

## Mercy Mercy Me: Social Injustice and the Prevention of Environmental Pollutant Exposures Among Ethnic Minority and Poor Children

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Children's lead and pesticide exposures are used as examples to examine social disparities in exposure reduction efforts as well as environmental policies impacting children in poverty and minority children. The review also presents an estimate of the effect of social disparities in lead exposure on standardized test performance. Because including measures of pollutants with potential behavioral effects can alter the observed effects of race/ethnicity, income, and other variables, suggestions are made for including measures of pollutants in longitudinal studies and studies of multiple and cumulative risk. Continued basic research on developmental correlates and effects of pollution exposure, participatory action-research with at-risk and underrepresented populations, and contributions to public awareness and education are important leadership areas for developmental researchers.

Poverty is a particularly salient, chronic, and uncontrollable source of stress that increases risk for negative physical and psychological health outcome of children across all ethnic and racial groups. And, although the majority of poor children in the United States are European American, the percentages of children living in poverty who are African American, Native American, Latino American, and recent immigrants are 2–3 times higher than their European American counterparts (McLoyd, 1998). In addition, poverty and ethnic minority status are both associated with higher exposure to certain harmful environmental pollutants such as lead exposure, particulate air pollution (Environmental Protection Agency, 2000; Evans, 2004) and ambient noise (Evans & English, 2002).

Poverty among ethnic minority children is more persistent than among their White counterparts, and the extent to which children live in low-income, high-risk areas also influences their degree of risk for exposure to potentially harmful environmental pollutants (Attar, Guerra, & Tolan, 1994; Schell, 1997). Using lead as a case example, we examine environmental policies and exposure prevention programs as they have impacted children in poverty and minority children. In addition to giving a brief overview of the routes of exposure and developmental effects of lead, we discuss two categories of prevention efforts: (a) national or local policies to reduce pollution sources and contact with sources of expo-

sure, and (b) educational programs that place the burden of preventing exposure on the individual and family. We consider how these approaches affect low-income minority families—the most highly lead-exposed segments of the U.S. population. The successes and failures of the lead exposure prevention efforts can be used to inform other pollution exposure prevention programs. After examining lead exposure, we briefly review pesticide exposure social disparities and exposure reduction efforts, indicating similarities and differences from lead and the significant policy gaps in prevention. We conclude with a discussion of the continuing role of developmental scientists in researching the developmental effects and correlates of pollution exposure, and we point to areas for future contribution to this area of study.

All children are at risk for contact with environmental toxins, but the burden of toxic exposures is disproportionately allocated to poor ethnic minorities (Moore, 2003; Schell, 1997). "Economic factors not only constrain choices but also inequitably distribute human made stressors..." (Schell, 1997, p. 67), and the psychosocial stress and environmental pollutants associated with poverty do not occur independently of one another. Rather, the effects may accumulate through risk focusing, a process by which exposures to toxic or infectious environmental materials are differentially allocated to a specific group partly because of previous exposure to those materials (Schell, 1997).

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### Case Example: Lead

Exposure to lead has been known to be neurotoxic at least since Roman times (Hamilton, 1943) and “is without a doubt the most widely studied pediatric neurotoxicant” (Dietrich, 1995, p. 224). In addition to its association with lowered intellectual function, lead has a variety of negative health effects, including association with cognitive decline in adulthood in former lead industry workers (Links, Schwartz, Simon, Bandeen-Roche, & Stewart, 2001), higher blood pressure, hearing loss, impaired renal function (Lippmann, 1990), spontaneous abortion (Borja-Aburto et al., 1999), and cardiovascular disease (Fewtrell, Pruss-Ustun, Landrigan, & Ayuso-Mateos, 2004). The study of the effects of lead exposure and the public policy efforts to prevent exposure are steeped in controversy (for a brief overview of the history of the controversies, see Moore, 2003).

The controversy over whether early life lead exposure is a developmental cause of intellectual deficit has centered on three alternative explanations: “reverse causality,” the roles of statistical power and confounding variables, and the possibility of “effect modification” or interactions between variables such as socioeconomic status and lead. The reverse causality hypothesis is that intellectual deficits promote behaviors that increase lead exposure; it has been stated most forcefully by DeSilva and Christophers (1997). The issue of statistical power and confounding variables and their appropriate treatment was hotly debated between Ernhart (1987) and Needleman (1987). The issue of effect modification has been presented by Bellinger (2000). A full review is beyond the scope of this paper, but even the severest detractors admit that early lead exposure shows a negative relationship to later IQ test scores even after adjusting for confounding variables (DeSilva & Christophers, 1997; for meta-analyses, see Needleman & Gatsonis, 1990; Schwartz, 1994). There has been less controversy about the association of lead exposure with teacher or parent ratings of behavior regulation such as aggression, restlessness, and inattention (Burns, Baghurst, Sawyer, McMichael, & Tong, 1999; Fergusson, Horwood, & Lynskey, 1993; Needleman et al., 1979), although concerns about the handling of confounds have been raised.

The latest data indicate that lead exposure, even at blood lead levels below the 10 µg/dl federal guideline (Centers for Disease Control and Prevention, 1991), is associated with lower IQ test scores and lower behavior regulation (Lanphear, Dietrich, Auinger, & Cox, 2000; Lanphear et al., 2005; Needleman, Schell, Bellinger, Leviton, & Allred, 1990). Canfield

et al. (2003) estimated that for 5-year-olds, there is a loss of about 1.4 IQ points for each increase of 1 µg/dl blood lead up to 10 µg/dl. Over the full range of lead exposure, 0.46 IQ point is lost for each 1 µg/dl increase in lead. Lead exposure is negatively associated with intellectual outcomes in studies in different countries as well as in studies with samples of different racial and ethnic composition in the United States: for example, samples of predominately white suburban children in Massachusetts (Needleman et al., 1990) as well as predominately Black economically disadvantaged urban children in Rochester and Cincinnati (Canfield et al., 2003; Dietrich, Berger, Succop, Hammond, & Bornschein, 1993).

Attempts to identify a sensitive period during which lead exposure has maximum effects on later behavioral outcomes have been unsuccessful (Dietrich, 1995). Nevertheless, early life lead exposure is related to later intellectual and behavioral outcomes. For example, in the Cincinnati lead study, lead exposure measured prenatally, at 78 months, and as the average of lead exposure from birth to 78 months were all significantly associated with higher self-reported delinquency at 15–17 years of age even though blood lead at age 15–17 had dropped to relatively low levels (Dietrich et al., 2001). Developmental continuity for the negative effect of early lead exposure is also well documented for intellectual outcomes (Schwartz, 1994).

