

# Preventing Environmental Health Risks to Children and Promoting Collaborative Research and Environmental Justice

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## **SUMMARY**

Research is needed on environmental health risks to children in disadvantaged, minority communities to improve environmental health policy and environmental justice. Multiple health effects in children have already been associated with environmental pollution, and minority communities tend to be both disproportionately exposed to pollutants and disproportionately affected by environmentally-related disease. Identifying environmental risks in these populations will lead to prevention of serious diseases including childhood asthma, developmental disorders, and cancer. Translation to policy requires the communication of scientific results to the health policy context. Effective community-academic partnerships to translate scientific data into public health policy are essential and will ensure major benefits to children's environmental health and greater environmental justice.

## **KEY WORDS**

Environmental health sciences, environmental justice, community-based participatory research, translational research, community outreach, partnership, health disparities, children's health, PAH, pesticides, ETS, asthma, cancer, developmental disability, adverse birth outcomes, WE ACT, CCCEH

## **INTRODUCTION\***

The theme of this article is twofold: I will elaborate on the need for more research on the environmental health risks to children in disadvantaged, minority communities to provide comprehensive and inclusive information for environmental health policy and environmental justice. Research reviewed here indicates that multiple health effects in children are associated with environmental pollution and that minority communities tend to be both disproportionately exposed to pollutants and disproportionately affected by environmentally-related diseases, including childhood asthma, developmental disorders, and cancer. In a second instance, I will describe the benefits and importance of collaborations as exemplified by the community-based participatory research model (CBPR) that our NIEHS-funded Center, Columbia Center for Children's Environmental Health (CCCEH), has assumed with West Harlem Environmental Action (WE ACT) in order for government agencies and academic institutions to better understand and incorporate community concerns into their research agendas (1).

## **NEEDED RESEARCH ON THE HEALTH BURDEN INCURRED BY MINORITY CHILDREN**

Childhood diseases impose significant burdens on families and society, have increased in recent decades, and are thought to be caused in substantial part by environmental factors such as toxic exposures due to lifestyle (smoking and diet) and pollutants in the workplace, ambient air, and water and food supply. The exogenous exposures can interact with "host" factors such as genetic susceptibility and nutritional deficits, and with psychosocial stressors associated with poverty, to cause disease or developmental impairment.

### **Common Diseases**

#### *Asthma*

Pediatric asthma is a serious and growing public health problem in the United States and in many other countries (2, 3). The percentage of

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children with asthma has doubled between 1980 and 1995 in the U.S. (from 3.6% in 1980 to 7.5% in 1995) (3). An estimated 8.7% (6.3 million) of children had asthma in 2001 (3). Rates vary widely by geographic area and ethnic group. New York City is one of four metropolitan areas in the country with the highest annual increase in asthma mortality (4). Five of the seven New York City zip code areas with the highest asthma hospitalization rates are located in Harlem (5). In the South Bronx in 1994, prevalence of asthma among children < 17 years of age was 17.9% in Hispanics, 11.6% among non-Hispanic blacks, and 8.2% among whites (6). A recent study found that over 25% of elementary school children in Harlem had asthma (7). Another study of pediatric asthma rates suggested that material and behavioral characteristics associated with poverty, such as parental smoking, air pollution, housing conditions, and allergens, may contribute to the disparities (8).

#### *Ongoing research*

While air pollution has long been known to be a trigger for asthma in children, more recent studies are pointing to an early causal role in the disease. Results from the CCCEH cohort study of African-American and Latina mothers and children in Northern Manhattan highlight the importance of the prenatal period of development, showing that difficulty breathing and probable asthma were reported more frequently among children exposed to prenatal PAHs and ETS (measured by a biomarker of nicotine exposure) postnatally (9). A parallel cohort study in Europe has found that prenatal PAH exposure was associated with an increased risk for respiratory symptoms during the course of the infant's first year of life (10). Together these research reports confirm that consideration both of ethnicity and social class is essential in understanding the determinants and distribution of asthma in populations, as well as in devising needed interventions in communities (1).

