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Dorothy L. McLeod

Loyola University Chicago, [dmcleod@luc.edu](mailto:dmcleod@luc.edu)

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# Children's Exposure to Environmental Toxins: Socioeconomic Factors and Subsequent Effects on Mental Health and Function

Dorothy McLeod, MA

**RESEARCH BRIEF:**

A non-exhaustive summary of peer-reviewed evidence related to a children’s rights topic, intended to highlight areas for policy and advocacy work.

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The physical environment in which children live, including the air they breathe and the water they drink, has a profound influence on their development. While children need many chemicals and nutrients to physically grow and develop normally, others, such as those deemed environmental toxins (e.g. pesticides, lead, mercury, and illicit substances) act instead as a threat to healthy development. These chemicals may have highly toxic effects, and while they are a threat to all individuals, they affect infants and children most severely.<sup>1,2</sup> This is because children's immature nervous and immune systems are highly susceptible to disruption during development, and their smaller size and higher metabolic rate means that they proportionally sequester higher toxin concentrations relative to adults. Furthermore, children's curiosity makes them more likely to encounter hazardous exposures as they explore their living environments.<sup>3</sup>

Environmental toxin exposure comes at a great cost not only to the healthy *physical* development, but also to the healthy *mental* development of an individual child. Though not the focus of this brief, there is much research documenting how environmental toxins have been linked to medical conditions like asthma, diabetes, and Parkinson's disease.<sup>4</sup> Furthermore, exposure to various toxins has been linked to many psychiatric and intellectual problems, including later diagnosis of Attention Deficit-Hyperactivity Disorder (ADHD), Specific Learning Disorders such as Dyslexia, conduct problems, and deficits in IQ.<sup>1,5-8</sup> All of these outcomes not only harm the individual, but also come at a high cost to society. As a result of

all of these outcomes, society incurs increased costs related to health care, hospitalization, joblessness, special education, and the juvenile and adult criminal justice systems.<sup>3</sup> Importantly, the contribution of environmental toxins to each of these outcomes is ultimately preventable through appropriate control and regulation.

Finally, exposure to environmental toxins contributes to social inequities and associated health disparities that are common in U.S. urban environments. The types of homes most likely to contain environmental toxins are also often older, and, particularly within urban environments, are concentrated in low-income neighborhoods that tend to be disproportionately inhabited by non-white residents.<sup>3</sup> Furthermore, low-socioeconomic status (SES) groups are exposed to greater amounts of environmental toxins not only in the home, but in school, in job-sites, and in neighborhoods.<sup>9</sup> Scientists have postulated that the difference in health between socioeconomic brackets is not due to a single type of exposure, but the cumulative exposure to various toxins across all of these environments.<sup>9</sup> These differences have also been tied to disparities in maternal and child health, including high rates of infant mortality, pre-term birth, and very low-birth-weight observed among black women and their infants.<sup>10</sup> Furthermore, these disparities in exposure may also contribute to disparities in child mental health and development, such as the increased rates of behavioral disorders among non-white populations.<sup>11</sup>

Because of environmental toxins' contribution to child physical and mental health disparities,

and the great subsequent cost of these disparities to society, these effects have become a focus of work of the Center for the Human Rights of Children at Loyola University Chicago. To support the view that toxin exposure is an issue of children's rights, key principles set out by the UN Convention on the Rights of the Child include a right to survival and (healthy) development, non-discrimination, and a developmental environment in the best interest of the child. In order to provide an introduction to the issue of toxin exposure and mental health outcomes, to highlight the relevant evidence-based research, and to build a foundation for policy change, we present the following brief review of the literature. Because children

are uniquely vulnerable to various environmental toxins prenatally and in the post-natal period, the literature is organized in these time frames. In addition, the specific toxins discussed below are not a comprehensive list, but represent an effort to focus attention on mental health outcomes of the more common environmental toxins. More research is undoubtedly needed to continue to support links between toxins and mental development, in addition to connections to physical development.

