

Evaluating Cumulative Risk Assessment for Environmental Justice: A Community Case Study

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A key feature of cumulative risk assessment (CRA) is the ability to estimate differential health risks from environmental exposures within populations. Identifying populations at increased risk from environmental exposures is the first step toward mitigating such risks as required by the fair treatment mandate of environmental justice. CRA methods remain under development except for a limited application in pesticide regulations. The goals of this research were to advance CRA methods and to test their application in a community case study. We compared cumulative risk and health assessments for South and Southwest Philadelphia communities. The analysis found positive correlations between cumulative risk and mortality measurements for total mortality in Whites and non-Whites when we conducted the risk assessment using a multi-end point toxicological database developed for this project. Cumulative risk scores correlated positively with cause-specific mortality in non-Whites. Statistically significant increases in total and respiratory mortality rates were associated with incremental increases in the hazard ratio cumulative risk scores, with ranges of 2–6% for total and 8–23% for respiratory. Regression analyses controlled for percent non-White population and per capita income, indicating that risk scores represent an environmental effect on health independent of race and income. This case study demonstrated the successful application of CRA at the community level. CRA adds a health dimension to pollutant concentrations to produce a more comprehensive understanding of environmental inequities that can inform decision making. CRA is a viable tool to identify high-risk areas and to guide surveillance, research, or interventions. *Key words:* community health, cumulative risk assessment, environmental justice. *Environ Health Perspect* 110(suppl 2):203–209 (2002).

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Cumulative risk assessment (CRA) is the U.S. Environmental Protection Agency's (U.S. EPA) most recent conceptual innovation to its primary decision-making tool, quantitative risk assessment. CRA represents a transition in methods from a focus on a single effect of a single chemical in a particular medium to the multiple ecological and human health effects of multiple exposures that may accumulate over time from multiple sources, pathways, and routes (1,2). The U.S. EPA concepts of CRA are similar to the framework of the multiple determinants of health currently in use in public health research and practice (3,4). The development of CRA offers an opportunity to address the lack of coordination between public and environmental health (5).

In addition, the U.S. EPA plans to apply CRA for community-based risk assessment and management. One key feature of CRA is the ability to estimate differential health risks from environmental exposures within populations. The principle of fair treatment in the U.S. EPA environmental justice policy mandates that no group experience a disproportionate burden of adverse environmental consequences such as health risks (6). The U.S. EPA envisions CRA as a tool to identify populations bearing a disproportionate health risk burden. The methods remain

under development, however, except for a limited application in pesticide regulations. This work examined CRA as a tool for health risk assessment to inform environmental justice concerns in communities. It focused on human rather than ecological health risk.

The aims of this research were to contribute to the development of methods and community applications for CRA. We achieved these aims by developing a CRA approach that included a broad range of publicly available toxicological information and testing the approach in a community case study (7). The case study presented below entailed the comparison of mortality measures of community health status to the potential cumulative health risks of inhalation of the Clean Air Act hazardous air pollutants (HAPs) (8).

Background

Cumulative Risk Assessment

The U.S. EPA approach to CRA acknowledges that there are contributors to human and ecological health risk beyond chemical exposure, and that the health of communities is influenced by economic, behavioral, social, and psychological stresses. Within the realm of chemical exposure, the cumulative

risk approach begins to tackle the reality of multiple exposures by all pathways, routes, and media and their contributions to health end points other than cancer. It considers the multiple contributors to health and is linked to the idea of differential susceptibility within populations or communities at risk. CRAs characterize those subpopulations that are susceptible to environmental exposures because of age, gender, disease history, ethnicity, or other characteristics. Holistic reduction of risk is the ultimate goal. However, existing single-substance techniques continue to be used while methods of CRA are developed (1).

Current Status of CRA

The strict and brief implementation timeline of the Food Quality Protection Act of 1996 (9) continues to drive progress in the development and application of cumulative risk concepts in the U.S. EPA pesticide programs. The other area of cumulative risk research comprises the public health-oriented community approaches that have been and are being piloted at the level of the U.S. EPA regional offices (10–12). The researchers using this approach are grappling with the multifactorial nature of health risk embodied in the cumulative risk definition. The multiple stresses of everyday life may include pesticides on foods, pollutants in the air, trihalomethanes in water, leaded paint in older houses, lack of health insurance, and occupational and behavioral risks.

The status of CRA within the agency as a whole remains uncertain. No legislation mandates its adoption. It appears that CRA will continue its slow, sporadic diffusion through U.S. EPA programs.

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Cumulative Risk and Environmental Justice

We undertook all three community-based studies mentioned above in response to concerns of environmental injustice. Therefore, the brief history of cumulative risk is closely tied to community environmental justice concerns. The U.S. EPA *Guidance on Cumulative Risk Assessment—Planning and Scoping* (1) establishes a framework to recognize and assess environmental risks that differentially manifest because of gender, ethnicity, geographic origin, age, or other characteristic. CRA is a necessary precursor to an environmental justice strategy, and lack of cumulative risk methods puts us at a loss to assess and ultimately manage disparities related to the environment.

Why South and Southwest Philadelphia?

The South and Southwest Philadelphia locations we chose for this study have complex social, physical, and chemical environments. They are places where cumulative exposures to environmental, economic, and social stresses occur. The residents' growing concerns about environment and health in their communities led to a descriptive research project conducted at the Johns Hopkins School of Hygiene and Public Health in cooperation with U.S. EPA Region III (10). Regional pilot studies are one avenue of policy development pursued by the U.S. EPA. We hope that building upon the regional study will facilitate consideration of these findings as CRA methods progress.

