Air Pollutants and Bay Area School Children: Exposure and Risks

Serena Chang
Environmental Sciences, University of California at Berkeley

Abstract

Due to their unique vulnerabilities, children are at greater health risk from environmental exposures to pollutants than any other segment of the population. Characteristics such as physical stature and behavior increase their chances of exposure. For young children generally, the school and home microenvironments contribute most to their exposure to environmental pollutants. Thus, it is important to be aware of the health risks that children potentially face as a result of their exposures at school. The goal of this project was to perform crude risk assessments for Bay Area school children based on their exposure to air pollutants. Air quality monitoring data from the California Air Resources Board (CARB) and the Bay Area Air Quality Management District (BAAQMD) were applied to schools located within three miles of each monitor. These concentrations were compared with both cancer and noncancer benchmarks for various compounds derived from EPA reference values. The results show that all the students in the study are potentially at health risk due to their exposures to air toxics while at school. The probability of developing cancer from exposure to metals was low, less than three per one million. The highest total risk from exposure to volatile organic compounds was approximately 400 per million. Because the study only focused on a select group of pollutants, the actual risks faced by these children are likely to be higher. In the future, a more extensive monitoring network with a wider range of pollutants measured would greatly improve the accuracy and usefulness of this type of risk assessment.

Introduction

The history of the Clean Air Act indicates that Congress made the protection of public health the sole determinant of acceptable air pollution levels (Committee 1996). The Act mandates that national ambient air-quality standards be set low enough to protect the health of all sensitive groups with the exception of those requiring life-support systems. A sensitive group is defined as one that exhibits "a response to a pollutant at a lower level or to a greater degree than the average response of the general population" (Committee 1996). Sensitivity varies among different ethnic groups, genetic backgrounds, as well as age and childhood experience and development. Age-related differences have a significant effect on metabolism, physiology, developmental stage, behavior, and diet (Goldman 1995).

Title III, Section 112 of the 1990 Clean Air Act Amendments (CAAA) defined a list of 189 hazardous air pollutants (HAPs), also called air toxics, (Kelly *et al*. 1994, Caldwell *et al*. 1998). They were designated as HAPs because they were known or suspected of causing cancer or other adverse human health effects or damage to ecosystems (Kyle *et al.* 1999). HAPs include certain volatile organic chemicals, pesticides, herbicides, and radionuclides and have been associated with effects impacting the neurological, reproductive, and developmental systems (Woodruff *et al*. 1998). They were listed by the EPA to identify source categories for which technology-based, toxic air emission reduction standards would be considered (US EPA 2000, elect. comm.) Title III required regulation of routine and accidental emissions of each HAP from large industrial sources and from small commercial sources (Kelly *et al*. 1994).

The greatest contributor to air pollution in the majority of the United States is the motor vehicle. Compounds such as benzene, an air toxic, are present in gasoline and are emitted to the air when gasoline evaporates or passes through the engine as unburned fuel (US EPA 2000, elect. comm.). In addition to being emitted in unburned fuel, a significant amount of automotive benzene is also released from the incomplete combustion of other compounds in gasoline such as toluene and xylene. Formaldehyde, acetaldehyde, and 1,3-butadiene are not present in fuel but are by-products of incomplete combustion. Stationary point sources of air pollution include power production plants utilizing coal combustion, oil refineries, and industrial engineering facilities (Mott *et al*.1997). Small businesses such as dry cleaners, auto body shops, and metal recycling plants also emit pollutants into the atmosphere.

As a group, children may be even more vulnerable than adults to deleterious health effects due to their unique exposures and susceptibilities (Goldman 1998). Their behavior patterns may put them in a position to receive greater exposures. Compared to adults, children are generally more active, with higher ventilation rates. Even at rest, due to their rapid growth, they have a higher resting metabolic rate and rate of oxygen consumption per unit body weight (International Programme 1986). Thus, even if concentrations of respiratory toxins in ambient air are low, the large quantity of air inhaled by a child may expose the child to large doses of the toxins (Gilliland *et al.* 1999). In addition, due to their physical stature and typical daily activity, young children generally spend more time close to the ground. Thus, they experience higher exposures to pollutants emitted near ground level such as car exhaust and pollutants with high densities (Mott *et al*.1997). The potential impacts of such exposure are exacerbated because growing lungs may be more vulnerable to permanent adverse effects (Gilliland *et al.* 1999). Children may also have decreased detoxification capacity for many chemicals because of metabolic enzyme differences present during development (Weaver *et al*. 1998). Furthermore, they often do not recognize the significance of respiratory symptoms and will not limit outdoor physical activity during periods of high ambient pollution (Mott *et al.* 1997). Thus, they are less likely to report exposure-related symptoms (Gilliland *et al.*1999).

