



Healthy Generations

Maternal & Child Health Program
School of Public Health

Children's
Environmental
Health

UNIVERSITY OF MINNESOTA

Children's Special Vulnerability to Environmental Health Risks

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Children's environmental health encompasses children's involuntary environmental exposures and susceptibilities to environmental hazards from conception through adolescence, and the policies and practices that are intended to protect them from harm

Special Concerns About Children

In recent years there has been much research to understand the reasons environmental contaminants may have a different effect on children compared to adults. Because children have a smaller body mass than adults, relative exposures to pollutants can be much larger. Also, human physiology is different at various life stages, thus children may respond to environmental insults differently from adults.

Children May Have Greater Exposure

Children have unique behaviors, diets, and physiologic characteristics that put them at greater risk for exposures to environmental contaminants. Children eat, drink, and breathe more per pound of body weight than adults. Very young children put non-food objects in their mouths, crawl on floors, and play in grass, increasing their contact with contaminants in soil and dust. Diets of children are also different from adult diets because they often eat a limited variety of foods. Adverse health effects can also result from parental exposures before conception, the time period near conception, and during pregnancy.¹ There are also a number of examples where early exposure to environmental agents results in adverse effects that are not seen until adolescence or adulthood.

Air intake. Pound for pound of body weight, children breathe more air than do adults. It is estimated that based on body weight, an infant's average air intake is twice the level of a resting adult's.^{2,3} In addition, the breathing zones of children



are closer to the floor than those of adults, where heavier chemicals such as radon tend to accumulate.^{4,5}

Food and drink consumption. In its 1993 landmark report, "Pesticides in the Diets of Infants and Children," the National Research Council (NRC) concluded that children eat more calories and drink more water than adults based on body weight.⁶ It has also been reported that, "children one through five years of age eat three to four (or more) times as much food per pound of body weight as average American adults."² Children tend to consume more of certain foods than do adults, such as fruit juice, fruit, breast milk, and cow's milk.⁶

Because children's diets are less varied and they consume more food than adults in proportion to their body weight, they may be exposed to higher levels of contaminants in food. There is a special concern about contaminants in breast milk and formula because these foods make up all or most of a young infant's diet. For some toxicants, the most significant exposure during

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Growing concern about the effect of the environment on children's health has led to increased awareness that early environmental exposures strongly influence long-term health. To paraphrase Dr. Maria Montessori, "...studies of early infancy leave no room for doubt that the first two years of life are important forever." From early stages of fetal development through adolescence, children are at particular risk of harm from environmental influences. Children differ from adults because they are undergoing dramatic changes in physiology associated with their development. Children have lower body mass than adults so they take in proportionately more food, drink, and air; thus, the dose of potentially toxic agents can be much higher than in adults. Children are always exploring their local environment and they lack avoidance of potential hazards that could imperil their health.

The articles in this issue focus on special concerns about children's exposure and sensitivity to the environment. An underlying theme is that our understanding of child-environment interaction is maturing, recognizing contributions of socioeconomic status, the built environment, man made and naturally occurring pollutants, personal care products, drugs, and nutrition to the overarching environment. As the song by the *House of Pain* implies, the environment influences us from the womb to the tomb. We thank the authors for sharing their insight and their commitment to creating a healthy environment for our children, who represent the future of humankind.

- William Toscano, Jr., PhD and Erica L. Fishman, MSW, MPH

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infant development is through breast milk. However, at this time, health scientists believe the benefits of breastfeeding outweigh the risks.⁷

Dermal exposure. Children have a higher skin surface area to body weight ratio and greater contact with their surroundings than adults.⁸ Therefore, a child may absorb through their skin a larger dose of a pollutant on a body weight basis than would an adult. Newborns, especially those who are preterm, experience increased absorption of some compounds through the skin.⁹

Transplacental exposure. Depending on the timing of the exposure, different health effects may result from *in utero* exposure to pollutants. Some chemicals that enter a pregnant woman's body will cross the placenta and may even concentrate in fetal tissue.¹⁰ Available data on fetal exposure to diethylstilbestrol (DES), thalidomide, alcohol, and tobacco smoke show significant adverse health effects.¹¹

Children have unique behaviors. As children pass rapidly through developmental phases, their activity patterns and behaviors change. Newborns spend prolonged periods of time in a single environment and are unable to voluntarily decrease exposure.⁴ Toddlers are more mobile and spend more time on the ground. Behaviors such as pica (desire to eat non-food substances), crawling, and oral exploratory activity increase ingestion of contaminants on surfaces. For example, children can experience greater exposure to pesticide residues from grass, lead dust from floors, volatile organic chemicals from carpets, and chromated copper arsenate (CCA) from wooden playground equipment.⁴ Of special concern are hazards in day care facilities, school buildings, gymnasiums, playgrounds, and teenagers' workplaces.

Children May Be More Sensitive

Children are still developing. From birth through childhood, children differ from adults in their ability to absorb, metabolize, and excrete contaminants. Detoxifying enzyme systems develop throughout childhood, so the ability to mitigate the effects of chemicals is age-dependent.¹¹ Children may be less able to eliminate some compounds because, for example, they are less efficient at excreting contaminants *via* the kidneys or bile.¹² Therefore, some contaminants have toxic effects during childhood that an adult does not experience.

Evidence also shows that the unique developmental stages experienced in childhood make children more vulnerable to harmful effects from exposures to certain hazards. As it passes through different stages of development, the brain is uniquely sensitive to some contaminants. For example, adverse effects from lead differentially affect children because their brain is developing and they are more exposed. The developing brain is also sensitive to exposure to organic mercury (methylmercury). *In utero* exposure to methylmercury can affect the developing brain of the fetus at the same time the mother will experience only mild adverse effects.¹³

Children have a longer future. Diseases requiring chronic exposures and long latency periods are likely to have more serious impacts when children are exposed throughout their lifetime beginning at an early age.^{14,15} Furthermore, cancer risk is believed to be "front-loaded." Some scientists have found that a large portion of a lifetime risk of cancer from some environmental carcinogens is actually due to exposures early in life rather than the total exposure throughout life.¹⁶

A Broader Look

According to the World Health Organization (WHO), "almost one third of the global burden of disease can be attributed to environmental risk factors, over 40% of this burden falls on children under 5 years of age...who constitute no more than 10% of the world's population."¹⁷ Around the world, the hazards that children face vary greatly and are largely dependent on geographic, economic, and social determinants. Poverty, especially, can exacerbate environmental risk factors.¹⁸

The WHO has categorized environmental health risks to children as "basic," "modern," and "emerging."¹⁸ Basic risks are unhealthy housing, unsafe water supply, lack of sanitation, indoor air pollution, and leaded gasoline. Risks that are considered modern are chronic respiratory illness and asthma, injuries from transport accidents, toxic chemicals, and neurodevelopmental and behavioral effects. Emerging risks are, for example, endocrine disruptors, environmental allergens, and UV radiation. Basic risks are more significant for children in poverty-stricken countries and modern risks plague children in industrialized countries. As populations make a transition from low to high income, basic risks decrease and modern risks increase. Emerging risks tend to affect all children.¹⁸ Because living conditions and environmental health risks vary so greatly, interventions and priorities for action need to be carefully tailored to be most effective.