Materials and Methods

Our study began with descriptive characterizations of the community health (mortality) status and the cumulative risk scores for the study area neighborhoods. The analytical phase also entailed two parts, nonparametric rank order correlation at the neighborhood level and Poisson regression at the census-tract level.

Sources of Data

We obtained mortality data by census tract from the city of Philadelphia. We used deaths for 1990 ($n = 3,151$) and for 1988–1992 ($n = 16,168$) to calculate the mortality rates. The additional information used for rate calculations included the 1990 census and the U.S. 1940 and 2000 standard populations for age adjustment (13).

The air pollutant data we used as a proxy for human exposure came from the U.S. EPA Cumulative Exposure Project (CEP) (14). The CEP developed a long-term Gaussian air dispersion model to produce annual average concentration estimates of the Clean Air Act HAPs for all census tracts in the continental United States for 1990. The CEP modeled

148 of the 188 HAPs because of availability of information in the source databases (14,15).

As described below, we conducted the cumulative risk scoring for this project using two toxicological databases, a multi-end point toxicological database developed specifically for this project (7) and the U.S. EPA Cumulative Exposure Toxicity Database (CETDB) (16). The former contains no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) for the HAPs and reflects the full range of the public, peer-reviewed toxicological literature through 1999.

Assessment of Community Health Status (Mortality)

We included four cause-of-death categories reflecting chronic disease processes in this analysis: all-cause mortality, total mortality excluding external causes, heart and vascular diseases (cardiovascular, cerebrovascular, and other arterial diseases), and respiratory diseases. These end points are consistent with previous studies of air pollution and mortality (17). The general community health characterization looked at all-cause mortality only. We excluded external causes of death, such as car crashes from the correlation and regression analyses because pollutant exposures are not thought to be associated with them.

We calculated directly age-adjusted cause-specific mortality and years potential life lost (YPLL) rates per 100,000 for the study area census tracts and neighborhoods for 1990 and 1988–1992. We based YPLL rates on the age 65 set point to facilitate comparisons with national data for the selected time periods (18,19). YPLL for the nation was not reported as White/non-White in 1990 (20). Therefore, we calculated national

YPLL by race in a manner consistent with the methods applied to the Philadelphia data.

Cumulative Risk Scoring

We conducted a noncancer CRA with census-tract-level estimates of the Clean Air Act HAPs and toxicological reference data using established procedures. We used the U.S. EPA database of reference concentrations and equivalents to calculate the hazard index and calculated the hazard ratio on the basis of a toxicological reference database we developed specifically for this project. Background and details on this approach have been published elsewhere (7,21,22).

Statistical Analyses

We assessed the hypothesis that cumulative risk measures will positively correlate with community health measures in two ways. Spearman's rank order correlation coefficients (CC) measured the alignment between high mortality and high cumulative risk scores at the neighborhood level. Using Poisson regression, we analyzed the associations between mortality measurements and cumulative risk scores at the census-tract level. Of the 60 residential census tracts in the South and Southwest Philadelphia study area, 53 had complete information on the outcomes and covariates of interest. Table 1 contains a description of the variables included in the regression analysis.

Results

We present the findings of this research in four sections. Neighborhood-level descriptions of community health and cumulative risk status are followed by the nonparametric and parametric analyses of relationships between the health and cumulative risk

Table 1. Description of regression variables.

Variable name	Number of observations	Mean	Range
1990			
Total YPLL rate	53	6,711.51	501–15,341
Total mortality rate	53	1,158.81	88–2,242
Heart/vascular YPLL rate	53	1,312.45	0–4,131
Heart/vascular mortality rate	53	469.45	0–1,007
Respiratory YPLL rate	53	552.26	0–3,491
Respiratory mortality rate	53	103.74	0–860
1988–1992			
Total YPLL rate	53	34,322.02	5,607–80,528
Total mortality rate	53	6,097.77	466–13,889
Heart/vascular YPLL rate	53	7,626.09	0–17,039
Heart/vascular mortality rate	53	2,523.17	0–5,634
Respiratory YPLL rate	53	1,174.00	0–4,706
Respiratory mortality rate	53	434.30	0–1,215
Total hazard index	60	55.86	27.2–130.46
Total hazard ratio	60	11.03	6.13–21.68
Heart and vascular hazard index	60	0.30	0.17–0.53
Heart and vascular hazard ratio	60	0.01	0.007–0.019
Respiratory hazard index	60	53.98	25.46–124.63
Respiratory hazard ratio	60	10.96	6.09–21.59
Per capita income	53	11,027.53	3,014–25,598
Percent non-White population	53	0.48	0–1

measurements at the neighborhood and census-tract levels, respectively.

Neighborhood Health Assessment: White and Non-White

Tables 2 and 3 present the 1990 neighborhood- and race-specific all-cause mortality and YPLL rates as well as rate ratios for both measurements in comparison with the U.S. national rates for the total population. The rates are age adjusted to the 1940 standard. As reported in the original study, the South and Southwest Philadelphia area as a whole experienced a 54% higher all-cause mortality rate than the nation for the period 1974–1993 (10). In the White population in 1990, the increased mortality rates range from 5% to nearly double the national average rate. In the non-White population in 1990, the increased mortality rates range from 42% to more than double the national average.

