Environmental exposures and hematologic cancers in Upstate New York

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Abstract:

Exposure to benzene can lead to the development of blood and bone marrow (i.e. hematologic) cancers in humans, including Leukemia. Physical science research on the deleterious effects of benzene exposure is substantial. Social science research suggests that some types of benzene exposure are inequitably distributed across the population. Using spatial analytic techniques and proprietary data from the New York State Department of Health SPARCS system, data on hazardous waste sites from the EPA, pesticide use data from the New York Department of Environmental Conservation and county level smoking rates, I examine the association between spatial exposure to benzene and age-, race-, and sex-specific zip-code level counts of leukemia in upstate New York. Preliminary findings suggest that for all standardized groups, the amount of pesticides per square mile is positively associated with higher counts of Leukemia. Other significant findings are reported within specific segments of the population.

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Background:
The causes for hematological cancers vary by the specific type of malignancy. Among them, are pesticide/herbicide exposure, smoking, dietary and lifestyle risk factors (e.g. smoking, hair color products), anti-cancer drugs (e.g. types of chemo and radiation), organ transplant/blood transfusions, genetic disorders (e.g. down syndrome), certain types of infections, ultra-violate exposure, genetic susceptibility, and immunodeficient disorders (e.g. HIV) (Rodriguez-Abreu et al 2007). Benzene exposure has consistently been used in empirical studies to understand the development of hematologic cancers and charge the chemical to be the leading risk factor for the development of hematologic cancers. Although the Agency for Toxic Substances & Disease Registry (ATSDR) outlines blood and the bone marrow organs that are generally affected, they state explicitly that "exposure to benzene has been associated with the development of a particular type of leukemia called acute myeloid leukemia (AML)" (Public Health, 2007).

Exposure to benzene can occur via air, water, and food, though the majority of studies have focused on air exposure. Benzene is a chemical that can be produced by natural events, but through this means only contributes a minimal amount of benzene to the environment. These natural events include forest fires, volcanic eruptions, and fumes from oil (Public Health 2007). Again, exposure to benzene via natural events is minimal. The Agency for Toxic Substances and Disease Registry states that approximately 50% of human exposure to benzene occurs through cigarette smoke, while 20% is from auto exhaust and industrial emissions (ToxProfile 2007). Other studies have verified this claim (McHale et al 2012), including a large study conducted by the EPA in the 1980's that found that more than half of American's exposure to benzene was experienced by smokers (Wallace 1996). Cigarette smoke, as well as vehicle exhaust are the primary sources of benzene exposure outside of occupational exposure (McHale et al 2012).

Beyond cigarette smoke, other high risk areas of exposure include exhaust and pesticides. In one Italian study, residence near road traffic emissions increased the risk of leukemia via benzene (Crosignani et al 2004). An American study found that those in the 90th percentile of traffic densities had rate ratios that were higher for leukemia than for the control group (Reynolds 2002). These impacts are not limited to traditional road traffic. A study conducted in the late 1990s in Great Britain found higher levels of leukemias and other tumors in residents near airfields, railways, motorways, and harbors (Knox 1997). Those working at or living near gas stations also present higher risk estimates for leukemia (Harrison et al 1999, ToxProfile 2007). With the amount of traffic and idling that occurs at gas stations, it makes sense that there would be a higher density of exhaust to which one could be exposed. A 2012 study also found that those with garages attached to their homes are also at risk of higher exposure levels via exhaust (Snyder 2012). Pesticides are also a concern for exposure to benzene. A study out of Texas using the state's Cancer Registry found that residence near crop land was associated with higher rates of some types of lymphoma. Additional sources of exposure that have been discussed in the literature and cited by the Center for Disease Control include gasoline-powered machines, warming cars in garages, showering in gasoline-contaminated water (Wallace 1996), manufacturing plants, and hazardous waste sites (ToxProfile 2007).

Though not seen as the primary source of benzene exposure, the ATSDR reports that hazardous waste sites are also a source of concern as many sites release and/or dispose of benzene (Public health 2007). Under the auspices of the Environmental Protection Agency (EPA), many different

types of facilities pose threats to nearby residents via benzene exposure. In a public health statement on Benzene from the ATSDR, as of 2007, of the 1,684 facilities on the National Priority List (NPL) under the superfund program, at least 1,000 of them were releasing benzene into the environment (Public Health 2007). And as the Boberg et al (2011) article found, this type of environmental exposure may result in negative health consequences like hematologic cancers for nearby residents.

Up to this point we have reviewed the literature that speaks to the wide assortment of ways through which one can be exposed to benzene. Now, we wish to introduce a demographic perspective of what is thus far known about spatial inequalities of hazardous waste sites in the US. This literature will set the stage for our accounting for community level characteristics in the analysis section.