**Children and infants are particularly vulnerable to environmental toxins while they are still developing in utero.** As such, this policy brief will describe some of the toxins that are the most common and most toxic to the mental health and function of the developing fetus, i.e., organophosphates, alcohol and nicotine. As noted earlier, prenatal exposure to environmental toxins is highly correlated with socioeconomic status, and is considered to be a contributor to continued health disparities between the poor and underserved and other populations. However, this is particularly true for the toxins discussed here. For one, Organophosphate exposure is most common in agricultural communities, which tend to be composed of low-SES Latino families.<sup>12</sup> Meanwhile, exposure to recreational drugs in pregnancy is associated with low SES environments due to SES-related stressors, little education, and limited substance abuse treatment options.<sup>13</sup> Therefore, many of the effects discussed below are most likely to be present among these less-resourced communities.

### **ORGANOPHOSPHATES**

**Exposure to organophosphates (OPs), common ingredients in insecticides in agriculture and in home/commercial pest control (another exposure that is linked to lower SES and substandard housing conditions), can cause mild to severe disruption of brain development.** OPs can disrupt a wide range of processes that are essential for the formation and function of brain circuits. Studies of functional outcomes in both animals and children demonstrate that modest changes in

brain architecture caused by exposure to Chlorpyrifos, an OP, can lead to measurable problems in learning, attention, and emotional control.<sup>14</sup>

### **OTHER INDUSTRIAL CHEMICALS**

**In industrialized societies like the US, there are several additional chemical additives that are known to have effects on child behavior following exposure in utero.** Two examples are Biphospheno A and Polycyclic Aromatic Hydrocarbons. Biphospheno A is commonly used in consumer products, including food/beverage containers and linings, medical equipment, and thermal receipts. Gestational exposure to Biphospheno A has been linked to increased hyperactivity and aggression scores for 2-year-old girls,<sup>6</sup> as well as increased anxiety, hyperactivity, and depression among 3-year-olds, particularly girls.<sup>7</sup> Meanwhile, Polycyclic Aromatic Hydrocarbons (PAHs) are a type of industrial toxin present in ambient air from incomplete combustion of fossil fuels, as well as in tobacco smoke. Prenatal air exposure to PAHs has been linked to slower cognitive development in early childhood, which in turn can lead to educational performance deficits in later childhood/adolescence.<sup>15</sup>

### **RECREATIONAL DRUGS**

**Recreational drugs may not seem at first to represent “environmental” toxins.** However, during the prenatal time period, this is essentially the role that they take on. The effects of in-utero exposure to illicit substances, such as cocaine and other illegal substances, is well documented and well recognized by the public. However, a frequent

misconception is that illicit/illegal substance use during pregnancy is more harmful than using legal substances. In fact, legal recreational drugs, such as alcohol and nicotine, also act as toxins to the developing fetus. Research indicates that each of these drugs may impact mental development in different ways, and that these impacts vary based on amount, timing, and duration of the exposure.<sup>16</sup> Though it is often difficult to separate fetal exposure to recreational drugs from the environmental stresses facing children of parents who use these drugs, there is rich scientific evidence that exposure to recreational drugs at sensitive periods of development may negatively impact mental development and later mental health outcomes.<sup>17</sup>

**ALCOHOL.** Alcohol is one of the best-documented of the teratogens, substances that can negatively influence fetal development. Exposure to alcohol is linked to a range of outcomes, many of which depend on the amount and timing of the exposure during pregnancy. However, in general, exposure to alcohol during pregnancy is related to the development of fetal alcohol spectrum disorders, a group of psychiatric diagnoses characterized by numerous physical and structural changes as well as deficits in several cognitive domains, including attention, language, memory, and motor skills.<sup>18,19</sup> Notably, these deficits are misdiagnosed as other psychiatric diagnoses, such as ADHD, when in fact they are a result of structural changes to the brain as a result of alcohol exposure in utero.<sup>19</sup> However, fetal alcohol exposure itself is related independently

to higher rates of other psychiatric disorders, such as depression and anxiety, in childhood and adulthood.<sup>20</sup> Therefore, exposure to alcohol in utero is highly linked to both cognitive and mental health outcomes for children.