### *Social Inequities in Exposure to Lead*

Despite three decades of public policy and lead exposure preventive efforts, poor African American children living in urban areas remain at an increased risk of exposure to lead compared with others. Figure 1 presents the distribution of lead exposure for race/ethnic and income groups at the two most recent time periods for which national survey data are available. First, the data show that children’s lead exposure continues to decline in the United States. Second, the data show persistent disparities by race/ethnic group and income. In the 1988–1991 data, Black children from families in poverty had 6.6 times the chance of having high lead compared with White children from families above the poverty level. The percentage of Black children in poverty with high lead declined such that in 1992–1994 the chance of having high lead was .57 that in 1988. However, the disparity from the White children above poverty actually increased. The 1992–1994 data show that a Black child in poverty had 14.7 times the chance of having high lead compared with a White child not in poverty. Also, the White children above poverty showed the largest

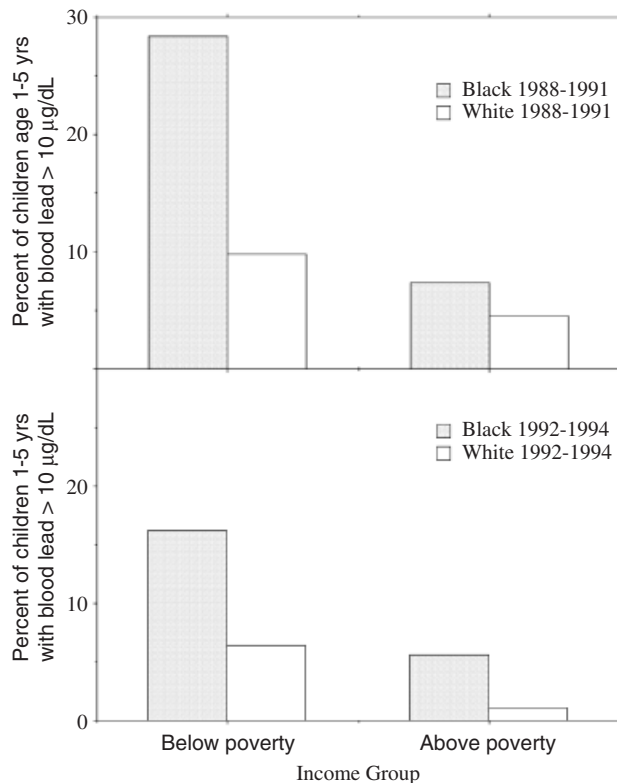


Figure 1. Percentages of children with blood lead above 10 µg/dl based on national surveys conducted by the Centers for Disease Control. The data for 1988–1991 were published by Brody et al. (1994), and the data from 1992 to 1994 are from EPA (2000).

reduction in risk such that in 1992–1994 a White child above poverty would have only .26 the chance of high lead compared with 1988–1991. In earlier data similar disparities occur. The 1984 national survey of blood lead found that 62% of Black children in poverty and 38% of Black children from families above the poverty level had blood lead greater than 15 µg/dl compared with 20% of White children in poverty and 10% of white children above the poverty level (Crocetti, Mushak, & Schwartz, 1990a).

It is clear that national lead abatement efforts have been effective, but they have been more effective for white and nonpoor children resulting in increasing racial/ethnic and income disparities. The racial and income disparities are especially disturbing, given that, in 2003, 58% of African American children lived in low-income homes (at 200% of the federal poverty level or less, the same criterion used in the lead exposure surveys) (National Center for Children in Poverty, 2004). This implies that the majority of African American children are at high risk for lead exposure compared with the rest of the population.

It is now accepted that the risk of various health problems can be transmitted across generations, al-

though there are likely to be multiple causal mechanisms (Chapman & Scott, 2001; Najman et al., 2004). Lead exposure risk can be transferred from parent to child across successive generations (Serbin et al., 1998; Serbin & Karp, 2003). In the case of lead, persistent poverty is a likely route of intergenerational transfer of lead exposure. In the 1984 national survey, racial and income disparities in lead exposure were found for both pregnant women and women of childbearing age, and it is well documented that lead crosses the placenta (Crocetti, Mushak, & Schwartz, 1990b). Breast feeding is another mode of intergenerational transfer (Ettinger et al., 2004; Moya, Bearer, & Etzel, 2004). Low-income mothers are likely to live in substandard housing with a higher presence of lead-based paint, plumbing, and lead-contaminated soil. These conditions increase lead exposure, which is then compounded by inadequate access to medical and nutritional information, and dietary habits (low-mineral, high-fat diets) that predispose both themselves and their children to higher lead absorption (Mahaffey, 1990). The same high fat/low mineral diet may predispose children to iron deficiency anemia (IDA). IDA, in turn, can facilitate additional lead absorption (Pollitt, 1994). Without proper nutritional intervention and lead abatement, these effects could continue throughout childhood and into adolescence, resulting in cumulative cognitive deficits such as lowered IQ and greater impulsivity. These cognitive effects, in turn, may contribute to risk for school dropout and early parenthood. Therefore, not only do lead exposure and IDA contribute to the poor health of a child, these conditions increase the likelihood that future generations will also live in poverty and, thereby, be further exposed.

Without proper lead abatement, nutritional intervention, and primary health care, these effects are likely to continue throughout childhood and into adolescence, resulting in cumulative deficits such as lowered IQ and diminished opportunities. Cognitive deficits, poor performance early in school, and poor behavioral regulation are all known risks for low educational attainment. In addition, at the national level it has been found that violent crime rates, the rates of pregnancy at age 15 or less, and unwed pregnancies are related to societal lead exposure over the last 50 years (Nevin, 2000).

#### National Policies

Three types of national policies that impact children's lead exposure have been enacted: (a) elimination of lead at the source, (b) regulations that

require testing children for lead exposure and abatement of lead in the homes of those children found to have high exposure, and (c) required information campaigns. We briefly review these programs and their effects.

*Elimination of lead sources.* At present, the environmental sources of most lead exposure in the United States are lead dust in older homes, lead in bare soil near homes that had lead paint, and lead in drinking water (Centers for Disease Control and Prevention, 1991; Lanphear, Burgoon, Rust, Eberly, & Galk, 1998). Federal regulations in the United States phased out lead from paint and tetraethyl lead from gasoline in the late 1970s. Lead in food containers also came under regulation in the early 1980s as did lead in plumbing solder and many industrial emissions from factories and smelters (Mushak & Crocetti, 1990). The phase-out of lead in gasoline and paint is often credited as having resulted in dramatic decreases in child lead poisonings, and resulted in a 37% decline in population mean blood lead levels between 1976 and 1980 (Lippmann, 1990). Other federal regulations to reduce lead exposure came considerably later. Lead abatement requirements in federally owned or subsidized housing were enacted only in the 1990s, and were tightened in 1999 (Housing and Urban Development, 2005). Federal regulations regarding lead in public utility drinking water were issued in 1991 (Environmental Protection Agency, 2005a); however, news reports have questioned the adequacy of water utility tests for lead in drinking water (Lennig, Becker, & Nakamura, 2004). Emissions from factories or smelters and "hot spots" from current and former lead industry sites continue to be a problem (Detroit Free Press, 2003; Kelly, 2004). Some organizations contend that enforcement of environmental regulations is inequitable and that it depends on factors such as income and the racial and ethnic composition of the community (Environmental Justice Resource Center, 1997).