Table 2. Comparison of White residents' all-cause mortality and YPLL rates to the United States, 1990 (rates per 100,000 population, age-adjusted to the 1940 standard).

Neighborhood ^a	Mortality rate	Mortality rate ratio (neighborhood/U.S.)	YPLL rate	YPLL rate ratio (neighborhood/U.S.)
Eastwick Elmwood	632.91	1.22	6,351.55	1.24
Paschall Kingsessing	891.78	1.71	10,105.14	1.97
Schuylkill Point Breeze	1,023.28	1.98	12,846.86	2.50
Southwark Bella Vista	626.03	1.20	5,472.80	1.07
Pennsport Queens Village	736.62	1.42	9,842.02	1.92
South Broad Girard Estates	569.40	1.09	5,167.90	1.01
Snyder Whitman	544.57	1.05	6,880.77	1.34
Grays Ferry Passyunk	545.63	1.05	5,881.32	1.15
United States	520.20	5,137.08		

^aAll neighborhoods are in Philadelphia, Pennsylvania.

Table 3. Comparison of non-White residents' all-cause mortality and YPLL rates to the United States, 1990 (rates per 100,000 population, age adjusted to the 1940 standard).

Neighborhood ^a	Mortality rate	Mortality rate ratio (neighborhood/U.S.)	YPLL rate	YPLL rate ratio (neighborhood/U.S.)
Eastwick Elmwood	736.10	1.42	8,173.05	1.59
Paschall Kingsessing	934.58	1.80	12,710.20	2.47
Schuylkill Point Breeze	966.17	1.91	14,173.56	2.76
Southwark Bella Vista	757.09	1.46	11,033.40	2.15
Pennsport Queens Village	1,065.54	2.05	13,652.69	2.66
South Broad Girard Estates	979.09	1.88	10,967.29	2.13
Snyder Whitman	805.55	1.55	14,518.34	2.82
Grays Ferry Passyunk	883.56	1.70	14,202.08	2.76
United States	520.20	5,137.08		

^aAll neighborhoods are in Philadelphia, Pennsylvania.

Table 4. Age-adjusted (1940) all-cause mortality and YPLL rate ratios by neighborhood, non-White/White, 1990.

Neighborhood ^a	Age-adjusted rate ratio mortality	Age-adjusted rate ratio YPLL
Eastwick Elmwood	1.16	1.29
Paschall Kingsessing	1.05	1.26
Schuylkill Point Breeze	0.97	1.10
Southwark Bella Vista	1.21	2.01
Pennsport Queens Village	1.45	1.39
South Broad Girard Estates	1.72	2.12
Snyder Whitman	1.48	2.11
Grays Ferry Passyunk	1.62	2.41

^aAll neighborhoods are in Philadelphia, Pennsylvania.

The YPLL rates in the neighborhoods exceed the national YPLL rates. In the White population, the excess ranges from a slight 1% greater to more than double the rates for the nation. The YPLL for the non-White populations in the neighborhoods exceeds the national rate, ranging from 59% higher to nearly triple the national average YPLL rate.

Within each population group and mortality measurement, we observed a considerable range in mortality experience. The White population in Schuylkill Point Breeze has mortality and YPLL rates about double those of the White residents of Snyder Whitman. The range of mortality and YPLL rates for non-Whites in the neighborhoods was not quite as large.

Disparities in Health

Table 4 presents the rate ratios comparing the non-White and White all-cause mortality and

YPLL rates for 1990. These rate ratios summarize and quantify the differences in mortality experienced by the two groups. With the exception of Schuylkill Point Breeze, where the non-White and White mortality rates were nearly the same (White mortality rates were slightly higher), the non-White population experienced higher mortality rates than the White population in the South and Southwest Philadelphia communities. Considering YPLL, which emphasizes mortality in younger age groups, the non-White populations' rates were consistently higher.

A sensitivity analysis assessed the impact of the new 2000 age adjustment standard on the community health characterization and subsequent hypothesis testing. The 2000 standard resulted in increased age-adjusted rates and slightly decreased non-White/White rate ratios. However, the year 2000 standardized rates reflected the higher mortality experienced by the non-White population in South and Southwest Philadelphia and did not substantially affect the neighborhood rankings based on the mortality indicators. The remaining analysis we conducted using rates adjusted to the 2000 population standard.

Neighborhood Cumulative Risk Characterization: Comparison of Risk Scores

We used both the U.S. EPA CETDB and the multi-end point NOAEL/LOAEL databases with the HAPs concentrations to calculate a total risk score for each neighborhood (Table 5). The hazard index calculated with the current CETDB yields a higher score because of the use of varying uncertainty and modifying factors in the calculation of reference concentrations in the CETDB.

The "adjusted" hazard ratio presented in the fourth column of Table 5 we calculated by applying the modal uncertainty and modifying factor (300) from approximately 40 HAPs with reference values in the U.S. EPA Integrated Risk Information System (IRIS) database to the total hazard ratios in the third column of the table (7,23). This

Table 5. Total risk scores for the HAPs by neighborhood and neighborhood/U.S. ratio.

