin	anomalies among the
		anomalies of	population	Bedouin population
		central nervous	(permanent	but not with the
		system,	localities and	Jewish population
		chromosomal	traditional tribal	
		anomalies and	settlements)	
		other major		
		congenital		
		malformations		
Brender et al.,	Texas (USA) live	Chromosomal	Race/ethnicity	Hispanic women who
2008	births and fetal	anomalies	(non-Hispanic	lived near hazardous
	deaths, 1996-	combined and	white, Hispanic,	waste sites 7.9 times
	2000	categorized	African-	more likely (95 % CI
		into nine	American, other)	1.1, 42.4) to have
		categories		offspring with
				Klinefelter variants
Genereux et	All live singleton	Preterm birth,	Maternal	Proximity to
al., 2007	births in	low birth	education (< 11	highways associated
	Montreal,	weight, and	years, 11 years,	with OR of 1.58 for
	Canada, 1997-	small-for-	12-13 years, >13	preterm birth, OR of
	2001	gestational age	years); census	1.81 for low birth
		(SGA)	tracts ranked into	weight births, and OR
			quintiles	of 1.32 for SGA
			according to	births among women
			neighborhood	living in the most
			poverty level	wealthy
				neighborhoods, but
				was not associated
				with these outcomes
				in less wealthy/poor
				areas; this residential
				characteristic was
				associated with
				preterm birth and low

Orr et al., 2002	California live births and fetal deaths, 1983- 1988	All congenital malformations combined and subcategorized into nine defects/defect groups	Race/ethnicity (Hispanic/Latino, black/African American, American Indian/Alaska Native, Asian/Pacific Islander)	birth weight births in the most highly educated women but not with the less educated Although the numbers of exposed cases and controls small, strongest association noted among American Indians/Alaska Natives between a maternal residence in a census tract with one or more National Priority List hazardous waste sites and birth defects
Sarov et al., 2008	Beersheba subdistrict, Israel, 1995-2000	Perinatal mortality (fetal deaths, intrapartum death, and postpartum death within 28 days after delivery)	Stratified by ethnicity (Jews and Bedouins) and by type of locality	Residential proximity to an industrial park was associated with increased rates of perinatal mortality among Bedouin births but not among Jewish births
Suarez et al., 2007	Texas live births and fetal deaths, 1996-2000	Neural tube defects	Ethnicity (non- Hispanic white, Hispanic)	Maternal residential proximity (within 1 mile) to one or more TRI industrial facilities associated with neural tube defects in offspring of white, non-Hispanic mothers (OR 1.8, 95% CI 1.1, 2.8) but not with births to Hispanic mothers (OR 1.1, 95% CI 0.8, 1.4)

CI = confidence interval; OR = odds ratio; TRI = Toxic Release Inventory

<u>Table 4. Studies of Residential Proximity to Potential Environmental Hazards and Adverse Pregnancy Outcomes and Childhood Cancer</u>

					Health outcome
Reference,	Study design,	Health outcomes	Exposure	Findings	associated with
Year,	Regional	included	description	Findings	proximity &
-	_	meraded	description		limitations
Country	description	· · · · · · ·	4 177 1 1 4 1	D 0	
4a.	Residential Prox		ntal Hazards and Ad	iverse Pregnancy Oi	itcomes
D 1	Б 1 :		tal malformations	D: 1 C : 1	D 11 /11
Bentov et al.,	Ecologic	Major congenital	Distance of	Risk of congenital	Residential
2006	study of 1995-	malformations	localities from	malformations	proximity to
Israel	2000 live	combined and	regional industrial	among Bedouin	industrial park
	births and	subcategorized	park and	populations higher	associated with
	stillbirths in	into anomalies of	predominant wind	in proximal than	increased rates of
	Beer-Sheva	central nervous	direction (17	distal localities	major congenital
	subdistrict in	system, anomalies	chemical plants	(RR 1.6, 95% CI	malformations
	Israel divided	associated with	and one industrial	1.4, 1.8)	among Bedouin
	into Jewish	chromosomal	toxic waste site)	especially risk of	populations;
	and Bedouin	anomalies, and		central nervous	limitations:
	localities	other major		system defects	potential for
		congenital		(RR 2.3, 95% CI	ecologic fallacy
		malformations		1.4, 3.6);	and residual
				congenital	confounding,
				malformations not	study did not
				associated with	include
				residential	information
				proximity to	about
				industrial park	pregnancies
				among births in	terminated
				Jewish localities	before 22 nd week
Boyle et al.,	Population-	Births with	Municipal landfill	In both area-level	Living near a
2004	based cohort	congenital	sites within 3 km	analyses and the	municipal
Great Britain	and case-	anomalies (all	(and other	case-control	landfill site was
	control	combined)	distances) of	study, congenital	not found a risk
	studies;	detected by the	district electoral	anomalies were	factor for
	Eastern	regional	divisions; distance	not found to occur	congenital
	Region of	congenital	of case and control	more commonly	malformations;
	Ireland births,	anomalies registry	addresses from	in proximity to	limitations:
	1986 - 1990		landfill sites	municipal	potential for
				landfills	residual
					confounding and
					addresses at
					registration used
					that did not
					account for
					residential
					mobility during
					pregnancy
Brender et	Population-	Live births and	Residence at	Neither residence	Findings

al., 2006a USA	based case- control study of live births and fetal deaths in Texas, 1996 - 2000	fetal deaths with cleft palate without cleft lip; cleft lip without or with cleft palate; isolated oral cleft (without any other major defect besides oral clefts)	delivery within 1 mile of NPL or state hazardous waste site and/or within 1 mile of industries with reported air emissions of chemicals; limited sample also available from the Texas portion of National Birth Defects Prevention Study (NBDPS) for residence during the periconceptional period (3 months before to 3 months after conception)	at delivery or during the periconceptional period associated with oral clefts if the mother lived within 1 mile of waste sites; among women < 35 years, no association between residence within 1 mile of industrial facilities and oral clefts in offspring; among women 35+ years, oral clefts in offspring associated with residence within 1 mile of industrial facilities (OR 2.4, 95% CI 1.3, 4.2) especially smelters (OR 15, 95% CI 2.8, 151)	suggested that maternal residential proximity to industries might be associated with oral clefts in births to older mothers; limitations: most analyses based on residence at delivery which did not account for residential mobility during pregnancy, potential for residual confounding and exposure misclassification
Brender et al., 2008a USA	Population- based case- control study of live births and fetal deaths in Texas, 1996 - 2000	Live births and fetal deaths with chromosomal anomalies (combined) and categorized into nine categories based on BPA codes	Residence at delivery within 1 mile of industries with reported air emissions of chemicals or residence at delivery within 1 mile of state or NPL hazardous waste site	Autosomal deletions in offspring (OR 1.5, 95% CI 1.0, 2.3) and Klinefelter syndrome (male births only, OR 2.9, 95% CI 1.1, 7.3) associated with maternal residence within 1 mile of industrial facility; among older women (35+ years), chromosomal anomalies in offspring associated with living near facilities with heavy metal or solvent emissions; maternal	Findings suggested some relation between residential proximity to industries with emissions of solvents or heavy metals and chromosomal anomalies in births to older mothers; limitations: exposure classification based on address at delivery, pregnancy terminations not included, potential for residual confounding

	1	1	T	T	
Cordier et al., 2004 France	Ecologic study of prevalence of birth defects in communities surrounding incinerators in southeast France, 1988- 1997	Malformations among livebirths, stillbirths, and medical terminations divided into minor, chromosomal, monogenic, and other major anomalies; other major anomalies subdivided into 23 different subgroups	Dioxin concentration estimates in 194 communities with municipal waste incinerators; residence in these communities	residence near waste sites not associated with chromosomal anomalies in offspring except Klinefelter variants among Hispanic births (OR 7.9, 95% CI 1.1, 42.4) RR for other major anomalies was 1.1 (95% CI 0.98, 1.2) with living in exposed communities relative to unexposed communities; some association seen for facial clefts (RR 1.3, 95% CI 1.1, 1.6) and renal dysplasia (RR 1.6, 95% CI 1.1, 2.2); in exposed communities, dose-response trend of risk for obstructive uropathies seen with increasing exposure	Rate of congenital malformations not significantly higher in exposed groups except for facial clefts and renal dysplasia; limitations: potential for ecologic fallacy, exposure misclassification, potential for residual confounding and ascertainment bias
Cresswell et al., 2003 United Kingdom	Ecologic study of prevalence of birth defects among livebirths in city of New Castle upon Tyne, United Kingdom, 1985-1999	Malformations among all livebirths, stillbirths, induced abortions, and fetal deaths after 14 weeks gestation categorized into chromosomal and non-chromosomal defects	Residence within 3 km of Byker waste combustion plant	Relative to living 3-7 km from plant, RR 1.11 (95% CI 0.96-1.3) for living within 3 km after site began operations; RR higher for non-chromosomal than for chromosomal defects among offspring of women living near waste combustion plant	Little evidence of relation between prevalence of congenital malformations and residence near waste combustion plant; limitations: exposure misclassification, potential residual confounding by maternal age

Croen et al.,	Population-	Neural tube,	764 hazardous	Little or no	Overall, results
1997	based, case-	conotruncal heart,	waste sites	increased risk	did not suggest
USA	control study	and oral cleft	classified with	noted for maternal	increased risks
CST	California live	defects	respect to human	residence in	for these defects
	births, fetal	derects	exposure potential,	census tract	for a maternal
	deaths, and		contaminated	containing one or	residence in a
	terminations,		environmental	more waste sites;	census tract with
	1989 - 1991		media, and	some association	one or more
	1909 1991		chemical	seen between a	waste sites, but
			contaminants	maternal	some association
			present; maternal	residence within	was seen
			exposure defined	½ mile of a	between a
			as residence in	National Priority	maternal
			census tract and	List site and	residence within
			within 1 mile or	neural tube	1/4 mile of an
			less of one or more	defects (OR 2.1)	NPL site and risk
			sites during	and heart defects	for NTD and
			periconceptional	(OR 4.2), but the	conotruncal heart
			period	95% CIs were	defects in
			period	compatible with	offspring;
				the null. Positive	limitations:
				associations were	potential for
				noted between a	selection bias
				maternal	due to
				residence within 1	differential
				mile of sites with	participation
				some heavy	between cases
				metals, polycyclic	and controls,
				hydrocarbons, and	potential for
				solvents and	recall bias, and
				neural tube	potential for
				defects in	exposure
				offspring.	misclassification
Czeizel et al.,	Retrospective	Categorized into	Three concentric	Decrease in risk	Overall, found
1999	cohort –	32 isolated and 5	bands around the	of undescended	little evidence to
	cluster	multiple	acrylonitrile	testis with	support an
Hungary	analysis of	congenital	factory	increasing	association
	live births,	anomaly groups	(Nyergesujfalu	distance from the	between living
	stillbirths, and	anomary groups	with factory,	acrylonitrile	near acrylonitrile
	terminations		within 5 km from	factory	factory and
	in surrounding		epicenter, and 5	ractory	congenital
	region of		and 25 km from		malformations in
	acrylonitrile		factory)		offspring with
	factory in		ractory)		the exception of
	Nyergesujfalu,				undescended
	Hungary				testis;
	Trungary				limitations:
					exposure misclassification,
					· .
					potential for residual
					restauat