The physical locations of exposure should also be considered. With school-age children who often spend a significant amount of time at school, the school's environment has a direct impact on their exposures. Due to economic reasons, schools are often built on relatively undesirable land where children may receive greater exposures to pollutants (Bearer 1995). The time of exposure may also be a factor; for example, children may receive a greater exposure to ozone due to the fact that they leave school in the afternoon, at a time when ozone levels may be at their highest (Bates 1995).

The occurrence of asthma is of particular importance when children are concerned. Between 1980 and 1993, asthma prevalence in children increased 58 percent while mortality due to asthma increased 78 percent (Clark *et al*. 1999). The greatest increase has been seen in young people living in the inner cities (Eggleston *et al*. 1999). In the U.S., hospitalization and morbidity rates due to asthma are two times higher in nonwhites than whites (Clark *et al*.

1999). These ethnic differences associated with asthma prevalence were significant even after adjusting for age, gender, and residence in the inner city (Eggleston *et al*. 1999).

Another factor to consider when assessing exposure may be sex. In a southern California study, Peters *et al*. (1995) noted that the girls in their study spent less time outdoors and less time exercising than boys, which should result in a lower exposure to ambient air pollutants.

For this project, monitoring data of air pollutant concentrations from various California agencies were used to assess whether Bay Area school children face health risks due to exposure to air pollutants. The data was geographically represented to determine how monitoring data would apply to school areas. Health benchmarks derived from EPA reference values were used to see the extent to which current ambient pollutant levels can affect children's health. I hypothesize that due to their special susceptibilities, Bay Area school children do face a health risk due to their exposures to air pollutants.

Description of Study Subjects

Public schools located in the Bay Area counties of Alameda, Contra Costa, Marin, Napa, San Francisco, San Mateo, Santa Clara, Solano, and Sonoma were included in our study. The students ranged in age from kindergarten to 12th grade. Data from the California Air Resources Board and the Bay Area Air Quality Management District collected in 1997 and the beginning of 1998 by monitoring stations located in Antioch, Concord, Fremont, Livermore, Martinez, Mountain View, Napa, Oakland, Pittsburg, Redwood City, Richmond, San Francisco, San Jose, San Leandro, San Pablo, San Rafael, Santa Rosa, Sausalito, Vallejo, and Walnut Creek were used in this project. The risk assessments were based on the children's exposure to ambient concentrations of 13 volatile organic compounds, acetaldehyde, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, ethylene dibromide, ethylene dichloride, formaldehyde, methylene chloride, *p*-dichlorobenzene, tetrachloroethylene, trichloroethylene, vinyl chloride and of the metals chromium (VI), cobalt, manganese, and nickel.

Methods

The addresses of the schools in the study were obtained from a CD-ROM purchased from the California Department of Education (CDE). The CDE data contained demographic

information on all public and private schools in the state (CDE 1999). Only the CDE data for public schools were used because they were more extensive, containing information on the lowest and highest grade levels taught at each school. All schools containing students in any of the grades from kindergarten through eighth grade taught were included. Thus, higher grades were also included at times where the lowest grade taught at a school is the 8th grade or lower. The addresses of the 29 air quality monitors located in the Bay Area were obtained from Amy D. Kyle of the School of Public Health (Kyle *et al.* 1999). Zip codes for the monitor locations were found using the US Postal Service website (USPS 2000, elect. comm.).

The locations of the schools and air monitors were mapped using Environmental Systems Research Institute's (ESRI) Business Map Pro Version 2.0. The mapping software was used to identify schools within 3 miles of each monitor. The CDE database contained 10,072 records of public schools in California. Queries were run using Microsoft Access to eliminate schools whose students did not include young Bay Area children. The Thomas Bros. Maps website, which contains address information on Bay Area public schools (Thomas Bros. 2000 elect. comm.), was used to find information missing from the CDE database. This source was also used to verify the locations mapped by the Business Map software. The Eagle Geocoder website was used to find latitude and longitude coordinates for addresses the software was unable to map (Etak 2000, elect. comm.). The nineteen schools for which the CDE database had insufficient address information and for which the necessary information could not be found, were eliminated. To further check the accuracy of the mapping software, a Rand McNally map of the Peninsula Cities was used to verify the scale and street locations of mapped addresses.

The 1,318 Bay Area schools and 29 monitors included in the study were first mapped separately to assess their distribution and then mapped together in order to identify their spatial relationship. In order to apply the monitoring data to schools, a cut-off distance between a monitor and school was determined. The data from a monitor only applied to schools located within that distance. Distances of one and five miles were considered; a three mile distance was then determined to be reasonable, capturing a significant number of schools while not resulting in too much overlap between them. Using the software, circles were drawn with three-mile radii with monitor locations as the centers. All schools within

those circles were then identified with that monitor. In instances where more than one monitor was within 3 miles of a school, the schools was associated with the closest monitor.

