CHILD DEVELOPMENT PERSPECTIVES

# Environmental Contaminants and Child Development

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**ABSTRACT**—***Developmental scientists have long been interested in how the environment influences children's development. However, with few exceptions, they have not researched how exposure to contaminants in the physical environment affects developmental processes. Children are uniquely at risk for exposure to contaminants because they drink more, eat more, and breathe more air than adults as a proportion of their body weight. In this article, we provide an ecosystems perspective to illustrate how contexts—from the prenatal environment and neighborhood-level exposure to laws and policies—contribute to children's exposure to contaminants. We also discuss four mechanisms that account for how and when exposure to contaminants affects children, and we provide examples to spur research on these mechanisms. We conclude with recommendations to foster integrative science where developmental science interacts with environmental health and toxicology.***

**KEYWORDS**—*contaminants; children; developmental science; environmental health; toxicology*

Environmental influences on children's development are well known; family, school, and neighborhood affect a range of developmental outcomes. The physical environment is also influential, yet developmental scientists have studied its impact less. Many chemicals that enhance the quality of life in industrialized societies are considered contaminants in the physical environment because they pose risks to health and development. These contaminants are especially risky for children because they drink more, eat more, and breathe more air than adults in proportion to their body weight (1). Infants and young children also spend more time close to the ground and engage in frequent hand-to-mouth behavior, and their metabolism is immature, rendering them less able to cope with toxic chemicals. Moreover, because of rapid growth and brain development in the early years, contaminants can contribute to potentially irreversible developmental delays.

Chemical contaminants include heavy metals like lead, mercury, and cadmium. Other contaminants are synthetic chemicals such as persistent organic pollutants (POPs) that are resistant to environmental degradation. POPs include intentionally produced chemicals like polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethane, and unintentionally produced chemicals from industrial processes. Other synthetic chemicals were developed for modern conveniences, including phthalates and other *plasticizers* used to soften materials as well as phenols such as bisphenol A (BPA) used in plastic storage containers and in the resins coating the inside of metal food cans.

Over the past several decades, research in toxicology and environmental health has uncovered links between exposure to these contaminants and impairments during childhood in cognition, behavior, and health (2, 3). For example, elevated levels of lead have long been associated with lower scores on tests of intelligence (4, 5). Moreover, conduct problems and other behavioral difficulties have been linked to exposure to heavy metals, pesticides, and other contaminants (6–8). Exposure to contaminants is also associated with problems in children's physical development, including obesity (9). Risks associated with contaminants have led to policies and laws, such as the U.S. Food and Drug Administration's ban on BPA in baby bottles and the packaging of baby formula. However, many contaminants persist in the environment, as evidenced by the crisis in Flint, Michigan, involving lead in water service lines and other aging infrastructure (e.g., paint in older buildings).

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Given the wide-ranging impacts of children's exposure to specific chemicals, research on exposure to contaminants must be informed by the science of child development, and environmental health and toxicology findings must advance developmental science. However, even though a few developmental scientists have tackled this topic, integrating developmental science and the fields of environmental health and toxicology is rare. As evidence of this problem, the flagship journals of developmental science, *Child Development* and *Developmental Psychology*, have not published recent articles on the developmental outcomes of contaminants—even though they once published articles on the impacts of contaminants on children (10). With these concerns in mind, in this article, we provide an ecological systems perspective on exposure to contaminants and discuss four mechanisms of exposure. We also recommend ways to integrate developmental science with relevant disciplines, including toxicology and environmental health.

## AN ECOLOGICAL SYSTEMS PERSPECTIVE ON EXPOSURE TO CONTAMINANTS

Bronfenbrenner's ecological systems theory is commonly used to describe how children's environmental context influences their development (11). Most often, this approach is applied to understanding the impact of the home, the school, the neighborhood, and the broader culture on psychosocial development, but Bronfenbrenner's perspective applies equally to influences from the physical environment. As shown in Figure 1, our ecological systems perspective on exposure to contaminants depicts how many layers of children's surroundings affect their exposure to contaminants. Children are exposed to contaminants through the microsystem, or their interactions with their immediate surroundings. The prenatal environment contributes to exposure to contaminants at the microsystem level (10). Within the postnatal environment, paint, dust, water, and building materials are potential sources of exposure to contaminants at home and at school (12–14). Within neighborhoods, soil and air pollution can be sources of exposure, too, especially in urban settings where, for example, rates of lead poisoning from soil are much greater than in nonurban settings (15). Disparities in the conditions of microsystem contexts might account for a portion of socioeconomic inequalities in childhood, including the achievement gap (16).