#### *Cancer*

The incidence of certain childhood cancers in US children has increased as well. The age-adjusted annual incidence of cancer in children increased from 128 to 161 cases per million children between 1975 and 1998 (3). Leukemia was the most common cancer diagnosis

from 1973-1998, representing about 20% of total cancer cases. Central nervous system tumors represented about 17% of childhood cancers.

#### *Ongoing research*

Environmental exposures are recognized as potentially important risk factors for childhood cancer (11); and again biomarkers are proving useful in assessing causal relationships. For example, carcinogen-DNA adducts are considered a biomarker of biologically effective doses of PAHs and of increased cancer risk (12). Research in African-American and Latina, Caucasians and Chinese mothers and newborns has shown that PAH-DNA adduct levels in the cord white blood cells of newborns are higher per estimated unit exposure than in the mother's blood, indicating greater fetal susceptibility to genetic damage (13).

Chromosomal aberrations have been associated with increased risk of cancer in multiple studies and are a well validated biomarker of the preclinical effect of carcinogens (14, 15). In the CCCEH newborns, maternal exposure to airborne PAHs during pregnancy was associated with increased frequency of chromosomal aberrations in WBC, suggesting that risk of cancer can be increased by exposure *in utero* (16).

#### *Adverse birth outcomes*

Low birth weight is the second leading predictor of infant mortality in the United States as well as a major cause of delayed development (17) and a risk factor for childhood asthma (18). In general, children in minority communities in NYC are at elevated risk for low birth weight and subsequent cognitive delay compared to other U.S. populations, but rates vary between them. In 1997, the incidence of low birth weight was 13.5% in central Harlem, 10.5% in the South Bronx, and 7.7% in Washington Heights, compared to 7.1% in whites in New York City (19).

#### *Developmental disorders*

Developmental disabilities, the name given to a broad group of conditions caused by learning or physical impairments, affect an estimated 17% of U.S. children under age 18 (20). The high rates of these childhood disorders have significant social impacts and medical costs for individual families and the country as a whole. Children in minority communities are also at elevated risk of subsequent cognitive delay compared to other populations. For example, 68% of elementary school children in Washington Heights and 74% in central Harlem are reading below grade level, compared with 46% city-wide (21).

*Ongoing research*

Experimental studies of prenatal and neonatal exposure to the organophosphate pesticide chlorpyrifos (CPF) have demonstrated neurochemical and behavioral effects as well as selected brain cell loss (22-26). Children in the CCCEH cohort who were prenatally exposed to high levels of CPF, as measured by high cord plasma CPF levels, were significantly more likely than children with low cord levels to experience delay in both psychomotor and cognitive development at 3 years of age (27). In addition, the highly exposed children were significantly more likely than less exposed children to manifest symptoms of attentional disorders, attention-deficit/hyperactivity disorder (ADHD), and pervasive personality disorder at age 3. Although the EPA banned residential use of CPF in 2001, this pesticide is still widely used in agriculture. In addition, cohort children with high prenatal exposure to airborne PAHs also had significantly lower test scores at age 3 on the Bayley test for cognitive development, after controlling for pesticide exposure (plasma CPF) (28).

Many cohort studies have demonstrated that low-level exposure to lead (even below 10ug/dL in blood) during early childhood is inversely associated with neuropsychological development through the first ten years of life (29-33). Prenatal exposure to PCBs and methylmercury, predominantly from maternal seafood consumption, has also been associated with neurocognitive deficits (34). In these studies, biomarkers (including blood concentrations) have been instrumental in quantifying the internal dose of the pollutants.

### **POSSIBLE FACTORS BEHIND INCREASED RISKS TO SPECIFIC COMMUNITIES**

These figures outline the disproportionately high rates of neurodevelopmental disorders, asthma, and cancer in underserved, minority populations in the United States.

These health disparities clearly reflect many factors but are thought to be due in part to greater exposure to certain environmental toxins (8). Many studies have reported the disproportionate exposure of minorities to air pollution, including during pregnancy (35, 36). In Harlem, Washington Heights, and the South Bronx minority communities in New York City (37, 38) where our partnership (WE ACT - CCCEH) conducts its work (1), poor quality housing, diesel bus

depots, a multitude of small industrial operations, and combustion-related pollution from the substantial network of highways are all to blame for this disproportionate exposures to environmental toxins. A further-reaching study operating at the county level for the United States found that Hispanic, African-American, and Asian/Pacific Islander mothers experienced higher mean levels of air pollution and were more than twice as likely to live in the most polluted counties compared with white mothers between 1998 and 1999 after controlling for maternal risk factors, region, and educational status (36).