Ambient concentrations of air pollutants in California from the Air Quality Data Review Section of the California Air Resources Board (CARB) and the Toxics Evaluation Section of the Bay Area Air Quality Management District (BAAQMD) were organized and analyzed using Microsoft Access software. Two data files were analyzed; one containing ambient metal concentrations and one containing concentrations of various volatile organic compounds (VOCs). The data was collected in 1997 and for some pollutants, the beginning of 1998. The monitoring data was linked to the schools by matching the monitor number in the data sets with the identifying monitor(s) of the schools. In instances where air pollutant concentrations fell below the detection limit of the monitor(s) the NDHalf value, which represents half of the detection limit, was used to represent the concentration. For a given monitor, all of the measured concentrations of a pollutant were averaged so that one value represented all of the concentrations taken during the year.

To fulfill our data completeness criteria, the concentrations for a pollutant had to have been collected at least 13 times, which was assumed to represent approximately one measurement per month, or over a minimum span of nine months. For monitors that measured the VOCs, there were six locations that contained two monitors at each location. For these monitors, the two sets of data were combined and the concentration values averaged. This combination of data resulted in a greater number of measurements for the pollutants at those monitors; this was used to fulfill the data completeness criteria.

In order to compare the VOCs concentrations at the schools in our study with the health benchmarks, a complete list of the schools and their associated VOCs concentrations was compiled. To do this, a unified list of all of the monitoring data was generated by combining the data from monitors with a duplicate at the same location and data from those without duplicates. The schools were first linked to the address of its identifying monitor. The two sets of monitoring data were then linked to the school names by matching the addresses of the monitors from the data sets to those on the school list. The concentrations of metals were also linked to schools in this manner.

Due to the fact that no national standards have been adopted, benchmarks had to be determined in order to assess whether ambient concentrations are a health risk (Kyle *et al*.

1999). For cancer risks, Caldwell *et al.* (1998) defined the benchmark as the concentration of a known, probable, or possible human carcinogen representing the upper bound of a one in one million probability of contracting cancer over a of lifetime exposure. The health benchmarks used were developed under EPA's Cumulative Exposure Project (Caldwell *et al.* 1998). For all schools, the theoretical cancer risk from exposure to individual pollutants were assessed by dividing the average concentrations of the pollutants by the benchmarks. Noncancer hazard indices were calculated by dividing the average concentrations of the pollutants by the noncancer benchmarks, derived from the EPA's Inhalation Reference Concentration (RfC). The RfC is defined as a "an estimate of a daily exposure to the human population that is likely to be without deleterious effects" for a lifetime exposure (Caldwell *et al*. 1998). Because the RfC was partly derived from science policy, it cannot be used to quantitatively estimate risk. Rather, a noncancer hazard index greater than one indicates that there is a potential health concern. The total cancer and noncancer risks for each school were derived by adding up the individual risks.

Results

Figure 1 displays the distribution of the Bay Area air quality monitors and the associated schools that were included in the study. Of the 23 sites originally mapped, 16 had sufficient data available for analysis. From the cancer benchmarks, it was possible to assess the theoretical cancer risks for the 344 schools associated with those monitors.

From the cancer benchmarks, theoretical cancer risks were assessed for the schools in our study. Figure 2 shows the total calculated risks and the number of schools with those risks. Each risk corresponds to the data from a specific monitor. Sixteen bars are shown, with the height of each bar representing the number of schools associated with that monitor. The geographic distribution of these risks is represented in Figure 3. The differences in magnitude of the risks are exaggerated for illustrative purposes. The calculated cancer risks varied throughout the Bay Area, with monitoring data from the South Bay resulting in the highest risks.

Table 1 shows each of the seven pollutants that were at some time not detected and the number of monitors, out of a total of 16 monitors, that did not detect it. In those instances where the concentration was not detectable, the NDHalf value was used to replace it, yielding

the contribution to total calculated risk. Five of these seven pollutants had detection limits higher than their benchmarks. Table 2 shows these pollutants and the differences between the detection limits and the health benchmarks. Note that the detection limit for 1,3-butadiene is more than two orders of magnitude higher than its health benchmark.

The health risks due to noncancer effects were roughly assessed. Figure 5 presents the noncancer hazard indices and the number schools associated with each value. The noncancer hazard index does not allow for a precise quantitative assessment of health risk. Rather, values higher than one are evidence that there are possible health concerns present due to chronic exposures. Results show that students from approximately 90 schools potentially face health risks other than cancer due to their exposure to the hazardous air pollutants included in the study.