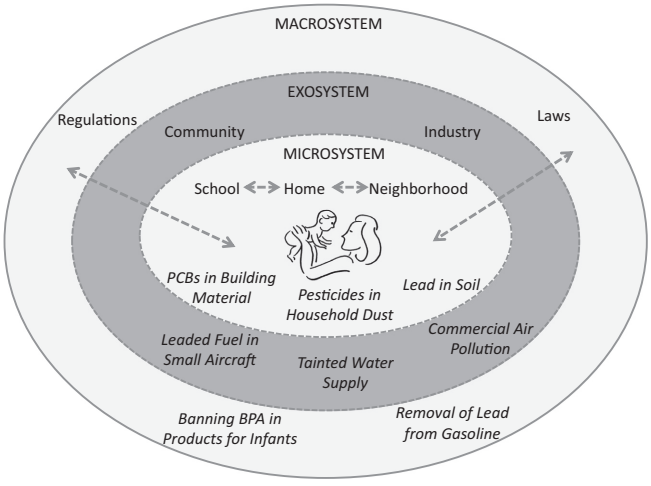


*Figure 1.* Ecological systems perspective on exposure to contaminants at the micro-, exo-, and macrosystem levels.

Sources of exposure can also be found within children's exosystem, the settings that do not directly include children but influence what they experience in their immediate surroundings. These exosystem settings may influence exposure by contaminating air, dust, water, and other sources in the immediate settings of children's microsystems. For example, living near sites of industrial pollution can expose children to heavy metals and other contaminants (17). Also, small aircraft use leaded fuel and children who live near airports in which airplanes use this kind of fuel have higher levels of lead in their blood than children who do not live in such proximity to planes (18). Municipalities can also be sources of contamination. For example, children who live in homes connected to wells with higher levels of manganese have greater concentrations of this chemical in their hair and more behavior problems in the classroom than children whose homes are not connected to wells with manganese (19).

At the macrosystem level, cultural values, policies, and laws contribute to children's risk for exposure to contaminants. For example, structural racism and discriminatory beliefs and practices can contribute to the likelihood that children will experience a substandard environment with higher levels of exposure to contaminants (20). As another example, lead-related policy changes in Massachusetts contributed to a drop in the proportion of children who tested in the unsatisfactory range on state academic tests (21). Policies and laws can also lead to regrettable substitutions when chemicals whose toxicity is less well understood replace prohibited chemicals (22). For example, bisphenol S is sometimes used when laws prohibit using BPA, resulting in products that are labeled BPA-free but still contain potentially toxic chemicals.

Applying the ecological systems perspective on exposure to contaminants to research on situations where children have experienced exposure to contaminants could help strengthen connections between developmental science and toxicology and environmental health. For example, during the water crisis in Flint, Michigan that began in 2015, young children were more likely to have elevated levels of lead in their blood after the community changed to a water source that was more corrosive to the lead pipes that supply water to homes (23). Many consider the change in water source and subsequent spike in blood lead levels a public health failure resulting from missteps at many levels of government and regulation (24). Thus, actions at the exosystem and macrosystem levels harmed children in this largely low-income and ethnic-minority community by tainting the

water in their homes. Developmental science can tell us why some children may be at greater risk from tainted water by focusing, for example, on microsystem-level variability in the family context, such as some parents being less attentive to the water their children were drinking (25). Such research could also improve coordinated responses to public health emergencies involving contaminants and vulnerable young children by helping identify the families who might benefit the most from immediate assistance.

Developmental science would also be strengthened if the ecological systems perspective on exposure to contaminants informed conceptual models of key indicators of child development. For example, growth in executive functioning is a marker of healthy development during the preschool years, and exposure to contaminants including lead and PCBs is related to less optimal executive functioning (26, 27). Thus, conceptualizations of the development of executive functioning should include these contaminants among the factors that interfere with normative development of working memory, attention, and other facets of executive functioning. Conceptualizations could also incorporate risk factors in children's immediate physical surroundings as well as their broader ecological context (including policies and laws) as factors that can impede the development of executive functioning by increasing children's exposure to contaminants.

## HOW EXPOSURE AFFECTS CHILDREN

In addition to identifying the main effects of exposure to contaminants, toxicologists and environmental health researchers have increasingly focused on how and when specific exposures affect children. This movement toward understanding how exposure affects children should be familiar to developmental scientists because it is similar to work elucidating when and how specific environmental contexts shape children's development. Next, we summarize four key mechanisms and provide examples to spur related research.