Environmental Health Hazards

There are various interpretations of what hazards are considered environmental in origin. Environmental factors include contaminants that are naturally present, manufactured or used by humans for a specific purpose, or by-products of an activity.¹⁹ The built environment, consumer products, and social factors may also affect health.

Examples of naturally-present hazards include radioactive materials such as rock and soil that emit radon, metals such as arsenic that contaminate drinking water, particulate matter that results from forest fires, wind, and erosion, infectious pathogens that contaminate food, and dust mites that can cause allergic responses in sensitized children. Examples of contaminants manufactured or spread through the environment by humans include pesticides used on food crops, lead used in paint or automobile gasoline, and phthalates used in children's toys. By-products of human activities and production processes include: chloroform in drinking water resulting from chlorination, nitrogen oxides from combustion, and many hazardous compounds in cigarette smoke.

The built environment and consumer products also affect child health and safety.²⁰ For example, toys and art and craft products intended for children can contain substances that can be toxic and may cause choking or injury.¹³ Products that are designed for adult use may also be hazardous to children. Poorly designed playground equipment or stairways may contribute to unintentional injuries. Urban planning that does not take into account the public's health may include unsafe cross walks that discourage walking. Greater automobile use, in turn, can result in higher levels of pollution from exhaust, greater incidence of injuries from collisions, and lower levels of physical activity.²¹

There are also many social factors that affect children's health and may act in combination with environmental hazards to exacerbate adverse health effects. For example, exposure to environmental contaminants likely varies based on the socioeconomic status of the child.²² Factors that may be affected by a child's socioeconomic status include the

distance he or she lives from the source of pollution, whether the home is in an urban, suburban, or rural area, the condition and age of the home, and the quality of the drinking water supply.²² In some cases, environmental health effects are moderated by factors such as nutritional status and level of parental stimulation during development.²³

The Future

As the ever-expanding population places more demands on natural resources and land use, we struggle with balancing environmental and health protection with development, technological advancements, and economic growth. Political representatives, government agencies, health organizations, public interest groups, and the general public have recognized and embraced children's environmental health as an important issue worthy of resource commitments and policy ingenuity. Outreach to parents, teachers, and childcare providers has increased and led to individual actions to reduce children's exposures to environmental dangers. Innovative research methods have expanded the ability to study environmental and health issues in fields such as toxicology, epidemiology, exposure assessment, genomics, and engineering. Greater attention is also being paid to legal, political, and ethical issues surrounding children's environmental health.

Today we have more resources to undertake quality research on exposures and health effects and more awareness of the role the environment plays in human health. Among the fairly new chemicals emitted into the environment, some have been identified as toxic and others will likely be identified as harmful in the future as more research is conducted. Evidence accumulates over time that the benefits of some products, such as lead paint, no longer outweigh the risk involved with that exposure, especially when affordable, accessible alternatives exist. As we look toward the future, we will need to find innovative ways to prevent harm before children are exposed to environmental hazards.

Exercising precaution starts at home. Individuals can take actions to make the environments where children play, learn, and live safer, become involved in political and community organizations, and support other cooperative efforts to improve children's health. Partnerships among multiple organizations will yield the most effective and protective actions to protect children's health.

Additional information is available from URL:
<http://www.health.state.mn.us/divs/eh/children/index.html>

References

1. Altshuler K, Berg M, Frazier LM, Laursen J, Longstreth J, Mendez W, et al. Critical periods in development: OCHP paper series on children's health and the environment. Paper 2003-2, U.S. Environmental Protection Agency; February 2003. Available from: URL:<http://yosemite.epa.gov/ochp/ochpweb.nsf/content/paperdownloads.htm>. Accessed December 23, 2003.
2. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services. Healthy children-toxic environments: acting on the vulnerability of children who dwell near hazardous waste sites. Report of the Child Health Workgroup Presented April 28, 1997 to the Board of Scientific Counselors. Available from: <http://www.atsdr.cdc.gov/child/chw497.html>. Accessed December 23, 2003.
3. U.S. Environmental Protection Agency. Child-specific exposure factors handbook. EPA-600-P-00-002B; September 2002.
4. Bearer CF. Environmental health hazards: how children are different from adults. The Future of Children: critical Issues for Children and Youths. 1995 Summer/Fall;5(2):11-26. Available from: http://www.futureofchildren.org/usr_doc/vol5no2ART2.pdf. Accessed February 2, 2004.
5. Goldman LR. Case studies of environmental risks to children. The Future of Children: critical Issues for Children and Youths. 1995 Summer/Fall;5(2):27-33. Available from: http://www.futureofchildren.org/usr_doc/vol5no2ART2.pdf. Accessed February 2, 2004.
6. National Research Council. Pesticides in the Diets of Infants and Children. Washington, DC: National Academies Press; 1993.
7. Pronczuk J, Akre J, Moy G, Vallen C. Global perspectives in breast milk contamination: infectious and toxic hazards. Environ Health Perspect 2002 June;110(6):A349-A351. Available from: URL: <http://ehpnet1.niehs.nih.gov/members/2002/110pA349-A351pronczuk/EHP110pA349PDF.PDF>. Accessed January 16, 2004.
8. Faustman EM, Sibernagel SM, Fenske RA, Burbacher TM, Ponce RA. Mechanisms underlying children's susceptibility to environmental toxicants. Environ Health Perspect 2000 March;108(Suppl.1):13-21.
9. U.S. Environmental Protection Agency. Dermal exposure assessment: principles and applications. EPA 600/8-91/011B; January 1992.
10. Timbrell J. Principles of biochemical toxicology. 3rd ed. Philadelphia: Taylor and Francis; 2000.
11. Selevan S, Kimmel CA, Mendola P. Identifying critical windows of exposure for children's health. Environ Health Perspect 2000 June;108(Suppl. 3):451-5.
12. Miller MD, Marty MA, Arcus A, Brown J, Morry D, Sandy M. Differences between children and adults: implications for risk assessment at California EPA. Int J Toxicol 2002;21:403-18.
13. Etzel RA, Balk SJ, editors. Handbook of pediatric environmental health. American Academy of Pediatrics, Committee on Environmental Health; 1999.
14. Landrigan PJ, Carlson JE, Bearer CF, Cranmer JS, Bullard RD, Etzel RA et al. Children's health and the environment: a new agenda for prevention research. Environ Health Perspect 1998 June;106(3):787-94.
15. Goldman L. Children's Environmental Health. Presented at Environmental Protection Agency Workshop on Information Needs to Address Children's Cancer Risk. Summary of the Workshop on Information Needs to Address Children's Cancer Risk, EPA/600/R-00/105; December 2000.
16. U.S. Environmental Protection Agency. Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens (External Review Draft). Risk Assessment Forum, Washington, DC. EPA/630/R-03/003: 2003.
17. World Health Organization. Press Release. Available from: URL: <http://www.un.org/News/Press/docs/2002/icef1858.doc.htm>. Accessed December 23, 2003.
18. World Health Organization. Healthy environments for children: initiating an alliance for action; 2002.
19. Carpenter DO, Arcaro KF, Bush B, Niemi WD, Pang S, Vakharia DD. Human health and chemical mixtures: an overview. Environ Health Perspect 1998 December;106(Suppl. 6):1263-70.
20. Tibbetts J. Building awareness of the built environment. Environ Health Perspect 2002 November;110(11):A670-2.
21. Cummins SK, Jackson RJ. The built environment and children's health. Pediatr Clin North Am 2001 October;48(5):1241-52.
22. Hubal EAC, Sheldon LS, Burke JM, McCurdy TR, Berry MR, Rigas ML, et al. Children's exposure assessment: a review of factors influencing children's exposure, and the data available to characterize and assess that exposure. Environ Health Perspect 2000 June;108(6):475-86.
23. Jacobson JL, Jacobson SW. Breast-feeding and gender as moderators of teratogenic effects of cognitive development. Neurotoxicol Teratol 2002;24:1-10.