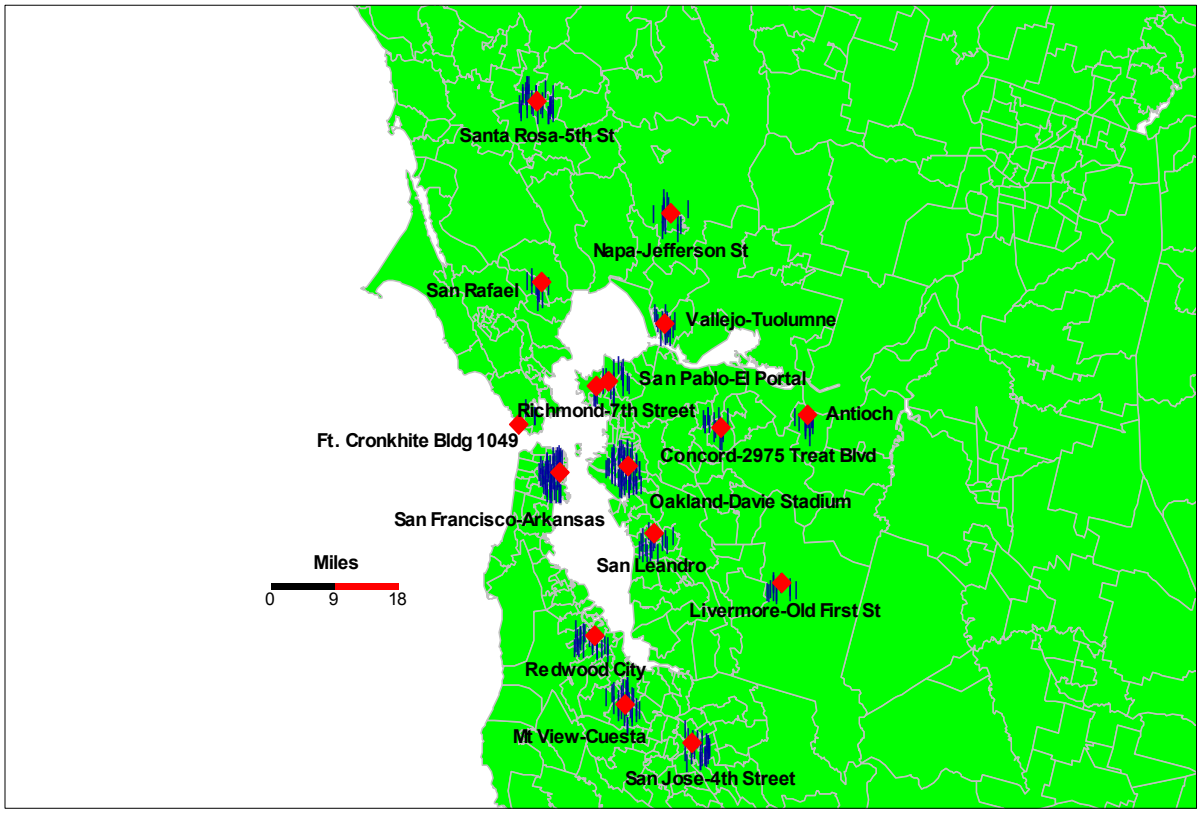

Figure 1. Distribution of Bay Area monitors and their associated schools. The monitoring stations are represented by the large red diamonds, the names of the stations are also given. Schools are represented by small blue diamonds. A total of 344 schools were represented by this study.