First, *mediating mechanisms* are increasingly studied to understand how exposure to contaminants is harmful. For example, exposure to chemicals including BPA is linked with metabolic changes and alterations in gene expression that are thought to lead to obesity (28). Other studies investigate possible neurobiological mediators that are proposed to link exposure to contaminants with impairments in cognition and behavior. For example, recent studies have focused on brain alterations, such as reduced volume of gray matter in adulthood, following exposure to lead in childhood (29). Researchers should test physiological and brain-based pathways as mediators of associations between exposure to contaminants and developmental outcomes. Thus, research on mediating processes should mirror ongoing efforts in developmental science, such as recent studies of the effects of differences in socioeconomic status on brain volume (30). Studies could incorporate exposure to contaminants with more traditional environmental measures (e.g., socioeconomic status) when examining paths to children's cognition via brain-based mediating processes.

Second, possible *moderators* are studied to understand the circumstances under which exposure to contaminants is most and least likely to harm children. Genetic variants have been examined as modifiers of the effects of contaminants in the physical environment. For example, in one study, carriers of a variant of the *APOE* gene were more vulnerable to the negative effects of exposure to mercury on behavior problems during childhood (31), with nutritional and family factors possibly mitigating the effect of exposure (3). In a study of Inuits who are at high risk of exposure to mercury from fish, preschoolers who ate more tomato products had lower levels of mercury (32). Large studies that include data on the physical and social environment in the home as well as genetic information could advance developmental science by focusing on the interplay among family context, exposure to contaminants, and genetic variants. For example, if a measure of parenting were included with assessments of *APOE* variants and exposure to mercury, researchers could investigate whether supportive parenting mitigates the impact of the combination of the *APOE* risk variant and mercury exposure on children's behavior problems.

Third, although earlier studies almost always examined exposure to a single contaminant, researchers are increasingly investigating the relative and joint impact of exposure to *many contaminants*. Initially, such studies focused on interactions between exposure to a few contaminants (3). For example, in one study (33), during early pregnancy, exposure to lead was associated with less optimal mental development among infants with lower exposure to cadmium, whereas during late pregnancy, exposure to lead was associated with less optimal mental development among infants with higher exposure to cadmium. Within the past several years, the term *exposome* was introduced to describe the range of exposure to contaminants an individual encounters across the life span (34). As technology and data collection improve, researchers will be able to assess exposure to a range of contaminants to closely approximate individuals' exposome. In the meantime, a summary score that tabulates exposure to a few contaminants could be created for developmental studies using the same approach as genetic risk scores and contextual risk indices (35, 36). Researchers could then assess the joint impact of cumulative contaminant risk, genetic risk, and social environmental risk on developmental outcomes.

Fourth, research on environmental contaminants and childhood outcomes has recently begun to address *multigenerational processes*, including epigenetic effects. Epigenetic changes include DNA methylation where methyl groups are added to DNA that can then lead to modifications of DNA function, including suppressed transcription of genes, which may be transmitted across generations. In mammals, the mother hosts the development of the offspring from the zygote stage to birth. As offspring develop, a separate lineage of cells, called the

primordial germ cells, migrate and differentiate into gamete precursor cells that become the *grand-offspring* generation. Thus, when a pregnant woman is exposed to a contaminant, it may directly affect not only her epigenome, but also the epigenome of her offspring and grand-offspring, producing intergenerational effects.

As an example of the importance of developmental exposure and intergenerational epigenetic effects in offspring, maternal exposure of mice to BPA was linked to changes in coat color and risk of obesity in offspring via decreases in DNA methylation (37). In addition, environmental exposures influenced what are referred to as transgenerational effects on the great-grand-offspring generation, in which no direct exposure occurred. In a review of studies on animals of transgenerational inheritance of diseases via epigenetic changes elicited by environmental contaminants, a key mechanism of transgenerational transmission of susceptibility to cancer, obesity, and other physical changes was incomplete or inaccurate reprogramming of DNA methylation of germ cells (sperm and egg) after exposure to contaminants (38).

Conducting studies of environmental contaminants and epigenetic changes in people is challenging, but evidence is mounting that exposure to specific contaminants is associated with DNA methylation in humans. For example, in one study, the level of preadolescent girls' exposure to BPA was associated with reduced DNA methylation at specific sites in genes linked to immune function, metabolism, and other functions (39). Researchers could assess exposure to contaminants along with aspects of the social environment (e.g., parenting) to investigate whether these features of the environment have unique or overlapping impacts on DNA methylation.