Nutritional deficits and genetic predisposition can increase the effect of environmental exposures. For example, by removing free radicals and oxidant intermediates, antioxidants protect DNA from the genotoxic, procarcinogenic effects of chemicals that bind to DNA (39, 40). Genetic susceptibility can take the form of common variants or “polymorphisms” that modulate the individual response to a toxic exposure such as organophosphate pesticides (41, 42), lead (43), tobacco smoke (44), or PAHs (40, 45, 46). The dietary habits of children in particular cause increased exposure to food-borne toxicants. U.S. children under 5 years of age eat three to four times more food per unit of body weight than the average adult American; and the average one-year-old drinks 10-20 times more juice than the average adult (47).

Adverse social condition at the individual and community-levels can produce profound effects on host susceptibility to disease (48). Recent studies have shown that women who live in violent, crime-ridden, physically decayed neighborhoods are more likely to experience pregnancy complications and adverse birth outcomes, after adjusting for a range of individual level sociodemographic attributes and health behaviors (49, 50). Other studies have suggested that the stresses of racism and community segregation are associated with lower birth weight (4). A recent study by CCCEH found that the risk of developmental delay among children exposed prenatally to maternal ETS was significantly greater among those whose mothers experienced material hardship during pregnancy (51).

Sensitive populations, either due to age, behavior, health status or a combination of these, are also populations of concern. Exposure to genotoxic and nongenotoxic chemicals as well as chemicals that exert both types of effects is at stake here. The latter include so-called “endocrine disruptors” that mimic or block natural hormones (52).

During pregnancy for example, toxicants to which the mother is exposed can reach the fetus via placental transfer; and toxicants stored in the bodies of mothers can become bioavailable, also exposing the fetus. Lactation is another potential source of exposure to infants. In addition, the *in utero* and childhood periods are characterized by rapid physical and mental growth and gradual maturation of major organ systems (53). Since cells are proliferating rapidly and organ systems are immature, they are sensitive to the potentially harmful effects of environmental toxins. The fetus and child also clear many toxicants less readily than the adult (54-58). Young children breathe air closer to the ground, exposing them to particles and vapors present in carpets and soil. While playing and crawling on the floor, children can inhale or dermally absorb toxicants which are subsequently absorbed more efficiently in children than in adults (53). In addition, infants have twice the breathing rate of the average adult. Hand-to-mouth behavior and thumb sucking habits can also increase exposure. Dermal exposures may also be higher, as a typical newborn has more than double the surface area of skin per unit of body weight than an adult (59). Experimental and human data indicate that the fetus and young child are indeed especially vulnerable to the toxic effects of environmental tobacco smoke (ETS), polycyclic aromatic hydrocarbons (PAHs), particulate matter, nitrosamines, pesticides, polychlorinated biphenyls (PCBs), metals, and radiation (54, 60, 61), as shown by the Center's own work.

Beyond infants and children, elderly and immuno-compromised individuals are also particularly vulnerable to exposure to environmental toxicants.

### **TRANSLATION IN PARTNERSHIP WITH THE COMMUNITY**

The research reviewed above has clear implications for environmental health policy. Translation to policy requires the communication and implementation of scientific results to the realm of health policy. Involving a diverse group of players active along the continuum from study idea to research and analysis to policy outcome is key and helps facilitate the entire process. Community-academic partnerships have demonstrated considerable potential for translation of science to policy. The importance in particular of community involvement in setting and implementing research agendas to address environ-

mental justice issues, such as the disproportionate burden that environmental degradation and pollution have had on the health and well-being of communities of color and low-income populations, cannot be denied (1).