Neighborhood ^a	Total hazard index ^b (neighborhood/U.S.)	Total hazard ratio ^c (neighborhood/U.S.)	Adjusted hazard ratio
Eastwick Elmwood	70.62 (5.10)	10.43 (3.82)	3,129
Paschall Kingsessing	44.93 (3.24)	10.48 (3.84)	3,144
Schuylkill Point Breeze	48.26 (3.48)	13.05 (4.78)	3,915
Southwark Bella Vista	45.36 (3.28)	11.57 (4.24)	3,474
Pennsport Queens Village	57.17 (4.13)	12.47 (4.57)	3,741
South Broad Girard Estates	54.13 (3.91)	11.76 (4.31)	3,528
Snyder Whitman	54.56 (3.94)	11.66 (4.27)	3,498
Grays Ferry Passyunk	67.59 (4.88)	9.38 (3.44)	2,814
United States	13.85	2.73	819

^aAll neighborhoods are in Philadelphia, Pennsylvania. ^bHazard index is based on the CETDB. ^cHazard ratio is based on the NOAEL/LOAEL database.

adjustment equilibrates the hazard index and hazard ratio formulas.

These data describe potential health risks in the neighborhoods not unlike that of the mortality analysis presented above. Regardless of database used, the neighborhoods' risk scores were 3–5 times the national average. Figures 1 and 2 illustrate the census-tract-level distributions of the total mortality excluding external causes and the total hazard ratio.

Correlation Analysis

Table 6 presents the CCs between rank ordering of the neighborhoods by the two CRA approaches and cause-specific mortality measurements (88–92) by race. A positive CC quantifies the similarities between the neighborhood lists rank ordered by cause-specific mortality or YPLL rate and each cumulative risk score. A CC of or near zero suggests no similarity between the rank-ordered lists. A negative CC reflects dissimilarity in rank. For example, the White population of Grays Ferry Passyunk ranks first in a CETDB CRA for heart disease but eighth for heart disease YPLL.

Regardless of race group or type of mortality measurement, the hazard ratio risk score based on the NOAEL/LOAEL database correlated positively with total mortality (excluding external causes). The CCs were consistently higher when we compared CRA based on the NOAEL/LOAEL database with cause- and race-specific mortality in the neighborhoods. This holds for both mortality and YPLL.

Regardless of database, with one exception, the CCs were highest between CRA scores and mortality in the non-White population. Considering age-adjusted mortality rates for total mortality (excluding external causes), the White and non-White CCs using the hazard ratio risk score from the NOAEL/LOAEL database were nearly identical (Whites, 0.33; non-Whites, 0.31).

The highest CC found, 0.81, was for respiratory disease YPLL in the non-White population when compared with the hazard ratio risk score from NOAEL/LOAEL CRA. Although this is consistent with the correlation results overall, the finding should be interpreted with some caution because respiratory disease was the most rare and statistically variable of the end points analyzed.

In this small sample, a rank order CC of 0.64 or higher would be needed to achieve statistical significance at the $p = 0.05$ level (for a one-sided test). Given the hypothesis that each risk score should be positively correlated with mortality, the only finding that achieves statistical significance is the 0.81 CC for respiratory YPLL rate and respiratory hazard ratio in non-Whites. Considering another example from these data, a sample

size of 30 neighborhoods would be required to establish statistical significance for the 0.31 rank order CC found for total mortality and total hazard ratio for non-Whites.

We conducted an additional neighborhood rank order correlation to examine a potential explanatory factor for the consistent positive correlations found for the non-White population; namely, was the non-White population exposed to higher concentrations of HAPs? We ranked the neighborhoods according to the total estimated 1990 HAP concentration and by percentage of non-White residents. The CC of -0.46 suggests that this simple measure of

total pollutant load does not explain the higher positive correlations between cumulative risk and mortality measures in non-Whites. This correlation does not address variations in the HAP mixture, which, in combination with the toxicological values, may explain the positive correlations between mortality and risk score for the non-White population.

Sensitivity Analysis on Correlation Results

In relative terms, the hazard ratios derived from the NOAEL/LOAEL database produced higher CCs than did the hazard index

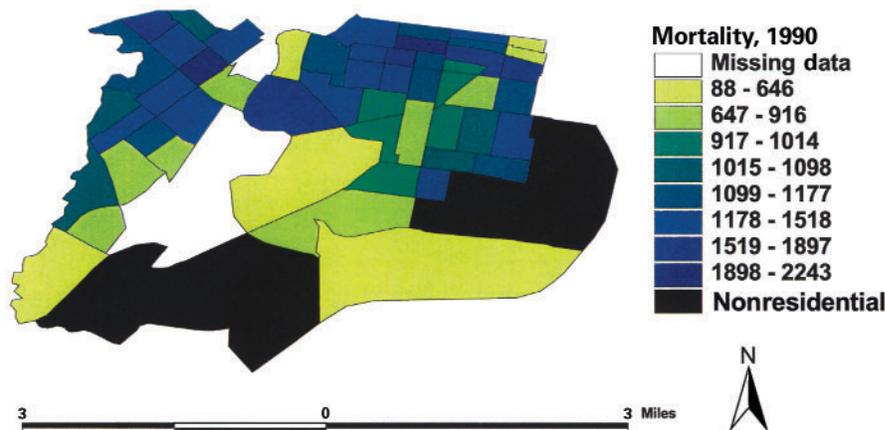


Figure 1. Distribution of age-adjusted total mortality (excluding external causes) rates by census tract, 1990.

