The Disproportionate Impact of Environmental Health Threats on Children of Color

Lawrie Mott
Natural Resources Defense Council, San Francisco, California

Children receive greater exposures to environmental pollutants present in air, food, and water because they inhale or ingest more air, food, or water on a body-weight basis than adults do. Communities of color are disproportionately exposed to hazardous wastes, dioxin, and air pollution. Existing data demonstrate that children of color are the subgroup of the population most exposed to certain pollutants, including lead, air pollution, and pesticides. Government regulations do not take into account children’s differential exposures or the cumulative nature of these exposures. Federal regulations fail to protect the most highly exposed and most sensitive subgroups of the population. More often than not this group is children of color.

—Environ Health Perspect 103(Suppl 6):33–35 (1995)

Key words: disproportionate impact, children of color, lead, air pollution, pesticides, drinking water

Introduction

Children receive greater exposure to environmental pollutants present or contained in the air, water, food, their homes, or their schools. Likewise, people of color are disproportionately exposed to pollution present in their communities. The likely confluence of these two trends is that children of color are the subgroup of the population most exposed to, and least protected from, environmental health threats.

Greater Childhood Exposures

It is a simple biological fact that children inhale or ingest more air, water, or food as a percentage of their body weight than adults do. For instance, the air intake of a resting infant is twice that of an adult under the same conditions (1). Infants and children drink more than 2.5 times as much water daily as adults do as a percentage of body weight (2). Children aged 1 through 5 eat three to four times more per unit of body weight than the average American (3). The average 1-year-old drinks 21 times more apple juice and 11 times more grape juice, and eats 2 to 7.5 times more grapes, bananas, apples, pears, carrots, and broccoli (3). For behavioral reasons as well, children are more exposed to environmental pollutants. They are more active and more curious. Children often play at ground level where pollutants can concentrate. According to a study in California, children spend more time out of doors (especially in the afternoon when air pollution levels peak) and are more active outdoors than adults, thereby increasing their exposure to air pollutants (1).

Communities of Color at Unique Risk

People of color are also more exposed to pollution hazards. Many studies have found a greater presence of environmental hazards in these communities. The best-known example of this problem is the siting of hazardous waste facilities. A landmark report by the United Church of Christ’s Commission on Racial Justice discovered that three of the five largest hazardous waste landfills in the United States are in black or Latino neighborhoods and that the mean percentage of people of color in areas with toxic waste sites is twice that of areas without toxic waste sites (4).

The situation is similar with air pollution. Scientists at the Argonne National Laboratory have found that African-American and Hispanic population subgroups experience greater exposure to substandard outdoor air quality. In particular, their research indicates that these minorities live in greater concentrations both in areas with above-average numbers of air polluting facilities and in air quality nonattainment areas. For instance, 52% of all whites live in counties with high ozone concentrations. For African-Americans the figure is 62% and for Hispanics 71%.

Population-group distributions were found to be similar for carbon monoxide, sulfur dioxide, nitrogen dioxide, lead, and particulate matter, with higher percentages of African-Americans and Hispanics than whites residing in counties with excessive levels of these pollutants (5).

Ethnic minorities are also more likely to be exposed to certain chemical contaminants in the food supply due to dietary differences. For example, Native Americans and subsistence fishing communities may be at much greater health risk from dioxin in fish. A draft EPA notice for a water permit to discharge effluent containing dioxins from a paper and pulp facility into the Penobscot River noted that the Penobscot Indian Nation had a fish consumption rate nearly twice the national average (6).

Children of Color at Greatest Exposure

Children of color are the obvious intersection of the two subgroups of the population, children and communities of color, now receiving the greatest exposures to environmental pollutants. Children of color may be at the highest risk of any segment of society. Data currently available clearly demonstrate this. The classic example is lead. The 1988 Centers for Disease Control report on lead poisoning estimated that 68% of poor, inner-city African-American children were lead poisoned as compared to 36% of poor, inner-city white children (7). In an earlier study conducted between 1976 and 1980, 9.1% of all preschool children in the United States had blood lead levels greater than 25 mg/dl, while 24.5% of black children exceeded this level (8). A detailed 1990 Centers for
Disease Control investigation found that the average blood lead level in poor Puerto Rican and Mexican–American children in the United States cities studied exceeded the federal definition of lead poisoning (12 mg/dl > 10 mg/dl) (9).

The same trend is apparent when active asthma cases are compared by race and ethnicity. Of Puerto Rican children less than 11 years old, 11.2% suffer from asthma, 5.9% for non-Hispanic black and 3.3% for non-Hispanic white children (10). Air pollution has been shown to aggravate asthma in a number of studies (11).

Pesticides provide another illustration that children of color are more exposed. A recent pilot project by the California Department of Health Services examined residential pesticide exposure of infants and children. Sampling was conducted in a small town in California’s San Joaquin Valley. All homes were within one-quarter mile of agricultural fields and approximately 50 agricultural pesticides were used within one mile of the town during the study period. Half of the homes sampled had at least one resident who was a farmworker. A total of 12 pesticides were detected in the house-dust samples. Although this pilot project was not large enough to draw conclusions, but was intended rather to test field and lab methods, it suggests a potential for higher residential exposure to some pesticides for children of farmworkers versus children of nonfarmworkers. The study did not look at ethnicity of the homes’ residents, but the vast majority of farmworkers are Hispanic (12).

Another issue worth noting is the racial differences in childhood cancer rates. For white children, cancer incidence rates increased 9.1% from 1973 to 1990, while for black children during the same period the increase was 23.9% (13). In adults, no such difference exists between blacks and whites. The incidence rates for all cancers in both sexes increased about 19% for both blacks and whites between 1973 and 1990 (13). The causes of this disparity in children are unknown but clearly warrant further investigation.