The information in this article was primarily compiled and condensed from the Minnesota Department of Health web site by Shawna Hedlund, MPH, a recent graduate of the Maternal and Child Health Program in the School of Public Health at the University of Minnesota. Other sources were also used.

Upcoming Event Environmental Threats to Children's Health: Legal and Policy Challenges

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School Health Initiative: Environment, Learning, and Disease (SHIELD) Study

John Adgate, PhD

It is widely believed that inner city settings have higher environmental risks than other urban and non-urban environments, but there are little data on children or for populations that make up the bulk of inner city residents.^{1,2} Children are more susceptible than adults to environmental hazards and children of low socioeconomic status have higher adverse health outcomes than other children.³ Concern about environmental equity prompts special interest in minority populations. To address these concerns scientists from the Division of Environmental Health Sciences at the University of Minnesota School of Public Health worked in partnership with the Minneapolis School District, Minnesota Department of Health, and other collaborators to examine environmental exposures in children.

Most school-age children spend about 20 hours per day, or about 85% of their time, indoors, primarily at home or in school. Indoor environments are thus important sources of potentially toxic exposures. Homes contain a broad range of potential hazards (e.g., environmental tobacco smoke [ETS], airborne particles from heating and cooking, allergens, lead from paint, bleaches, organic solvents, and pesticides), but are difficult to study in sufficient numbers and detail to quantify these risks for a substantial number of children. Schools also present various hazards but unlike homes, schools offer an opportunity to study large numbers of children at fixed locations where the indoor environment can be assessed comparatively easily. Schools therefore present possibilities for quantifying exposures, doses, and health effects among children, and for examining multiple exposures to toxic hazards at relatively low exposure levels.

Data Moment *Teratogens*

Several articles in this issue discuss teratogens. Teratogens are external agents (chemical or physical) that damage embryonic or fetal development. Our modern understanding of such agents is short. It was in 1941 that an Australian ophthalmologist first showed that infection with Rubella was associated with birth defects. An important indicator of environmental toxins occurred in Japan in 1956 with mercury exposure in fish, resulting in Minamata Disease. Thalidomide, which was prescribed to pregnant women in the early 1960s, showed us that a non-toxic drug could cause specific malformations. However, not all drug-induced teratogens can be identified because of obvious, and specific, birth defects. Experiences with DES, prescribed in the 1940s and 1950s to reduce fetal loss in high-risk pregnancies, showed that the effects of drug exposure *in utero* may not all manifest at birth. By 1970, a clear association between *in utero* exposure and adenocarcinoma of the vagina in women was established. Later work suggested a relationship between reproductive cancers and men exposed *in utero*. The researcher has several challenges in studying the teratogenic effects of prescribed drugs. First, as with DES, the outcomes may not manifest at birth—but occur decades later in adult offspring. Second, the careful researcher must also consider *why* drugs are prescribed. For example, an association between an antibiotic and a birth defect may not be associated with the medication, but rather to the infection for which the drug was prescribed.

The School Health Initiative: Environment, Learning, and Disease (SHIELD) is one of the first to systematically explore children's exposure to complex mixtures of a wide array of environmental agents, including volatile organic chemicals (VOCs), ETS, allergens, bioaerosols, metals, and pesticides. SHIELD explored links between these various classes of pollutants to



identify highly exposed subpopulations so future studies can explore links between real world mixtures of pollutants and disease outcomes. In a pilot epidemiology module SHIELD investigators are exploring links between these exposures and effects on both respiratory health (assessed by spirometry and peak flow data) and learning outcomes (using standardized test scores, measures of academic performance, and attendance). SHIELD was designed to take advantage of a unique opportunity using a natural experiment that exists in the Minneapolis Public Schools. Children in grades 2-5 from two inner-city schools serving predominantly low-income households (~80% qualified for free or reduced price meals in the National School Lunch/Breakfast Program) were recruited between November 1999 and January 2000 and monitored for two years. One school (Whittier) was recently constructed and particular attention was given to using building materials that were unlikely to present environmental health risks, by avoiding carpets and other floor coverings that were likely to retain dust and other hazardous agents, and by ensuring adequate ventilation. The second school (Lyndale) was built in the 1970s when building construction emphasized energy efficiency. It was thought that the older school likely presented greater environmental health risks to children.

This study will provide important new information concerning: (i) school environments as a possible source of toxic exposures and health risks; (ii) rates of illnesses among urban minority children; and (iii) exposure-response information linking multiple environmental risks to respiratory health and learning outcomes. To date investigators have completed analyses on the student recruitment process² and exposure to ETS.⁴ Statistical analyses of exposure to specific classes of chemicals (i.e., school indoor air quality; in-school, in-residence, and personal VOC levels), analyses of associations between classes of chemicals (i.e., pesticides and metals), and exploration of the associations between these exposures to health effects and learning outcomes are pending.

The overall enrollment rate in year 1 was 57% of all students. There was a substantial disparity between children from English-speaking (42%) versus non-English-speaking (71%) families. Of the students enrolled in the study, 85% remained in the study after year 1. Personal VOC levels were collected using small diffusion samplers, and health

related samples or data were obtained from most subjects. Study personnel or parents helped with some data collection, such as time-activity diaries, and children were active participants in the study. For example, nearly all indoor air samples in both schools were valid, a relatively high percentage of children provided both blood (82%) and urine (86%) samples in year 1, and 90% provided a valid spirometry sample. These results indicate that a school-based research design makes it feasible and practical to conduct probability-based assessments of children's environmental health in economically disadvantaged and ethnically diverse neighborhoods. There is an ongoing need, however, to improve understanding of the cultural, economic, psychological, and social determinants of study participation among this population.

Exposure assessment results indicate that, for most measured indoor air quality parameters (i.e., building comfort parameters, VOCs, carpet allergens, and fungal counts) the two schools were not substantially different. This somewhat surprising finding is because most pollutant concentrations were very low in both schools, likely due to good building control and maintenance practices by the Minneapolis School District. Some of our other results point to the home environment being a major source of exposure to other chemical hazards. For example, in our analysis of urinary biomarkers we found widespread exposure to tobacco-specific lung carcinogens, and an analysis of VOC levels indicates that VOC levels in both schools were low and that children's personal exposures are much more strongly influenced by the residential

environment. Further analyses will explore these questions in more depth, as well as potential links to health and learning outcomes.