					confounding
Dolk et al., 1998 Europe	Population-based case-control study Belgium, Denmark, France, Italy, UK Live births, fetal deaths, and pregnancy terminations	Non-chromosomal congenital anomalies	21 landfill sites with hazardous waste zone within 3 km radius of site was defined as "proximate zone"	Significant associations noted for residence within 3 km of site and neural tube defects (OR 1.9, 95% CI 1.2, 2.8), cardiac septal defects (OR 1.5, 95% CI 1.1, 3.2), and anomalies of the great arteries and veins (OR 1.8, 95% 1.0, 3.2). Elevated odds ratios were also found for tracheoesophageal anomalies, hypospadias, and gastroschisis, although estimates were consistent with the null.	Results indicated small, excess risk of non-chromosomal defects in offspring among women who lived near hazardous waste sites; limitations: addresses not determined for the periconceptional period, potential for residual confounding
Eizaguirre- Garcia et al., 2000 United Kingdom	Population- based descriptive geographical study of birth defect cases and births during 1982 – 1989 in Glasgow and nearby areas	Congenital anomalies combined into one group	Residence in a circle within 10 km in radius around former site of factory and area contaminated by chromium; areas divided into 2 km area containing site and 8 concentric rings around it, each 1 km wide	Relative risk highest in an area 2-4 km away from pollutant (RR 1.5, 95% CI 1.2, 1.8); referent category 0-2 km from center of polluted area	Investigators concluded that results did not point to possible teratogenic effect of chromium waste; limitations: used residence at birth, congenital malformations combined together, potential for residual confounding by maternal age
Elliott et al., 2009 Great Britain	Ecologic study of births in England, 1983-1998	Congenital malformations included hypospadias and epispadias, cardiovascular defects, neural tube defects, and	Divided England into a grid of 5x5 km squares in which births in each square were classified in terms of proximity (< 2 km, 2+ km) to a	Noted slightly positive associations for all congenital anomalies combined (OR 1.08) and cardiovascular	Weak associations noted between risk of all anomalies combined and selected anomalies and

		abdominal wall defects; also combined all defects	landfill site 1 year previously; landfill exposure index developed with four categories of intensity	defects (OR 1.16) with landfill exposure index in third quartile for special waste sites and for hypospadias and epispadias for third and top categories (all ORs < 1.25)	geographic density of special wastes sites at the level of 5x5 grid squares; limitations: potential for ascertainment bias, exposure misclassification, and residual confounding; residential mobility not taken into account
Geschwind et al., 1992 USA	Population- based, case- control study; New York State live births, 1983 – 1984	All birth defects combined; malformations grouped into seven general categories	Exposure risk index that incorporated distance from and the hazard ranking score for each hazardous waste site within 1-mile radius of birth residence	Maternal proximity to waste sites slightly associated with congenital malformations in offspring (OR 1.1, 95% CI 1.1, 1.2) and high exposure risk was more strongly associated with these defects (OR 1.6, 95% CI 1.3, 2.0), especially for defects of the musculoskeletal and integument systems; proximity to sites with pesticides associated with defects of musculoskeletal system (OR 1.2, 95% CI 1.1, 1.4); proximity to sites with plastics associated with chromosomal anomalies (OR 1.5, 95% CI 1.0, 2.1). Modest effect for central nervous system	Results suggested small, statistically significant additional risk for birth defects with maternal residential proximity to toxic waste sites; limitations: birth defects ascertained among live births only; maternal addresses based on residence at delivery, potential exposure misclassification and residual confounding

				defects in offspring with maternal residence within 1 mile (OR 1.3, 95% CI 1.1-1.6) as well as high exposure risk category (OR 1.5, 0.5% CI 0.7.3.2)	
Jarup et al., 2007, Great Britain	Ecologic study of births in England and Wales 1989-1998	Down syndrome	Maternal addresses linked by year (2-year lag) and postcode to landfill data; exposure defined as an address within 2-km zone of a landfill site	No excess risk of Down syndrome noted in populations living within 2 km of a landfill site, regardless of site type	No association found between a residence within 2 km of landfill site and Down syndrome in offspring; limitations: potential exposure misclassification, ecologic fallacy, and residual confounding; residential mobility not taken into account
Kloppenborg et al., 2005 Denmark	Population- based cohort of live births in Denmark, 1997 - 2001	Congenital anomalies combined and sub-grouped as defects of the nervous or cardiovascular systems in live births	Distance of maternal residence from waste landfill sites in three buffer zones: 0-2, 2-4, and 4-6 km	No association noted between landfill location and congenital malformations combined or nervous system anomalies; noted small excess risk for anomalies of the cardiovascular system	Other than anomalies of the cardiovascular system, no excess risk noted between maternal residential proximity to landfills and congenital malformations; limitations: congenital anomalies restricted to live births, potential residual confounding by maternal age and other unmeasured variables,

					residential mobility during pregnancy not taken into account
Kuehn et al., 2007 USA	Population- based case- control study of live births in Washington State, 1987 - 2001	Cases identified from linked birth-hospital discharge records and categorized into 11 groups based on system and type	Distance of maternal residence at delivery from hazardous waste sites; proximity defined as various distances up to 5 miles; waste sites categorized as high or low priority based on types of contaminants present and media contaminated	Relative to living > 5 miles from a site, living within 2 and 5 miles (OR 1.2), 1 and 2 miles (OR 1.3), 0.5 to 1 mile (1.3), and less than 0.5 miles (1.3) were significantly associated with increased risk of any congenital malformations in offspring; most associations with specific effect groups were modest except for birth defects involving skin if the mother lived within 1 mile of a site (OR 2.4, 95% CI 2.2, 2.7); associations for malformations stronger with sites in urban areas than in rural areas	Results suggested an increased risk of congenital malformations among offspring of women living in close proximity of hazardous waste sites; limitations: congenital malformations restricted to live births, exposure based on maternal address at delivery only; potential for exposure misclassification and residual confounding
Langlois et al., 2009, USA	Population- based case- control study of Texas live births and fetal deaths, 1996-2000	Conotruncal heart defects with and without chromosomal anomalies and truncus arteriosus, transposition of the great vessels, and tetralogy of Fallot separately	Residential proximity (maternal address at delivery within 1 mile) to hazardous waste sites and industrial facilities with reported air emissions of chemicals	Only truncus arteriosus associated with a maternal residence within 1 mile of any waste site (crude OR 2.80, 95% CI 1.19, 6.54) and with NPL sites (adjusted OR 4.99, 95% CI 1.26, 14.51)	In this population, residential proximity to waste sites or industrial facilities not associated with conotruncal heart defects with the exception of truncus arteriosus; limitations: potential for exposure

Malik et al., 2004 USA	Population- based case- control study Dallas County, Texas live births 1979 - 1984	Live births diagnosed with congenital heart disease at any age	Mothers' residence at delivery within 1/4 and 1 mile of hazardous waste site	Maternal residence within one mile of hazardous wastes site was slightly associated with congenital heart disease in offspring (OR 1.2, 95% CI 1.1, 1.4)	misclassification, ascertainment bias, and residual confounding; use of maternal address at delivery to assign exposure Results of study suggested small, but statistically significant, additional risk for congenital heart disease among offspring of women who lived near a hazardous waste site; limitations: congenital heart malformations restricted to live births, exposure based on maternal address at delivery, potential for residual confounding
Marshall et al., 1997 USA	Population- based, case- control study 18 counties in New York State, 1983 – 1986 live births	Central nervous system and musculoskeletal system defects	Proximity and related exposure index of mother's address at delivery to waste sites with solvents, metals, and pesticides; proximity (within 1 mile) of maternal residence at delivery to industrial sources of air emissions; industrial sources identified from 1988 Toxic Release Inventory (TRI); general dispersion model used for solvent emissions	Minimal or no association was noted between maternal residential proximity to waste sites (in general and those sites with solvents or metals) and central nervous system and musculoskeletal system defects; central nervous system (CNS) defects in offspring associated with maternal residence within 1	No increased risk noted between women living in areas with a medium or high probability of exposure to chemicals from hazardous waste sites and CNS and musculoskeletal birth defects in offspring; however, association seen between living in close proximity to industrial facilities with emissions of

				mile of TRI facility with air emissions of solvents (OR 1.3, 95% CI 1.0, 1.7) and metals (OR 1.4, 95% CI 1.1, 1.7).	solvents or metals and CNS defects; limitations: birth defects ascertained among live births only, maternal addresses based on residence at delivery, potential exposure misclassification (used data from 1988 TRI) and potential residual confounding
Ochoa-Acuna & Carbajo, 2009 USA	Retrospective cohort study of rural women in Indiana and respective births conceived during spring-summer months, 2000-2004	Births from Indiana Birth Records Database and birth defects divided into abdominal cavity, craniofacial, heart, limb, neural tube, other nervous system, and urogenital defects	Developed land cover metric of pasture, soybeans, and corn crops; exposure defined as living within 500 meter radius to a given crop that exceeded the median of the area planted with the crop for the entire dataset	Only limb malformations associated with exposure to cornfields (OR 1.22, 95% CI 1.01, 1.47 per additional 10 hectares planted with corn within 500 meters); no associations found between maternal residential proximity near soybean crops and birth defects	Significant association noted between increase in area planted in corn around maternal residences and risk of limb birth defects; limitations: address at delivery used to assign exposure, elective terminations not included in birth defects, possible exposure misclassification and residual confounding
Orr et al., 2002 USA	Population- based, case- control study; California (24 counties) births and fetal deaths, 1983 – 1988; focused on minority births	All birth defects combined; musculoskeletal, central nervous system, integumental, heart or circulatory, and oral cleft defects; chromosomal anomalies	Maternal address at child's birth (obtained from birth certificate) in census tract with one or more National Priority List (NPL) hazardous waste sites (n = 84 sites)	Modest effects observed for NTDs (OR 1.5, 95% CI 0.93, 2.6), anencephaly (OR 1.9, 95% CI 0.91, 3.8), and spina bifida (OR 1.3, 95% CI 0.61, 2.5) though estimates compatible with the null;	Modest association observed between a maternal residence in a census tract with one or more NPL sites and birth defects in offspring across all racial/ethnic

Palmer et al., 2005 Great Britain	Population- based cohort of live births in Wales 1983-1997	Congenital anomalies combined and sub-grouped as chromosomal anomalies, cardiovascular defects, and abdominal wall defects in live births	Expected rates of congenital anomalies in births to mothers (at time of delivery) living within 2 km of landfill sites, before and after opening of the sites, with referent group living at least 4 km away from these sites	associations also noted with trisomy 13 (OR 2.7, 95% CI 1.5, 4.6), trisomy 18 (OR 2.7, 95% CI 1.4, 5.1), and sex chromosome anomalies (OR 3.1, 95% CI 1.0, 9.6); strongest association between birth defects and maternal proximity to NPL site was among American Indians/Alaska Natives Ratio of observed to expected rates of congenital malformations before landfills opened was less (0.87) than after their opening (1.2) giving a standardized risk ratio of 1.4 (95% CI 1.1, 1.7); although risk ratios for the subcategories of malformations also elevated, the 95% confidence intervals around these estimates included 1.0 Elevated risks for	groups studied; limitations: exposure based on maternal residence at delivery, used census tracts to assign exposure, potential for residual confounding, small numbers of exposed cases and controls available for study, congenital malformations restricted to those that resulted in live births and fetal deaths 20+ weeks gestation Found increased risk of congenital anomalies after the opening of landfill sites from 1983-1997 but increase did not persist during 1998-2000; limitations: congenital anomalies restricted to live births, potential for residual confounding and exposure misclassification Some
2006, USA	based case- control study of live births, fetal deaths, and terminations	defects combined and anencephaly and spina bifida separately	geographic metric based on linkage of pesticide-use reports with land- use survey maps of crops; proximity	NTDS and anencephaly and spina bifida subtypes were associated with pesticides	associations noted between proximity to certain pesticide applications and NTD risk;

	in California, 1987-1991		defined as maternal residence within 1000 m pesticide applications	classified as amide, benzimidazole, methyl carbamate, or organophosphorus pesticides and with increasing pesticides; NTD risk also associated with benomyl and methomyl applications	potential for recall bias of residential addresses and exposure misclassification
Suarez et a 2007 USA	nl., Population-based case-control study of live births and fetal deaths in Texas, 1996 - 2000	Live births and fetal deaths with neural tube defects	Residence at delivery within 1 mile of state or NPL hazardous waste site or within 1 mile of industries with reported air emissions of chemicals	No association noted between maternal residence near waste site and neural tube defects in offspring (OR 1.0, 95% CI 0.6, 1.7); modest risk seen for neural tube defects in offspring with maternal residence at delivery within 1 mile of industrial facility (OR 1.2, 95% CI 1.0, 1.5) with a stronger association among mothers 35 years and older (OR 2.7, 95% CI 1.4, 5.0) and among non-Hispanic white mothers (OR 1.8, 95% CI 1.1, 2.8)	No excess risk noted for NTDs in offspring among women living near hazardous waste sites; however, close proximity to industrial facilities with chemical air emissions associated with NTDs in several subgroups; limitations: congenital anomalies did not include terminations, used residence at delivery to assign exposure, potential for residual confounding and exposure misclassification
Vinceti et 2001 Italy	al., Ecologic study of prevalence of birth defects during 1982- 1986, 1987- 1990, and 1991-1995 in Provinces of	All malformations combined and specific malformations divided into 18 groups	Prevalence of birth defects in Ceramic District (contaminated with lead) with the remainder of the two Provinces containing this District, but	Relative to the unpolluted areas, excess risk of cardiovascular defects observed in lead-polluted area that decreased over time as pollution	Parental residence in lead- contaminated area associated with increased risk of all malformed births combined and

