US Metropolitan Areas - 2000 Air Toxic Concentrations and Racial Differences in

Exposure to Air Toxics - Methodology

Russ Lopez, MCRP, DSc

Department of Environmental Health
Boston University School of Public Health
715 Albany St., Talbot 2E
Boston, MA 02118

Rptlopez@bu.edu
(617) 414-1439 phone
(617) 638-4857 fax

Introduction

       While it is possible to study the distribution of individual types of pollution sources (coke oven, trash transfer stations, etc the environment is the sum of many sources of risk and benefit. Individual case studies can assist in understanding how present-day pollution and residential patterns have became established; however, focusing on overall air quality might provide a better portrait of pollution burden than analyzing a particular industry type. The Environmental Protection Agency (EPA) sets ambient air standards for criteria air pollutants (Ozone, Carbon Monoxide, Oxides of Nitrogen, Sulfur Dioxide, Lead and Particulates) and localities use air monitoring to assess their compliance with these regulations. The term "air toxics" is used to describe all the other potentially hazardous chemicals that may be present in outside air, such as benzene and chromium. These comprise the air toxics database of the EPA   The EPA sets no regulatory limits on ambient air toxics concentrations and monitoring data are sporadic.   As an alternative to monitoring, the EPA produces periodic estimates of concentrations based on a detailed modeling methodology that incorporates large stationary sources, small area sources, mobile sources and background levels of pollutants. Lead is the only criteria air pollutant included in the air toxics database. The first estimates for 140 air toxics were prepared for 1990 and issued in 1997. They were based on air dispersion modeling, using the then current database of potential sources of these pollutants.

       The EPA committed itself to periodic updates of the estimates. The 1996 version of the EPA's modeled air toxics contained estimates of 41 chemicals, chemical compounds, metals and chemical groups at the census tract level. The data were prepared for each of the approximately 64000 census tracts in the continental United States and was released in 2000. Because of the changes in methodology between 1990 and 1996, the results are not comparable; and more recent data are not available at this time. However, subsequent updates of the 1996 data, scheduled to be released every three years, may be comparable; and trend analysis might be possible at a later date.

Methodology

Modeled Air Toxic Data

       The data are available on CD-ROM from the EPA which consists of a series of files on individual air toxics that must be combined to give a complete portrait of the range of modeled air toxics in each census tract. Aggregation of the data is easily achieved by commercially available software. This analysis used Access and Excel to translate the CD-ROM data into a format usable by statistical analysis software.

Conversion to 2000 Census Tracts

The 1996 Modeled Air Toxic Concentrations were estimated based on 1990 census tracts. While the majority of 1990 census tracts were unchanged for the 2000 Census, a subset was modified. Most of these were minor changes to smooth out tract boundaries or split single tracts into multiple tracts that collectively approximated the original tract. In order to facilitate translation of tracts between 1990 and 2000, the Census publishes a Tract Relationship File. Using this file, every 2000 tract was assigned to the 1990 tract that would have comprised the majority of the population of the 2000 tract.

Summing of Air Toxics Data

1996 census-tract based estimates were combined with 2000 Census data to produce a modeled estimate of exposure for the population in every census tract in the continental United States. Each census tract's total potential exposure was tabulated in three ways. First, the unadjusted modeled concentrations of air toxics were summed to give an estimate in micrograms per cubic meter of total air toxics concentrations (called total unweighted here). This crude methodology does not account for toxicity differences between air toxics. While perhaps an appropriate measure of overall air quality, it says nothing about potential health risks.

Second, the total sum of the lifetime total predicted probability of cancer occurrences for each air toxic concentration was calculated (called cancer-weighted risk here). For those air toxics (except diesel emissions) determined by the EPA to have potential carcinogenic effects, the Agency has determined a benchmark level concentration that potentially may result in one cancer occurrence per million persons over a lifetime of exposure. This methodology excludes those air toxics that the EPA does not consider to be carcinogenic or for which the EPA has yet to set benchmark levels. The actual modeled concentration for each toxic divided by the benchmark concentration gives a predicted lifetime cancer incidence rate due to modeled ambient concentrations. The cancer-weighted risk score is the sum of these predicted excess occurrences. This methodology has been previously used to sum the data. It must be noted that this represents a theoretical risk and it is not an estimate of any individual's actual lifetime cancer risk. The cancer-weighted risk score is best interpreted as a measure of relative levels of carcinogens in ambient air.

Finally, the ratio of estimated concentrations to the EPA's reference doses for non-cancer effects for each air toxic was summed (non-cancer weighted). The EPA sets reference doses. They represent a lifetime level of continuous exposure below which sensitive populations (children, asthmatics, etc.) should be without appreciable health risks. Only air toxics with reference doses are included in this measure. Again, this methodology is commonly used A particular problem in interpreting this measure is how to compare potential health effects. For

example, are liver effects more problematic than lung effects? How might one weight neurological effects versus kidney effects? So again, the best use of this measure is that of population exposure to relative levels of air quality rather than as a predictor of individual risk.

Measuring Racial Differences in Exposure to Air Toxics - Net Difference Scores

The net difference score is a statistical measure used in income inequality analysis that is adapted here to measure inequality in exposure. Based upon cumulative frequency distributions, the advantage of using a net difference score is that it measures inequality over the whole range of exposures. It is calculated using the following formula (using Black/White exposure differences as an example):

$$ND_{wb} = \sum_{i=1}^{I} \sum_{j=1}^{I} (X * W_i * B_j)$$

Where, if i > j, X = +1; if i = j, X = 0; if i < j, X = -1; W = Non-Hispanic Whites; B = Non-Hispanic Blacks; i = exposure level of non-Hispanic Blacks and j = exposure level of non-Hispanic Whites.

The net difference score as used here can be described as the following: If a Black person and a White person are randomly picked in a given metropolitan area, the net difference score is the probability that the Black person is exposed to a higher level of total air toxics than the White person minus the probability that the White person is exposed to a higher level of total air toxics than the Black person. A score of 1 would mean that all Blacks are breathing more polluted air than all Whites. A score of –1 means that all Whites are breathing more polluted air than all Blacks.   A score of 0 means that Whites and Blacks are breathing similar levels of pollutants.