In 1994, Anderton et al published a piece called Environmental Equity: The Demographics of Dumping. The 80s and 90s had been a period of realization of the environmental inequalities that might be associated with hazardous waste and dumping. This was also the era when environmental racism and environmental justice literature started gaining ground. But what Anderton et al (1994) found was an underwhelming amount of evidence for environmental inequity. In fact, in their study using census tract level data and facilities that treat, store, and dispose of hazardous wastes (TSDFs), they found no statistical significant evidence to suggest that racial or ethnic differences could account for the location of TSDFs. Hispanics were the only group they found to have slightly higher exposure to dumping. Six years later, Davidson and Anderton (2000) took another stab at this analysis, but broadened it to include all facilities that are governed by the Resource Conservation and Recovery Act (RCRA). They found that although severe environmental inequalities on a national scale do not necessarily exist, RCRA facilities are more likely to be found in working class communities, and in communities with higher proportions of minorities.

There are sound empirical findings that smoking, exhaust emissions, pesticide use, and even hazardous waste sites are to blame for the development of hematologic cancers. And while we attempt to provide a sociological perspective by contextualizing the methodology with community level characteristics and more precise measurement of benzene exposure, it is also our responsibility to ask if the effects of human action are disproportionately felt by those in different positions. While cultural anecdotes now tote the “cancer knows no race or class”, the demographic disparities across space may present a different picture. To address this issue, the present paper examines the zip-code level rates of leukemia. While the full paper includes control variables (e.g. percent poverty, percent college educated, percent black, and percent non-hispanic white) for both Black and White individuals over the age of 39, this abstract highlights the main effects of the relationship between leukemia and variables that measure exposure to benzene.

Data:

This paper uses rich data from the New York Statewide Planning and Research Cooperative System (SPARCS) that include all diagnoses of hematologic cancers in Upstate New York State health facilities from 2000 through 2011. In addition to reporting basic demographic data (e.g. race, age, gender), each patient is linked to a home zip code. Using this information, counts for Leukemia diagnoses were calculated for each standardized group (i.e. gender, race, and age group) of individuals in each zip code. We excluded zip codes in New York City, Long Island, and Staten

Island. This exclusion has been followed in other studies (Boberg et al 2011) as individuals in zip codes in these three regions are exposed to significantly more exhaust.

The independent variables for this analysis include: percent of the zip code that is urban, pounds of benzene applied via pesticide application per square mile for each county, a dummy variable for if the zip code has any industrial disposal of benzene, and the county level estimate of percent of population that smokes. The percent of the zip code that is urban was obtained from the 2000 decennial census. The zip codes where industrial benzene was disposed was obtained from the EPA Toxic Release Inventory website. The pounds of benzene applied via pesticide application were obtained from the New York State Department of Environmental Conservation Pesticide Reporting Law[1]. And smoking rates for each county were obtained from County Health Rankings[2]. Control variables include percent female, percent African American, percent non-Hispanic white, percent poverty, and percent with a college degree. All of the control variables are reported at the zip code level and were obtained from the decennial census for 2000.

Methods:
This analysis uses multilevel (zip codes and counties) Poisson regressions for count data. I employ Exploratory Spatial Data Analysis (ESDA), which is an advancement of traditional Exploratory Data Analysis (EDA). In ESDA, I use Moran statistics to detect if spatial patterning is occurring in particular variables of interest (Anselin et al 2006). If spatial autocorrelation is detected these influences will need to be accounted for in any models conducted. Even if spatial autocorrelation is not detected, environmental exposures to benzene (percent urban, pesticide applications, and benzene disposal) are spatially weighted to account for their ability to have spillover effects. GeoDa software is used to conduct the ESDA.

Next, I use a Poisson regression for count data and for a multilevel model (zip codes and counties) using PROC GLIMMIX in SAS software. These models examine if the four types of exposure are significant predictors of leukemia diagnoses even when controlling for community level characteristics. By using these methods, we contribute a sociological perspective to the discussion of the relationship between the development of leukemia and exposure to benzene. Not only do we control for key individual level and community level variables, but we will also account for density of exposure rather than simply the presence of exposure. Only the models without the control variables are reported in this abstract.

Results:
The regressions reported below are for populations of non-Hispanic white men and women over the age of thirty-nine. The variables that are proceeded by a "W" indicate that they are the spatial weight of the proceeding variable. The values reported in the table are the coefficients for each of the variables.

The results indicate that the rate of pesticides applied at the county level have a significant positive effect on the incidence of Leukemia at the zip code level. This effect is consistent across sex and age groups. For the oldest age group of women, there is a significant and positive effect on leukemia counts. And for women between the ages of 40 and 59, there is a significant positive

[1] http://www.dec.ny.gov/chemical/97575.html
[2] http://www.countyhealthrankings.org/our-approach/health-factors/tobacco-use

effect between the county-level percent of smokers and the rate of leukemia. For several of the spatially weighted variables (first order queen contingency – i.e. an average of all adjacent counties), however, there was a negative effect on incidence of Leukemia. For example, for populations of men ages 60 and older, there is a significant or nearly significant negative effect of surrounding zip codes with benzene disposal on the incidence of leukemia. In addition, for urbanicity of surrounding zip codes has a negative effect on the number of leukemia cases in a zip code.