References

1. Sexton K, Greaves IA, Church TR, Adgate JL, Ramachandran G, Tweedie RL, et al. A school-based strategy to assess children's environmental exposures and related health effects in economically disadvantaged urban neighborhoods. *J Expo Anal Environ Epidemiol* 2000;10:682-94.
2. Sexton K, Adgate JL, Church TR, Greaves IA, Ramachandran G, Fredrickson AL, et al. Recruitment, retention, and compliance results from a probability study of children's environmental health in economically disadvantaged neighborhoods. *Environ Health Perspect* May 2003;111(5):731-6
3. Institute of Medicine (IOM). *Toward Environmental Justice: Research, Education, and Health Policy Needs*. Washington, D.C.: National Academies Press; 1999.
4. Hecht SS, Ye M, Carmella SG, Fredrickson A, Adgate JL, Greaves IA, et al. Metabolites of a tobacco-specific lung carcinogen in the urine of elementary school-aged children. *Cancer Epidemiol Biomarkers Prev* Nov 2001;10(11):1109-16.

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Interested in making a difference?

Consider a Master's in Public Health (MPH) Degree in Maternal and Child Health (MCH)

First year MCH student Liz Radel was drawn to the Maternal and Child Health program because of its focus on women's health issues, especially reproductive and sexual health, and the emphasis on social justice. After looking into schools of public health throughout the country, she chose this program because it allowed her to pursue her interests in MCH and data analysis through the optional MCH epidemiology curriculum.



In one semester, Liz has had numerous opportunities for hands-on experiences learning about the field. Through her research assistantship, she completed a drug pricing survey for a group of Minnesota family planning providers, and reported the findings. Liz participated in the School of Public Health's mentor program where students are matched up with public health professionals. "The mentor program has allowed me to understand some of the challenges and benefits of working in the field of public health through the eyes of my mentor," said Liz. "I feel much better prepared to enter the field from this experience." Liz has also had the opportunity to attend conferences sponsored by the University for health professionals and interview staff of public health agencies as a part of her coursework.

In addition to the hands-on learning experiences, classroom instruction is an important part of the curriculum. "Our faculty provide each student with many opportunities to gain a greater understanding about the field of public health," Liz reports. "I have been pleasantly surprised to see how much I've learned from my fellow classmates. MCH students enter the program with experiences in fields including social work, psychology, medicine, and public health. In our interactive, discussion-oriented classes, we review MCH public health issues, benefiting from the expertise of our professors and each other. In this program, we build solid personal and professional relationships with our classmates - the people who will soon be our colleagues in the field." For more information about the MCH program at the School of Public Health, read on.

What is the MCH Program? It is a training program for MPH students who are interested in promoting and preserving the health of families, including women, children, and adolescents. The Program is in the Division of Epidemiology in the School of Public Health at the University of Minnesota.

Who should apply? People who care about vulnerable populations and want careers in program planning and development, evaluation, surveillance, assessment, teaching, or research. The program offers a special emphasis on MCH epidemiology for interested students. Clinical professionals, and others with advanced degrees who are interested in administering MCH-related health programs or conducting research projects are also encouraged to apply. Individuals with advanced degrees may have the option of completing the two-year MPH Program in one year.

For further information about the MCH Program. Call 612-626-8802 or 1-800-774-8636; email gradstudies@epi.umn.edu; or visit <http://www.epi.umn.edu/mch> and <http://www.umn.edu/sph>.



Children Living in a Perilous Environment

William Toscano, Jr., PhD, Charity Hovey, Jody Hulne, Jeff Sturm, Elizabeth Thompson

Ancient civilizations recognized that the environment was involved in human disease.¹ As experimental science advanced, microorganisms and viruses were found to play a role in some human disease, which led to the concept known as the *Germ Theory of Disease*.² As the advances of modern science allowed researchers to delve into the cellular and molecular basis of disease, the idea of the environment playing a direct role in human diseases became increasingly ignored.³ By the second half of the twentieth century, much of the thinking changed to focus only on our genetic makeup and that genes were solely responsible for human diseases. Thus, it was proposed by some that there were “bad genes” that ran in families and they were the cause of diseases such as cancer or birth defects.⁴

Recently, a greater appreciation for the role of the environment in disease etiology has resurfaced.⁵ Our health is the summation of not only our genetic makeup (*genome*) but also multiple exposures from our surroundings (*envirome*). Everything we ingest and where and how we live and behave has an effect on our health. We are exposed to about 90,000 different compounds in our daily lives. Approximately 488 of these compounds are known or suspected cancer-causing (*carcinogenic*) agents. Another 250 of those compounds are known to have a detrimental impact on developing fetuses.⁶ An unknown number of the 90,000 compounds are hormone-active agents that may also cause cancers or birth defects.^{7,8}

It is now widely recognized that many diseases result from interactions of the environment with our genes.^{9,10} The field of *Enviromics* is about how the environment interacts with genes at critical life stages to cause disease. The environment can interact with human genes either directly or indirectly. Direct interaction often results in a mutation in the DNA. Indirect interactions usually involve processes that affect expression levels of specific genes. Both direct and indirect gene-environment interactions can lead to complex human diseases such as spina bifida, cleft palate, obesity, diabetes, a variety of cancers, lung and cardiovascular diseases, and neurological diseases.^{11,12}

The question many people have is “how does the environment affect our children’s health?” There is no simple answer to this question. To provide answers to this question, environmental health scientists are using an integrated approach, called *Systems Biology*, to link data from the environment, exposure, and biology. This approach will yield a clear understanding of the action of environmental agents on child health and a determination at which life stages particular genes are most vulnerable to exposures.

Children are vulnerable to environmental insults *in utero*, as newborns, and during their development into adolescence. These are all developmental stages of dramatic change in physiology; however, the most vulnerable stage is *in utero*. This is a time of sequentially tightly

regulated growth and cell division. In some instances, environmental interactions in the womb have dramatic effects on the newborn and appear to exert health effects that transcend generations.

Some examples of *in utero* exposure to external foreign compounds leading to disease are well established. Thalidomide, a drug used by pregnant women in Europe during the 1950s to prevent nausea in early pregnancy, caused limb and other specific deformations in their offspring. The particular deformity depended on the stage of fetal development at which the drug was ingested.¹³

Another example of chemically induced disease comes from children whose mothers were prescribed diethylstilbesterol (DES) during pregnancy. DES is a compound that was supposed to prevent miscarriage. In the United States DES was widely used from 1941 through the early 1970s.^{7,14} The children at birth were “normal,” with no apparent birth defect. However, as the children grew, reproductive problems presented themselves. Daughters of women exposed to DES have higher rates of vaginal cancers than non-exposed women and they have high infertility rates.^{15,16} Sons of DES-exposed women are less well studied than their daughters, but increased rates of testicular cancer have

been reported.¹⁷ The transgenerational effects of DES are of great concern. For example, it has been reported that boys born to DES daughters are at a higher risk for hypospadias, a condition in which the urethra does not properly exit the penis at the tip.¹⁸

Thalidomide and DES are examples of prescription drugs that had dramatic and sinister effects on child health. There is growing concern that chronic low-dose exposures to other environmental agents such as pesticides, plasticizers, and food additives may have similar effects on human development.¹⁹ Data from animal

studies suggest chemicals in our everyday environment can have deleterious effects accounting for common diseases that are increasing in incidence among humans. Examples include obesity, type II diabetes, Parkinson’s disease, and endometriosis.^{20,21} However, relating exposures to specific outcomes and identifying susceptible individuals are among the most difficult problems in child environmental health.²²

Like mind and body or mother and fetus, humans and environment are not isolated systems; what affects one has a direct impact on the other. Our ancestors recognized that the quality of the environment could affect health, although their environment was considerably less complex than ours. Thalidomide and DES provide poignant examples of the direct impact of maternal exposure on offspring. The DES example also suggests that these effects may transcend generations. Our lack of knowledge about the subtler effects of human-made chemicals in the light of transgenerational effects of DES should signal a cause for concern and a desire to become more aware about specific exposures from the world around us. Increasing our understanding of the effects of exposure from the envirome will allow us to pinpoint human predisposition to deleterious effects and will help us predict more accurately risks to child health.