*Required blood lead testing and action.* A program of testing and reporting cases is considered to be indispensable for any public health problem because testing and reporting allow assessment of the extent of a public health problem and aids in tracking the effectiveness of prevention efforts. Lead exposure testing has been required by law since 1991 for children less than 5 years of age from families with income less than twice the poverty level (Centers for Disease Control and Prevention, 1991; but see Needleman, 1998, for a discussion of the abandonment of primary prevention). In some areas of the country, head start centers require lead tests as a condition of enrollment, but this is not the case in all areas. For

example, the California Department of Health Services was sued in 2000 for failing to make regulations that would require doctors to test children for lead as mandated by federal law (Miller, 2005). Compliance with the lead screening requirements of federal law is mixed. For example, in the State of Wisconsin approximately 50% of Medicaid-eligible preschoolers are tested for lead exposure (J. Schirmer, personal communication, 2002). In Rhode Island approximately 80% are screened (Vivier, Hogan, Simon, Leddy, & Alario, 2001). However, a report to Congress found that only 19% of Medicaid children between 1 and 5 years are tested nationwide (General Accounting Office, 2001). It is apparent that compliance varies widely across states. The General Accounting Office report concluded that compliance and blood lead reporting could be improved by better systems for informing Medicaid-eligible families about the services available to them, including ways of notifying eligible families about blood lead testing other than when they seek medical services. Compliance and reporting could also be improved through better access to health care for Medicaid-eligible families and better coordination between managed care and community-based health services (General Accounting Office, 2001).

An elevated blood lead value in a child triggers required lead assessment and abatement actions if housing and urban development (HUD) financing is involved in the dwelling. For children from families that are not receiving any sort of federal housing subsidy (either directly or through the property owner's financing), high blood lead does not necessarily trigger enforcement action, depending on the state and local regulations as well as funding for investigating cases (Housing and Urban Development, 2005). HUD also makes grants to state and local governments for lead paint abatement in housing that is occupied by low-income renters. HUD estimated that the number of homes with lead hazards was reduced by 26 million between 1990 and 2003. In spite of these efforts, the socioeconomic and ethnic disparities in lead exposure persist.

In a study of lead-related housing policy, Brown et al. (2001) found that strict policy enforcement is an effective means of preventing additional lead exposures once an exposed child is identified. The study examined adjacent areas in two northeastern states from 1993 to 1998. One state engaged in "strict" policy enforcement techniques once a lead-poisoned child was identified that included criminal and civil penalties for failing to abate, inspection of all units at the address where lead poisoning was discovered, reporting the lead hazard to housing owners and to

all tenants, and referral to the state lead poisoning prevention program. The “limited” enforcement state conducted inspections only in the unit in which a lead-poisoned child resided, did not initiate civil or criminal action against owners, and did not inform other tenants. Abatement upon identification of lead hazard rarely occurred in limited enforcement areas. The authors determined that homes in areas of strict policy enforcement were 4.6 times less likely than homes in the area of limited enforcement to house children with blood lead levels above Centers for Disease Control (CDC) guidelines in the 5 years following the identification of a lead-exposed child (Brown et al., 2001).

Brown (2002) also showed that strict enforcement of lead in housing would be cost effective. Using the 2001 Consumer Price Index, Brown estimated that the societal cost of limited policy enforcement after identification of a lead-poisoned child was \$1,01,999 over the lifetime of *each* subsequently exposed child. Strict policy enforcement, conversely, has an estimated cost to society of \$56,639. The approximately \$46,000 savings from strict enforcement are due to lower costs for short- and long-term medical care, special education, and higher work productivity (Brown, 2002). Proper lead abatement saves society money and will save some families the heartbreak of dealing with special needs in a developing child.

*Parent lead hazard awareness and home risk reduction educational programs.* One aspect of federal lead prevention is “the importance of educating parents and children about the dangers of lead paint hazards in housing” (Environmental Protection Agency [EPA], 2005a). As of 1996, property owners of homes built before 1978 are required to provide buyers or renters with the EPA information pamphlet on lead hazards in the home, and a lead hazard declaration form. Declarations about lead in drinking water by local water utility companies have been required since 1991. In addition to these legally required declarations of lead hazards, a number of clinical trials have been conducted of in-home methods of lead reduction by parents. Is the information in the legally required declarations getting through, and do actions based on the information actually reduce the lead exposure of children?

A study in Milwaukee of knowledge of lead hazard from drinking water found that two thirds of the sample of 610 adults said they did not recall reading the information pamphlet (Griffin & Dunwoody, 2000). Also, self-reported reliance on the information pamphlet was not associated with knowledge of preventative behaviors. Ethnic minority group members were more likely to perceive a hazard from

lead and to believe that their home drinking water was contaminated, but they had less knowledge about exposure prevention measures than majority group members. Those who relied on health professionals for information about lead in drinking water had higher risk perceptions due to lead but also had a higher perception of personal control, although relying on health professionals was not associated with higher knowledge of preventative behaviors. The authors concluded that pamphlets about lead in drinking water are unlikely to yield effective preventative action.

Surveys to examine whether parents are aware of effective lead exposure preventive techniques and how to perform them have shown mixed results (Mahon, 1997; Mehta & Binns, 1998; Polivka, 1999; Porter & Severtson, 1997). In a study of primarily European American rural residents in Ohio, Polivka (1999) found that people could identify risk factors, means, and consequences of lead exposure, but were less knowledgeable of the importance of preventive efforts. However, of 70 women, infants, and children (WIC) participants without phones who were predominantly White (94%), unemployed (70%), and having only a high school degree or equivalent (75%), 29% were unaware that lead paint was still present in many homes and 71% did not know of the risk of lead-contaminated residential drinking water. Moreover, 90% of the entire sample was unaware of the preventive benefits of good nutrition and high calcium (e.g., drinking milk; eating greens high in calcium such as collards) and 55% did not know that regularly cleaning windowsills could help reduce exposure risk. Almost 22% of WIC participants did not know that children could be exposed to lead yet not appear ill, and 1 in 7 were unaware of the linkages between blood lead and learning problems or that lead exposure has been associated with long-term health effects. Low levels of knowledge about prevention strategies and the role of adequate nutrition in risk reduction were also observed in a sample of 2,225 Chicago area parents (80% Caucasian) (Mehta & Binns, 1998).

If pamphlets are unlikely to help, will a more intensive home intervention help? The Phillips Lead Project was a randomized trial of an intensive, culture-specific peer education program aimed at reducing blood lead levels among poor, urban children from birth to 36 months of age living in the Phillips neighborhood of Minneapolis, MN (Jordan, Yust, Robison, Hannan, & Deinard, 2003). The final sample included 378 mothers and their children, 184 intervention and 194 control, after a total attrition rate of approximately 40%. The attrition rate was equivalent

across the control and intervention conditions, and was partly due to the inability to obtain venous blood samples from all children. Seventy-eight percent of the sample was racial/ethnic minority (African American, Native American, Latino, Hmong, Cambodian, and Laotian), and 71% of participating children lived in poverty during the intervention. All participants had lead exposure below the CDC threshold of 10 µg/dl at the start of the study. The goal was to test an intervention to maintain low blood lead readings. The program provided basic lead exposure prevention information to all participants, but the intervention group received 20 bi-weekly, in-home, peer education sessions that included information about possible symptoms of lead exposure and strategies for reducing exposure such as housekeeping, nutrition, safe water usage, and hygiene information. The intervention was done carefully as shown by the fact that 90% of the final sample of intervention group mothers completed at least 19 of the 20 sessions. In addition, half of the intervention mothers completed 1 year of quarterly "booster" sessions. Despite many positive features that are important to successful prevention programming such as matching of peer educators' racial/ethnic and linguistic backgrounds with those of participants, intensive education sessions that included key information for reducing exposure to lead, and "booster" sessions (Nation et al., 2003), the intervention was only marginally effective. Blood lead rose above the CDC threshold in 19% of intervention group children compared with 27% of children of control group mothers ( $p = .08$ ). With a larger sample the results could have reached statistical significance, but it is still notable that even with an intensive targeted intervention 19% of the sample ended up with blood lead over the CDC cutoff. This compares to the national average of 16% for Black children living in poverty.