Table 1: Spatially Weighted Multilevel Poisson Regression Models for variables associated with rates of Leukemia in New York State^

	Male			Female		
	(40-59)	**(60-79)**	**(80+)**	**(40-59)**	**(60-79)**	**(80+)**
Intercept	-7.3080***	-7.1152***	-6.6292***	-9.3597***	-6.8505***	-6.7641***
W Dependent Variable	-0.2021	-0.06562	-0.1711	-0.1094	-0.1024	0.121
Applied Pesticides	1.448E-7***	1.903E-7 ***	2.999E-7***	1.136E-7***	9.756E-8***	1.253E-7***
W Applied Pesticides	-0.00009	0.000055	-0.00004	-0.00010	0.000072	0.000187
Benzene Disposal (dummy)	0.00865	0.04334	-0.2013	-0.4215	-0.01595	0.1761
W Benzene Disposal (dummy)	0.5569	-0.8625+	-1.6589*	0.7968	0.551	0.2884
% Smokers	-0.02807	0.0207	0.02624	0.04849*	-0.00624	-0.00250
% Urban	0.000346	0.002826	0.004399	-0.00007	0.001771	0.006609*
W % Urban	-0.00169	-0.00183	-0.00192	0.00362	-0.00258	-0.00810*
-2 Res Log Pseudo-Likelihood	6071.57	5147.7	7014.28	6098.65	5644.27	6936.38
Convergency Criterion satisfied (PCONV=1.11022E-8)	Yes	Yes	Yes	Yes	Yes	Yes

*+ approaching sig., * sig at .05 level, ** sig at .01 level, *** sig at the .001 level*

^ Excluding New York City, Staten Island, and Long Island

Conclusion:

This paper contributes to growing evidence of a relationship between exposure to benzene and the development of hematologic cancers, specifically Leukemia. My findings contribute to the knowledge of this field by examining the relationship between environmental exposures and race, gender and age standardized zip code level counts of leukemia, while controlling for demographic level characteristics. The initial findings do show strong support for the positive relationship between pesticide application and counts of leukemia. Complete findings for all standardized groups using the full models will be presented in the final paper and at the conference. The findings of this paper contribute to conversations about the role of spatial and demographic techniques in understanding the variability in hematologic cancer prevalence.

Work Cited:

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological profile for Benzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Anderton, D., Anderson, A., Oakes, J. M., Fraser, M. (1994) Environmental Equity: The Demographics of Dumping. *Demography*, 31(2): 229-248.

Atlas, M. (2002). Few and Far Betwee? An Environmental Equity Analysis of the Geographic Distribution of Hazardous Waste Generation. *Social Science Quarterly*, 83(1): 365-378.

Boberg, E., Lessner, L., Carpenter, DO. (2011) The role of residence near hazardous waste sites containing benzene in the development of hematologic cancers in upstate New York. International Journal of Occupational Medicine and Environmental Health, 24: 327-338.

Crosignani, P., Tittarelli, A., Borgini, A., Codazzi, T., Rovelli, A., Porro, E., Contiero, P., Bianchi, N., Tagliabue, G., Fissi, R., Rossitto, F., Berrino, F. (2004). Childhood leukemia and road traffic: A population-based case-control study. *International Journal of Cancer*, 108 (4): 596-599.

Davidson, P., Anderton, D. L. (2000) Demographics of Dumping II: A national environmental equity survey and the distribution of hazardous materials handlers. *Demography*, 37(4):461-466.

Knox, A., Gilman, E. (1997). Hazard Proximities of childhood cancers in Great Britain from 1953-1980. *Journal of Epidemiological Community Health*, 51: 151-159.

Harrison, R., Leung, P., Sommervaille, L., Gilman, E. (1999) Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations. *Occupational & Environmental Medicine*, 56: 774-780.

McHale, C. M., Zhang, L., Smith, M. T. (2012) Current understanding of the mechanism of benzene-induced leukemia in humans; Implications for risk assessment. *Carcinogenesis*, 33(2): 240-252.

Public Health Statement for Benzene. Aug 2007. Agency for Toxic Substances & Disease Registry. http://www.atsdr.cdc.gov/phs/phs.asp?id=37&tid=14

Rodrigueq-Abreu, D., Bordoni, A., Zucca, E. (2007) Epidemiology of hematological malignancies. *Annals of Oncology*, 18(Supplement 1): i3-i8.

Snyder, R. (2012) Leukemia and Benzene. *International Journal of Environmental Research and Public Health*, 9: 2875-2893.

Tox Profile. Public Health Statement for Benzene. Aug 2007. Agency for Toxic Substances & Disease Registry. http://www.atsdr.cdc.gov/phs

Toxic Release Inventory Program. Environmental Protection Agency. Accessed on 17 December 2013. http://www2.epa.gov/toxics-release-inventory-tri-program/learn-about-toxics-release-inventory US Environmental Protection Agency. Toxic Release Inventory, TRI Explorer. http://www.epa.gov/triexplorer

Wallace, L. (1996) Environmental Exposure to Benzene: An Update. *Environmental Health Perspectives*, 104 (6): 1129-1136.