	Reggio Emilio		outside of the lead-	decreased (1982-	several specific
	Reggio Emilia and Modena, northern Italy		contaminated area serving as the unexposed population	1986: RR 2.6, 95% CI 1.7, 3.8; 1987-1990: RR 1.2, 95% CI 0.62, 2.1; 1991-1995: RR 0.97, 95% CI 0.57, 1.5); also found higher risks of oral clefts and musculoskeletal defects in lead- contaminated area with decreasing risk over time	groups of defects in offspring; limitations: potential for ecologic fallacy- misclassification of exposure at the individual level, potential for residual confounding
Vinceti et al., 2009, Italy	Population- based case- control study of live births, fetal deaths, and terminations in a northern Italy community, 1998-2006	All anomalies combined, anomalies classified by system, chromosomal anomalies, oral clefts, eye anomalies	Used a dispersion model to estimate concentrations of dioxins and furans emitted from municipal solid waste incinerator and designed maps of low, intermediate, and high ground level exposure to these compounds	With adjustment for education and maternal age, OR for congenital anomalies was 1.49 (95% CI 0.70, 3.19) in the medium exposure group and 0.66 (95% CI 0.25, 1.79) in the high exposure group; chromosomal defects only specific group associated with exposure (medium) with OR 2.53 (95% CI 0.88, 7.24)	Maternal exposure to emissions from municipal solid waste incinerator not associated with excess risk of congenital anomalies in offspring (ORs consistent with unity) in this population and setting; limitations: potential for exposure misclassification and residual confounding
Vrijheid et al., 2002 Europe	Population- based case- control study Belgium, Denmark, France, Italy, UK live births, fetal deaths, and pregnancy terminations	Chromosomal congenital anomalies further classified as either Down's syndrome or non-Down's syndrome	23 landfill sites with hazardous waste; zone within 3 km radius of site was defined as "proximate zone"	With adjustment for maternal age and socioeconomic status, women who lived within 3 km of hazardous waste site more likely to have a birth with a chromosomal anomaly than women who lived 3-7 km (OR 1.4, 95% CI 1.0, 2.0)	Results indicated an increased risk of chromosomal anomalies with a maternal residence near hazardous waste landfill sites; limitations: potential misclassification of exposure, used maternal residence at birth to assign exposure

Wulff et al.,	Population-	All congenital	Persons living in	Slightly larger	No significantly
1996	based	malformations	parishes within 20	proportion of	increased risk of
Sweden	retrospective	grouped into 13	km from a copper	malformations	birth defects
	cohort study	categories; heart	smelter; parent	seen among	noted in
	of 1973-1990	defects sub-	employed at	exposed children	offspring of
	births in	categorized into	smelter	in cohort than	persons living in
	selected	17 subcategories		among reference	the vicinity of a
	parishes of			population (RR	smelter or who
	Sweden			1.2, 95% CI 0.95,	were employed
				1.4) with	at the smelter;
				chromosomal	limitations: small
				anomalies more	exposed
				common among	population (N =
				exposed group	2604), potential
				(RR 2.6, 95% CI	surveillance bias,
				0.90, 6.7).	residual
				Authors attributed	confounding, and
				chromosomal	misclassification
				association to	of environmental
				under-reporting in	and occupational
				reference area	exposures
				and/or active	
				surveillance in	
XX 1 1	D 1.1	Y	X	exposed area.	3.6
Yauck et al.,	Population-	Live births	Mother's address	Among older (>	Maternal
2004	based case-	diagnosed with	at delivery within	37 years) mothers,	residential
USA	control study;	congenital heart	1.32 miles of waste	CHD in offspring	proximity to
	Milwaukee,	defect (CHD)	sites and industrial	associated with a	waste sites and
	Wisconsin	based on	facilities with	maternal	industries with
	live births	echocardiography,	emissions of	residence within	TCE emissions associated with
	1997 – 1999	surgical findings,	trichloroethylene	1.32 miles of	CHD in
		and autopsy	(TCE)	TCE-emitting	
		reports		sites (OR 3.2,	offspring of older but not
				95% CI 1.2, 8.7); no relation found	
					younger women;
				between living near these sites	limitations: maternal address
				and CHD in	at birth used to
				offspring among	assign exposure,
				younger women	potential
				younger women	exposure
					misclassification
					and residual
					confounding,
					pregnancy
					terminations not
					included in case
					group
	L	Fetal/N	Neonatal Deaths	L	
Bell et al.,	Population-	Fetal and neonatal	Linked TRS	Largest risks for	Excess risk of
2001a	based case-	deaths within 24	(township, range,	fetal death due to	fetal death due to
	•				

USA	control study of 1984 fetal deaths and live birth controls in ten California counties	hours of birth due to congenital anomalies	section) data from the state Pesticide Use Report database to maternal addresses; exposure defined as maternal residence within a TRS and/or within any of the surrounding 8 TRSs; daily exposure estimated for each woman's pregnancy	congenital anomalies were from pesticide exposure during the 3 rd – 8 th week of pregnancy especially to halogenated hydrocarbon pesticides (OR 2.2, 95% CI 1.3, 3.9); odds ratios for all pesticide classes increased when exposure occurred within same square mile as residence and with exposure to multiple pesticide classes	congenital anomalies noted with potential environmental exposure to pesticides during the 3 rd to 8 th week of pregnancy; limitations: maternal address at delivery used to assign exposure, potential exposure misclassification and residual confounding
Bell et al., 2001b USA	Population- based case- cohort study of 1984 fetal deaths and random sample of live births in ten California counties	Fetal and neonatal deaths within 24 hours of birth due to causes other than congenital anomalies, multiple births, umbilical cord compression, and factors not likely to be influenced by environmental exposures	Linked TRS (township, range, section) data from the state Pesticide Use Report database to maternal addresses; exposure defined as maternal residence within a TRS and/or within any of the surrounding 8 TRSs; daily exposure estimated for each woman's pregnancy	No strong associations noted between residential proximity to pesticide applications and fetal deaths not due to congenital anomalies; slightly elevated hazard ratios (1.3 – 1.4) were noted between a residence near applications of several types of pesticides and fetal deaths	Overall, minimal or no association noted between residential proximity to pesticide applications and risk of fetal death due to causes other than congenital malformations; limitations: address at delivery used to assign exposure, potential exposure misclassification and residual confounding
de Medeiros et al., 2009 Brazil	Population- based case- control study of 2000-2001 perinatal deaths and births in 14 districts located in the	Perinatal deaths (fetal and early neonatal)	Distance-weighted traffic density (DWTD) metric in the vicinities of maternal residences using a 750 feet radius around the homes; DWTD values	Adjusted odds ratios for fetal and neonatal deaths in the highest quartile of DWTD (relative to the lowest quartile) were 1.20 (95% CI 0.65, 2.24) and	Some association noted between exposure to pollutants from heavy-traffic roadways and perinatal deaths but adjusted ORs consistent with

	south region of Sao Paulo, Brazil		grouped into quartiles based on the distribution for all subjects	1.47 (95% CI 0.67, 3.19) respectively	unity and p- values for trend not significant; limitations: potential exposure misclassification and residual confounding, did not account for residential
Mueller et al., 2007 USA	Population-based case-control study, Washington State vital records, 1987-2001	Fetal deaths 20+ weeks gestation and further grouped to early (< 28 weeks gestation) and late (28+ weeks gestation) death	Measured straight- line distances in miles between the mother's residence at the time of live delivery or fetal death and the nearest hazardous waste site; hazardous waste sites were classified as high- priority or low priority; also classified sites by contaminants and contaminated media	Risk of fetal death not elevated with maternal residence • 0.5 mile relative to greater than 5 miles from hazardous waste site (adjusted OR 1.06, 95% CI 0.90, 1.25). With the exception of women residing within 1 mile of a site contaminated with pesticides (OR for fetal death 1.28, 95% CI 1.13, 1.46), no association noted between fetal deaths and a maternal residence within 5 miles of sites with contaminated air, soil, water, solvents, or metals	mobility during pregnancy With the exception of pesticide-contaminated sites, fetal death not associated with maternal residential proximity to hazardous waste sites; limitations: used maternal residence at delivery to assign exposure although did have information on length of residence, potential for exposure misclassification, underreporting of early fetal deaths, and residual confounding, higher proportion of control births than case births successfully geocoded
Sarov et al., 2008 Israel	Ecologic study of births in Beersheba subdistrict, 1995-2000	Perinatal mortality divided into three categories: fetal death before	Residential distance from industrial park that contained industries	Overall, rates of perinatal mortality did not vary by distance (< 20 km with > 20 km as	Increased risk of perinatal mortality for Bedouin but not Jewish births

		delivery, intrapartum death, and postpartum death within 28 days after delivery	(chemical, pharmacochemical, and heavy industry) and a hazardous waste disposal site; exposure defined as living within 20 km of the industrial park	referent) to industrial park; with stratification by ethnicity, rates of perinatal mortality did not vary for Jewish births with proximity to the industrial park, but did vary for Bedouin births for perinatal mortality (RR 1.45, 95% CI 1.22-1.72), postpartum deaths (RR 1.32, 95% CI 1.02, 1.71) and fetal deaths before delivery (RR 1.57, 95% CI 1.23, 2.00)	observed with maternal residence within 20 km of an industrial park; limitations: potential for ecologic fallacy, misclassification of exposure, and residual confounding, maternal residential mobility not taken into account in assignment of exposure
			Veight/Preterm Birth		
Baibergenova et al., 2003 USA	Ecologic study of New York State births during 1994-2000 (excluding New York City)	Low birth weight (1500 to < 2500 grams) and very low birth weight (< 1500 grams)	Exposure defined as maternal residence at birth in a zip code that contained or was adjacent to a PCB-contaminated site	Birth weight in the PCB zip codes on the average 21.6 g less than in other zip codes (p < 0.001); adjusted OR for low birth weight with maternal residence in PCB zip code 1.04, 95% CI 1.02, 1.07 (association only noted for male births) and for very low birth weight 0.95, 95% CI 0.88, 1.02	Slight association noted for risk of low birth weight in male births and maternal residence in zip code with one or more waste sites contaminated with PCBs; limitations: potential for ecologic fallacy, exposure misclassification, and residual confounding
Genereux et al., 2007 Canada	Retrospective cohort study of live singleton births in Montreal, Canada, 1997- 2001	Preterm birth (gestational age < 37 weeks), small- for-gestational age births (< 10 th percentile birthweight for gestational age), and low birth weight (< 2500	Distance between residence at delivery and nearest highway; defined residential proximity as distance of 200 m from highway	Proximity to highways associated with preterm birth (adjusted OR 1.14, 95% CI 1.02, 1.27) and low birth weight births (adjusted OR 1.17, 95% CI	Proximity to highways associated with preterm and low birth weight births – effects mainly confined to wealthy neighborhoods and highly