A similar resident dust control trial was carried out in a low-income sample in Rochester, NY (Lanphear et al., 1999). In addition to information about lead risk and home cleaning techniques, study participants were provided with cleaning equipment (i.e., a broom with dust pan, sponge mop with replacement heads, rubber gloves, double bucket, and Lead Away containing trisodium phosphate) and were visited by a "dust control advisor" over the course of the 18-month study. The authors concluded that home dust control strategies, even when monitored by a trained advisor, are ineffective without concerted lead abatement efforts such as replacing windows and old carpets containing lead dust (Lanphear et al., 1999).

### *Discussion: Public Policy and Family-Based Lead Exposure Prevention*

Family-based lead exposure prevention programs make several problematic assumptions about both the program content and the target population. First, there is the assumption that if the actions recommended in the educational program are carried out then the exposure hazard will be reduced. Lead dust control programs have been shown to be effective for reducing very high lead exposure ( $>30$  µg/dl), but the effectiveness of such programs for the typical levels of lead exposure at present in the United States is marginal (Charney, Kessler, Farfel, & Jackson, 1983; Hiltz, Hertzman, & Marion, 1995; Lanphear, Winter, Apetz, Eberly, & Weitzman, 1996). Even the intensive Phillips program failed to keep the blood lead levels of a high-risk sample at the national average for Black children in families living in poverty.

Second, family-based lead reduction programs include the assumption that parents have the time and resources to monitor risk and carry out the home exposure reduction behaviors on a regular basis. Among the challenges inherent in targeting the family of the child is that the responsibility for lead exposure reduction is placed on the individuals least prepared to accomplish the task—families living in poverty (e.g., McLoyd, 1998; McLoyd & Flanagan, 1990).

Parents living in impoverished conditions are at greater risk than their more affluent counterparts for a variety of environmental stresses such as limited accessibility to employment-based and public and private services, inadequate informal social supports that would help improve the families' living conditions, substandard and overcrowded housing, insufficient funds, unemployment, substance abuse, lack of child care, lack of food, and inadequate social outlets. These conditions can lead to negative psychological and health outcomes in adults (Attar et al., 1994; McLoyd & Flanagan, 1990; Pelton, 1978). Poor single mothers living alone with their children, in particular, are at a greater risk for anxiety, depression, and somatic complaints, and are more likely to have experienced the illness and death of a child or the imprisonment of a husband or boyfriend (McLoyd, 1998). Given the adverse effects of stress on learning and memory (Kuhlman, Piel, & Wolf, 2005; Payne, 1991; Shors, 2004) and the multitude of stressors associated with living in poverty, it seems that the likelihood that low-income parents will be able to adapt successfully to the addition of yet another stress, carrying out an in-home lead dust abatement program, is low.

Residential stability is the third assumption inherent to these family-based programs. Low-income families move quite frequently and many of the lowest income families experience repeated periods of homelessness (Bassuk, 1987; Wong, Piliarin, & Wright, 1998). Therefore, the strategies of an effective lead exposure intervention may be applicable to almost any dwelling, but parents must do their own informal risk assessment and adapt the intervention strategies every time they move.

In summary, enforcement and lead abatement have been shown to reduce the societal cost of lead exposure (Brown et al., 2001), but in-home dust control has limited efficacy. The federal phase-outs of lead from gasoline, paint, and food containers have been highly effective in reducing average lead exposure nationwide, but racial and income disparities persist. The national policies such as Medicaid lead screening and lead abatement in the homes of high-lead children have been successful, but only to the extent of compliance. Consistent child lead screening followed by lead removal or abatement targeted in high-risk residential areas of older homes appears to be needed to reduce the lead exposure of the highest risk segments of the U.S. population.

The long history of investigation into the developmental effects of lead exposures and public policy and preventive efforts to reduce children's lead burdens provides a benchmark against which to judge other pollutants to which children are exposed. This benchmark can also be used to evaluate progress in studying the developmental effects and reduction of children's exposure to other pollutants. In the next section, we review the research on the developmental effects of organophosphorus and carbamate pesticides as well as efforts to reduce exposures among children.

### **Case Example: Pesticide Exposures Among Children of Farm Workers**

The main similarity between pesticide exposure of farm workers' children and lead exposure of children living in poverty is that the substances are present in the home, are difficult for the family to control, and are inequitably distributed across ethnic and socioeconomic groups. Unlike lead, the potential developmental effects of childhood exposure to many types of pesticides are greatly understudied. Some pesticides have been shown to cause behavioral effects in rodents such as hyperactivity, learning and memory problems, and altered habituation (Icenogle et al., 2004).

In spite of the paucity of research on the effects of pesticides on human neurobehavioral development, there are reasons to be concerned about children's exposure. First, two widely used classes of insecticides, organophosphates and carbamates, inhibit cholinesterase. Cholinesterase inhibition leads to excess acetylcholine at the synapse, which in turn causes overactivation of cholinergic neural pathways. There is evidence that organophosphate and carbamate pesticides can negatively affect early life rodent brain development by interfering with gene signaling by cholinesterases as well as by inducing faulty wiring of the brain via other mechanisms (Aldridge, Meyer, Seidler, & Slotkin, 2005; Slotkin, 1999). The gene signaling functions of cholinesterase are sensitive to the concentration and developmental timing of release. Hence, cholinesterase-inhibiting pesticides have the potential to function as neurodevelopmental teratogens (for overviews, see Abou-Donia & Lapadula, 1990; Lauder & Schambra, 1999). Second, the potential of chemicals that alter cholinergic systems to function as neurobehavioral teratogens is supported by extensive data indicating that prenatal nicotine is harmful to children's development, including negative effects on habituation in infancy (Fried & Makin, 1987), attention, activity level, and standardized reading scores in childhood (Butler & Goldstein, 1973; Naeye & Peters, 1984). Nicotine is a cholinergic agonist. Because both nicotine and two major classes of insecticides increase the activity of cholinergic neural systems, there is reason to be concerned about the effects of insecticides that alter concentrations of cholinergic chemicals. Third, some types of pesticides are regarded as endocrine disruptors because they can bind to hormone receptor sites or alter the production of different hormones (Riegart & Roberts, 2001). Thyroid hormone function is critical to early brain development (Kilby, 2003), and some pesticides may disrupt thyroid function (Garry, Holland, Erickson, & Burroughs, 2003). Androgen or estrogen disruptors have the potential to alter sexual development and gender-related behaviors (Pierik, Burdorf, Deddens, Juttman, & Weber, 2004; Porter, Jaeger, & Carlson, 1999).