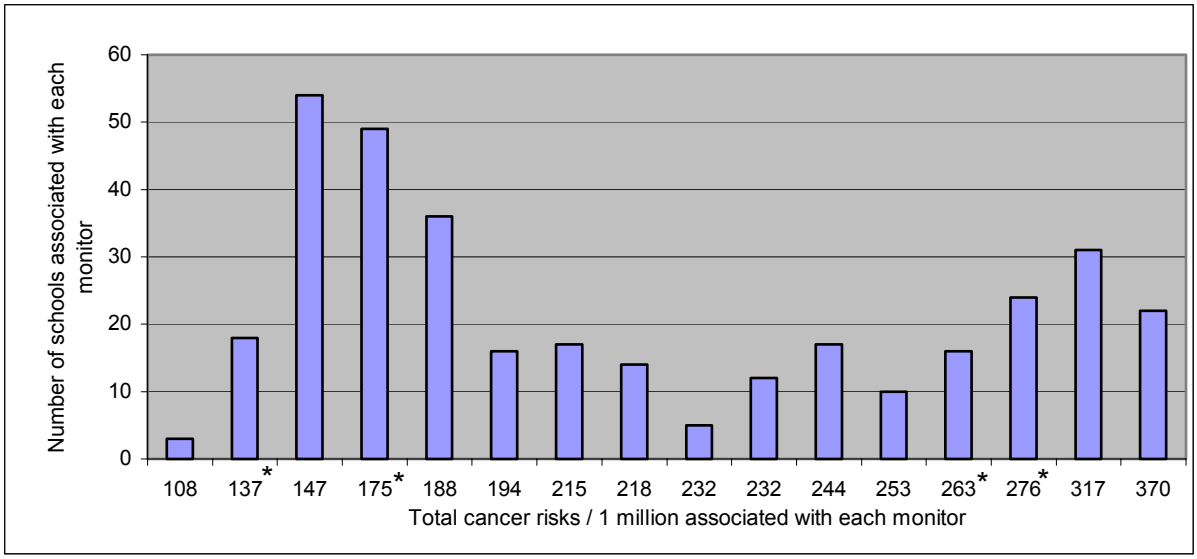

Figure 2. Total cancer risks out of a population of 1 million associated with each monitor. The numbers on the x-axis are the actual calculated cancer risks while the y-axis displays the number of schools with those risks. Each bar corresponds to the data from a specific monitor. Only the total risks shown with an * include risks from exposure to acetaldehyde, formaldehyde, and *p*-dichlorobenzene.

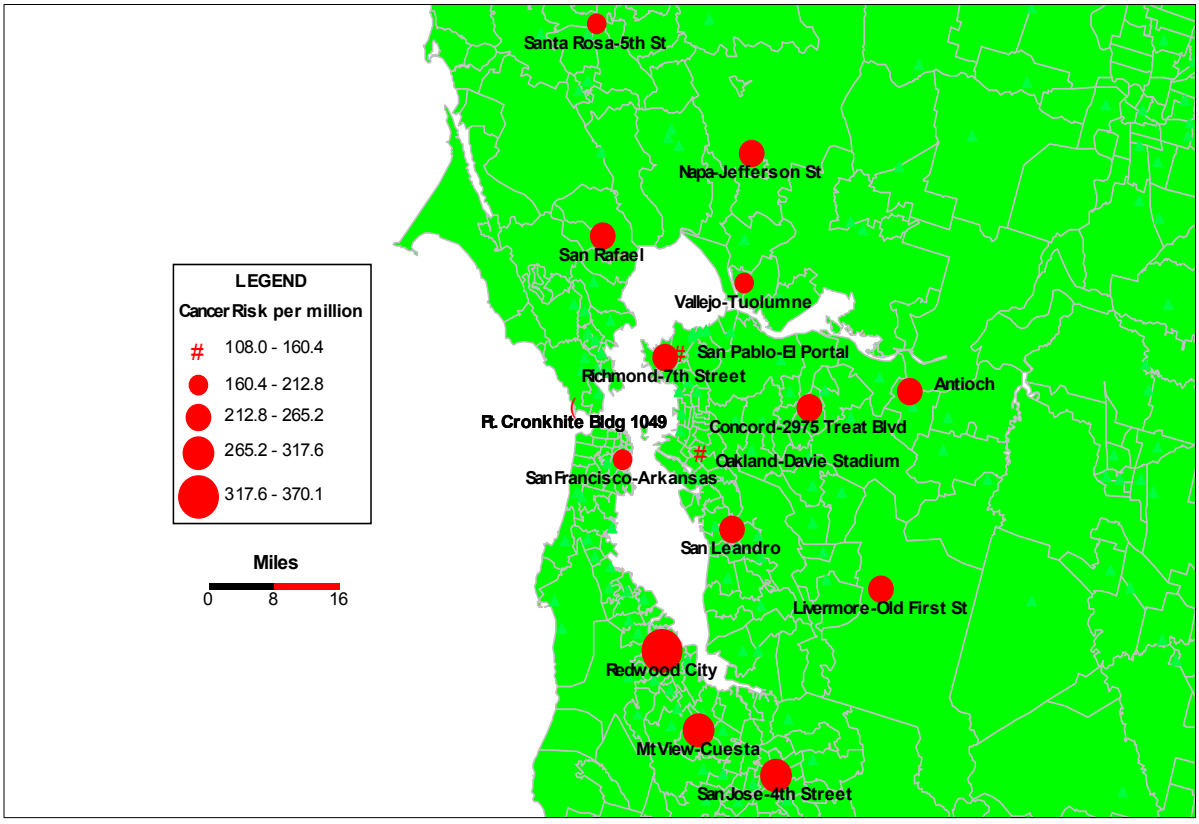

Figure 3. Variation of total calculated risks throughout Bay Area. Differences in risks represented by the red circles are exaggerated for illustrative purposes.