Goldberg et al., 1995 Canada	Population- based, case- control study of live births to residents on Island of Montreal, 1979-1989	Low birth weight, very low birth weight, preterm births, small-forgestational age (less than or equal to the third percentile weight for gestational age)	Defined three exposure zones representing areas proximal and distal to a municipal solid waste landfill site; high exposure zone divided into two subzones to account for prevailing winds	1.04, 1.33); effects of proximity to highways strongest for preterm and low birth weight births among highly educated women and wealthy neighborhoods Significant excess of between 11 and 20% in low birth weight and between 8 and 13% in small for gestational age noted among births to mothers who resided adjacent to the landfill	educated mothers; limitations: used address at delivery to assign exposure, potential exposure misclassification with reliance on postal codes, and residual confounding Increased risks for low birth weight and small for gestational age births noted among mothers who lived near landfill site; limitations: used maternal residence at delivery to assign exposure, potential for exposure misclassification and residual confounding from unmeasured risk factors
Morgan et al., 2004 United Kingdom	Retrospective cohort study of singleton live births in England, 1986-1999	Low birth weight births	Residence within 3 km of a landfill; for all study areas pooled, defined 1-km distance bands with 6-7 km as baseline	Adjusted pooled OR for residence within 3 km of hazardous waste landfill site 1.03 (95% CI 0.98, 1.08); with adjustment of ORs, all ORs compatible with null in individual study areas; no trend of increased risk noted with closer proximity to sites	No significant increase of low birth weight associated with a maternal residence near hazardous waste sites; limitations: assessed for limited number of confounders which might have led to residual confounding; potential for exposure

					misclassification
Wilhelm and	Population-	Term low birth	Calculated a	Noted an elevated	Results
Ritz, 2003	based case-	weight, preterm	distance-weighted	RR (1.08, 95% CI	suggested
USA	control study	and low birth	traffic density	1.01, 1.15) for all	exposure to
	of births to	weight, and all	value (DWTD) for	preterm births in	traffic-related
	residents in	preterm births	each subject by	relation to	pollution might
	112 zip code		constructing 750-	maternal	be risk factors
	areas in Los		foot radius buffer	residence in the	for term low
	Angeles,		around each	highest DWTD	birth weight,
	1994-1996		subject home and	quintile; stronger	preterm and low
			estimated	associations for	birth weight, and
			dispersion of	all outcomes	all preterm
			motor vehicle exhaust from	noted for women whose third	births; limitations:
				trimester fell	address at
			roadways in this region	during fall/winter	delivery used to
			region	months in the	assign exposure,
				highest DWTD	potential for
				quintile;	exposure
				significant trend	misclassification,
				noted between	selection bias,
				increasing DWTD	and residual
				in the fall/winter	confounding
				months during the	
				third trimester and	
				risk of preterm	
				birth and low birth	
				weight births	
Bhopal et al.,	Population-		regnancy Outcomes Residential	No significant	With exception
1999	based,	All congenital abnormalities	proximity to major	differences in	of low birth
United	ecologic study	(excluding	steel and	congenital	weight, no
Kingdom	of births,	isolated minor	petrochemical	malformation	excess risk of
Kingdom	stillbirths, and	congenital	industries in	rates (combined)	adverse
	terminations	abnormalities),	Teesside divided	found either	pregnancy
	in Teesside	low birth weight,	into three zones	across the	outcomes
	and	stillbirth, sex ratio	based on distance	Teesside zones or	associated with
	Sunderland,	, , , , , , , , , , , , , , , , , , , ,	with Sunderland	between these	living near major
	United		serving as the	zones combined	steel and
	Kingdom,		reference	and Sunderland,	petrochemical
	1986 - 1993		population	the reference	industries;
				population (OR	limitations:
				ranged from 0.87	potential for
				– 1.2 with all 95%	ecologic fallacy
				CI including the	and residual
				null); % low	confounding,
				birthweight higher	maternal
				in Teesside than	residential
				Sunderland (OR	mobility during
				1.20 95% CI 1.09,	pregnancy not
				1.33); no	taken into

				association noted with sex ratio	account
Dodds and Seviour, 2001 Canada	Population- based cohort study Nova Scotia, Canada Live births and stillbirths, 1988-1998	All major anomalies combined and nine anomaly subgroups, low birth weight, preterm delivery, intrauterine growth retardation (IUGR)	Rates for malformations and other adverse pregnancy outcomes compared by maternal address at the time of delivery in Sydney (site of hazardous waste site), Nova Scotia, and Cape Breton County (excluding Sydney)	Residents in Sydney (with hazardous waste site) were 1.3 times more likely (95% CI 1.0, 1.5) to have births with a major congenital anomaly than Nova Scotia residents; also relative to Nova Scotia, rate ratio for NTDs elevated in births to Sydney residents (RR 1.8, 95% CI 1.1, 3.1); no excess risk noted in Sydney for low birth weight, preterm birth, or IUGR	Small statistically significant increase in rate of major congenital malformations in community with a hazardous waste site; limitations: used residence at delivery to assign exposure, potential exposure misclassification
Dummer et al., 2003 United Kingdom	Retrospective cohort study Cumbria (northwest England), 1956-93	Deaths from congenital anomaly (ICD 740 – 749), stillbirth, neonatal death	Distances of maternal address at child's birth (obtained from birth certificate) from incinerators and crematoriums	Risk of lethal congenital anomaly significantly increased (p < 0.01) with maternal address closer to incinerators (restricted to heart defects and spina bifida); increased risk of anencephaly, other congenital anomalies, and stillbirth with maternal address near crematoriums	Significant increased risk of spina bifida and heart defects with maternal proximity to incinerators and increased risk of anencephaly and stillbirth with maternal proximity to crematoriums; limitations: congenital malformations restricted to deaths, pregnancy terminations not included, used maternal addresses at delivery to assign exposure, potential for

Elliott et al., 2001 Great Britain	Ecologic study of live births, stillbirths, congenital malformations	All congenital anomalies combined; neural tube, cardiovascular, and abdominal	Mother's address (unclear when ascertained or from what sources); distance to hazardous waste	Unadjusted and adjusted relative risks close to 1.0 for all defects studied; modest association	exposure misclassification and residual confounding Found small excess risk of congenital anomalies and low and very low birth weight in
	including terminations; Great Britain, 1983-1998	wall defects; hypospadias and epispadias; surgical correction of hypospadias and epispadias; surgical correction of gastroschisis and exomphalos; still births; low and very low birth weight	sites; within 2 km categorized as exposed	observed for surgical correction of gastroschisis and exomphalos (RR 1.2, 99% CI 1.1, 1.3) if mother lived within 2 km of site relative to living farther away; adjusted RR for low and very low birth weight 1.05 (99% CI 1.047, 1.055) and 1.04 (99% CI 1.03, 1.05) respectively	populations living within 2 km of landfill sites; limitations: potential for ecologic fallacy, exposure misclassification, and residual confounding
Fielder et al., 2000, United Kingdom	Ecologic study of population in South Wales who lived near a landfill site, 1983-96	All congenital anomalies combined and anomalies of the abdominal wall, low birth weight, spontaneous abortion	Exposure defined as living in electoral wards within 3 km of the landfill; examined rates before and after site opened	Increased risk for congenital malformations in births among residents living near the site both before opening (RR 1.9, 95% CI 1.3, 2.85) and after opening (RR 1.9, 95% CI 1.23, 2.95); cluster of gastroschisis detected after the site opened; neither hospitalization rates for spontaneous abortion or percentage of low birth weight births differed between the populations	Increased rate of congenital malformations (combined) found in population living near landfill which predated opening of landfill; limitations: potential ecologic fallacy and exposure misclassification

				living near and farther away from the site	
Gilbreath & Kass, 2006 USA	Population- based retrospective cohort study of live births and fetal deaths in Alaska Native villages, 1997 - 2001	Fetal deaths 20 weeks of gestation or greater; neonatal deaths; observable congenital anomalies as recorded on the birth records grouped into five categories including central nervous system, circulatory and respiratory, gastrointestinal, urogenital, and musculoskeletal or integumentary defects	Exposure variables obtained from hazard rankings of dumpsites; residence in village with open dumpsites ranked as lower or higher hazard	The 95% confidence limits for crude and adjusted rate ratios for nearly all outcomes of interest were consistent with the null, although the adjusted point estimates of the rate ratios (rate in villages with higher hazard dumpsites relative to rate in villages with lower hazard dumpsites) were positive for all congenital anomaly categories except gastrointestinal defects; infants born to mothers residing in high hazard dumpsites were 4.27 times (95% CI 1.76, 10.36) more likely to have anomalies classified as "other defects"	With the exception of one group of congenital anomalies, no significant excess risk was found for fetal deaths, neonatal deaths, or congenital anomalies with a maternal residence in Alaska Native villages with higher hazard dumpsites; limitations: potential for exposure misclassification and residual confounding from unmeasured confounders; maternal address at delivery used to assign exposure; terminations not included in birth defects group
Morris et al., 2003 Great Britain	Ecologic study that included all births, stillbirths, and termination registries in Scotland between 1982 and 1997	All congenital anomalies combined; neural tube, cardiovascular, and abdominal wall defects; hypospadias and epispadias; surgical correction of hypospadias and epispadias; surgical surgical	Mother's address (unclear when ascertained or from what sources); distance to hazardous waste sites; within 2 km categorized as exposed	No statistical excess was found for all congenital anomalies combined (RR 0.96, 99% CI 0.89-1.02) or for any of the specific anomalies studied; no excess risks were found for low and very low birth weight or still births with a	No excess risks of adverse pregnancy outcomes detected in population living within 2 km of a hazardous waste site; limitations: potential ecologic fallacy, ascertainment bias, misclassification

		correction of gastroschisis and exomphalos; low and very low birth weight; still births		residence within 2 km of a hazardous waste site	of exposure, and residual confounding from unmeasured variables such as maternal age
Shaw et al., 1992 USA	Population- based, case- control study Five-county San Francisco Bay Area live births and fetal deaths 1983 - 1985	All congenital malformations (grouped in 10 malformation groups) except those considered to be inherited or could be attributed to another exposure; birth weight	Mother's residence at the time of delivery in a census tract with one or more sites with documented environmental contamination	Few associations noted between malformations studied and maternal residence within a census tract with site(s) containing environmental contaminants; elevated risk (OR 1.5, 95% CI 1.1, 2.0) for heart/circulatory defects in offspring of mothers who resided in census tracts with sites with evidence of potential human exposure; minimal effects noted on birth weight with this exposure	No excess risks found for reduced birth weight or congenital malformations with the exception of heart/circulatory defects; limitations: potential for exposure misclassification given varying land area of census tracts, address at delivery used to assign exposure, potential residual confounding
Sosniak et al., 1994 USA	Population- based case- control study of births from the 1988 National Maternal and Infant Health Survey conducted in 48 states	Low and very low birth weight, congenital anomalies, infant deaths, fetal deaths	Distance between zip code centroids of maternal residences and National Priority List (NPL) sites; a distance of 1 mile or less from nearest NPL site was classified as exposed	Adjusted OR for low birth weight in relation to residential proximity to NPL site was 0.99 (95% CI 0.86, 1.16). Residential proximity to NPL sites as defined in this study not associated with congenital anomalies, fetal deaths, infant deaths, or very low birth weight	Maternal residential proximity to NPL sites not associated with adverse pregnancy outcomes; limitations: distance measures based on zip code centroids that could have led to exposure misclassification
Tango et al., 2004	Retrospective cohort of	Infant, neonatal, and fetal deaths	Distance of street addresses (from	No significant excess noted in	Peak-decline in risk noted with