**NICOTINE.** Similar to alcohol, nicotine's effects on fetal development are often underestimated or ignored. Further, much attention is given to the physical effects of nicotine exposure in childhood (e.g., asthma) over the potential cognitive and mental health effects it may produce when exposure occurs during gestation. In fact, nicotine use during pregnancy may be related to severe antisocial behavior in later life.<sup>21</sup> Furthermore, children of mothers who used nicotine during pregnancy are more likely to struggle with the development of "theory of mind," a skill related to empathy.<sup>22</sup>

This is a short list of some of the toxins that are most prevalent and harmful to infant cognitive and mental health development in utero. However, there are other toxins that may impact infants at this stage, and the toxins described here may also have effects if ingested by children after birth. This is particularly true for alcohol and nicotine, for which even second-hand exposure can have devastating effects on the developing brain even into adolescence.<sup>23-25</sup>

### POLICY RECOMMENDATIONS

Grason and Misra<sup>26</sup> provide excellent documentation of potential policy strategies that may help to reduce prenatal toxin exposure. In terms of short-term strategies, they recommend: 1) capitalizing

on existing public notification requirements that stem from environmental legislation, 2) continuing and enhancing use of the news media, 3) increasing product labeling guidelines, 4) promoting improved healthcare provider counseling, and 5) increasing surveillance and research. In the long-term, they recommend 1) creating an organized system of information and care specific to these exposures, 2) undertaking a scientific/political initiative that unites health and environmental concerns. All of these efforts are particularly important in light of current policy weaknesses, including the fact that manufacturers of commercial chemicals, including pesticides, are required to supply only minimal toxicity data before selling their products. An obligation to supply premarket toxicity and exposure data is necessary to ensure that children will be protected from exposure and potential harm.



As much as prenatal conditions of vulnerability, postnatal conditions and influence have a great deal of impact on mental health and development. Also similar to prenatal conditions, postnatal conditions are largely a factor of socioeconomic status, with by far the largest burden placed on those who live in underserved neighborhoods and communities. Though there are many toxins that affect children's development postnatally, most of the research with regards to effects on cognitive/mental development focuses on lead. While other toxins have these effects, lead is certainly that which is most widely understood, and can provide context for other toxins for which research is still being developed. Unfortunately, concentrations of lead are highest within older homes, which, particularly in large cities, often represent the homes of those who are already financially or otherwise disadvantaged.<sup>3</sup> Furthermore, low-SES children are more likely to live in urban neighborhoods with greater soil and dust lead concentrations from traffic and industrial activities, and to have nutritional deficiencies that increase lead absorption.<sup>27</sup> This disparity has been recently illuminated by the tragedy in Flint, Michigan that has been making headlines since 2015. In this community, the incidence of blood lead concentrations above the recommended amount rose from 2.4% to 4.9% after a change in water supply.<sup>28</sup> Notably, these statistics ignore the fact that there is no known "safe" level of lead exposure, and that even low levels of exposure can have negative impacts.<sup>29</sup> Fortunately, this tragedy, though deeply concerning, has widely publicized the degree to which environmental

health adversities disproportionately impact underserved communities.

### LEAD

Lead is harmful to children both in utero and following birth. In fact, experts have noted that the greatest risk of lead in water may be to infants consuming reconstituted formula.<sup>28</sup> Outcomes of lead exposure, in addition to the highly publicized decrease in general IQ, include deficits in executive functioning, a set of processes that guide advanced cognitive functions, such as attention, inhibition, working memory, and cognitive flexibility.<sup>5</sup> Furthermore, lead exposure has also been linked to conduct disorder, a behavioral disorder associated with antisocial behaviors.<sup>8</sup> To date, no effective treatments have been found to "cure" the permanent developmental effects of lead toxicity.