The EPA reference doses for pesticides are based primarily on animal research showing gross malformations, fetal loss, or tumor formation. Calculated separately for acute and chronic exposures, the reference dose is the highest daily intake that is estimated to produce, at most, a minimum level of adverse outcomes. Neither the neurodevelopmental altering nor endocrine disrupting potential of pesticides have yet been fully incorporated in the

estimated reference doses. The EPA is still in the process of reregistering a large number of pesticides following changes in some regulations.

### *Inequities in Pesticide Exposure*

Pesticide exposure is higher in children whose parents work in agriculture than in comparison children, and is estimated to exceed the EPA reference doses for some chemicals for at least a small percentage of children (Fenske et al., 2000; O'Rourke et al., 2000). Figure 2 presents data from a study of agricultural families in Washington compared with families of comparable ethnic and socioeconomic background (approximately 70% of both groups were Hispanic) (Fenske et al., 2000). The data show that for some pesticides the chronic reference dose is exceeded by many times for the children from agricultural families. Also, the median concentration of pesticides in the bodies of children from agricultural families is approximately 7 times the median concentration of pesticides in nonagricultural children. In a study of a Yuma, Arizona Hispanic agricultural community, the maximally exposed child in each age group (3-, 4-, and 5-year-olds) was found to have exposure ranging from 23 to 125 times the EPA reference dose for diazinon and methyl parathion, two cholinesterase-inhibiting insecticides (O'Rourke et al., 2000). In the locations where pesticide exposures of children have been measured, pesticide exposure in inner city children is also higher than those who live in the suburbs (Adgate et al., 2001; Landrigan et al., 1999). This is likely due to the relatively

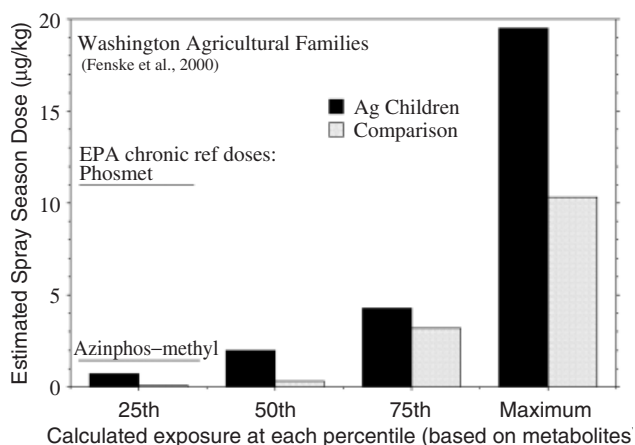


Figure 2. Spray season dose ( $\mu\text{g}/\text{kg}$ ) for children from families with a member working in agriculture and comparison children. Dose was estimated from urinary metabolites that could derive from either phosmet or azinphos methyl. The Environmental Protection Agency chronic reference dose for each pesticide is shown by the horizontal lines. Data are from Fenske et al. (2000).

heavy use of insecticides in many inner city dwellings in urban locations such as the boroughs of Brooklyn and Manhattan in New York City (Landrigan et al., 1999).

The research to date shows that the children of farm workers are more highly exposed to pesticides than others. Although data on the exact pathways of exposure are incomplete, several studies have concluded that workers are bringing pesticide residues home in their vehicles, on their clothes, and on their hair and skin (Curl et al., 2002). There is also spray drift from nearby fields, and some farm workers may use pesticides in their homes more frequently than others because of the presence of pests in "labor camp" type of housing (Quandt et al., 2004). Schools in agricultural communities may also have higher exposure to pesticides than in nonagricultural areas, although thorough studies have not yet been done. Registration of one soil fumigant was canceled in California because the levels detected in a nearby school were approximately 800 times the acceptable exposure level (National Academy of Sciences, 1993). Pesticide drift from a nearby cotton field was reported to have caused acute illnesses in both teachers and students in a recent incident in Texas (Tanner, 2005).

### *National and State Pesticide Exposure Reduction Policies*

Pesticide policy became a hot issue in the United States after the publication in 1964 of Rachel Carson's *Silent Spring*, a well-researched book about the indiscriminate use of pesticides and the scientifically documented negative effects on both people and nontarget organisms (Carson, 1964). DDT, a widely used insecticide that is environmentally persistent, was banned from use in the United States in 1972. Recent years have seen many changes at the national policy level, but most of the activity is in the area of exposure monitoring and poisoning incident reporting with less attention to reducing children's exposure (see Wargo, 1996, for an overview of pesticide policy in the United States). The U.S. Department of Agriculture, Environmental Protection Agency, and the Food and Drug Administration began a program of monitoring pesticide residues in foods in 1991 in cooperation with state agencies (U.S. Department of Agriculture, 2005). The National Academy of Sciences issued a report on pesticide residues in children's diets in 1993. The report recommended that in order to estimate the risks of pesticide residues in foods, three kinds of additional information were needed: (a) the food consumption patterns of infants and chil-



dren, (b) the pesticide residues that are found in the foods that are most consumed by infants and children, and (c) the developmental toxicities of the pesticides found in those foods (National Academy of Sciences, 1993). Progress has been made in these areas. For example, some surveys of pesticide exposure are being conducted by the CDC, and there are targets for reduced exposure by 2010 for all Americans (Centers for Disease Control and Prevention, 2005). The Food Quality Protection Act of 1996 includes an additional safety factor for pesticide exposure in children's foods (Environmental Protection Agency, 1996). In 1999 the EPA cancelled the use of two organophosphorus pesticides on foods commonly eaten by children. The agency, supposed to rereview existing pesticides by 2006, says it has licensed new pesticides that are intended to be lower risk, and is encouraging the development of low-toxicity natural substances that have pesticidal characteristics (Environmental Protection Agency, 2005b).

In some places, employers are required to offer medical monitoring to workers that handle pesticides. The first year of a mandatory monitoring program found that one in five pesticide handlers in the state of Washington had significant cholinesterase inhibition, and that about 4% had enough reduction in cholinesterase to require them to be removed from pesticide handling (Beecher, 2005). Being removed from work, or reassigned to a lower paying job than pesticide handling, is obviously a hardship for the worker. Testing is not currently required for field workers who are not directly applying pesticides.

Methods of reducing the pesticide exposure of workers were suggested by a National Academy of Sciences panel in 2000: addition of odors to pesticides so that workers can be certain when pesticides are present, restriction of certain pesticides to use by "prescription" only (similar to the way in which antibiotics are used in medicine), and improvement in the packaging and application technologies so that lower volumes are used and drift is reduced (National Research Council, 2000b). Another potential policy change to protect both farm workers and their families would be to extend the field reentry times after pesticide applications. Lower levels of pesticide metabolites in urine were found in apple thinners in Washington when they entered the orchards after a longer time period (Fenske, Curl, & Kissel, 2003). Increasing the buffer distance from residences for application of pesticides might also help, but compliance with this would require monitoring similar to the monitoring of compliance with the Worker

Protection Standards (these standards are described and discussed below). Of course, an even more extreme step is to ban a pesticide, either totally or from a specific use. Levels of banned pesticides do decline over time in farm worker homes (Fenske, Lu, Barr, & Needham, 2002), but the safety of the alternatives to the banned substances must also be considered.