Japan	births and fetal deaths in Japan, 1997- 1998	due to congenital malformations (all combined), male/female sex ratio, low and very low birth weight, neonatal deaths and infant deaths, fetal deaths	vital records) from municipal solid waste incinerators divided into ten sub-areas delimited by ten circles of radii of 1,2,, 10 km.	deaths due to congenital malformations with address within 2 km of municipal solid waste incinerator; statistical significant peak decline in risk of infant deaths and infant deaths due to congenital malformations (combined) with distance from the incinerators up to 10 km with peak at 1-2 km	distance from municipal solid waste incinerators for infant deaths and infant deaths with all congenital malformations combined; limitations: potential for exposure misclassification, maternal addresses at registration used which did not account for residential mobility during pregnancy, potential for residual confounding
Vinceti et al., 2008, Italy	Retrospective cohort study of women 16- 49 years of age who resided in Modena, northern Italy and surrounding areas, 2003- 2006	Spontaneous abortions and all birth defects combined	Residential proximity to municipal waste incinerator with two zones delineated based on predicted mean annual atomospheric concentrations of dioxins and dibenzofurans; also considered occupational exposures	No excess risk of miscarriage (RR 1.00, 95% CI 0.65, 1.48) or birth defects (RR 0.64, 95% CI 0.20, 1.55) noted in women residing in two zones close to the incinerator plant; in women working in plant, no excess risk for spontaneous abortions noted, but increase prevalence of birth defects found (RR 2.26, 95% CI 0.57, 6.14)	No statistically significant excess risk for spontaneous abortions or birth defects noted among women residing near a municipal waste incinerator; limitations: small sample size, all birth defects combined, possible exposure misclassification and residual confounding
	•		rds and Childhood C		
Carozza et al., 2008, USA	Ecologic study; U.S. cancer cases ages 0-14	All cancers combined and specific cancers diagnosed among	Percent of cropland for each county based on 1997 U.S. Census	All cancers combined showed no association with percent	Counties with a higher percentage of cropland showed

	years diagnosed between 1995- 2001 and reported to member registries of the NAACCR	children 0-14 years	of Agriculture; divided into <20% cropland (referent); 20 - <60% (medium), and 60+% (high); also examined six leading U.S. crops	cropland in counties; incidence rates of several specific cancers showed an association with medium and/or high levels of agricultural activity; risk estimates for	a higher incidence of several childhood cancers; limitations: potential for ecologic fallacy, use of county of residence at time of diagnosis,
Corogra et	Donulation	Childhood	Fields identified	childhood cancer varied by type of crop grown with elevated risks noted in counties with corn, oats, and soybeans	potential for residual confounding
Carozza et al., 2009, USA	Population- based case- control study; Texas childhood cancer cases and controls born 1990- 1998	Childhood cancers reported among children < 15 years of age to the Texas Cancer Registry	Fields identified from digital orthophoto quadrangle data and Field Mass Index created to incorporate land area (cropland) and distance to each field from birth residence listed on birth certificate	No association between a birth residence within 1000 m of agricultural land use and all cancers combined. A birth residence near cropland showed some association with germ-cell tumors, non-Hodgkin lymphoma, and Burkitt lymphoma, but ORs based on few cases	Minimal associations found between birth residence near cropland and childhood cancer; limitations: small numbers of exposed cases and potential for residual confounding
Choi et al., 2006, USA	Population-based case-control study; cases < 10 years of age at time of diagnosis during 1993-1997 and residents of Florida, New Jersey, New York (excluding NYC), or	Incident cases of primary brain cancer	Residential proximity to Toxic Release Inventory (TRI) during pregnancy (less than or equal to 1 or less than or equal to 2 miles), whether carcinogens were emitted, and a comparative ranking system for TRI releases that combined toxicity	Increased risk for brain cancer among children less than 5 years of age at diagnosis observed for mothers living within 1 mile of a TRI facility (OR 1.66, 95% CI 1.11, 2.48) and living within 1 mile of a facility releasing carcinogens (OR	Results suggestive of relation between living in close proximity of TRI site emitting carcinogens during pregnancy and childhood brain cancer; limitations: quality of exposure data, potential for

	Pennsylvania at diagnosis		information and total mass of release	1.72, 95% CI 1.05, 2.82)	residual confounding from parental occupational exposures
Crosignani et al., 2004, Italy	Population- based case- control study; Varese Province, Italy	Childhood leukemia cases diagnosed diagnosed 1978- 1997	Exposure distances of childhood addresses from highly trafficked roads and also traffic densities in surrounding area; estimation of benzene concentrations with Gaussian diffusion model	Relative to children whose homes were not exposed to road traffic emissions (< 0.1 ug/m³ benzene as estimated by the model), risk of leukemia with benzene concentration > 10 ug/m³ (OR 3.91, 95% CI 1.36, 11.27)	Results suggest that motor vehicle emissions might be etiologic factor for childhood leukemia; potential for residual confounding from unmeasured confounders (parental occupation) and imperfect measurement (SES assigned based on municipality of residence)
Harrison et al., 1999, United Kingdom	Population- based case- control and retrospective cohort study designs, United Kingdom West Midlands	Childhood leukemia cases diagnosed between 1990- 1994	Exposure defined as an address at the time of diagnosis within 100 m from petrol station or a zone 100 m from a main road	Odds ratios from case-control study 1.61 (95% CI 0.90, 2.87) and 1.99 (95% CI 0.73, 5.43) for living within 100 m of a main road or petrol station respectively; incidence ratios from cohort analysis 1.16 (95% CI 0.74, 1.72) and 1.48 (95% CI 0.65, 2.93) for proximity to roads and petrol stations respectively.	Results suggestive of association but CIs around risk estimates compatible with null; limitations: risk estimates not adjusted for age or sex
Jarup et al., 2002, Great Britain	Ecological study that included cancer cases	Childhood and adult leukemia; adult bladder cancer, brain	Constructed 2 km buffer zones around 9565 landfill sites using	With rate ratios adjusted for age, sex, year of diagnosis, no	No association found between living within 2 miles of landfills

	diagnosed from 1983- 1997	cancer and hepatobiliary cancer	GIS techniques. Postcodes lying outside 2 km buffer were the referent areas	excess of any cancer was found in relation to living within the 2-mile buffer of landfills	and cancer; limitations: potential for exposure misclassification to chemicals in landfills and potential for ecological fallacy
Kaatsch et al., 2008, Germany	Population- based case- control study of 41 counties in the vicinity of 16 West German power plant sites	Leukemia and other cancers that were diagnosed in children less than 5 years of age	Distance of residence at the time of diagnosis from the chimney of the nearest nuclear plant; residential proximity within 5 km and within 10 km	For all leukemia cases combined, a dose-response effect was noted in which cases lived closer to sites than controls; residential proximity within 5 km was associated with an odds ratio of 2.19 (lower 95% CL: 1.51)	Positive relationship found between diagnosis of childhood leukemia and residential proximity to the nearest nuclear power plant; limitations: potential selection bias due to differential response rates between cases and controls and between those who lived within 5 km and outside the buffer zone; potential residual confounding
Knox, 2000, Great Britain	Migration study of 4385 children who died from cancer before age 16 in Great Britain, 1953-1980	Tumors were classified into 11 groups	Migration asymmetries of birth and death addresses and proximity of these addresses to municipal and hospital waste incinerators and landfill sites	No systematic migration-asymmetries were noted for landfill sites; highly significant excesses of migrations away from birth places close to municipal and hospital incinerators	Children with cancer more likely to live near incinerators at birth than at death; limitations no external control group nor control for demographic characteristics or proximity to other environmental hazards, deaths instead of incident cases

					were used
Knox, 2006, Great Britain	Migration study of 5,663 children who died from cancer before age 16 in Great Britain, 1953-1980	Tumors were classified into 10 diagnostic subtypes	Birth and death addresses linked to locations of railway stations, bus stations, ferry terminals, railways, roads, canals, and rivers and migration asymmetries of birth and death addresses examined	Significant migration asymmetries (close residential proximity at birth but not at death) noted for residential proximity to bus stations, railway stations, ferries, railways and roads	Children with cancer more likely to live near roads and railways at birth than at death; limitations include no external control group nor control for demographic characteristics or proximity to other environmental hazards, cancer deaths instead of incident cases were used
Knox & Gilman, 1997, Great Britain	Retrospective cohort study with 22,448 addresses at death (and available birth addresses) of children ages 0-15 years who died of leukemia and other cancers in England, Wales, and Scotland	Deaths from leukemia and other childhood cancers	Radial distances of home address postcodes (birth and death) from potential hazards including industries, railway lines, motorways, airfields, and harbors	Relative excesses of leukemias and solid tumors were found with residences close to a variety of industries and airfields, railways, motorways, and harbors; hazard proximities for birth addresses were stronger than for death addresses; among children who moved between birth and death, the proximity effect was limited to birth addresses	Authors concluded that childhood cancers were geographically associated with industrial atmospheric effluents that contained 1) petroleum derived volatiles and 2) kiln and furnace smoke and gases, and effluents from internal combustion engines; limitations: population at risk not enumerated at various distances, no adjustment for age or sex, analyzed cancer deaths instead of incident cases
Langholz et al, 2002,	Population- based case-	Incident cases of childhood	Integrated distance-weighted	Although unadjusted ORs of	No evidence found between

USA	control study in Los Angeles County, CA, USA	leukemia diagnosed during 1978-1984 among children 0 to 10 years of age	traffic density was computed for the residence of longest duration	the relation between quintile of traffic density and risk of leukemia suggested a linear trend, this trend was confounded by wire code	traffic density and childhood leukemia with adjustment for wire code; limitations: potential exposure misclassification since traffic counts were obtained for 1990-1994
Liu et al., 2008, Taiwan	Population- based case- control study of 226 Taiwan municipalities	Brain cancer deaths from Bureau of Vital Statistics occurring in persons 0 – 29 years of age; controls included deaths from all causes other than cancer and diseases with respiratory complications	Proportion of municipality's total population employed in petrochemical industry used as indicator of petrochemical air emissions at residence at death	With the petrochemical indicator variable divided into tertiles, persons in the highest tertile (with the lowest tertile as referent) had a significantly higher OR for death from brain cancer (OR 1.65, 95% CI 1.0, 2.73) with adjustment for age, gender, urbanization level of residence, and nonpetrochemical air pollution (p-value for trend < 0.01)	Risk of brain cancer associated with metric of residential exposure to petrochemical air pollution; used brain cancer deaths instead of incident cases and not clear if data available to distinguish primary from metastatic brain cancers; exposure metric based on municipality instead of individual distance from industry thereby introducing exposure misclassification; death address may not be relevant for cases who changed residences between birth and death
Raaschou- Nielsen et al., 2001 Denmark	Population- based case- control study of Danish children < 15	Leukemia, tumors of the central nervous system, malignant lymphoma	Addresses from nine months before birth to diagnosis of cancer or similar date for the	Risks of leukemia, CNS tumors, and all selected cancers combined not linked to	Risk of Hodgkin's lymphoma increased in offspring of

	years diagnosed with selected cancers 1968- 1991 and control children	diagnosed in children < 15 years of age	matched controls linked to average concentrations of benzene and nitrogen dioxide at the front door of dwelling with use of Operational Street Pollution Use Model	exposure to benzene or nitrogen dioxide estimates; risk of Hodgkin's lymphoma associated with highest categories of exposure for benzene and nitrogen dioxide (ORs respectively 4.3 [95% CI 1.5, 12.4] & 6.7 [95% CI 1.7, 26.0]	mothers who resided in areas with high outdoor levels of traffic-related air pollution; limitations: parental occupation not taken into account in the analyses
Reynolds et al., 2002a, USA	Ecologic study using 1988-1994 childhood cancer incidence rates in California	Cases of invasive cancer diagnosed in children less than 15 years of age during 1988-1994	Assigned census block groups to case residences at diagnosis; for each block group, estimated pesticide use density in pounds per square mile for four toxicologic groups, four chemical classes, and seven individual pesticides	For all cancers combined, the RR for block groups with high propargite usage was 1.25 (95% CI included 1.0); with leukemia, the RR associated with propargite usage was 1.48 (95% CI 1.03, 2.13); no association noted between usage density of pesticides classified as probable carcinogens at or above the 90 th percentile and all types of childhood cancer combined (RR 0.95, 95% CI 0.80, 1.13)	Study found little evidence of association between residence at diagnosis in areas with high pesticide usage and childhood cancer incidence rates; limitations include potential for ecologic fallacy, potential for residual confounding, and exclusive use of residence at diagnosis to assign exposure
Reynolds et al., 2002b, USA	Ecologic study using 1988-1994 childhood cancer incidence rates in California	All childhood cancers combined; leukemias; gliomas (brain cancer) diagnosed in children < 15 years of age	Assigned census block groups to case residences at diagnosis and used GIS to match addresses with a road network; estimates developed for vehicle, road, and	Rate ratios at the 90 th percentile of traffic density were 1.08 (95% CI 0.98, 1.20) for all childhood cancers combined, 1.15 (95% CI 0.97, 1.37) for leukemia, and	Results of study showed minimal/no association between high traffic, vehicle, or road density and childhood cancer; limitations:

Reynolds et	Population-	Childhood cancer	traffic density; these three metrics were correlated with ambient measurements of carbon monoxide, nitrogen dioxide, PM10, benzene, and 1,3-butadiene	1.14 (95% CI 0.90, 1.45) for gliomas; minimal/no evidence of rate differences in these cancers in census block groups with high vehicle or road density; results were suggestive of an association between traffic density and Hodgkin's lymphomas but a dose-response pattern was not observed For all cancers	potential ecologic fallacy, lacked data on potential confounding factors, used residence at diagnosis to assign exposure which might not be relevant for children who changed residences
al., 2004, USA	based case- control study; California state-wide	combined, leukemias, & central nervous system tumors diagnosed in children < 5 years of age	maternal residential address at delivery linked to road and traffic density in 500-foot radius of residence	combined, OR for highest road density exposure category compared with lowest was 0.87 (95% CI 0.75-1.0), OR for leukemia was 0.80 (95% CI 0.64, 1.01), and OR for CNS tumors was 1.03, 95% CI 0.75-1.43). Similar ORs were found with traffic density although OR for CNS tumors was 1.22 (95% CI 0.87, 1.70)	evidence of increased cancer risk in young children born in high traffic density areas; limitations: assignment of exposure limited to residence at birth and potential for residual confounding due to lack of information on parental occupational exposures, exposure to secondhand smoke, etc.
Reynolds et al., 2005, USA	Population- based case- control study; California state-wide	Childhood cancer combined, leukemias, & central nervous system tumors diagnosed in children < 5 years of age	Case and control maternal residential addresses at birth linked to pesticides used on land area (pounds per square mile) within one-	No clear risk patterns noted although mildly elevated ORs for leukemia associated with pesticides that were probable and	No specific patterns of risk noted between living near pesticide applications and childhood cancer;

			half mile of residence	possible carcinogens, and use of organochlorines or organophosphates	limitations: small numbers of children exposed to high-use areas, exposure assessment restricted to birth address, potential exposure misclassification
Rull et al., 2009, USA	Population- based case- control study; selected counties in northern California	Incident cases of childhood acute lymphoblastic leukemia diagnosed in children < 15 years 1995-2002	Case and control lifetime and first year of life residences linked to pesticides used on land area within one-half mile; Pesticides categorized by toxicological effects, physicochemical properties, and target pests or uses	Noted increased risk of acute lymphoblastic leukemia (ALL) with lifetime moderate exposure to pesticide applications of organophosphates, chlorinated phenols, and triazines and with pesticides classified as insecticides and fumigants; elevated risk not consistent with high exposures	Elevated ALL risk with moderate but not high exposure; limitations: small numbers of exposed cases and controls, climatic conditions not considered
Sharp et al., 1996, Scotland	Ecological study of populations near seven nuclear sites in Scotland, 1968-93	Incident cases of leukemia & non- Hodgkin's lymphoma in children < 15 years of age	For each nuclear site, study zone constructed with a population centroid within 25 km; each nuclear site examined separately; small area statistical methods used	No evidence of general increased incidence of childhood leukemia and non-Hodgkin's lymphoma noted around nuclear sites; only one site had appreciably more cases observed than expected (O/E 1.99)	No notable increased incidence of childhood leukemia & non-Hodgkin's lymphoma in children living near nuclear sites; limitations: minimal information on confounding factors, residence at diagnosis used
Spix et al., 2008, Germany	Population- based case- control study around all 16 major nuclear power plants	Leukemia including specific forms, central nervous system tumors diagnosed in children < 5	Metric of 1/(distance in km) used as measure of proximity; categorical analyses of 5- and	Effects modest except for the association between living in the inner 5-km zone and	Results showed some increased risk for cancer among young children who lived within 5

Steffen et al.,	in Germany Multi-center,	years of age during 1980-2003	10-km zones versus outer zones History of	leukemia (OR 2.19, lower one- sided 95% CI 1.51)	km of nuclear power plants; limitations: other sources of potential radiation exposure not accounted for, potential unmeasured confounders Childhood
2004 France	hospital- based, case- control study (four centers) in France of newly diagnosed cases during 1995-1999	children ages 0-14 years	exposure to hydrocarbons (residential proximity to roadways, car repair garage, petrol station) from date of conception to date of diagnosis (cases) or interview (controls); proximity information obtained by in- person interview; also obtained information about parental occupation	between residential proximity to a petrol station or repair garage during childhood and risk of childhood leukemia (OR 4.0, 95% CI 1.5, 10.3) which was stronger for acute non-lymphocytic leukemia (OR 7.7, 95% CI 1.7, 34.3)	residence near petrol station or automobile repair garage associated with childhood leukemia; limitations: proximity to hazards ascertained by self-report which could have introduced recall bias and inflated risk estimates
Tsai et al., 2006, USA	Population- based case- control study of residents of California, Florida, New Jersey, Michigan, North Carolina, and Pennsylvania	Wilms' tumor diagnosed in children through 9 years of age during 1992 - 1995	Maternal and paternal addresses in close proximity to a National Priority List (NPL) site during the 2-year period before the child's birth; residential history determined by parental interview	OR of 0.35 (95% CI 0.12, 0.99) for Wilms' tumor with a maternal residence within 1 mile of NPL site during pregnancy and OR 0.39 (0.16, 0.98) with a residence within 1 mile of NPL site during 2 years prior to birth; no association noted for paternal residence	Wilms' tumor was not associated with a maternal or paternal residence near NPL site in two- year period before birth; limitations: small numbers of exposed cases and controls and potential selection bias with African Americans more likely to not participate than

					Whites
Von Behren et al., 2008, USA	Population- based case- control study in northern California counties	Leukemia diagnosed in children < 15 years of age during 1995-2002	Traffic density within a 500-foot radius buffer determined for each address at diagnosis, birth, and lifetime average	OR of 1.17 (95% CI 0.76, 1.81) for acute lymphocytic leukemia with residential traffic density above 75 th percentile (0 traffic density as referent) for residence at diagnosis, OR 1.11 (95% CI 0.70, 1.78) for residence at birth, and 1.24 (95% CI 0.75, 2.08) for average lifetime traffic density	No association noted between a residence in areas of high traffic density during any of the exposure periods and childhood acute lympocytic leukemia; limitations: potential selection bias with control subject families having higher household incomes than case families
Weng et al., 2009, Taiwan	Population- based case- control study of all deaths in Taiwan residents	Leukemia deaths in children < 15 years of age, 1996-2006	Petrol station density in municipalities that the residents lived in at the time of death	Adjusted OR for leukemia (death) was 1.91 (95% CI 1.29, 2.82) in association with living in municipalities with the highest petrol station density; a significant trend was noted between increasing petrol station density and risk of death from childhood leukemia	Increased risk for death from childhood leukemia noted with death address in a municipality with high petrol station density; limitations: studied deaths instead of incident cases of leukemia; exposure classification restricted to municipality of residence at death; petrol station density metric based on 2008 data which might have introduced exposure misclassification; potential for residual confounding
Yu et al.,	Population-	Incident leukemia	Exposure	No overall	Results varied by

2006, Taiwan	based case- control study in Kaohsiung, southern Taiwan, 1997- 2003	diagnosed in persons < 30 years of age 1997-2003	opportunity score assigned, based on residences up to two years before birth, that took into account residential mobility, length of stay in each residence, distance to petrochemical plants, monthly prevailing wind direction, and multiple petrochemical pollution sources	association noted with acute lymphocytic leukemia in the group 0-19 years of age who had a higher exposure opportunity score OR 1.21 (95% 0.89, 1.65); positive association seen between residential petrochemical exposure and leukemia among 20-29 year olds (one unit increase in log-transformed exposure score: OR 1.54, 95% CI 1.14, 2.09)	age group. A positive relation found between residential exposure to petrochemicals and leukemia in persons 20-29 years of age but no association seen with leukemia in younger persons with this exposure; limitations: potential for selection bias because of differential participation between cases and controls
--------------	--	--	--	--	--

CI = confidence interval

NAACCR=North American Association of Central Cancer Registries

OR = odds ratio RR= relative risk

Table 5. Studies of Residential Proximity to Potential Environmental Hazards and Cardiovascular, Respiratory, and other Chronic Diseases

Exposur e	Outcome	Reference, Year, Country	Study design, Regional description	Health outcomes included	Exposure description	Target Population	Geospatial Methods		Health outcome associated with proximity & limitations (blue=significant associations purple=mixed evidence, black=no
Industrial Plants	ry	Aylin et al., 2001, England & Wales	Small area study (England and Wales), proximity analysis	Emergency hospital admissions primary diagnosis of respiratory or cardiovascular diseases	Industrial plants: Distance (buffers up to 7.5km buffers) from operating coke works facility	Adults 65 years and over (n=87,760), Children under 5 years (n=43,932)	Distance decline model based on concentric areas around the facility	Older adults: only sign. regression result- coronary heart disease near Teesside plant RR=1.04 (1.00,1.08); no sign. findings for coke works combined Children: respiratory disease RR=1.08 (0.98,1.20); asthma RR=1.07 (0.98,1.18); at Teeside plant gradation of declining risk with distance for both respiratory illness and asthma	sign associations) Possible elevated risk of respiratory disease and asthma in children with proximity to Teesside coke works. Migration and mobility not controlled, use of a simple radial dispersion-decline model for estimating exposure.
Hazardou s Waste sites	PBC levels	Choi et al., 2006, USA	Small area study (New Bedford, Acushnet, Fairhaven, and Dartmouth, Massachusetts), proximity and multivariate regression analysis	Cord serum polychlorinated biphenyl (PBC) levels in infants (collected at birth)	Exposure to superfund sites: Residences within 5 mile radius of superfund hot spot	Infants born to mothers residing near contaminated New Bedford Harbor	Residential distance to superfund hot spot	No association found between cord serum PCB levels and distance to hot spot. Maternal age and	No evidence that living near New Bedford superfund site is associated with increased cord serum PCB. But, higher levels found in children born before and during dredging of harbor. Exposure measurement simplified (pathway), cross-sectional study design.
Air pollution	Respirato ry	Edwards et al., 1994, UK	Case-control study (Birmingham, UK), proximity analysis	Hospital admission for asthma	Mobile source air pollution: Residential proximity (within 200 and 500 meters) to major roads and traffic flow (>24,000 vehicles per hour)	Children under 5 years (cases: n=715, hospital controls: n=736, community controls: n=?)	Distance decline model based on fixed distance buffers around major roads (200, 500 m) and high traffic flows	Sign. association between exposure to traffic and asthma hospitalization versus community control group: for distance to mj road: OR=1.52 (1.22,1.90, p<0.0002); for high traffic flow roads: OR=1.40 (1.13,1.74, p<0.002), also between hospital controls: OR=1.29 (1.04,1.50, p<0.02); evidence of a dose-reponse relationship for traffic flow	Evidence of increased odds of asthma hospitalization with proximity to major roads and high traffic flow areas. Possible confounding (no controls for SES), measurement error (single exposure measure).
Air pollution	ry	USA	Case-control study (San Diego County, CA), proximity analysis with logistic regression	Hospital admissions for asthma	Mobile source air pollution: Residential proximity to high traffic flow (within 550 ft of residence)	Children 14 years or younger (cases: n=5,996, controls: n=2,284)	Fixed 550 ft buffer around residence, and actual distance from residence to street, traffic flow dispersion model	Only sign. results: among cases, those residing within high traffic flow areas more likely to have 2 or more visits than only 1 visit per year: OR=2.89 (1.07,7.40, p<0.05)	No evidence of increased hospital visits for asthma with higher traffic counts near residence. Among asthmatic children, greater number of visits associated with higher traffic counts (contributing rather than causal). Possible confounding (smoking), exposure misclassification.
Hazardou s Waste sites	stage renial disease	Hall et al., 1996, USA	Ecological case-control study (20 counties, New York State), logistic regression analysis	disease (ESRD)	Exposure to hazardous waste sites: Listed on NY inactive hazardous waste site registry	, ,	buffers around each site, 25 sections classified within each buffer as high, medium, low, and unknown liklihood of exposure	Elevated associations found between residence within buffer, number of years at residence, high/medium exposure and ESRD but ORs not significant	No evidence of increased odds of living near hazardous waste facility and ESRD. Exposure measurement errors (residential vicinity as proxy for actual exposure measurement), small sample size).
Air pollution	Stroke Mortality	Hu et al., 2008, USA	Ecological study (Northwest Florida, Escambia and Santa Rosa counties), Bayesian hierarchical model	Stroke mortality (age- adjusted death rate) at census tract level	Air pollution (recorded point and mobile sources): Toxic Release Inventory (TRI) facilities, dry cleaning, sewer treatment, solid waste disposal superfund sites, and vehicular traffic	Residents of Escambia and Santa Rosa counties	Dasymetric mapping for environmental exposure value and spatial interpolation to create air pollution density surfaces	Elevated risk of stroke mortality in areas with high pollution, low income and low level of green space: 95% credible sets for traffic: 0.034, 0.144; monitored point sources: 0.419, 1.495; unmonitored point sources: 0.413, 1.522	Increased risk of stroke mortality in high pollution areas. Measurement error (ischemic vs. hemorrhagic stroke and individual exposure assessments), ecological fallacy.
Industrial Plants	Cancer	Johnson et al., 2003, Canada	Case-control study (Canada), residential distance and logistic regression analysis	Non-Hodgekin Iymphoma (NHL)	Industrial plants: residential proximity (0.5 - 2 miles) to industrial plants: copper smelters, lead smelters, nickel smelters, steel, petroleum refineries, kraft pulp mills, and sulfite pulp mills	Cases of NHL (newly diagnosed) reported to provincial cancer registry (n=1,499) and population controls (n=5,039)	Residential distance to industrial plants (lat/long), distance categories of <0.5, 0.5-2, >2 miles	No sign. association found between proximity to industrial plant (all categories) and NHL. But sign. findings for 1) residing within 2 miles and follicular NHL in women: OR=1.48 (1.10-1.99, p<0.05); 2) residing within 2 miles of copper smelter: OR=5.1 (1.5,17.7, p<0.05); and 3) within 0.5 miles of sulfite pulp mill: OR=3.7 (1.5, 9.4, p<0.05)	No evidence of increased odds of NHL with residential proximity to industrial plants. Some significant finding with specific industry and types of NHL but sample sizes small, need for further research. Recall bias in exposure assessment possible, measurement error (emissions from plants not measured), potential confounding.