Not only is lead itself related to negative impacts on mental health and development, but the knowledge of disparities in lead exposure may also impact mental health throughout communities even among those unexposed. For example, a qualitative study of residents in the Flint, MI area noted increased stress, anxiety, and depression among the city's population, and hypothesized that these effects were the most severe among low-income, African American populations in the city. The participants stated that these mental health consequences were related not only to the water contamination but to increasing distrust of public officials related to the crisis.<sup>30</sup> Furthermore, it is often ignored that the treatment and additional supports that may be necessary to

overcome outcomes of lead exposure (e.g., tutoring, specialized school-based services) themselves may cause additional stress among the families of affected children.

### **POLICY RECOMMENDATIONS**

Policy changes regulating exposure to lead are often touted as a success story, since regulations prohibiting the inclusion of lead in gasoline, household paint, and other consumer products have resulted in significant decreases in childhood lead exposure overall.<sup>29</sup> However, it is clear from the Flint, MI crisis that lead exposure has not been eradicated completely, and that those who continue to receive lead exposure are also disadvantaged systemically. Furthermore, as noted above, there is no “safe” level of lead exposure for children, and current federal standards for lead in house dust, water, and soil remain too high to protect children adequately. Further reduction in lead exposure could have critical social impact, since the reductions that have been accomplished so far have been linked to such wide-reaching factors as an overall decrease in crime.<sup>31</sup>

Environmental health policy experts believe that to continue improving our approach protecting children from known toxins like lead, the US EPA and FDA need more authority and resources to further regulate and reduce emissions and exposures.<sup>32</sup> In addition to federal oversight, lead abatement programs by municipal/county governments and community organizations are critical since the leading sources of lead poisoning are found in homes (old paint, lead pipes).

In conclusion, in spite of increased awareness of the common presence of environmental toxins in many children's early environments, a number of children remain at risk for pre- and postnatal exposure to environmental toxins that will eventually impact their mental health and development. Such exposure may have societal impacts (e.g. crime reduction) and increase socioeconomic disparities (e.g. most heavily affect poor communities). Therefore, public and private systems have an obligation to protect our children from these known toxins, as well as any newly created substances that may be toxic. At a policy level, federal agencies such as the EPA and FDA need additional funding and enhanced ability to regulate exposure at both the pre- and post-natal stages. However, as events in Flint have demonstrated, these issues also can and should be addressed through increased regulation more locally. Particularly in the United States, where many policies guiding the oversight of hazardous substances are developed at a municipal and state level, grass-roots efforts to affect change in these policies may be effective. From a children's rights perspective, all of the policy recommendations outlined above are in line with efforts to promote the internationally adopted standards to consider the best interest of the child, and the safety, survival, and healthy development of children. One way to facilitate many of these short- and long-term goals would be for the U.S. to ratify the UN Convention on the Rights of the Child (CRC). Ratification of this convention would provide both moral and political authority to exercise greater consideration of toxin exposure

as a violation of children's human rights, to monitor such effects, and to engage in greater prevention efforts. In summary:

- Federal agencies such as the EPA and FDA need additional funding and enhanced ability to regulate exposure at both the pre- and post-natal stages.
- Grass-roots efforts to effect policies guiding the oversight of hazardous substances at a municipal and state level are also needed.
- National ratification of the UN Convention on the Rights of the Child (CRC) would allow greater moral and political authority to combat toxin exposure.