POLLUTANTS NOT DETECTED	# OF MONITORS NOT DETECTED	APPROX. CONTRIBUTION TO TOTAL CALCULATED CANCER RISK
ETHYLENE DIBROMIDE	16	17
ETHYLENE DICHLORIDE	16	5
VINYL CHLORIDE	16	30
TRICHLOROETHYLENE	7	0.4
CHLOROFORM	2	1
METHYLENE CHLORIDE	2	0.4
1,3-BUTADIENE	1	94

Table 1. Pollutants not detected by various monitors. # of monitors not detected shows the number of monitors, out of a total of 16, that never detected the pollutant. Contribution to total calculated risk gives the risk calculated for each pollutant when not detected.

POLLUTANTS NOT DETECTED	DETECTION LIMIT (PPB)	BENCHMARK (PPB)	DIFFERENCE BETWEEN BENCHMARKS AND DETECTION LIMITS
ETHYLENE DIBROMIDE	0.02	0.00059	>1 order of magnitude (OM)
ETHYLENE DICHLORIDE	0.1	0.0094	>1 OM
VINYL CHLORIDE	0.3	0.005	>1 OM
CHLOROFORM	0.02	0.0088	<1 OM
1,3-BUTADIENE	0.3	0.0016	>2 OM

Table 2. Those pollutants not detected by monitors with detection limits higher than health benchmarks. Difference between benchmarks and detection limits gives the order of magnitude by which the detection limit is higher than the benchmark.

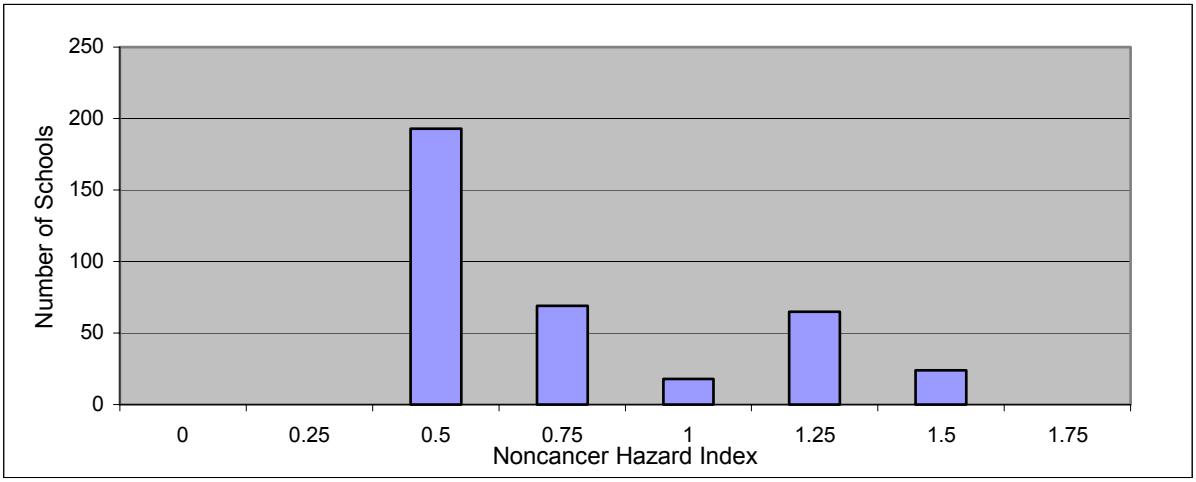

Figure 4. The noncancer hazard indices and the number of schools associated with each value. Risks associated with exposure to ethylene dibromide, ethylene dichloride, and vinyl chloride have been excluded because they were never detected. The values included in each column are bounded by the x-axis value directly below the column and the previous value on the axis (e.g. the bar above the 0.5 noncancer hazard index represents all values greater than 0.25 up to and including 0.5).

Discussion

Due to the mapping scheme that was used to identify schools with monitors and the quantity of air quality monitoring data, risks were assessed for 344 schools, roughly one fourth of the public schools originally in the study. The results show that all of the children attending these schools encounter some health risks due to their exposure to hazardous air pollutants. The calculated risks of developing cancer based on chronic exposures to the volatile organic compounds (VOCs) in the study ranged from 108 to 370 out of one million. The calculated cancer risks from exposures to the metals in the study, chromium (VI), cobalt, manganese, and nickel, ranged from approximately 1.6 to 2.5 / 1 million. Thus, ambient metals did not appear to pose a health risk to schoolchildren while exposure to VOCs may result in serious adverse health effects.