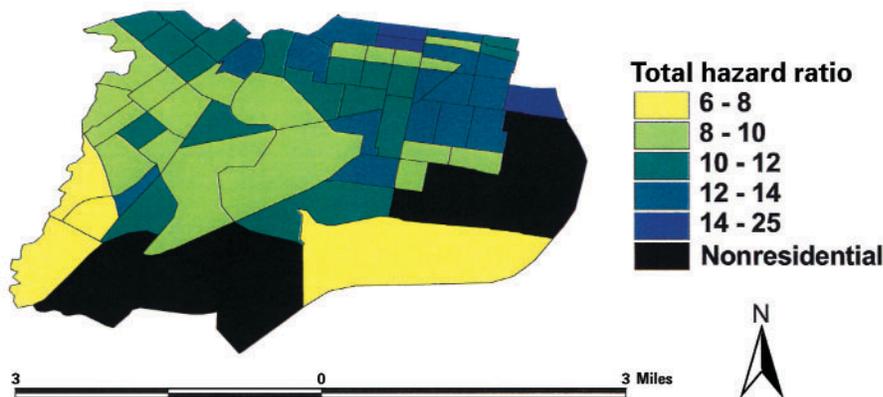


Figure 2. Distribution of total hazard ratio by census tract.

Table 6. Correlation coefficients for comparison of neighborhood rankings on cause-specific mortality and cumulative risk score by population group.

Population group	HR ^a versus mortality rate	HR versus YPLL rate	HI ^b versus mortality rate	HI versus YPLL rate
Whites				
Total excluding external causes	0.33	0.55	-0.40	-0.60
Heart disease	-0.07	-0.02	-0.66	-0.76
Respiratory	-0.02	-0.31	-0.52	-0.76
Non-Whites				
Total excluding external causes	0.31	0.57	0.14	0.12
Heart disease	0.43	0.52	0.05	0.17
Respiratory	0.33	0.81	0.31	0.17

^aHazard ratio (HR) is based on the NOAEL/LOAEL database. ^bHazard index (HI) is based on the CETDB.

based on the U.S. EPA CETDB. This does not address the question of whether the hazard ratio risk scores identify the highest risk areas, however. A high positive correlation could be produced for similarity of rank in the lower-risk rather than higher-risk areas. An additional analysis on exact rank matches assessed the ability of the two hazard scores to identify the highest-risk areas as measured by mortality. We drew the exact rank matches from the 96 neighborhood-level rank comparisons (rankings of the 8 neighborhoods on hazard score and mortality under 12 different conditions: two population groups, two mortality measurements, and three health end points) in the overall correlation analysis.

Table 7 presents results of the analysis on exact rank matches. Of the 96 neighborhood rank comparisons, 19 (about 20%) were of the same rank when we used the hazard ratio, and 13 exact matches (14%) resulted when we used the hazard index. Of these exact matches, 9 of the 19 (47%) with the hazard ratio were in the top four (highest risk), whereas 8 of the 13 (62%) with the hazard index score were in the top four.

Regression Results

Total mortality. We conducted the regression analysis to examine associations between cumulative risk scores and mortality at the census-tract level on the complete data set, then identified outliers by visual assessment of plots of the covariates and of regression residuals for the total and cause-specific mortality end points. We identified 14 outlier census tracts that had either very high or

very low mortality and YPLL rates or risk scores that could drive the regression findings. We repeated the regression series after removing these census tracts. The results are presented below. In these regression models, the cumulative risk scores, hazard ratio, or hazard index, were statistically significant at $p < 0.001$.

A one-unit increase in the total hazard ratio was associated with a 1.6% increase in the total YPLL rate for 1990 and slightly larger mortality increases, approximately 4–6%, for total mortality rates in 1990 and total mortality and YPLL rates in the 5-year time period (Table 8). We repeated the analyses using the total hazard index without the same consistency in results. An increase of one unit in the total hazard index was associated with a slight 0.3% increase in the total YPLL for 1990 and 1988–1992.

Heart and vascular mortality. The cause-specific analysis for heart and vascular mortality showed highly variable estimates of association between the heart hazard ratio and mortality and YPLL measurements (Table 9). This is likely due to the lack of variability in the heart hazard ratio scores. With the exception of the heart and vascular YPLL rate for 1988–1992, we found incremental increases in the heart hazard index to be negatively associated with heart disease mortality and YPLL rates after controlling for per capita income and percent non-White population.

Respiratory mortality. Consistent positive associations between increasing risk score and mortality and YPLL rates were demonstrated in the regression analysis of

respiratory mortality for the respiratory hazard ratio derived from the multi-end point toxicological database but not for the hazard index (Table 10). After controlling for per capita income and percent non-White population, a one-unit increase in the respiratory hazard ratio was associated with 23 and 17% increases in YPLL and mortality rates, respectively, for 1990 and 6 and 8% increased YPLL and mortality rates for 1988–1992. The respiratory hazard index had no association with respiratory mortality.

Discussion

Viewing the study area as a whole, the findings of the descriptive community health and CRAs show all-cause mortality rates and total cumulative health risks to be greater than, and sometimes up to triple, the national averages for both Whites and non-Whites.

The hypothesis that the cumulative risk scores would be positively correlated (or associated) with mortality at the neighborhood and census-tract levels was demonstrated for both the total hazard ratio (derived from the multi-end point NOAEL/LOAEL database) and total mortality and YPLL rates. It was also demonstrated for the respiratory hazard ratio and respiratory mortality and YPLL rates at the census-tract level. Although the CCs were not statistically significant because of the limited sample of eight neighborhoods, the cumulative risk score variables were statistically significant ($p < 0.001$) in the regression analysis. The associations found between the mortality end points and cumulative risk scores do not imply causality.