- <sup>1</sup> National Scientific Council on the Developing Child. *Early Exposure to Toxic Substances Damages Brain Architecture*; 2006. <http://developingchild.harvard.edu/resources/early-exposure-to-toxic-substances-damages-brain-architecture/>. Accessed January 24, 2017.
- <sup>2</sup> Weiss B. Vulnerability of children and the developing brain to neurotoxic hazards. *Environ Health Perspect*. 2000;108(Suppl 3):375-381.
- <sup>3</sup> Center for the Human Rights of Children. *A City and Countywide Summit to Advance Healthy Homes and Healthy Communities and Chicago and Cook Counties, Illinois*. Chicago, IL: Loyola University Chicago; 2014. <http://luc.edu/media/lucedu/chrc/pdfs/A%20City%20and%20Countywide%20Summit%20to%20Advance%20Healthy%20Homes%20&%20Healthy%20C.pdf>. Accessed January 24, 2017.
- <sup>4</sup> Muir T, Zegarac M. Societal costs of exposure to toxic substances: economic and health costs of four case studies that are candidates for environmental causation. *Environ Health Perspect*. 2001;109(Suppl 6):885-903.
- <sup>5</sup> Canfield RL, Gendle MH, Cory-Slechta DA. Impaired neuropsychological functioning in lead-exposed children. *Dev Neuropsychol*. 2004;26(1):513-540. doi:10.1207/s15326942dn2601\_8.
- <sup>6</sup> Braun JM, Yolton K, Dietrich KN, et al. Prenatal Bisphenol A exposure and early childhood behavior. *Environ Health Perspect*. 2009;117(12):1945-1952. doi:10.1289/ehp.0900979.
- <sup>7</sup> Braun JM, Kalkbrenner AE, Calafat AM, et al. Impact of early-life Bisphenol A exposure on behavior and executive function in children. *Pediatrics*. 2011;128(5):873-882. doi:10.1542/peds.2011-1335.
- <sup>8</sup> Marcus DK, Fulton JJ, Clarke EJ. Lead and conduct problems: A meta-analysis. *J Clin Child Adolesc Psychol*. 2010;39(2):234-241. doi:10.1080/15374411003591455.
- <sup>9</sup> Evans GW, Kantrowitz E. Socioeconomic status and health: The potential role of environmental risk exposure. *Annu Rev Public Health*. 2002;23(1):303-331. doi:10.1146/annurev.publhealth.23.112001.112349.
- <sup>10</sup> Morello-Frosch R, Shenassa ED. The environmental “riskscape” and social inequality: Implications for explaining maternal and child health disparities. *Environ Health Perspect*. 2006;114(8):1150-1153.
- <sup>11</sup> Siegel CE, Laska EM, Wanderling JA, Hernandez JC, Levenson RB. Prevalence and diagnosis rates of childhood ADHD among racial-ethnic groups in a public mental health system. *Psychiatr Serv Wash DC*. 2016;67(2):199-205. doi:10.1176/appi.ps.201400364.
- <sup>12</sup> Bouchard MF, Chevrier J, Harley KG, et al. Prenatal exposure to organophosphate pesticides and IQ in 7-year-old children. *Environ Health Perspect*. 2011;119(8):1189-1195. doi:10.1289/ehp.1003185.
- <sup>13</sup> Forray A. Substance use during pregnancy. *F1000Research*. 2016;5. doi:10.12688/f1000research.7645.1.
- <sup>14</sup> Needleman HL. The neurotoxic properties of pesticides. In: Davidson PW, Myers GJ, Weiss B, eds. *Neurotoxicity and Developmental Disabilities*. Elsevier Academic Press; 2006.