The average enrollment of the Bay Area schools in the study was found to be roughly 500 students, resulting in an estimated student population in the 344 schools included in this study to be approximately 170,000 students. These students face a minimum cancer risk of 108 per million. At this rate, which is more than 1 in 10,000, almost 20 of these students could theoretically develop cancer. For schools associated with the highest calculated risk, the cancer risk is three times greater.

Total cancer risks were calculated as the sum of the individual risks associated with each pollutant in our study. Figure 4 illustrates the range of calculated cancer risks from exposure to VOCs and the number of schools with those risks. However, not all of the monitors measured all 13 of the VOCs. Three pollutants, acetaldehyde, formaldyde, and *p*-dichlorobenzene, were only measured by six monitors. The data from two of those monitors were not used due to incomplete data availability. Thus, only the risks calculated from the data of the remaining four monitors included those pollutants. The basis for the determination of total risks was not equal for all schools. Despite this discrepancy, the risks for the schools associated with monitors that measured more pollutants were not always higher than those calculated for schools associated with the other monitors. The contribution of those pollutants to the total risk did not skew the results to one side. Each risk shown in Figure 4 is associated with one monitor; consequently, the risks should not be used as a basis from which to compare various schools, but rather as rough estimates of the risks of the schools that are associated with the monitor. Figure 3 shows that the total risks are

distributed relatively evenly throughout the Bay Area. There is a slight concentration of the highest calculated total risks in the South Bay. This may be due to geographical and environmental factors or perhaps is a product of the industrial processes that take place in that area.

Seven of the compounds analyzed in the study were at times not detected; in those instances, their NDHalf values were used to represent their ambient concentrations. Therefore, for schools represented by monitors that did not detect a compound, the risk from exposure to that compound was derived from a value that was never actually measured. This process was considered to be reasonable because of the low frequency at which the pollutants were not detected. This was true for four of the non-detected compounds: butadiene, chloroform, methylene chloride, and trichloroethylene. However, the concentrations of ethylene dibromide, ethylene dichloride, and vinyl chloride were never detected at any of the monitors. Using their NDHalf values resulted in a calculated risk of approximately 52 based on compounds not detected in the study site during the time period for which ambient concentrations included in this study were measured. Presumably, these compounds were never detected due to low ambient levels, and so were not included in the risk calculations.

The schools associated with the monitor located in Fremont were also omitted from Figure 4. For that monitor, all pollutants except acetaldehyde and formaldehyde had been excluded because of data availability. Thus, the total risks calculated for those schools were solely based on the concentrations of acetaldehyde and formaldehyde. To include those schools in Figure 4 would have a misrepresentation of the risks at those schools.

Taking into account pollutants that were not measured at certain monitors and the exclusion of compounds that were never detected, cancer risks for all the schools were therefore based on data for 7 to 10 pollutants. The three schools with a calculated risk of 108, shown by the first bar in Figure 4, were associated with the monitor located at Fort Cronkhite in Marin County. Ambient concentrations of 1,3-butadiene were never detected at this monitor. This non-detected compound was found to contribute approximately 94 to the total calculated risk of 108. However, because the detection limit for 1,3-butadiene was much higher than the health benchmark, it was considered to be reasonable to include the risks determined from the monitor's data.

Ambient concentrations measured during 1997 and, for some cases, early 1998 were averaged to yield the concentrations for each monitor that were compared to the benchmarks for each compound. Thus, all of the potential inconsistencies of the data that may have been attributed to factors such as the accuracy and precision of the monitoring equipment or to seasonal variation were internalized into the risk assessments. Furthermore, these potential effects were compounded in the cases where data from duplicate monitors were averaged, where assessments were based on a value that actually represented two sets of data. These data manipulations call to attention the fundamental issue concerning the applicability of monitoring data in assessing risks; that monitoring data may not accurately measure exposures because their locations often differ from those of the study subjects and because behavioral pattern are not taken into account. Despite these issues, the use of monitoring data was appropriate for the scope of this project.

In conclusion, Bay Area school children do face health risks due to their exposure to hazardous air pollutants. They encounter these risks daily depending on where they attend school. While the assessed risks of developing cancer may not seem very high taken out of a population of one million, it must be noted that those risks were calculated for exposures to only 7 to 10 pollutants. This study points outs that a monitoring system with an increased number of monitors and pollutants measured is necessary for more precise risk assessments. Also crucial is the use of monitors with lower detection limits, so that potentially hazardous ambient concentrations can be detected.