### **Community-based Participatory Research Model (CBPR)**

The past two decades have witnessed a rapid proliferation of community-based participatory research (CBPR) projects (62). CBPR is a model rooted physically and conceptually in community. In CBPR, scientists work in close collaboration with community partners involved in all phases of the research, from the inception of the research questions and study design, to the collection of the data, monitoring of ethical concerns, and interpretation of the study results. Importantly, in CBPR, the research findings are communicated to the broader community – including residents, the media, and policymakers – so they may be utilized to effect needed changes in environmental and health policy to improve existing conditions. Building upon existing strengths and resources, CBPR seeks to build capacity and resources in communities and ensure that government agencies and academic institutions are better able to understand and incorporate community concerns into their research agendas (1).

The National Institute of Environmental Health Sciences (NIEHS), the premier biomedical research facility for environmental health, has taken a lead in promoting the use of CBPR in instances where community-university partnerships serve to advance our understanding of environmentally related disease. CBPR can be an effective tool to enhance our knowledge of the causes and mechanisms of disorders having an environmental etiology, reduce the adverse health outcomes through innovative intervention strategies and policy change, and address the environmental health concerns of community residents (62).

Currently absent from CBPR research, however, is work on the crucial global environmental justice topics, including deforestation and loss of biodiversity, agriculture and soil erosion, climate change and stratospheric ozone depletion, and stockpiling of nuclear weapons and wastes (1).

### **WE ACT – CCEH Partnership**

One such partnership between West Harlem Environmental Action,

Inc. (WE ACT), an environmental justice organization, and the CCCEH at the Mailman School of Public Health exemplifies the emerging model of community-based action designed to advance environmental health policy and improve the quality of life in New York City and throughout the United States. This eight-year collaboration has had multilevel impacts on a variety of environmental justice achievements: air monitoring studies published in peer-reviewed journals, training courses for community leaders on environmental health topics, educational forums for community residents on environmental justice issues, and meaningful input into policy decisions that have addressed issues like diesel exhaust in Northern Manhattan. WE ACT has provided strategic leadership in translation of the scientific findings to environmental health policy and practice. This partnership has been given substantial credit for the conversion of the NYC bus fleet to clean diesel and the installation by the EPA of permanent air monitors in Harlem and other “hot spots” (1, 63).

#### **CCCEH: Other impacts on policy**

Several of the CCCEH’s other findings, in concert with the efforts of other research centers and environmental organizations, have had an impact on environmental health policy in low-income neighborhoods of NYC and beyond. For instance, CCCEH data on health effects associated with prenatal pesticide exposure helped pass two landmark pesticide bills in NYC to reduce residential exposure to pesticides. WE ACT was instrumental in making the New York City Council aware of the findings. CCCEH investigators also provided their research results in expert testimony during NYC’s development of mandates for city-funded construction projects to use vehicles with low-sulfur fuel and particle traps. Through its role as a mediator and frequent meetings with community residents to address concerns about pollution, the partnership convinced Mayor Bloomberg to keep the 135th Street Marine Waste Transfer Station closed in Harlem because of health concerns, keeping 320 polluting diesel garbage trucks out of the area every day. The partnership also supported the negotiation of contracts with the New York City Transit Agency to retrofit old diesel buses with emission reduction technology in Northern Manhattan, was engaged in keeping the Amsterdam Bus Depot closed, and advocated for New York State penalties on diesel vehicle idling.

## CONCLUSION

Effective community-academic partnerships to translate data into public health policy will ensure major benefits to children's environmental health and greater environmental justice. The high rates of asthma, certain cancers, and developmental disability in children, the disproportionate burden borne by minorities, and the growing evidence that risk of disease is associated with *in utero* and childhood environmental exposures support an "early focus" in prevention. When preventive measures have been enacted based on this knowledge, children's health has benefited. The disproportionate burden of pollution across communities, the impacts of multiple and cumulative exposures – including the potential for synergistic effects – and the special concerns of susceptible populations, including children, the immuno-compromised, and the aged, are important concerns. To truly affect meaningful change in the environments and health of communities of color and low-income communities, academic institutions and community-based organizations and leaders must engage the larger public and work in coalition with government agencies, public and private foundations, policymakers, legal experts, and local businesses (1).

It is important to have diverse institutions and groups of people engaged in this type of research because research results underlie regulatory decisions that affect many different parties in different ways. The agendas of the different stakeholders in environmental health and more broadly in the environmental movement must be synchronized as much as possible in order to obtain the most focused and effective outcomes.

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