- <sup>15</sup> Perera FP, Rauh V, Whyatt RM, et al. Effect of prenatal exposure to airborne Polycyclic Aromatic Hydrocarbons on neurodevelopment in the first 3 years of life among inner-city children. *Environ Health Perspect*. 2006;114(8):1287-1292. doi:10.1289/ehp.9084.
- <sup>16</sup> Selevan SG, Kimmel CA, Mendola P. Identifying critical windows of exposure for children's health. *Environ Health Perspect*. 2000;108:451-455. doi:10.2307/3454536.
- <sup>17</sup> Stanwood GD, Levitt P. Drug exposure early in life: functional repercussions of changing neuropharmacology during sensitive periods of brain development. *Curr Opin Pharmacol*. 2004;4(1):65-71. doi:10.1016/j.coph.2003.09.003.
- <sup>18</sup> Golding J, Emmett P, Iles-Caven Y, Steer C, Lingam R. A review of environmental contributions to childhood motor skills. *J Child Neurol*. 2014;29(11):1531-1547. doi:10.1177/0883073813507483.
- <sup>19</sup> American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. 5th ed. Washington, D.C: American Psychiatric Association; 2013.
- <sup>20</sup> Rangmar J, Hjern A, Vinnerljung B, Strömland K, Aronson M, Fahlke C. Psychosocial outcomes of fetal alcohol syndrome in adulthood. *Pediatrics*. 2015;135(1):e52-58. doi:10.1542/peds.2014-1915.
- <sup>21</sup> Wakschlag LS, Pickett KE, Cook E, Benowitz NL, Leventhal BL. Maternal smoking during pregnancy and severe antisocial behavior in offspring: A review. *Am J Public Health Wash*. 2002;92(6):966-974.
- <sup>22</sup> Reidy RE, Ross RG, Hunter SK. Theory of mind development is impaired in 4-year-old children with prenatal exposure to maternal tobacco smoking. *Int Neuropsychiatr Dis J*. 2013;1(1):24-34. doi:10.9734/INDJ/2013/3916.
- <sup>23</sup> Witt ED. Research on alcohol and adolescent brain development: opportunities and future directions. *Alcohol*. 2010;44(1):119-124. doi:10.1016/j.alcohol.2009.08.011.
- <sup>24</sup> Squeglia LM, Jacobus J, Tapert SF. The influence of substance use on adolescent brain development. *Clin EEG Neurosci*. 2009;40(1):31-38. doi:10.1177/155005940904000110.
- <sup>25</sup> Dwyer JB, McQuown SC, Leslie FM. The dynamic effects of nicotine on the developing brain. *Pharmacol Ther*. 2009;122(2):125-139. doi:10.1016/j.pharmthera.2009.02.003.
- <sup>26</sup> Grason HA, Misra DP. Reducing exposure to environmental toxicants before birth: Moving from risk perception to risk reduction. *Public Health Rep*. 2009;124(5):629641.
- <sup>27</sup> Bellinger DC. Lead contamination in Flint — An abject failure to protect public health. *N Engl J Med*. 2016;374(12):1101-1103. doi:10.1056/NEJMp1601013.
- <sup>28</sup> Hanna-Attisha M, LaChance J, Sadler RC, Champney Schnepf A. Elevated blood lead levels in children associated with the Flint drinking water crisis: A spatial analysis of risk and public health response. *Am J Public Health*. 2015;106(2):283-290. doi:10.2105/AJPH.2015.303003.
- <sup>29</sup> Council on Environmental Health. Prevention of childhood lead toxicity. *Pediatrics*. June 2016:e20161493. doi:10.1542/peds.2016-1493.

<sup>30</sup> Cuthbertson CA, Newkirk C, Ilardo J, Loveridge S, Skidmore M. Angry, scared, and unsure: Mental health consequences of contaminated water in Flint, Michigan. *J Urban Health Bull N Y Acad Med*. 2016;93(6):899-908. doi:10.1007/s11524-016-0089-y.

<sup>31</sup> Reyes JW. *Environmental Policy as Social Policy? The Impact of Childhood Lead Exposure on Crime*. National Bureau of Economic Research; 2007. <http://www.nber.org/papers/w13097>. Accessed February 21, 2017.

<sup>32</sup> Lanphear BP, Vorhees CV, Bellinger DC. Protecting children from environmental toxins. *PLOS Med*. 2005;2(3):e61. doi:10.1371/journal.pmed.0020061.