References

1. Binswanger HC, Smith KR. Paracelsus and Goethe: founding fathers of environmental health. *Bull World Health Organ* 2000;78:1162-4.
2. Bynum WF. The evolution of germs and the evolution of disease: some British debates, 1870-1900. *Hist Philos Life Sci* 2002;24:53-68.
3. Prost A. From disease to health: the individual, society, environment and culture. *Sante* 1995;5:331-3.
4. Epstein RJ. Bad genes, bad diseases, and bad luck. *Quart J Med* 1998;91:861-4.
5. Schell LM, Denham M. Environmental pollution in urban environments and human biology. *Annu Rev Anthropol* 2003;32:111-34.
6. COEHHD. Proposition 65 List of Chemicals. 2003. Available from: http://www.oehha.ca.gov/prop65/prop65_list/Newlist.html. Accessed January 26, 2004.
7. McLachlan JA. Environmental signaling: what embryos and evolution teach us about endocrine disrupting chemicals. *Endocrine Rev* 2001;22:319-41.
8. Schettler T, Solomon G, Valenti M, Huddle A. Generations at risk: reproductive health and the environment. Cambridge, MA: MIT Press; 1999.
9. Olden K, Gutherie J. Genomics: implications for toxicology. *Mut Res* 2001;473:3-10.
10. Olden K, Wilson S. Environmental health and genomics: visions and implications. *Nat Rev Genet* 2000;1:149-53.
11. Bianchi F, Calzolari E, Ciulli L, Cordier S, Gualandi F, Pierini A et al. Environment and genetics in the etiology of Cleft Lip and Cleft Palate with reference to the role of Folic Acid. *Epidemiol Prev* 2000;24:21-7.
12. Schork NJ. Genetics of complex disease. *Amer J Respir Crit Care Med* 1997;156:S103-9.
13. Nathanielsz PW. Life in the womb: the origin of health and disease. Ithaca, NY: Promethean Press; 1999.
14. Braun ML, Stuart NM. DES Stories. Rochester, NY: VSW Press; 2001.
15. Fowler WC Jr, Edelman DA. In utero exposure to DES: evaluation and follow-up of 199 Women. *Obstr & Gynecol* 1978;51:459-63.
16. Kruse K, Lauver D, Hanson K. Clinical implications of DES. *Nurse Pract* 2003;28:26-32.
17. Vessey M P. Epidemiological studies of the effects of Diethylstilbesterol. *IARC Sci Publ* 1989;96:335-48.
18. K lip H, Verloop J, van Gool JD, Koster ME, Burger CW, van Leeuwen FE et al. Hypospadias in sons of women exposed to Diethylstilbesterol *in utero*: a cohort study. *Lancet* 2002;359:1102-7.
19. Guillette EA, Meza MM, Aguilar MG, Soto AD, Garcia IE. An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico. *Environ Health Perspect* 1998;106:347-53.
20. Francis GA, Fayard E, Picard F, and Auwerx J. Nuclear receptors and the control of metabolism. *Annu Rev Physiol* 2003;65:261-311.
21. Heindel JJ. Endocrine disruptors and the obesity epidemic. *Toxicol Sci* 2003;76:247-9.
22. Longnecker MP, Wolff MS, Gladen BC, Brock JW, Grandjean P, Jacobson JL et al. Comparison of Polychlorinated Biphenyl levels across studies of human neurodevelopment. *Environ Health Perspect* 2003;111:65-70.

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News From the Minnesota Department of Health Environmental Health Division

Minnesota Birth Defects Prevention and Information System

It is estimated that each year, over 2,000 babies in Minnesota and an estimated 150,000 babies nationwide are born with serious birth defects. Birth defects are the leading cause of death in children less than one year of age, causing one in every five deaths. Birth defects include a variety of conditions with many different manifestations and with many different etiologies. Approximately 20% of birth defects may be attributed to genetic factors, another 10% attributed to environmental factors (including drug or alcohol abuse, infections, or exposure to certain medications or other chemicals), and the causes of the remaining 70% are currently unknown.

The MDH Environmental Health Division has received a three-year planning and implementation grant from the CDC to establish the Minnesota Birth Defects Prevention and Information System. Current activities include a comprehensive review of available data for quality, pilot studies to refine methods, formation of a multi-disciplinary Birth Defects Advisory Workgroup, and the development of plans for creating primary prevention programs and assuring that children with birth defects receive appropriate services in a timely manner. The long-term goals of the program will be to:

- ◆ monitor incidence trends of birth defects to detect emerging health concerns and identify affected populations,
- ◆ provide services to affected families,
- ◆ prevent birth defects through targeted education, and
- ◆ stimulate research on risk factors, treatment, prevention, and cure of birth defects.

Better tracking of when and where birth defects occur and potential links to environmental and other factors would provide critical information to help prevent them. Without a system in place, the assessment of disease trends is very difficult and the development of sound public health policy based on scientific data is nearly impossible. For more information on Minnesota's programs, contact Daniel Symonik at MDH at 651-215-0776 or at daniel.symonik@state.mn.us. Information on national surveillance efforts can be accessed at <http://www.cdc.gov/ncbddd/bd/bdsurv.htm>.

Children's Environmental Health Economics

Environmental regulations applied to hazardous chemicals are intended to reduce health risks associated with exposures. Regulators must consider the health costs and benefits of reducing exposures to chemical hazards. Many people who advocate for children's health feel that benefits to children should be considered separately from the general population and that our environmental health regulations should focus on protecting children—and protecting them to a greater extent than we protect adults.

Economics is used to analyze the costs to achieve health benefits and to determine the monetary value of health benefits such as the quality or length of life. The use of economics in environmental decision-making is growing and even mandated by the federal government. However, the environmental health economics work that has been conducted does not take into account whether there is a greater perceived societal benefit or value from protecting children than protecting adults; and, if so, the extent and magnitude of this perception.

In 2003 the Minnesota Department of Health sought and received special funding from the state legislature to study how environmental health economics could be used to place a monetary value on the societal value for protecting children and adults. The U.S. Environmental Protection Agency's National Center for Environmental Economics and economists from academic institutions around the state worked with the Minnesota Department of Health to develop a "willingness-to-pay" analysis for the children versus adult valuation question. The collaborators are designing a survey, using standard valuation techniques, to determine the dollar amounts that adults are willing to pay to protect children compared to themselves or other adults from risks of cancer. The intent is to survey a representative sample of adult Minnesotans about protecting their family or the general population. The survey will be carried out in 2004.

Using environmental health economics to answer policy questions such as these is controversial. However, the data collected from the survey will be only one of many necessary and useful pieces of information that help the Minnesota Department of Health develop rules and guidelines for environmental health hazards. Additional information is available at: <http://www.health.state.mn.us/divs/eh/children/environmental.html>.