#### *Family Education and Exposure Reduction Programs*

The EPA has developed a number of information pamphlets such as "Pesticides and food: What your family needs to know" distributed to grocery stores, and "Citizens guide to pest control and pesticide safety" (Environmental Protection Agency, 2005b). These pamphlets are directed toward the general population. The "Citizens guide" pamphlet has suggestions for avoiding pesticide exposure for people who live near fields (stay indoors and close windows and doors), as well as "safe" use of pesticides at home (emphasis on reading and following the directions on the label). EPA also has information on the web on preventing accidental pesticide poisonings at home, including keeping pesticides out of reach of children.

*The EPA worker protection standards:* The most widely disseminated federal pesticide exposure prevention effort targeted at farm workers is the EPA's worker protection standards (WPS) which were developed in 1992 and implemented in 1995 (Arcury, Quandt, Austin, Preisser, & Cabrera, 1999; Environmental Protection Agency Pesticide Worker Protection Standard, 40 CFR Part 70.130, 1992). Agricultural workers, mostly Spanish speaking or local dialect speaking migrants originally from Mexico and Central America, typically have income several thousand dollars below the federal poverty level, and live in housing with inadequate plumbing or heating, a high likelihood of peeling paint, and pests (Applied Survey Research, 2005). These living conditions affect the ability of a family to reduce exposure by their own actions at home. The purposes of the WPS are to "reduce the risks of illness from workers' and handlers' occupational exposures to pesticides. . . and also from the accidental exposure of workers and other persons to such pesticides" (Environmental Protection Agency, 1992, 170.1, p. 211). Hence, a primary focus is avoiding acute poisoning symptoms. The WPS requires exposure prevention training for all farm workers who enter areas within 30 days of pesticide treatment or areas subject to a period of restricted entry. Workers must receive training in 11 content areas by a certified trainer in a manner understandable by workers (i.e., use of

Table 1  
*Environmental Protection Agency (EPA) Worker Protection Standard  
 Required Content Areas (EPA 170.130, 1992; Arcury et al., 1999)*

1. Where and in what form pesticides may be encountered during work activities.
2. Hazards of pesticides resulting from toxicity and exposures, including acute and chronic effects, delayed effects, and sensitization.
3. Routes through which pesticides can enter the body.
4. Signs and symptoms of common types of pesticide poisoning.
5. Emergency first aid for pesticide injuries or poisonings.
6. How to obtain emergency medical care.
7. Routine and emergency decontamination procedures, including emergency eye flushing techniques.
8. Hazards from chemigation<sup>a</sup> and drift.
9. Hazards from pesticide residues on clothing.
10. Warnings about taking pesticides or pesticide containers home.
11. Requirements . . . designed to reduce the risks of illness or injury resulting from workers' occupational exposures to pesticides, including application and entry restrictions, the design or the warning sign, posting of warning signs, oral warnings, the availability of specific information about applications, and the protection against retaliatory acts.

<sup>a</sup>Chemigation refers to the application of pesticides through the use of irrigation systems.

translators or material written in agricultural workers' dominant language) within 5 days of working in a pesticide treated area (see Table 1 for a summary of the key aspects of the WPS). Very little of the information pertains directly to prevention of indirect or direct exposure of one's family members, but there is the implication that one could inadvertently carry pesticides home on one's person.

Recent evaluations of the implementation and the effectiveness of the WPS produced disappointing results (National Research Council, 2000b, Chap. 3). In North Carolina only about one third of workers said they received the training and most workers could not recall methods for protecting themselves from exposure (Arcury et al., 1999). In Washington, approximately two thirds of workers said that pesticides touch their clothes daily and slightly over half said that they got pesticides on their skin daily and that they breathed pesticide dust daily (Thompson et al. 2003). Approximately, one in four field workers said that water for hand washing was not available, and two out of five said there was either no soap or towels. Showers were reported to be available by only 45% of pesticide applicators even though showers are required for applicators under WPS. This study in Washington also found that only about half of the workers said they washed their hands

immediately after work, removed their work boots before entering the home, or showered within 1 hr of returning home. Approximately three out of four say they removed their work clothes before holding children. In a California study, pregnant women who lived with a farm worker (or were themselves workers) reported a variety of behaviors that were considered a risk for pesticide exposure, such as eating while in the fields without hand washing, eating produce from the fields without washing it, and wearing work clothes inside the home (Goldman, Eskenazi, Bradman, & Jewell, 2004).

#### *Discussion: Pesticide Risk Awareness and Exposure Prevention*

The lessons learned from the inequities in lead exposure and prevention have not yet been applied to pesticide exposure. Just as lead exposure was presumed during the first part of the 20th century to be safe below certain thresholds, pesticides are presently presumed by many to be safe and there is little concerted effort to reduce societal exposure (Richter, 2002). There has been progress in pesticide exposure monitoring, but little of that progress has extended to exposure prevention for the most highly exposed and vulnerable segment of the population, the children of farm workers who live near active conventional agricultural areas. Although there is considerable debate about the levels at which exposure to specific pesticides may be harmful, there is little doubt about the social inequities in exposure. Behavioral effects are not yet incorporated in the EPA reference doses, and behavioral effects would be expected to occur at lower levels of exposure compared with acute illness (Weiss, 1983).

Programs aimed at the general population, such as the EPA pesticide information pamphlets and strict pesticide labeling requirements, will be unlikely to succeed when applied to farm worker families because the routes of exposure are slightly different—workers tracking the substances home and drift from fields rather than trace exposure in foods and home use of pesticides (Lu et al., 2004). As with lead exposure, programs that encourage workers to alter their personal habits may also be expected to be only minimally effective because the pesticides are present in their environments in relatively high concentrations. Worker awareness and exposure reduction programs are unlikely to help unless they are supported by workplace changes such as better access to water, soap and towels for routine hand washing at work, a requirement and facilities for changing clothes and shower before

going home, wearing special clothes at work, and compliance with the existing WPS. Other policy changes that reduce pesticide presence in farm workers' environments, such as longer field reentry periods, will await better risk assessment data.

### Next Steps for Developmental Research

There are at least two leadership areas for developmental researchers in the study and prevention of low-income and ethnic minority children's exposure to environmental pollutants. First, developmental researchers are poised to direct basic and applied research about the effects of pollutant exposures and ways to reduce children's pollutant burdens. Second, by bringing awareness of the potential developmental effects of pollutants into undergraduate and graduate level teaching, developmental scholars can significantly contribute to promoting public awareness of both risks and preventive strategies.

#### *Continued Basic and Applied Research*

With the exception of studies with an explicit focus on environmental toxicology, developmental researchers tend to neglect aspects of the physical environment that transmit toxic environmental substances contemporaneously and across generations. The psychosocial stressors of poverty and minority status do not occur independently of exposure to environmental pollutants, but these influences on child development are rarely studied together. In studies of lead exposure in economically disadvantaged populations, the social quality of the home (measured by the Home Observation for the Measurement of the Environment [HOME], Bradley & Caldwell, 1977) correlated  $-.23$  to  $-.46$  with lead exposure (Bornschein, 1985; Ernhart, Morrow-Tlucak, Wolf, Super, & Drotar, 1989). Similarly, single-parent family, maternal education, and maternal intelligence are variables that show significant correlations with children's lead exposure in the expected directions, with a magnitude ranging from  $.2$  to  $.35$  (Ernhart et al., 1989; Lanphear & Roghmann, 1997; McMichael et al., 1992). Such correlations of a pollutant with factors regarded by the field of developmental psychology as causes of intellectual and behavioral outcomes demonstrate the potential impact of pollutants on child outcomes and their importance to furthering our understanding of developmental processes.