Table 7. Proportions of exact matches for neighborhood rankings on mortality measurements and risk scores.

	Hazard ratio ^a versus mortality and YPLL rates	Hazard index ^b versus mortality and YPLL rates
Total exact matches	19/96 (20%)	13/96 (14%)
Matches in top four	9/19 (47%)	8/13 (62%)

^aHazard ratio is based on the NOAEL/LOAEL database. ^bHazard index is based on the CETDB.

Table 9. Heart and vascular mortality and YPLL rate ratios and 95% confidence interval by time period for heart cumulative risk scores.

Heart and vascular disease	Heart hazard ratio ^a score rate ratio (95% CI)	Heart hazard index ^b score rate ratio (95% CI)
1990		
YPLL rate	1.72×10^{-10} (1.59×10^{-12} – 1.86×10^{-8})	0.2592 (0.2169–0.3098)
Mortality rate	2.44×10^{-25} (1.08×10^{-28} – 9.7×10^{-11})	0.1800 (0.1353–0.2395)
1988–1992		
YPLL rate	2.76×10^5 (4.2×10^4 – 1.8×10^6)	1.533 (1.426–1.648)
Mortality rate	1.26×10^{-10} (4.9×10^{-12} – 3.24×10^{-9})	0.5268 (0.4663–0.5951)

^aHazard ratio is based on the NOAEL/LOAEL database. ^bHazard index is based on the CETDB.

Table 8. Total mortality and YPLL (excluding external causes) rate ratio and 95% confidence interval associated with cumulative risk scores by time period.

Total mortality	Total hazard ratio ^a score rate ratio (95% CI)	Total hazard index ^b score rate ratio (95% CI)
1990		
YPLL rate	1.016 (1.014–1.019)	1.003 (1.0027–1.0035)
Mortality rate	1.064 (1.058–1.071)	0.9951 (0.9942–0.9960)
1988–1992		
YPLL rate	1.039 (1.038–1.040)	1.003 (1.0026–1.0029)
Mortality rate	1.055 (1.052–1.058)	0.9979 (0.9975–0.9983)

CI, confidence interval.

^aHazard ratio is based on the NOAEL/LOAEL database. ^bHazard index is based on the CETDB.

Table 10. Respiratory mortality and YPLL rate ratio and 95% confidence interval by time period for respiratory cumulative risk scores.

Respiratory disease	Respiratory hazard ratio ^a score rate ratio (95% CI)	Heart hazard index ^b score rate ratio (95% CI)
1990		
YPLL rate	1.23 (1.22–1.25)	0.9827 (0.9808–0.9845)
Mortality rate	1.17 (1.14–1.20)	0.9856 (0.98190–0.9892)
1988–1992		
YPLL rate	1.06 (1.057–1.072)	0.9802 (0.9792–0.9813)
Mortality rate	1.08 (1.07–1.10)	0.9961 (0.9946–0.9977)

^aHazard ratio is based on the NOAEL/LOAEL database. ^bHazard index is based on the CETDB.

Results for the correlation analyses for the other end points were mixed. The negative correlations across all health effects between the cumulative hazard index (U.S. EPA CETDB database) and mortality in the White population at the neighborhood level are likely to be due at least partly to scant representation of the multiple effects of the HAPs in that database. We also observed negative correlations for neighborhood rank ordering between heart and vascular and respiratory mortality and the relevant hazard ratios (NOAEL/LOAEL database) in Whites. The positive correlations between mortality and risk scores in non-Whites are not explained by exposure to higher concentrations of HAPs overall. Further sensitivity analysis is planned on the neighborhood-level findings.

With one exception, heart and vascular risk scores showed negative associations with measures of mortality, in contrast to the more consistent positive results for the total and respiratory end points. This suggests that these associations reflect effects separate from individual-level social and behavioral risk factors. The inconsistent findings for respiratory and heart and vascular mortality would seem to argue against confounding by smoking. Again, we need further research with alternative data and techniques to resolve this question.

Caveats

This work tested the U.S. EPA CRA methodology through an evaluation of community health indicators. It did not assess etiology. Several assumptions in the risk assessment and limitations of data sources must be acknowledged.

This noncancer CRA employed the default assumption of additivity. Potential interactions among the HAPs could not be modeled with this approach.

The toxicological values applied in this risk assessment we abstracted from studies of chronic effects on target organs or systems. The dosing regimens in these studies correspond with the longer-term potential exposure to the annual average HAP concentrations from the CEP. Toxicological studies of mortality determine the lethal dose for acute exposures (24).

The U.S. EPA CEP estimated the HAP concentrations for 1990. Validation studies comparing model estimates to monitored values suggested that the model tended to underpredict actual pollutant concentrations (14). Risk scores based on the model results would be less than those calculated from measured concentrations.

At the time of this analysis, the HAPs estimates from the CEP were the most comprehensive example of cumulative chemical

exposure data available, containing information on 148 of the 188 HAPs. The U.S. EPA conducted a limited update for 34 of the HAPs (25). The food portion of the CEP evaluated 37 chemicals (26), but this became available after this risk assessment was completed. The U.S. EPA did not produce a cumulative exposure assessment for drinking water for 1990 because of inadequate data (27).