Hazardou s Waste sites	Diabetes	Kouznetsova et al., 2007, USA	Ecological study (New York State), negative binomial regression analysis	Diabetes inpatient hospitalization rates at ZIP code level	Exposure to hazardous waste sites by ZIP code: POP sites (dioxins/furans, PCBs, persistent pesticides), other sites (volatile organics and metals, etc.), and clean sites	Adults age 25-74 residing in NYS	Residence within ZIP code containing a hazardous waste site	Sign. association between diabetes hosp. rates for those residing in POP ZIP codes versus those in clean sites: Rate Ratio (IRR)=1.23 (1.15,1.32, p<0.05); and those in non-POP sites: IRR=1.25 (1.16,1.34, p<0.05)	Evidence of increased rates of diabetes hospitalizations in adults residing in POP ZIP codes. Exposure measurement error (only residential proximity measured), unit of analysis large, risk factors at individual-level not taken into account.
Air pollution	Respirato ry	Livingstone et al., 1996, UK	Case-control study (London, UK), proximity analysis	Hospital admissions for asthma	Mobile source air pollution: Residential proximity (within 150 m) to high traffic flow (1,000 vehicles per hour at peak) by post code	Patients age 2-64 years of age (cases: n=978, controls: n=5,685)	Shortest distance from residence post code and point process methods (distance decline)	No difference in odds of asthma hospital admissions for those living 150m or less from high traffic flow for any age group (adults or children under 16); no association for point process methods	No evidence of an association between living near high traffic flow and asthma admissions. Exposure measurement error (distance from road crude approximation of exposure to traffic pollution).
Air pollution	ry	Maantay, 2007, USA	Small area study (Bronx, NY), cross- sectional proximity analysis	Asthma-related Hospitalization rates	Air pollution: distance (buffers up to 0.5 miles depending on source) from known noxious land uses (TRI facilities, heavily trafficked roadways, other point sources of pollution)	Adults 16 years and older, Children under 16 years	Fixed distance buffers, areal interpolation by census block group	Combined exposure: Adults: OR=1.28-1.30 (p<0.01), Children: OR=1.11-1.17 (p<0.01), Standardized incidence ratios (SIRs over 5 years) inside and outside exposure buffers are sign. different (p<0.05)	Elevated incidence of hospitalizations due to asthma in children and adults with proximity to air pollution sources. Data errors, exposure measurement errors, assumes everyone in buffer is impacted equally.
Air pollution	Respirato ry	Maantay et al., 2009, USA	Small area study (Bronx, NY), cross- sectional proximity and multiple regression analysis	Asthma-related Hospitalization rates	Air pollution (PM ₁₀ , PM _{2.5} , NO _x CO, SO ₂): distance from plume buffers of stationary point sources (21 facilities); Source Impact Index (SII) for exposure to combined pollutants	Residents of the Bronx	Air dispersion modeling (AERMOD) using loose coupling with GIS	Regression analysis: sign. relationship between asthma rates and cumulative SII quantile groups (R2=0.305, •=0.553, p<0.05); Sign. differences between asthma rates inside buffers and outside (OR)	Sign. differences between asthma rates inside buffers and outside. Data errors, exposure measurement errors (only NEI stationary point sources included in exposure), assumes everyone in buffer is impacted equally.
Air pollution	Stroke Mortality	Maheswaran and Elliott, 2003, England and Wales	Small area ecological study (England and Wales), proximity analysis using log- linear poisson regression	Stroke mortality (age- adjusted death rate) at census enumeration district level (CED)	Mobile source air pollution: Proximity to main roads (from centroid of CED)	Adults age 45 and older	Distance decline model based on centroid of CED distance from main roads (<200, 200-<500, 500- <1,000, 1,000+ m from road)	Sign. associations between stroke mortality and distance to main roads (<200m versus 1,000+m) for: men IRR=1.07 (1.04,1.09, p<0.05); women IRR=1.04 (1.02,1.06, p<0,05), dose-response demonstrated for distance categories	Evidence of increased rate of stroke mortality for adults residing near main roads. Exposure measurement error (proximity to roads as proxy for mobile air pollution), imprecision of centroid analysis, no controlling for individual risk factors, ecological fallacy.
Nuclear Plant	Cancer	Morris and Knorr, 1996, USA	Case-control study (Plymouth, MA), proximity analysis with logistic regression	Adult Leukemia	Radioactive emissions from nuclear plant: Individual summary exposure scores and residential proximity (within 4 miles) of Pilgrim's nuclear plant	Adults over 13 years of age (cases: n=105, controls: n=208)	Fixed buffers around plant <4, 4-12.9, 13-22.9, 23 plus miles)	No sign. association found between proximity to plant (all categories) and adult leukemia (but odds increased with proximity to plant). For exposure score, evidence of dose-response relationship, each score category sign. higher than lowest level. When stratified, results sign. for highest exposed women versus lowest exposed: OR=5.19 (1.83-14.7, p<0.0.5)	Some evidence of sign. association between higher levels of exposure and adult leukemia. Small sample size, stratification further reduces statistical power, exposure measurement errors (not actual individual radiation doses), possible confounding and bias.
Air pollution	ry	Oosterlee et al., 1996, The Netherlands	Small area study (Haarlem, The Netherlands), cross- sectional study with logistic regression	Chronic respiratory symptoms (self- reported by parents)	Mobile source air pollution: Residential proximity (lives on high traffic density street) to high traffic flow (10,000- 30,000 vehicles per 24 hours)	Children under 16 (exposed: n=106, unexposed: n=185); Adults (exposed: n=673, unexposed: n=812)	Mobile (traffic) air pollution (street dispersion model)	Children: only sign. association for use of respiratory medications: OR=2.2 (1.1,4.6, p<0.05); stratification results: girls (ORs significant: wheeze, attacks, respiratory medicine) and children age 6-10 higher ORs. Adults: only sign. association for dyspnoea-occasional: OR=1.8 (1.1,3.0, p<0.05)	Evidence of increased odds for some asthma symptoms for children living on a high traffic flow road (effects greater for girls). Exposure measurement crude, possible information bias, small sample size for stratification analyses.
Air pollution	Respirato ry	Smargiassi, 2009, Canada	Ecological study (Montreal, Canada), case-crossover (time- series) proximity and logistic regression analysis	Asthma-related ED and hospital admissions	Air pollution (SO ₂): exposure to levels above the daily mean within 0.5-7.5 km of refinery stacks	Children age 2-4 years (n=3,470)	Air dispersion modeling (fixed monitoring sites and AERMOD) estimating daily SO ₂ levels at the centroid of residential postal codes	Same-day ED visits, peak levels: OR=1.10 (1.00,1.22), p<0.05; sameday hospital admissions, peak-levels: OR=1.42 (1.10,1.82), p<0.05	Short-term increases in SO ₂ (above mean) are sign. associated with a higher number of asthma related episodes in children residing near refineries (lag of 0 days). Data errors, exposure measurement errors (modeling, estimated exposure at residence may not be accurate), associations may represent pollutate mixture.