Pollutant exposure levels are often included as essential covariates in epidemiological studies of the effects of toxic exposures such as maternal alcohol use (S. W. Jacobson & J. L. Jacobson, 2000). Maternal

smoking and/or alcohol use are often entered as covariates in studies of lead exposure as well as Polychlorinated Biphenyl (PCB) or mercury exposure from fish, and methylmercury is used as a covariate in studies of PCB exposure (Canfield et al., 2003; J. L. Jacobson & S. W. Jacobson, 1996; Stewart, Reihman, Lonky, Darvill, & Pagano, 2000). However, it is exceedingly rare to see lead or any other pollutants used as covariates in studies of sociodemographic influences on intellectual outcomes, or in studies of personality characteristics such as temperament, aggression, antisocial behavior, or impulsivity.

Exposures to pollutants could be included in studies of multiple and cumulative risk (Sameroff & Seifer, 1995; Seifer & Sameroff, 1987). The concepts of risk and resilience could be expanded to include pollutant exposure as a potentially significant risk for children. To date, there is virtually no information about potential intraindividual, family, school, or community factors that offset the negative effects of pollutant exposures or if any such factors exist. By expanding the concepts of risk and resilience to include pollutant exposures, developmental researchers can fruitfully consider topics such as whether the relations between environmental pollutant exposures and adverse behavioral or academic outcomes are mediated or moderated by preexisting health and social conditions or risks.

Inclusion of environmental pollutants in developmental studies can also contribute new perspectives on racial/ethnic and income differences in developmental outcomes. The school achievement gap between African American and White students has been well documented (e.g., Campbell, Hombo, & Mazzeo, 2000; Jencks & Phillips, 1998, but see National Center for Education Statistics, 2005), and conditions related to lack of wealth, racial discrimination, parents' ability to assist with homework, and school quality are among the likely causes (Lubiencki, 2002; Obed, Ault, Bentz, & Meskimen, 2001; Orr, 2003; Zady & Portes, 2001). But a thumbnail estimate of the potential impact of social disparities in lead exposure on the cognitive performance of children provides a different look. Canfield et al. (2003) found that each increase of  $1\ \mu\text{g}/\text{dl}$  blood lead (below  $10\ \mu\text{g}/\text{dl}$ ) affected the IQ test scores of preschoolers by approximately  $-1.4$  points, *after* adjusting for maternal IQ, race, maternal education, tobacco use during pregnancy, family income, HOME score, child sex, birth weight, and the child's iron status. Combining this with Brody et al.'s (1994) national survey data showing that the geometric mean blood lead levels of Black children were  $5.9$  versus  $2.9\ \mu\text{g}/\text{dl}$  for the Whites translates into an average group

difference of approximately 4.2 IQ test points, or approximately one quarter of a standard deviation. This effect size, although slightly above “small” (Murphy & Myers, 1998), creates a noticeable difference in the tails of the distribution (see Needleman, Leviton, & Bellinger, 1982, for an example). Assuming a normal distribution of IQ, a shift of .25 standard deviation increases the percentage of children scoring below 80 from approximately 9.3% to 14.2%. Brody et al. (1994) did not report means broken down by both income and racial/ethnic group.

Recall that lead exposure is itself correlated with maternal IQ scores, maternal education, income, and race, many of the variables that were partialled out by Canfield et al. (2003). The raw regression slope between IQ test scores and lead was approximately  $-2.5$  at age 5. Thus, lead exposure could be responsible for a group difference of up to 7.5 IQ test points, a “medium” effect size that would be expected to more than double the percentage of children expected to score at an IQ of 80 or below from 9.3% to 20.3%. Therefore, the societal inequities in lead exposure could be expected to yield a group difference of approximately one quarter to one half of a standard deviation on IQ tests and associated cognitive outcomes. However, it is important to note that Canfield et al.’s (2003) findings partialled out maternal factors and race, which may contain additional effects of lead exposure in addition to the direct effect. In keeping with the risk focusing premise (Schell, 1997), it is possible that children are indirectly affected by their mothers’ previous lead exposure through a chain of events in which maternal lead exposure would be expected to lower maternal educational attainment which, in turn, would lead to worse employment opportunities and subsequent low income, as well as lower quality home environments.

Exposures to environmental pollutants are not the only mechanism driving racial/ethnic differences in intellectual and other outcomes. Lack of resources, inadequate health care, and racial discrimination are also contributors to these differences (Attar et al., 1994; Epstein, Griffin, & Botvin, 2002; Obed et al., 2001; Prelow, Danaff-Burg, Swenson, & Pugliano, 2004). Nevertheless, the question of whether exposures to environmental pollutants may be among the causes of racial/ethnic and income differences in child outcomes has not yet been addressed systematically.

#### *Methodological Approaches for “Pollutant-Informative” Studies of Child Development*

A pollutant-informative design would be one in which the effects of key exposures would be re-

moved from the dependent variable before assessing the contribution of other sources of influence on development; thus, such a design would yield estimates of the effects of other variables over and above the key pollutants. As illustrated above with our calculations, such designs may considerably alter the field’s estimates of the influences of sociodemographic variables on development. A similar point has been made recently in a major review of the developmental effects of micronutrients, social variables, and heavy metal exposure (Hubbs-Tait, Nation, Krebs, & Bellinger, in press).

The ideal pollutant-informative design would measure targeted exposures both prospectively and contemporaneously. It is usually best to have some history of exposure rather than only concurrent exposure because pollutant exposures may have different toxic properties at different points in development. But which pollutants should be measured? Social science research occurs in specific locations with specific communities of people (Becker, 1998). Careful consideration of the contexts in which research is conducted can inform the selection of important pollutants for inclusion in research designs. Table 2 presents examples of internal and external pollutant exposure measurement techniques that researchers might incorporate into developmental studies. Internal assessment techniques involve collecting samples of tissues or bodily products that can be assayed for specific chemicals. The best practice is to use an internal assessment of exposure where possible, because internal assessments yield estimates of the concentrations of pollutants that have actually entered the body. Just as developmental researchers interested in psychosocial stress routinely collect saliva and assay it for cortisol, it is relatively easy to collect overnight urine samples, hair samples, and deciduous teeth (i.e., shed baby teeth).