The 5-year aggregated time period reduced the variability of the small-area mortality statistics. In comparing the aggregated mortality rates to risk scores based on the 1990 pollutant concentration, we assumed that the potential exposures and population in residence in the focal time period were representative of the longer term. Toxic releases began to change during the 1988–1992 time period with the implementation of the Emergency Planning and Community Right to Know Act, a provision of the Superfund Amendments and Reauthorization Act of 1986 (28). Mobile source emissions would be expected to be stable. Agreement between the regression results for the 1990 and 1988–1992 time periods suggests that the assumption was reasonable.

There was a major loss of population and little in-migration in the study area from 1974 to 1993 (10). The population in the 1988–1992 period comprised long-term residents. Given the latter, the findings reported may overestimate the associations between HAP exposures and mortality for shorter term or transient residents of the study area.

Although this study was primarily concerned with potential chemical exposures, we included available socioeconomic variables of income and minority population distribution in the analysis. The very simple treatment of socioeconomic measures as control variables utilized here is not representative of current approaches to the analysis of socioeconomic status and health inequalities. Examples of these more sophisticated measurements and analyses can be found in Breeze et al. (29), Blakely et al. (30), and Kennedy et al. (31).

In addition, the indicators of mortality present a limited picture of the range of potential community health effects related to air pollution. To maximize its preventive potential, associations between CRA and morbidity or earlier indicators of health status and disease development should be established. At present, however, mortality surveillance remains the most readily available source of health data for small-area analysis that is accessible nationwide.

Conclusions

The U.S. EPA considers cumulative exposure and risk-based methodologies among other analytical tools for investigating environmental

justice complaints (32). Our case study provides strong evidence in support of these methodologies as community-level health assessment tools. Simple assessment of pollutant loads or the distribution of potential exposures based on pollution monitoring are of limited value. The HAP concentration data do not reflect an overall increased exposure to the non-White population in the South and Southwest Philadelphia area, yet the cumulative risks of the HAPs correlate positively with mortality in the non-White population. Increased mortality was associated with increasing cumulative risk scores in a regression analysis that controlled for both per capita income and percent non-White population at the census-tract level, suggesting that the health risks of the HAPs are separate from income and race.

Cumulative risk assessment adds a health dimension to simple pollutant concentrations and will produce a more comprehensive understanding of environmental inequities. Identifying populations at increased risk from environmental exposures is the first step toward mitigating such risks as required by the fair treatment mandate of environmental justice. However, given the limitations of the current toxicological inputs, CRA should not be the only measure of disparate environmental impacts. The best indicators of the potential for adverse health impacts remain measures of health status.

This research successfully applied CRA in the communities of South and Southwest Philadelphia. Total cumulative risk scores based on a multi-end point toxicological reference database were associated with total mortality (excluding external causes) at both the neighborhood and census-tract levels. Incremental increases in risk scores were associated with 2–6% increased total (excluding external causes) mortality and YPLL rates. Respiratory risk scores based on the multi-end point database were consistently associated with increased respiratory mortality and YPLL rates, ranging from 6% to 23%. Results of the cause-specific analysis of heart and vascular disease mortality and cumulative risk were mixed.

At present, then, CRA may be best applied as a screening tool to identify high-risk areas to guide research, surveillance, and intervention. We need further development of the various inputs (toxicological reference values, information on other sources, media and routes of chemical exposure) before CRA is appropriate for detailed investigations, and additional studies to establish whether the associations found here for mortality end points also hold for morbidity.

This work made a small step toward the integration of epidemiologic and environmental health assessment tools. A primary

impetus for the Food Quality Protection Act (9) and the U.S. EPA articulation of CRA was the National Research Council's age-specific approach to health risks from pesticide residues on food (33). The research and development of cumulative risk methods present an opportunity to the public health community to continue to bring sophisticated epidemiologic analyses to bear on questions of environmental health risk.