									,
Air	ry .	Venn et al., 2001, UK	Small area study (Nottingham, UK), cross- sectional study with logistic regression		Mobile source air pollution: Residential proximity to main road (from centroid of postal code)	Children 4-11 years old (n=6,147), children 11-16 years old (n=3,709)	based on centroid of residential post code distance from main roads (quartiles)	Children 4-11: No sign effect found by quartile but for children living within 150m of road, increase in odds of wheeze with increasing proximity to roads: OR for 30m-increment=1.08 (1.00,1.16, p<0.05); stronger effect for girls: OR for 30m-increment=1.17 (1.05,1.31, p<0.05) but absent for boys. Children 11-16: No sign effect found by quartile but for children living within 150m of road, increase in odds of wheeze with increasing proximity to roads: OR for 30m-increment=1.16 (1.02.1.32, p<0.05)	
Air pollution	ry	Netherlands	Small area study (South Holland, The Netherlands), cross- sectional study with logistic regression	symptoms (self- reported by parents)	Mobile source air pollution: Residential proximity (within 100m, 100-1,000m) from freeway (80,000-150,000 vehicles per 24 hours, ambient air pollution)	Children 7-12 years old attending 13 different schools (n=878)	freeway, distance categories of <100m, 100- 1,000m	wheeze: OR=3.05 (1.11,8.41, p<0.05)	between exposure variables and asthma symptoms except when straffied by sex (girls). Possible information bias and confounding, long-term exposure measurement missing.
Industrial Plants	Cancer	Wilkinson et al., 1997, England	Small area study (Waltham Abbey, Essex England), proximity analysis	Cancer incidence and mortality	Industrial plants: Exposure to Pan Britannica Industries factory (pesticides and fertilizers), residential proximity by electoral ward within 7.5 km radius	Residents of Waltham Abbey	based on concentric areas around the facility	Sign. association between exposure to the plant and cancer (as determined by observed versus expected values): Incidence: 0-7.5km distance O/E ratio=1.04 (1.02,1.06, p<0.05); 0-1km distance O/E ratio=1.10 (1.00,1.22); Deaths: 0-7.5km distance O/E ratio=1.04 (1.02,1.06, p<0.05); 0-1km distance O/E ratio=1.04 (1.02,1.06, p<0.05); 0-1km distance O/E ratio=1.24 (1.11,1.39); inconsistent evidence of dose-response relationship	Some evidence of increase incidence and death from cancers for residents residing near the Pan Brittanica factory but inconsistent evidence of dose-response relationship (also, increase of noncancer mortality found). Measurement errors (population around facility, cancer records), potential confounding, effects from other exposures nearby.
Air pollution	Respirato ry	Wilkinson et al., 1999, UK	Case-control study (North Thames, UK), proximity analysis with logistic regression	Asthma and respiratory illness-related hospital admissions	Mobile source air pollution: residential proximity (within 150 m) to high traffic flow (1,000 vehicles per hour at peak) and volume (from centroid of postal code)	Children 5-17 years old (asthma cases: n=1,380, resp illness cases: n=2,131, controls: n=5,703)	based on centroid of residential post code	No difference in odds of asthma or respiratory illness hospital admissions for those living within 150m from main roads (for distance as dichatomous and continuous variable)	No evidence of an association between living near high traffic flow and volume roads and asthma or respiratory illness admissions. Exposure measurement error (distance from road crude approximation of exposure to traffic pollution), potential confounding.
Air pollution	Respirato ry	Wjst et al., 1993, Germany	Small area study (Munich, Germany), cross-sectional with logistic regression	Pulmonary function (via pulmonary function test) and respiratory symptoms (self- reported by parent)	Mobile source air pollution: Residence in school districts with high traffic flow (district represented by top value of census traffic count)	Children in 4th grade, age 9-11 years (n=4,678)		Odds of reduced peak flow (pulmonary function) and recurrent dyspnoea sign. associated to residing in school zone with greater traffic counts (top third) versus bottom third zone (no diff between middle third and bottom): peak flow %change=2.18% (3.3,1.04%, p<0.001); recurrent dyspnoea OR=1.40 (1.03,1.91, p<0.05); also odds of some respiratory symptoms sign. associated with increase of 25,000 cars: recurrent wheezing OR=1.08 (1.01,1.16, p<0.033); recurrent dyspnoea OR=1.10 (1.00,1.20, p<0.039), but not asthma	Evidence of reduced pulmonary function (and increase of some respiratory symptoms) with increased traffic counts. Possible reporting bias, exposure measurement area (school zone as proxy for individual exposure).

Appendix B – Figures

Figure 1	Spatial Coincidence Approach: Selection of Host Census Units
Figure 2	Circular Buffers of Uniform Radius around Facilities of Concern
Figure 3	Cumulative Distribution Functions for Hazard Proximity: Comparing Racial Characteristics of the Population
Figure 4	A Typical Plume Footprint for a Hypothetical Chlorine Release Scenario using the ALOHA Model
Figure 5	Selection of Census Units with a Circular Buffer using the Polygon Containment Method
Figure 6	Selection of Census Units with a Circular Buffer using the Centroid Containment Method
Figure 7	Selection of Census Units with a Circular Buffer using the Buffer Containment or Areal Apportionment Method
Figure 8	Cadastral Dasymetric Mapping: Estimating Households within a Circular Buffer using Land Parcels
Figure 9	Using Geographically Weighted Regression to Explore Relationships between Cancer Risk from Other (Minor) Point Sources of Air Toxics and Various Explanatory Variables in Florida: Distribution of Local t-statistic by Census Tract

Hazardous Facility
Host Census Tract
Non-Host Tract

1 2
Miles

Figure 1. Spatial Coincidence Approach: Selection of Host Census Units

Figure 2. Circular Buffers of Uniform Radius around Facilities of Concern

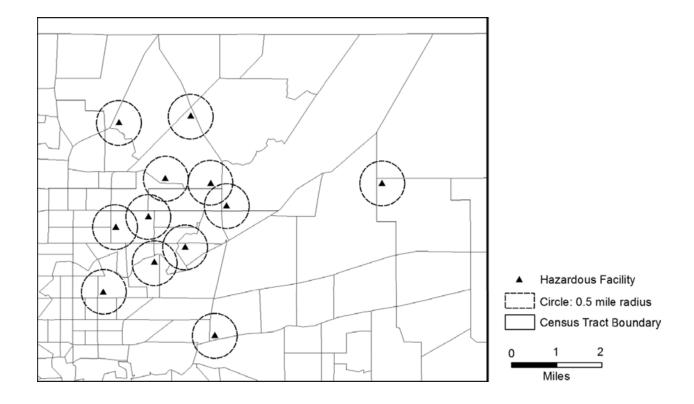


Figure 3. Cumulative Distribution Functions for Hazard Proximity: Comparing Racial Characteristics of the Population

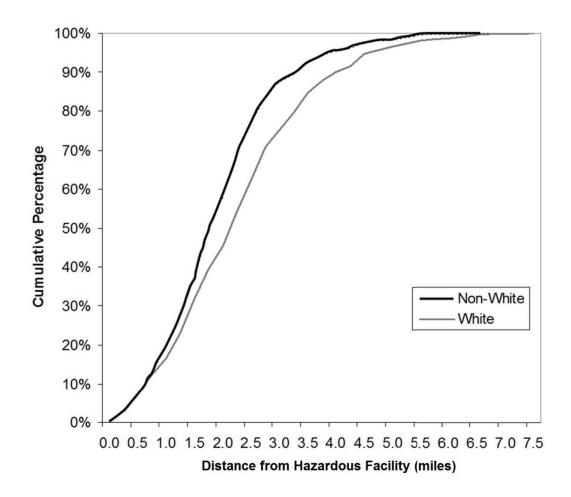


Figure 4. A Typical Plume Footprint for a Hypothetical Chlorine Release Scenario using the ALOHA Model

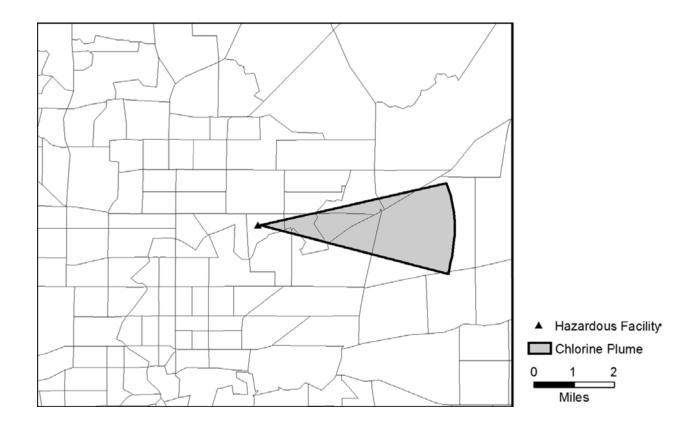


Figure 5. Selection of Census Units with a Circular Buffer using the Polygon Containment Method

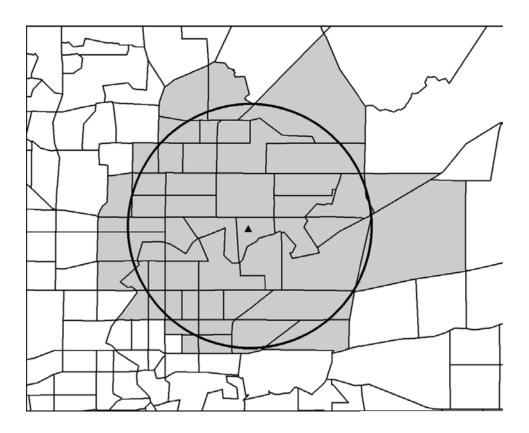


Figure 6. Selection of Census Units within a Circular Buffer using the Centroid Containment Method

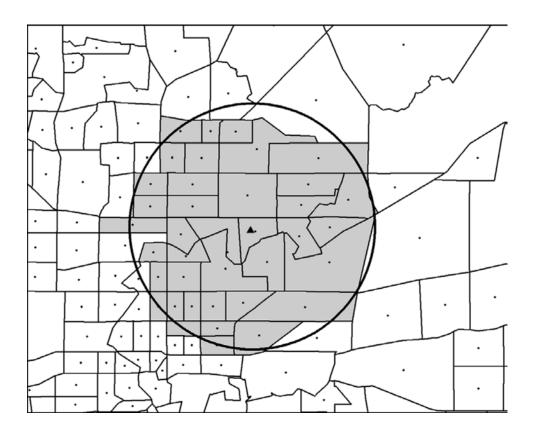


Figure 7. Selection of Census Units using a Circular Buffer using the Buffer Containment or Areal Apportionment Method

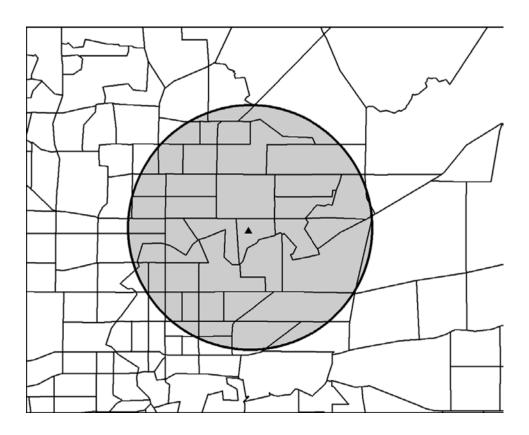


Figure 8. Cadastral Dasymetric Mapping: Estimating Households within a Circular Buffer using Land Parcels

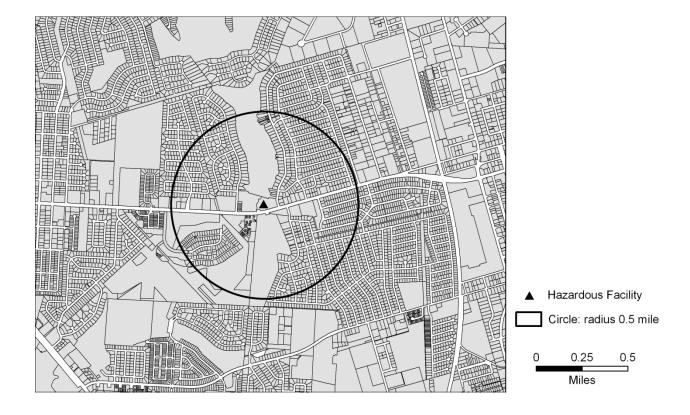
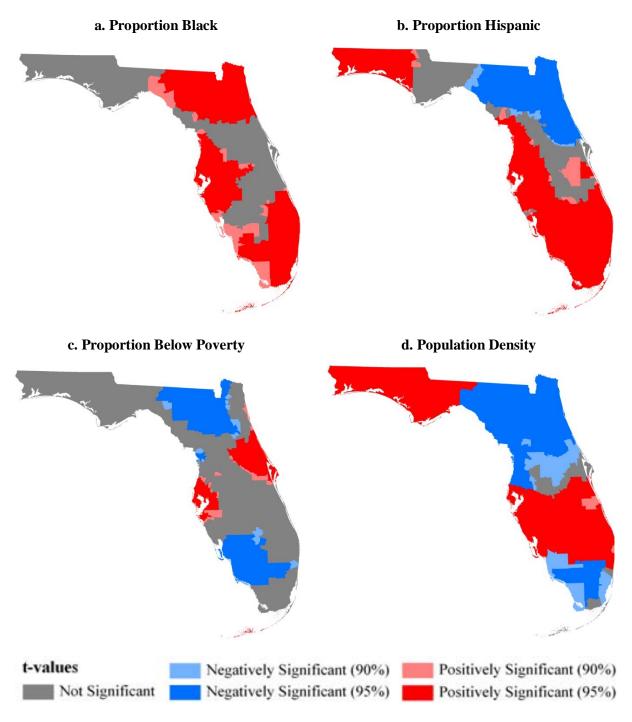


Figure 9. Using Geographically Weighted Regression to Explore Relationships between Cancer Risk from Other (Minor) Point Sources of Air Toxics and Various Explanatory Variables in Florida: Distribution of Local t-statistic by Census Tract



Source: Gilbert, 2009.