**Drinking Water Exposure Scenario**

To establish a real world framework for considering the proposition that children of color are the subgroup of the population most exposed to environmental contaminants, we present a hypothetical scenario involving drinking water contamination in a rural agricultural area with a largely Hispanic community, a good portion of whom are farmworkers.

Consider the following factors. A unique state law requires monitoring of drinking water for chemical contaminants not currently regulated under the federal Safe Drinking Water Act. As a result of the monitoring, several pesticides and other chemicals are detected in drinking water wells, including a number of pesticides. Some of the pesticides detected are still in use, and others have been banned. In addition, xylene is detected but no obvious industrial source of the compound exists in the area. However, it has been used as an inert ingredient in pesticides applied nearby.

The community discovers results of the drinking water monitoring through the news media and is understandably scared and angry. Members of this community have voiced long-standing concerns about the health effects of pesticides, but an earlier epidemiologic investigation was inconclusive, primarily due to a small study population and lack of data on pesticide levels in the air and water.

The disclosure of the drinking water monitoring results is occurring in the midst of the state’s revision of its drinking water action level standard for one of the pesticides detected in the community’s wells. State government data also demonstrate that this pesticide is being used in fields adjacent to town. Therefore, the chemical is likely to be present in air and food as well as in the water. Also, farmworkers fortunate enough to be able to leave their children at home when they go to the fields expose their children to residues of the chemical present on their clothes when they return home. Other farmworkers may have to bring their children to the fields, providing another exposure to the pesticide.

The state decides to lower the drinking water standard for the pesticide based on new scientific evidence showing the chemical is more hazardous than previously thought. But the standard assumes the only route of exposure to the pesticide is drinking water and does not consider children’s higher intake of drinking water. The standard does not reflect the real world multiple and cumulative exposures to the pesticide for children. The farmworkers’ children in this community are the most exposed and the least protected.

**Recommendations**

Several reforms are necessary to protect children of color from environmental health threats. These changes involve immediate efforts to protect children of color from known hazards, strengthening government standards, and further research.

First, we must immediately mitigate the environmental exposures that disproportionately affect children of color. This includes lead, pesticides, and air pollution where existing data demonstrate children of color are at greatest risk.

Second, whenever federal or government agencies regulate contaminants in our air, food, water, or homes, the standards established should protect the most sensitive and most highly exposed subpopulations. More often than not, this group will be children of color. The standards must also take into account cumulative exposure to pollutants. In the real world, we are not exposed to just one chemical in one environmental medium.

Finally, we need more data to demonstrate that children of color are at disproportionate risk from environmental health threats. Additional research studies should look at representative community exposures, taking into account children’s differential exposures and the cumulative exposures. Particular sensitivity in these research efforts should be paid to involving and informing the community in an appropriate fashion.

In conclusion, children are the most vulnerable and least protected members of our society. This is especially true for children of color, who face an array of formidable challenges in their lives. Ultimately, we must work together to protect all children from environmental threats to their health. This symposium and the Children’s Environmental Health Network are critical in identifying the measures to safeguard children of color and all others from pollution.

**REFERENCES**

1. Kun V, Gordon W, Lavelle K, Mott L. Out of Breath: Children’s Health and Air Pollution in Southern California. Los Angeles: Natural Resources Defense Council, 1993.
2. Plunkett L, Tumbul D, Rodericks J. Differences between Adults and Children Affecting Exposure Assessment. In: Similarities and Differences between Children and Adults: Implications for Risk Assessment (Guzelian P, Henry C, Olin S, eds). Washington: International Life Sciences Institute, 1992: 79–94.
3. Wiles R, Campbell C. Pesticides in...
Children’s Food. Washington: Environmental Working Group, 1993.
4. Commission for Racial Justice, United Church of Christ. Toxic wastes and race in the United States: a national report of the racial and socioeconomic characteristics of communities with hazardous waste sites. New York: United Church of Christ, 1987.
5. Wennetter DR, Nieves LA. Breathing polluted air. EPA J 18(1):16–17 (1992).
6. Petition to Prohibit the Discharge of 2,3,7,8-Tetrachlorodibenzo-P-dioxin by Pulp and Paper Mills. U.S. EPA Water Management Division, Compliance Branch, Region 1, and Maine Department of Environmental Pollution, Bureau of Water Quality Control, August 23, 1993 as cited in Natural Resources Defense Council and Natural Resource Council of Maine. September 14, 1993.
7. ATSDR. The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta: Agency for Toxic Substances Disease Registry, 1988.
8. Landrigan P, Graef J. Pediatric lead poisoning in 1988: the silent epidemic continues. Pediatrics 79:582–583 (1987).
9. Carter-Porkras, O. Blood levels of 4-11 year old Mexican American, Puerto Rican, and Cuban children. Public Health Rep 388:388–395 (1993).
10. Guarnaccia P. Asthma, the Puerto Rican child and the school. In: Puerto Rican Children on the Mainland: Interdisciplinary Perspectives. As cited in Hispanics and Environmental Equity: Key Indicators. Washington: National Coalition of Hispanic Health and Human Services Organizations, 1994.
11. Bates, DV. The effects of air pollution on children. Environ Health Perspect 103(Suppl 5):115–119 (1995).
12. California Department of Health Services. Childhood Pesticide Exposure Methods Development. Memo to S. Kimberly Belshe, Director, California Department of Health Services, 24 December 1993.
13. Miller BA, Ries LAG, Hankey BF, Kosary CL, Harras A, Devesa SS, Edwards BK, eds. SEER Cancer Statistics Review: 1973–1990. National Cancer Institute. NIH Publ No 93-2789, 1993.