REFERENCES AND NOTES

- U.S. EPA. Guidance on Cumulative Risk Assessment. Part 1. Planning and Scoping. Washington, DC:U.S. Environmental Protection Agency, July 1997. Available: <http://www.epa.gov/swerosps/bf/html-doc/cumrisk2.htm> [accessed 10 September 1997].
- U.S. EPA. Framework for Cumulative Risk Assessment. Washington, DC:U.S. Environmental Protection Agency, August 2, 2001. Available: http://www.epa.gov/NCEA/raf/pdfs/Draft_Framework_0802_2001.pdf [accessed 24 September 2001].
- Birch S, Stoddart G, Beland F. Modelling the community as a determinant of health. *Can J Public Health* 89(6):402–405 (1998).
- Institute of Medicine, Committee on Using Performance Monitoring to Improve Community Health. Improving Health in the Community: A Role for Performance Monitoring. Washington, DC:National Academy Press, 1997.
- Pew Environmental Health Commission. America's Environmental Health Gap: Why the Country Needs a Nationwide Health Tracking Network. Baltimore, MD:Johns Hopkins University School of Public Health, 2000.
- U.S. EPA, Office of Enforcement and Compliance Assurance. Environmental Justice. Washington, DC:U.S. Environmental Protection Agency, 6 September 2001. Available: <http://es.epa.gov/oeca/main/ej/index.html> [accessed 17 November 2001].
- Fox MA. Identifying Public Health Tools for Environmental Policy: A Case Study of Cumulative Risk Assessment in South and Southwest Philadelphia [PhD thesis]. Baltimore, MD:Johns Hopkins University, 2001.
- Clean Air Act Amendments of 1990. Public Law 101-549, 1990.
- Food Quality Protection Act of 1996. Public Law 104-170, 1996.
- Burke TA, Shalauta NM. Pilot Multi-media Environmental Health Characterization Study of South and Southwest Philadelphia. Philadelphia:U.S. Environmental Protection Agency, Region III, 1997. Available: <http://www.epa.gov/region03/sswphil/index.htm> [accessed 9 February 2001].
- U.S. EPA. Chicago Cumulative Risk Initiative. Chicago:U.S. Environmental Protection Agency, Region V, December 1998. Available: http://www.epa.gov/reg5oopa/agenda99/goal_8.htm [accessed 9 February 2001].
- U.S. EPA. Cumulative Exposure Project Community Specific Study: Greenpoint/Williamsburg. Washington, DC:U.S. Environmental Protection Agency, August 2000. Available: <http://www.epa.gov/cumulativeexposure/communit/communit.htm> [accessed 9 February 2001].
- Anderson RN, Rosenberg HM. Age standardization of death rates: implementation of the year 2000 standard. *Natl Vital Stat Rep* 47(3):1–20 (1998).
- Rosenbaum AS, Ligocki MP, Wei YH. Revised Final Report. Modeling Cumulative Outdoor Concentration of Hazardous Air Pollutants, Vol 1, Text. San Rafael, CA:Systems Applications International, 1999.
- Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. *Environ Health Perspect* 106(5):245–251 (1998).
- U.S. Environmental Protection Agency. Cumulative Exposure Toxicity Database. Cambridge:Industrial Economics, 1998. Available: <http://www.epa.gov/cumulativeexposure/resource/resource.htm> [accessed 18 September 1999].
- Kelsall JE, Samet JM, Zeger SL, Xu J. Air pollution and mortality in Philadelphia, 1974–1988. *Am J Epidemiol* 146(9):750–762 (1997).
- Centers for Disease Control and Prevention. Premature mortality in the United States: public health issues in the use of years potential life lost. *Morb Mortal Wkly Rep* 35(suppl 2S):1S–11S (1986).
- Centers for Disease Control and Prevention. Introduction to Table V: Premature Deaths, Monthly Mortality, and Monthly Physician Contacts—United States. *Morb Mortal Wkly Rep* 46(24):556–561 (1997).
- National Center for Health Statistics. Vital Statistics of the United States, 1990, Vol. 2, Mortality, Part A. Washington, DC:U.S. Public Health Service, 1994.
- U.S. EPA, Office of Health and Environmental Assessment. The Risk Assessment Guidelines of 1986. EPA/600/8–87/045. Washington, DC:U.S. Environmental Protection Agency, 1987.
- Morello-Frosch RA, Woodruff TJ, Axelrad DA, Caldwell JC. Air toxics and health risks in California: the public health implications of outdoor concentrations. *Risk Anal* 20(2):273–291 (2000).
- U.S. EPA. Integrated Risk Information System: List of Substances on IRIS. Washington, DC:U.S. Environmental Protection Agency, 1999. Available: <http://www.epa.gov/iris/subst/index.html> [accessed 18 September 1999].
- Eaton DL, Klaassen CD. Principles of toxicology. In: Casarett & Doull's Toxicology: The Basic Science of Poisons, 5th ed (Klaassen CD, ed). New York:McGraw-Hill, 1996;13–34.
- U.S. EPA. National-Scale Air Toxics Screening Assessment. Washington, DC:U.S. Environmental Protection Agency, 1996. Available: <http://www.epa.gov/ttn/uatw/nata/nsata1.html> [accessed 27 February 2001].
- Doughtery CP, Hotlz SH, Reinert JC, Panycosit L, Axelrad DA, Woodruff TJ. Dietary exposures to food contaminants across the United States. *Environ Res A* 84:170–185 (2000).
- U.S. EPA. Cumulative Exposure Project: Drinking Water. Washington, DC:U.S. Environmental Protection Agency, August 2000. Available: <http://www.epa.gov/cumulativeexposure/water/water.htm> [accessed 27 February 2001].
- Superfund Amendments and Reauthorization Act of 1986. Public Law 99-499, 1986.
- Breeze E, Fletcher AE, Leon DA, Marmot MG, Clarke RJ, Shipley MJ. Do socioeconomic disadvantages persist into old age? Self-reported morbidity in a 29-year follow-up of the Whitehall Study. *Am J Public Health* 91(2):277–283 (2001).
- Blakely TA, Kennedy BP, Kawachi I. Socioeconomic inequality in voting participation and self-rated health. *Am J Public Health* 91(1):99–104 (2001).
- Kennedy BP, Kawachi I, Glass R, Prothrow-Smith D. Income distribution, socioeconomic status, and self-rated health in the United States: multilevel analysis. *Br Med J* 317:917–921 (1998).
- U.S. EPA. Draft Title VI Guidance for EPA Assistance Recipients Administering Environmental Permitting Programs (Draft Receptient Guidance) and Draft Revised Guidance for Investigating Title VI Administrative Complaints Challenging Permits. Available: http://www.epa.gov/civilrights/t6guidedoc_june2000_frtxt.pdf [accessed 6 March 2002].
- National Research Council. Pesticides in the Diets of Infants and Children. Washington, DC:National Academy Press, 1993.