## FOSTERING AN INTEGRATIVE DEVELOPMENTAL SCIENCE OF EXPOSURE TO CONTAMINANTS

Advancing developmental science and spurring innovative, developmentally informed research on environmental contaminants require integrative approaches and collaboration across disciplines. Broadly speaking, the focus on integration and

**Table 1**
*Integrating Developmental Science With Toxicology and Environmental Health.*

| Strategies to integrate developmental science with toxicology and environmental health | Examples of how to implement each strategy |
|---|---|
| 1. Increase developmental scientists' awareness of cutting-edge research on environmental contaminants | Organize a special section in a leading developmental science journal (e.g., *Child Development*) focusing on childhood outcomes of contaminant exposure. |
| | Plan an invited symposium for an upcoming meeting of the Society for Research in Child Development (SRCD) focused on childhood contaminant exposure. |
| | Ensure SRCD members are aware of results from the National Institute of Environmental Health Sciences (NIEHS) Core Centers as well as initiatives such as the NIEHS Children's Health Exposure Analysis Resource through webinars and presentations at SRCD meetings. |
| 2. Promote scholarly collaboration across disciplines | Add environmental health specialists and toxicologists to the editorial teams of developmental science journals. |
| | Organize a multiday SRCD Special Topic Meeting on the interface between developmental science and environmental health and toxicology. |
| | Increase cross-disciplinary centers or institutes or leverage existing cross-disciplinary institutes at universities to foster collaborative research projects. |
| 3. Provide developmental scientists with intensive training on key methods in toxicology and environmental health | Engage developmental scientists in workshops in exposure assessment offered by the NIEHS Core Centers. |
| | Ensure developmental scientists are aware of continuing education and summer workshops in environmental health that are regularly offered by Schools of Public Health. |
| | Develop innovative training on methods at the interface between developmental science and environmental health and toxicology. |
| 4. Train the next generation of integrative developmental scientists | Offer graduate coursework on contaminants and their impact on child development that are team taught by faculty from across disciplines. |
| | Develop cross-disciplinary pre- or postdoctoral training programs that focus on the integration of developmental science, toxicology, and environmental health. |
| | Facilitate joint appointments of mentoring faculty across disciplinary schools and departments (e.g., School of Public Health and Department of Psychology). |

collaboration fits with the mission statement that the Society for Research in Child Development (SRCD) "advances developmental science and promotes its use to improve human lives." More specifically, one of SRCD's strategic goals focuses on supporting "researchers' efforts to collaborate, integrate, and communicate research across disciplines." Several strategies and approaches are needed to integrate developmental science with disciplines that focus on environmental contaminants, including toxicology and environmental health (see Table 1).

First, developmental scientists need to be more aware of current research on environmental contaminants. One of the flagship journals of developmental science could publish a special section on childhood outcomes of exposure to contaminants with articles on mechanisms of exposure's associations with developmental outcomes, including mediating processes, moderators that mitigate or exacerbate the effects of exposure, and multigenerational impacts. In addition, the biennial meeting of SRCD could feature an invited symposium on the impact of exposure to contaminants on many domains of child development that includes presentations by environmental scientists, toxicologists, and policy experts as well as developmental scientists.

Second, developmental scientists need to collaborate more with researchers in environmental health and toxicology. SRCD could host a special topic meeting on the interface between developmental science and the other disciplines. A targeted meeting would allow researchers from all three fields to present findings to members of the other disciplines and forge professional connections. Universities could also integrate these disciplines by creating cross-disciplinary centers or institutes that span these disciplines or tapping into existing institutes to fund collaborative research.

Third, developmental scientists need intensive training in relevant methods. The Core Centers of the National Institute of Environmental Health Sciences host workshops on assessment of exposure. Efforts could be made to increase developmental scientists' awareness of these workshops, and funding could be provided to support the participation of developmental science trainees. In addition, trainings could be created on methods at the interface between developmental science and environmental health and toxicology. For example, webinars or workshops could focus on how to use biological specimens (e.g., blood) from longitudinal studies of children and families to assess exposure to contaminants.

Fourth, the next generation of integrative developmental scientists needs to be trained so a cadre of young scholars will be prepared to conduct cutting-edge research at the interface of these disciplines. To accomplish this goal, cross-disciplinary pre- and postdoctoral training programs could be developed at leading universities that have strengths in developmental science, toxicology, or environmental health. These programs could include graduate coursework on contaminants and their impact on children's development. Such coursework could be taught by teams of faculty from these disciplines, and trainees could be mentored by scholars in each discipline.

The success of these strategies would hinge on the involvement of leading scholars in developmental science, environmental health, and toxicology, as well as representatives from professional organizations, including SRCD. Although the prospects for lasting integration across these fields may seem daunting, similar approaches have integrated developmental science with genetic and neuroscientific perspectives (40). Integration across relevant fields will help fulfill the intent of Bronfenbrenner's ecological systems perspective in which studying the impact of contexts and systems on children was viewed as essential to advancing developmental science. Given evidence of the role of environmental contaminants in children's development, developmental scientists must pursue the proposed strategies to generate an integrative developmental science of exposure to contaminants.

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