David Wallinga, MD, MPA and Kathleen Schuler, MPH

Children are especially vulnerable to environmental toxicants because of diet, physiology, and behaviors that may expose them to higher levels of toxicants than adults, and the inherent susceptibility of their developing organs.¹ Of those developing organs, the brain may be particularly vulnerable. In 2000, industrial releases of the nation's top 20 chemicals alone included about two billion pounds of known or suspected chemical neurotoxicants.²

An estimated 17% of U.S. children under age 18 have one or more developmental disabilities.³ New evidence indicates that these disabilities are caused by interaction between social factors, genetic factors, and exposures to some of the better-tested environmental neurotoxicants, like lead, mercury, polychlorinated biphenyls (PCBs), as well as alcohol and other solvents.³ Thousands of other neurotoxic pollutants have never been tested for their impact on early brain and nervous system development. Animal and human studies have found that even very low-level exposures to these compounds early in life can disrupt brain development, with later impacts on intelligence, behavior, learning, and attention. These common, widespread pollutants are important, preventable contributors to learning and developmental disabilities in children. This article focuses specifically on two ubiquitous environmental neurotoxicants, methylmercury and flame retardant chemicals.

Mercury readily crosses the placenta, while flame-retardants are soluble in fat—rapidly accumulating in breast milk—ensuring early childhood exposure. Prudent public policy to protect children demands not only reducing a child's exposure to these agents, but also primary prevention to prevent emission of these neurotoxicants into the environment in the first place.

These neurotoxic agents present a special public health challenge because exposure is associated with behaviors that are promoted in public health: fish consumption and breastfeeding. Fish is generally a lean protein source that contains heart-healthy fatty acids. Breast milk is known to be the finest food source for infant health and development and the act of breastfeeding may have positive effects on maternal-infant bonding.

Scope of the Problem

Methylmercury contaminates much of our supply of salt and freshwater fish, including canned tuna.³ Eating fish is the chief route of human exposure. The CDC recently reported that 8% of women of childbearing age currently carry a body burden of mercury that could put their offspring at risk for adverse developmental effects, potentially affecting 325,000 newborns a year.⁴ Results from large-scale epidemiological studies suggest that fetal and neonatal mercury exposure through maternal fish consumption can adversely affect children's brains, resulting in impaired brain function; specifically impaired are motor, attention, visuospatial, language, and memory functions.⁵

Mercury is ubiquitous in the environment. Human industrial activities like coal-fired power plants, taconite processing, and municipal and medical waste incineration are the major sources of elemental mercury emitted into the environment. Upon release, mercury can travel globally and settle into aquatic environments where bacteria convert it into the more toxic methylmercury, which accumulates in fish.

Virtually every fish, whether caught in Minnesota waters or bought at the grocery store, has some level of mercury. No cooking or cleaning method can reduce the mercury in fish. Because methylmercury passes easily across the placenta and to breastfeeding infants, women of childbearing age and young children are advised to follow fish consumption advice to avoid harmful exposure to this toxicant. Obviously, asking women and children to forego or reduce fish consumption is not a good long-term solution to this public health problem. (Please visit the Minnesota Department of Health at: <http://www.health.state.mn.us/divs/eh/fish/index.html> for information on safe levels of fish consumption.)

Lesser-known is the public health threat posed by flame retardant chemicals—the polybrominated diphenyl ethers (PBDEs)—used in foam products, textiles, lubricants, electrical equipment, building materials, and transportation. Because the chemical structures of PBDEs are similar to PCBs, there is great concern about PBDE exposure. PCBs were banned in 1976 due to their high toxicity and environmental persistence. Neurotoxicity of PCBs is well established. In one large cohort study, children born to women who consumed Lake Michigan fish prenatally and who had the highest serum and breast milk levels of PCBs, demonstrated lower IQ scores and worse performance on tests of reading comprehension, attention, and memory. Some of the findings occurred at PCB levels only slightly higher than that found in the general population.⁶

Like PCBs, PBDE flame-retardants have accumulated in the environment in fish and meats, breast milk, and humans. Laboratory studies in animals indicate that PBDEs, like PCBs, are neurodevelopmental toxicants⁷ and disruptors of hormone function including thyroid hormone.⁸ While PCB levels in fish and breast milk have slowly declined since being banned, PBDE levels are increasing at an exponential pace as they are still largely unregulated in the U.S. One study showed a 100-fold increase in PBDEs in Lake Ontario trout between 1978 and 1998.⁹ Likewise, PBDEs in human milk are steadily increasing. Recent research found levels of PBDEs in U.S. women's breast milk to be 10–100 times higher than reported levels in European women.¹⁰ We are learning that PBDEs are leaving a lasting toxic legacy in the environment and in human beings.⁸

Public Health Prevention Means Source Reduction

The main thrust of current public policy at both the state and national levels is to attempt to reduce exposure to contaminants like methylmercury. This takes two forms: (i) setting health-based standards for contaminant levels and (ii) providing safe fish consumption advice. In the former case, state and federal agencies determine a “safe” level of contaminant, as dictated by risk assessment, which determines fish consumption advice by state and federal agencies. Agencies target their advice toward high-risk groups like women of childbearing age and children.

A critical flaw in this process is that the safe levels of these toxicants are unknown. In fact, with mercury and PBDEs—as many now believe is true for lead and PCBs—there may be *no* level of exposure that is “safe” for early brain development. The history of scientific inquiry around lead, mercury, and PCBs shows that as methods to measure toxic injury from low-level exposures have improved, we observe adverse effects at lower and lower levels.³

While reducing exposures is necessary, it is not enough. Primary public health prevention requires eliminating the pollution that is contributing to these health problems. To make breast milk safer for infants, and to make fish safer for women to eat, we need to reduce neurotoxic pollutants at their source. For mercury, this means public health professionals ought to consider advocating for public policies that encourage non-polluting energy sources like wind or sun, and moving away from fossil fuels—a major source of asthma-worsening pollutants like sulfur dioxide, nitrogen dioxide, ozone, and particulate matter—and mercury. Public health voices are essential for the success of campaigns to reduce industrial emissions. Eric Pianin, writing recently in the *Washington Post*, notes:

The levels of mercury contaminant found in largemouth bass and other wildlife of the Everglades declined by 60 to 75 percent since state and federal agencies began waging an aggressive campaign in the early 1990s to close or modernize municipal and medical-waste incinerators that emitted mercury gases.¹¹

Similarly, public health professionals can advocate for replacing mercury-containing products with non-polluting alternatives. Nationally, Health Care Without Harm, a coalition of 431 organizations in 52 countries (www.noharm.org), has led the way in eliminating mercury-containing thermometers, blood pressure devices, and other products from health care facilities, thereby reducing mercury pollution by keeping them out of medical incinerators.

Mercury-Free Minnesota (www.mercuryfreemn.org), a coalition of public health, environmental, and consumer groups, is calling for a 90% reduction in mercury emissions by 2010, and a complete phase-out by 2020. At the federal level, the proposed energy bill and the proposed “Clear Skies Initiative” both further fossil fuel reliance while failing to address the public health impacts of mercury pollution. If these proposals become federal policies, we will see more pollution and increased health problems.

Global production of PBDEs is over 40,000 tons a year. Eliminating most uses of PBDE flame-retardants is a possible and prudent primary prevention step whose time has come. Sweden’s swift action to enact regulatory controls on PBDEs in the late 1990s has resulted in significant declines in levels of PBDEs in breast milk in just a few years.⁸ The European Union, Germany, and the Netherlands have also taken regulatory action to reduce the use of PBDEs. If the U.S. banned PBDEs today, we could see a similar decline within just a few years. In the interim, scientific study should continue to confirm preliminary findings that these

persistent compounds have low-level toxicity to the developing brain and nervous system. We must prevent a potential PCB scenario in the U.S., where it took 50 years of exposures and injured children before they were banned.