Risk assessments for children need to take their special vulnerabilities into account. Studies on the impact of ozone on pulmonary function have shown that exercising young adults or children make up the susceptible population and the magnitude of their response differs from that of the general population (US EPA 1995). Although ozone is not an air toxic, factors such as respiration rate are common to exposures of all air pollutants. Therefore, in the future, to be able to accurately assess risks for children, it will be essential to define benchmarks suited to their physiology, stage in development, and activity patterns.

Acknowledgments

I would like to thank Kevin Kennedy for helping me find a project that fit with my interests. I am also very grateful to him for his advice throughout the year. I would also like

to thank Amy Kyle for her great insight and contribution throughout every step of the research.

References

Bates, D.V. 1995. The effects of air pollution on children. Environmental Health Perspectives 103: 49-53. (suppl. 6).

Bearer, C.F. 1995. How are children different from adults? Environmental Health Perspectives 103: 7-12. (suppl. 6).

Caldwell, J.C., T.J. Woodruff, R. Morello-Frosch, D.A. Axelrad. 1998. Application of health information to hazardous air pollutants modeled in EPA's Cumulative Exposure Project. Toxicology and Industrial Health 14:429-454.

California Department of Education (CDE). 1999. California Department of Education Public School Directory Database. Public and Private Mailing Excel 97 #9746.

Clark, N.M., R.W. Brown, E. Parker, T.G. Robins, D.G. Remick Jr., M.A. Philbert, G. J. Keeler, and B.A. Israel. 1999. Childhood asthma. Environmental Health Perspectives 107:421-429 (suppl. 3).

Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. 1996. Health effects of outdoor air pollution. American Journal of Respiratory and Critical Care Medicine 153:3-50.

Eggleston, P.A., T.J. Buckley, P.N. Breysse, M. Wills-Karp, S.R. Kleeberger, and J.J.K. Jaakkola. 1999. The environment and asthma in U.S. inner cities. Environmental Health Perspectives 107:439-450 (suppl. 3).

Etak Inc. Testdrive Eagle Geocoding Web Page. Allows you to find the latitude/longitude coordinates for a specified address. http://www.geocode.com, accessed March, 2000.

Gilliland, F.D., R. McConnell, J. Peters, and H. Gong Jr. 1999. A theoretical basis for investigating ambient air pollution and children's respiratory health. Environmental Health Perspectives 107:403-407 (suppl. 3).

Goldman, L.R. 1995. Children-unique and vulnerable. Environmental risks facing children and recommendations for response. 103:13-18. (suppl. 6).

___. 1998. Chemicals and children's environment: what we don't know about risks. Environmental Health Perspectives 106:875-880 (suppl. 3).

International Programme on Chemical Safety. 1986. Principles for evaluating health risks from chemicals during infancy and early childhood: the need for a special approach. Environmental Health Criteria 59:3-66.

Kelley, T.J., R. Mukund, C.W. Spicer, and A.J Pollack. 1994. Concentrations and transformations of hazardous air pollutants. Environmental Science and Technology 28:378A-387A.

Kyle, A.D., C.C. Wright, J.C. Caldwell, P.A. Buffler, and T.J. Woodruff. 1999. Characterizing the health significance of toxic air pollutants in California: the contribution of monitoring networks. Unpublished report.

Mott, L., D. Fore, J. Curtis, and G.S. Solomon. 1997. Our children at risk. Natural Resources Defense Council.

Peters, J.M., E. Avol, W. J. Gauderman, W.S. Linn, W. Navidi, S.J. London, H. Margolis, E. Rappaport, H. Vora, H. Gong Jr., and D.C. Thomas. 1995. A study of twelve southern California communities with differing levels and types of air pollution. American Journal of Respiratory and Critical Care Medicine 159: 768-775.

Thomas Bros. Maps. Points of Interest Web Page. http://www.thomas.com/poi/bam/, accessed March, 2000.

United States Environmental Protection Agency (US EPA). Unified Air Toxics Website. http://www.epa.gov/ttn/uatw/basicfac.html, accessed April 3, 2000.

___. 1995. Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. US EPA Office of Air Quality Planning and Standards, Research Triangle Park, NC.

United States Postal Service**.** ZIP+4 Code Look-up Web Page. Allows you to find a zip code for a specified address. http://www.framed.usps.com/ncsc/lookups/lookup_zip+4.html, accessed March, 2000.

Weaver, V.M., T.J. Buckley, and J.D. Groopman. 1998. Approaches to environmental exposure assessment in children. Environmental Health Perspectives 106:827-832 (suppl. 3).

Woodruff, T.J., D.A. Axelrad, J. Caldwell, R. Morello-Frosch, and A. Rosenbaum. 1998. Public health implications of 1990 air toxics concentrations across the United States. Environmental Health Perspectives 106:245-251.