External measures of pollutant exposure that rely on either self-report, observation by members of the research team, or collection of data about characteristics of housing, neighborhoods, and schools can also be useful. For example, studies of the effects of aircraft and road traffic noise on children’s reading, attention, and school performance have used the proximity of housing to expressways (Cohen, Glass, & Singer, 1973) or proximity of neighborhoods to air flight paths as their primary indices of noise exposure (Hygge, Evans, & Bullinger, 2002). Further, a widely cited study of multiple pesticide exposure in children in Mexico used location of residence as a proxy for pesticide exposure after establishing that two communities of the same ethnic background had

Table 2  
*Examples of Ways to Measure Environmental Pollutants in Child Development Research*

Internal measures of exposure	External measures of exposure
<ul style="list-style-type: none"> <li>• Urine—pesticide metabolites, lead, mercury</li> <li>• Deciduous teeth—lead, strontium 90</li> <li>• Hair—lead, mercury, cadmium and other heavy metals</li> <li>• Blood—cholinesterase inhibition, lead, mercury, PCBs, dioxins, organochlorine pesticides (e.g., DDT)</li> <li>• Breast milk—organochlorine pesticides (e.g., DDT), other organochlorine and lipophilic chemicals (e.g., PCBs, PBDEs), lead, mercury</li> <li>• Personal air sampler—particulate air pollution</li> </ul>	<ul style="list-style-type: none"> <li>• Public record review—air pollution records; age of individual buildings or average age of buildings by neighborhood; building inspection violations; building permits for renovations or lead removal (lead)</li> <li>• Geocoding—proximity to bus depot, freight train station, major highways, airport flight paths (noise, particulate air pollution); operating or former points sources for pollutants (gas pipeline terminals, lead smelters, mines, or factories)</li> <li>• Self-report—dietary habits (lead, mercury, pesticides); home use of pesticides; recreational habits (sport fishing, hobbies that involve toxic substances such as lead or mercury); children’s normal play areas (arsenic from treated wood decks or play sets, lead in soil); nuisance level, paint condition, proximity to bus depot (lead, mercury, particulate air pollution, noise)</li> <li>• Observer report—pesticides in containers in the home; proximity to factories, major highways, airport flight paths (particulate air pollution, lead, mercury, noise)</li> <li>• School record review—blood lead levels for head start students</li> <li>• Medical record review—blood lead tests</li> </ul>

Note. PBDE = Polybrominated Diphenyl Ether

vastly different pesticide exposure because of proximity to conventional agriculture (Guillette, Meza, Aquilar, Soto, & Garcia, 1998). The landmark studies of the effects on development of maternal smoking or alcohol use during pregnancy were based on self-reported substance use by the pregnant women. This illustrates how even simple measures can sometimes be very informative in discovering new effects.

*Participatory Action Research to Inform Future Studies and Preventive Interventions*

Participatory action research is now regarded as an important approach in fields such as epidemiology, public health, community psychology, and natural resource management. A central tenet of the approach is that community members can present concerns to the researchers that might otherwise be overlooked. Partnerships with communities using action research techniques not only increase the likelihood of successful access to the population of interest but can also inform the creation of relevant research questions and set the stage for later community action, intervention, and prevention programs (Dalton, Elias, & Wandersman, 2001; Elias & Dilworth, 2003). The approach is especially useful for work with historically underrepresented or at-risk populations (Dalton et al., 2001), and has been suc-

cessfully used to study a wide range of phenomena including poverty (e.g., Collins, 2005) and immigration experiences (e.g., Prilleltensky, 1993). It has also been a vital component in the development of interventions to promote successful social and emotional development in adolescence (e.g., Small, 1995) and school-based violence prevention (e.g., Hunter, Elias, & Norris, 2001).

Participatory action research has already been successfully used in the study of the effects of environmental pollutants. For example, Thompson et al. (2003) used input from farm workers to create a culturally specific pesticide exposure prevention program for Hispanic migrant workers in Washington State. Similarly, researchers in Chicago surveyed community members concerning a highway reconstruction project and used community input to develop measures of air quality (Dorevich, Persky, Scheff, Erdal, & Conroy, 2005). In another study of air quality during the demolition of high-rise public housing, the Chicago researchers were able to open communication channels with both city officials and community groups, and, based on air quality measures, demolition activities were modified for public safety (S. Dorevitch, personal communication, May 30, 2005). There is always a trade-off in such research between using a “gold standard” design versus accomplishing a research project that yields some new

knowledge plus benefits for the community of research participants (McCall & Green, 2004). However, failing to involve participants in the research can backfire by creating resentment. There are also serious ethical issues about providing full information to participants about potentially harmful pollutants where those substances are being studied (Bellinger & Dietrich, 2002; Needleman, 2002).

Developmental researchers can contribute to the reduction of exposure risk among low-income and ethnic minority children through research that couples action research and community-based preventive interventions with longitudinal developmental studies about the potential effects of pollutants and potential protective factors. Developmental scientists possess both the knowledge and the expertise to provide developmentally appropriate interventions to ameliorate the negative effects of pollutant exposure. Such future intervention programs must move beyond short-term, tenant-, family-, and worker-focused approaches to longer term interventions that incorporate multiple domains, are implemented in a manner relevant to the cultural context, and promote collaboration between tenant or employee, landlords or employers, and governmental agencies.

#### *Education and Promoting Public Awareness*

Developmental scientists are in an optimal position to take the lead in educating the public about potential environmental risks for child development through their classroom teaching. The latter goal is beginning to be addressed in undergraduate textbooks. McDevitt and Ormord (2004), for example, briefly discuss the developmental risks of lead exposure in toddlerhood and early childhood. Feldman (2004) goes further, briefly mentioning both lead and the relationship between particulate air pollution and asthma risk among low-income children. Laura Berk's widely used developmental text includes discussion of the effects of environmental pollutants on prenatal development and the relationship between particulate air pollution and asthma (Berk, 2005). Similarly, some undergraduate texts on atypical child development, although focusing heavily on family and community level poverty, violence, and risks associated with single parenthood, do mention that pollutants can be associated with deleterious psychological and behavioral outcomes (e.g., Mash & Wolfe, 2005; Wicks-Nelson & Israel, 2003). The textbooks mentioned here, while good examples of ways to connect the dots between pollution and child development, typically cover only one or two pollutants, spend very little time discussing the

specific deleterious effects of pollutants, and have little information on how to prevent pollutant exposure.

#### **Conclusion**

Exposures to some environmental pollutants and risk for subsequent developmental deficits are burdens disproportionately borne by low-income, ethnic minority children. Unfortunately, low-income, predominately ethnic minority neighborhoods are often already sources of higher risk in the forms of limited resources, high crime, unemployment or underemployment, and poor educational opportunities (Attar et al., 1994). However, the social issue of children's exposures to environmental pollutants is a context for the advancement of developmental science (Pedersen & Totten, 2001). By incorporating pollutant-informative designs into developmental research, developmental scientists can increase knowledge about important developmental processes such as the acquisition of cognitive abilities and behavioral self-regulation.

We examined inequities in environmental pollutant exposure and exposure prevention with a particular focus on the increased risk of lead and pesticide exposures among specific subgroups of low-income and ethnic minority children. Similar inequities have been found for other environmental pollutants, including noise, particulate air pollution, industrial waste, and, in some locations, PCBs (Moore, 2003). It is only recently that toxic exposures such as mercury and a variety of pesticides have been included in national health surveys conducted by the Centers for Disease Control and Prevention. The extent of the risk to lower income children has yet to be discovered in many areas. Developmental researchers have an important role in documenting and correcting the inequitable risks of exposure to environmental pollutants through basic and applied research, community collaborations to develop effective interventions, and public education.

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