The state of California has already enacted a phase-out by 2008 of penta and octa-BDEs. Penta-BDE is the most bioaccumulative form, with the highest production in North America. California’s action provides a model for possible regulatory action.

Health professionals should advocate for environmental standards that better protect children. Ultimately, we need to stop the pollution and make public health considerations paramount in chemical regulation. Such an approach is needed to protect the health of today’s children and the health of future generations.

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References

1. National Research Council. Pesticides in the diets of infants and children. Washington, DC: National Academies Press; 1993.
2. United States Environmental Protection Agency. 2000 toxics release inventory (TRI) public data release report: executive summary. Available from: URL:http://www.epa.gov/tri/tridata/tri00/press/execsummary_final.pdf. Accessed January 26, 2004.
3. Stein J, et al. In harm’s way: toxic threats to child development. *J Dev Behav Pediatr* 2002;23(1S):S13-22.
4. Centers for Disease Control and Prevention, National Center for Health Statistics. National health and nutrition survey: 2003. Available from: URL:<http://www.cdc.gov/nchs/nhanes.htm>. Accessed January 26, 2004.
5. Grandjean P, et al. Cognitive deficit in 7-yr old children with prenatal exposure to methylmercury. *Neurotoxicol Teratol* 1997;19(6):417-28.
6. Jacobson JL, Jacobson SW. Intellectual impairment in children exposed to PCBs *in utero*. *NEJM* 1996;335:783-9.
7. Eriksson P, et al. Brominated flame retardants: a novel class of developmental neurotoxicants in our environment? *Environ Health Perspect* 2001;109(9):903-8.
8. Darnerud PO, Eriksen GS, Johannesson T, Larsen PB, Viluksela M. Polybrominated diphenyl ethers: occurrence, dietary exposure, and toxicology. *Environ Health Perspect* 2001;109(S1):49-68.
9. Luross JM, et al. Spatial and temporal distribution of polybrominated diphenyl ethers in lake trout from the Great Lakes. *Organohalogen Compounds* 2000;47:73-6.
10. Schecter A, et al. Polybrominated diphenyl ethers (PBDEs) in U.S. mother’s milk. *Environ Health Perspect* 2003;111(14):1723-9.
11. Pianin E. Mercury rules work, study finds EPA, Florida cite emissions regulation. *Washington Post*; 6 November 2003.



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Environmental Justice: Disparities in Health Effects and Exposures

Kathleen Schuler, MPH

Environmental justice is the fair treatment and meaningful involvement of all people regardless of race, color, national origin, culture, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies. For more information, visit the EPA website at <http://www.epa.gov/compliance/environmentaljustice/>

The U.S. Environmental Protection Agency (EPA) recently reported on the state of children's environmental health in their 2003 publication, *America's Children and the Environment, Measures of Contaminants, Body Burdens, and Illness*.¹ This report reveals that children, the group most vulnerable to the effects of toxic pollution, are widely exposed to contaminants in air, water, soil, food, and indoor environments like homes and schools. Although environmental toxicants have an affect on all children, a closer look at this report reveals wide disparities in both health outcomes and exposures by income and race.

Lead and exposure to tobacco smoke are two measures that show general improvement, but children of color and poor children fare worse than other children. Although the average blood lead levels of all children age 1–5 declined 85% from 1976–80 to 1999–2000, concentrations of lead in children's blood differ by race/ethnicity and family income.² In the most recent period, African American and low-income children had median blood lead levels 37% higher (2.8 µg/dL-micrograms per deciliter of blood), than those of white Hispanic and non-Hispanic children (at 2 to 2.1 µg/dL).²

Fewer children are exposed to cigarette smoke than to lead. Nineteen percent of homes of children under age 7 had a smoker in 1999, down from nearly 29% in 1994. This reduced exposure is borne out in reduced concentrations of cotinine in blood, a biomarker of tobacco smoke. From 1999–2000, median (50th percentile) levels of cotinine measured in children were 56% lower than they were between 1988–91. However, higher concentrations of cotinine in blood were correlated with lower income. Children living below the poverty level had concentrations over 2.5 times higher than all income groups combined.¹

The asthma rate doubled between 1980 and 1995.³ Black and low-income children fare worse, as they are most likely to suffer asthma attacks and experience twice the hospital admissions and emergency room visits for asthma and respiratory problems as white children.⁴

Neurodevelopmental disorders may have environmental causes. Poor and African American children have higher rates of mental retardation (12/1,000 for those below the poverty level, 10/1,000 for black non-Hispanic children compared with 6/1,000 for all children). Rates of reported attention-deficit/hyperactivity disorder in children aged 5 to 17 in 1997–2000 were 6.7% overall, but increased to 7.7% for children with family incomes below the poverty level, with poor white children having the highest rate at 13.6%.¹

The EPA report reveals something about greater exposures to hazardous chemicals among low-income children and children of color, which could account for some of the observed disproportionate health effects. These groups of children are more likely to live in inner-city neighborhoods that have greater air pollution from automobiles, incinerators, and diesel buses. For example, based on the 1996 cancer risk benchmark of 1/10,000, 28% of black non-Hispanic, 31% of Hispanic, and 34% of Asian or Pacific Islander children, compared with 12% of white non-Hispanic children lived in counties where hazardous air pollution concentrations put them at increased risk for cancer.¹

Low-income children and children of color are more likely to reside in older housing and attend school in older buildings where they can be exposed to lead in paint, soil, and water.^{5,6,7} Older schools tend to be located in inner city lower income neighborhoods.

In 2000, 1.3% of all children lived within one mile of a high priority hazardous waste site designated by the EPA as a Superfund site.¹ Superfund sites are considered by the EPA to contain chemicals that pose the greatest health threats. There is evidence that low-income individuals and those of color and are more likely to live in areas adjacent to toxic waste sites and polluting industries.^{8,9,10} Finally, low-income individuals and people of color are potentially more affected by fish contaminants like mercury, because they often consume the fish they catch. Overall, an estimated 8% of women carry a body burden of mercury that could put their offspring at risk of harmful effects on the developing brain.¹¹

The World Health Organization Constitution states: "The enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being without distinction of race, religion, political belief, economic, or social condition."¹² Public health professionals can help make these rights a reality for all children by working for environmental justice. For more information, see: *Preventing Harm Minnesota*, www.preventingharmmn.org, or *Environmental Justice Advocates of Minnesota*, www.ejadvocates-mn.us.

References

1. US Environmental Protection Agency (EPA). *America's children and the environment, measures of contaminants, body burdens, and illness* (EPA 240-R-03-001);2003. Available from URL: <http://www.epa.gov/envirohealth/children>. Accessed January 27, 2004.
2. Meyer PA, Pivetz T, Dignam TA, Homa DM, Schoonover J, Brody D. Surveillance for elevated blood lead levels among children in the United States, 1997–2001. *MMWR* 2003; 52:1–21.
3. Storey E, Cullen M, Schwab N. A survey of asthma prevalence in elementary school children. Online report. New Haven Connecticut: Environment and Human Health, Inc. 2003;58. Available from: URL: <http://www.ehhi.org>. Accessed January 23, 2004.
4. Federico MJ, Liu AH. Overcoming childhood asthma disparities of the inner-city poor. *Pediatr Clin North Amer* 2003;50:655–75.
5. Mielke HW, Powell ET, Shah A, Gonzales CR, Mielke PW. Multiple metal contamination from house paints: consequences of power sanding and paint scraping in New Orleans. *Environ Hlth Perspectives* 2001;109:973–8.
6. Mielke HW, Gonzales CR, Smith MK, Mielke PW. The urban environment and children's health: soils as an integrator of lead, zinc, and cadmium in New Orleans, Louisiana, USA. *Environ Res* 1999;81:117–29.
7. Mielke HW, Blake B, Burroughs S, Hassinger N. Urban lead levels in Minneapolis: the case of the Hmong children. *Environ Res* 1984;34:64–76.
8. Bullard RD. *Dumping in Dixie: race, class, and environmental quality*. Boulder, CO: Westview;1990.
9. U.S. General Accounting Office. *Siting of hazardous waste landfills and their correlation with racial and economic status of surrounding communities*. Washington, D.C.: General Accounting Office;1983;1.
10. Greenberg MR, Anderson RE. *Hazardous waste sites: the credibility gap*. New Brunswick, NJ: Rutgers University Center for Urban Policy Research. 1984;158.
11. U.S. Environmental Protection Agency. *Integrated Risk Information System (IRIS) risk information for Methylmercury (MeHg)*. Washington, DC: National Center for Environmental Assessment;2001. Available from URL: <http://www.epa.gov/iris/subst/0073.htm>. Accessed January 23, 2004.
12. World Health Organization. *Health as a human right*. Available from: URL: <http://www.who.int/archives/who50/en/human.htm>. Accessed January 23, 2004.

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Use “KARE”: A Community Reponse to Keeping Children Safe From Toxic Substances



Marjorie Vigoren, BS

In 2000, the City of Plymouth, Minnesota, took on the challenge to increase awareness of proper use, disposal, and reduction of household hazardous waste. A grant from Hennepin County’s Department of Environmental Management allowed Plymouth’s Recycling Program to tackle the perplexing issue of reaching the public with the primary message to reduce exposure to household toxicants.

Focus groups conducted with randomly-selected citizens to determine how to best deliver important and often complicated environmental messages confirmed that people don’t take the time to read detailed information. A woman in one of the focus groups who commented on a bumper sticker-sized notice emblazoned with three simple words, “I like this one, I can read it as I throw it away” exemplifies this point. Believing this attitude to be more the rule than the exception, we decided that the key to being heard was to find people who *want* the information. They would be people who:

- ◆ have something personal at stake related to the information;
- ◆ would be venturing into some new chapter of their lives—open to guidance as they face unfamiliar ground; and
- ◆ would belong to a group, so we could identify them and address several people at a time.

Applying these criteria, the Recycling Coordinator looked for potential groups that had a stake in our message. The group that satisfied all the criteria was parents in our local Early Childhood Family Education (ECFE) parenting classes. Many were first-time parents, eager for information to guide them through the maze of parenting skills. All of them had the safety and health of their infant or young children in mind. They gathered regularly with the goal of gaining skills to raise their young offspring.

With this group in mind we developed a one-hour class for parents and caregivers of infants and small children. The class focused on reducing exposure to toxic substances in homes. The presentation was called “Use KARE to Keep Children Safe From Toxic Substances.” The acronym KARE represents four factors that are important in keeping children safe from toxicants: Know, Assess, Remediate, and Educate. Our message focused on how parents can reduce the odds of their children being harmed by toxic substances using KARE. Presentations consisted of PowerPoint slides, a bag of “look-alike” products, and a worksheet to help participants understand what labels tell them about product safety.

Program Components of KARE

Know. Parents need to know why exposure to toxic substances pose a greater risk to children than to adults so they can begin to consider these factors in managing their child’s environment. Parents learn that the size, rate of development, behavior characteristics, and cognitive development of children make them particularly vulnerable to toxic substances. Parents also learn how children are exposed to toxic

substances and how to assess product risks. An exercise in reading and evaluating product labels introduces parents to the skills necessary to assess product safety. Parents also learn that some products, like medications, alcohol, and gasoline do not necessarily carry warnings, but can be toxic nonetheless. After completing the label-reading exercise participants discuss alternative products and methods to reduce the use of toxic chemicals.

Assess. Participants learn how to assess their child’s environment for toxic substances. They receive a checklist that identifies places where toxicants are likely to be found and are encouraged to perform an inventory of their homes and daycare centers. Each point of concern on the checklist has a corresponding action to reduce or eliminate the threat.

Remediate hazards or exposure. It is critical that parents are given the tools to limit exposure and reduce its harmful consequences because the potential for accidental exposure can never be completely eliminated. Information about the Poison Control Center, correct use of syrup of Ipecac, intact product labels, and proper clean-up techniques are introduced as vital tools to effectively deal with a child’s exposure to toxic substances. Participants are also given information to help them dispose of any household hazardous waste they may have in their homes.

Educate. Information from sources like the U.S. Environmental Protection Agency (EPA), the Poison Control Center, and the Extension Service of the University of Minnesota are gathered into packets for each participant. Participants are encouraged to read the information and then to share it with their child’s caregivers.

Project Outcome

ECFE classes have been an excellent choice for the implementation of the program. Since the program began, presentations have been made to over fifty classes. Parents are engaged and often offer stories from their experiences that become illustrations for other classes. Twenty-five participants have

taken action on the advice from the presentation to order radon detection kits at a cost of \$7 each. Teachers of the ECFC parenting classes report that parents remain interested in the topic of child exposure to household toxicants after the presentation. The ECFC Program Director and parenting class teachers continue to request the presentation.

Plymouth’s Recycling Program continues to publish brochures, fact sheets, and newsletters to alert people to the potential hazards and environmental consequences of various household products, but we are convinced that face-to-face encounters through the ECFC classes are by far our most effective outreach.

Marjorie Vigoren, BS is the Solid Waste and Environmental Education Coordinator for the City of Plymouth, Minnesota.

City of Plymouth is a Leader in the Field of Environmental Education

Environmental education can be found at the core of the City of Plymouth’s plans for its programs in water resources, forestry, and solid waste. In an unusual move for a city, they added three workdays and new duties to an existing part-time position to create the environmental education coordinator position. In collaboration with schools, community organizations, and citizens—the coordinator carries out a public education campaign to promote environmental stewardship through events, programs, and a variety of media.

Healthy Generations Videoconference

Children's Environmental Health
Tuesday, March 2, 2004
1-3 pm

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Hennepin County
 MN Dept. of Health
 Room 118B
 717 Delaware St. SE
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 Health and Human Services Bldg.
 2200 23rd St. NE
 Willmar

Nobles County
 Courthouse, Room 111
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 Worthington

Polk County
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St. Louis County
 Government Services Center
 Room 709
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 Human Services, ITV Room
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Registration is free and limited by site. To register, contact Jan Pearson by email (pearson@epi.umn.edu) and provide your name, mailing address and the site you plan to attend. Certificates of Attendance will be provided. Please visit: <http://www.epi.umn.edu/mch/events/index.shtm> for any changes to these sites.

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