

Testimony in Support of the Child Safe Playing Fields Act

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We are pediatricians and specialists in pediatric environmental health. We are part of the Children's Environmental Health Center based in Mount Sinai Medical Center in New York City. The mission of the Children's Environmental Health Center is to educate health care providers and others about the scientific and medical aspects of environmental health problems impacting children and to provide clinical consultation to families, health care professionals, public health officials, and community organizations with concerns regarding children's exposure to environmental health hazards.

Introduction

School-aged children can spend anywhere from 35 to 50 hours per week in and around school facilities for at least 12 years of their life. After school, many children are involved in extracurricular activities that take place in public park playgrounds and ball fields.

A good education is essential in preparing children to become active members in their communities. It is crucial that schools and public park areas provide a safe environment for children to foster their growth and development. Unfortunately, schools and local parks may contain environmental hazards that can negatively impact children's health and learning. Pesticides are a group of hazardous chemicals used in these environments that can have both short-term and long-term effects on children's health and development.

There are, however, well-studied, effective approaches to pest management that minimize children's exposure to pesticides. We will review children's vulnerability to pesticides, health effects from exposure to pesticides, and strategies to minimize pesticide exposure to provide all children with a safer learning and playing environment.

Children's and Adolescents' Vulnerability to Pesticides

Children of all ages are susceptible to environmental toxins. Children and adolescents are exposed to pesticides through inhalation, ingestion, and dermal absorption. Children have greater exposure to pesticides because they drink more water, eat more food and breathe more air relative to their size than adults. Adolescents may also be disproportionately affected to pesticides exposures on playing fields due to participation in organized sports. Children's

growth and development and adolescents' pubertal development may be affected by exposure to pesticides. Children and adolescents have more future years of life and therefore more time to develop chronic diseases that have been triggered by environmental exposures.^{1,2}

Health Effects of Pesticide Exposure on Children and Adolescents

Pesticides are a group of diverse chemical compounds that are among the most toxic chemicals that children are commonly exposed to. Pesticides include insecticides, herbicides, fungicides, rodenticides, fumigants, and insect repellants. The mechanism by which pesticides kill pests is frequently similar to that which harms or kills humans.³ For example, a major class of insecticides is the organophosphates. Organophosphate pesticides target the nervous systems of insects and other pests through the inhibition of acetylcholinesterase, an enzyme that degrades the neurotransmitter acetylcholine, resulting in a buildup of acetylcholine in the neuronal junction. Acute poisonings in humans (via the same mechanism) leads to a spectrum of cholinergic symptoms, including excess shedding of tears, abdominal cramps, vomiting, diarrhea, and profuse sweating, with more severe cases progressing to respiratory arrest and death.

Children and adolescents are exposed to pesticides by their use in homes, gardens, food supplies, schools, and playing fields. The resulting cumulative burden of pesticide exposure in children and adolescents is potentially very high given the ubiquity of pesticides in the environment. The National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC) conducts The National Health and Nutrition Examination Survey (NHANES), which assesses the health and nutrition status of the civilian U.S. population. As part of this survey, metabolites of pesticides (breakdown products that are markers of exposure) are measured in the US population. Using this data, Barr et al. found that children and adolescents often had higher levels of organophosphate metabolites than adults; children from 6-

¹ American Academy of Pediatrics Committee on Environmental Health. Etzel, RA, ed. *Pediatric Environmental Health*, 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003.

² Landrigan PJ, Goldman LR. Protecting children from pesticides and other toxic chemicals. *J Expo Sci Environ Epidemiol*. 2011 Mar;21(2):119-20.

³ American Academy of Pediatrics Committee on Environmental Health. Etzel, RA, ed. *Pediatric Environmental Health*, 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003; p. 323.

11 years of age had especially higher levels of organophosphate metabolites than adults.⁴ These findings suggest that the general child and adolescent populations have greater levels of exposure to pesticides than the adult population.

Exposure to pesticides can cause both acute and chronic health effects. Acute exposure to pesticides can lead to asthma exacerbations, cough, shortness of breath, nausea, vomiting, eye irritation, and headaches. Pesticide exposures in schools have been documented to cause acute illness among students and school employees.⁵ Low dose pesticide exposure in children and in pregnant women has also been linked to some cancers⁶ and birth defects⁷, respectively. The effects of low-dose exposure to pesticides, both prenatally and postnatally, have also been shown in many studies to negatively affect children's development.^{8,9,10,11,12,13,14} At this low-dose level of pesticide exposure, no acute effects are seen but nonetheless organ systems (in this case, the nervous system) are still affected. In one study, children of agriculture workers in Oregon and North Carolina were found to have deficits in coordination as well as visual memory, in comparison to children of non-agricultural workers.¹⁵ Another study compared the urinary concentrations of a metabolite of organophosphates in the general population as measured by the National Health and Nutrition Examination Survey (2000–2004) and the prevalence of attention-

⁴ Barr DB, Bravo R, Weerasekera G, Caltabiano LM, Whitehead RD, Jr, Olsson AO, Caudill SP, Schober SE, Pirkle JL, Sampson EJ, Jackson RJ, Needham LL. Concentrations of dialkyl phosphate metabolites of organophosphorus pesticides in the U.S. population. *Environ Health Perspect* 2004;112:186.

⁵ Alarcon WA, Calvert GM, Blondell JM, Mehler LN, Sievert J, Propeck M, et al. Acute illnesses associated with pesticide exposure at schools. *JAMA* 2005;294(4):455-65.

⁶ American Academy of Pediatrics Committee on Environmental Health. Etzel, RA, ed. *Pediatric Environmental Health*, 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003; pp. 340.

⁷ Garry, V. F. , Schreinmachers, D. , Harkins, M. E. and Griffith, J. (1996) Pesticide applicers, biocides, and birth defects in rural Minnesota. *Environ. Health Perspect* 1996; 104: 394-399.

⁸ Grandjean P, Harari R, Barr DB, Debes F. Pesticide exposure and stunting as independent predictors of neurobehavioural deficits in Ecuadorian school children. *Pediatrics* 2006;117(3):e546–e56.

⁹ Rohlman DS, Arcury TA, Quandt SA, Lasarev M, Rothlein J, Travers R, et al. Neurobehavioural performance in preschool children from agricultural communities in Oregon and North Carolina. *Neurotoxicology* 2005;26:589–98.

¹⁰ Rauh VA, Garfinkel R, Perera FP, Andrews HF, Hoepner L, Barr DB. Impact of prenatal chlorpyrifos exposure on neurodevelopment in the first 3 years of life among inner-city children. *Pediatrics* 2006;118(6):1845–59.

¹¹ Ruckart PZ, Kakolewski K, Bove FJ, Kaye W. Long-term neurobehavioural health effects of methyl parathion exposure in children in Mississippi and Ohio. *Environ Health Perspect* 2004;112(1):46–51.

¹² Young JG, Eskenazi B, Gladstone EA, Bradman A, Pedersen L, Johnson C. Association between in utero organophosphate pesticide exposure and abnormal reflexes in neonates. *Neurotoxicology* 2005;26(2):199–209.

¹³ Eskenazi B, Marks AR, Bradman A, Harley K, Barr DB, Johnson C. Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environ Health Perspect* 2007;115(5):792–8.

¹⁴ Engel SM, Berkowitz GS, Barr DB, Teitelbaum SL, Siskind J, Meisel SJ. Prenatal organophosphate metabolite and organochlorine levels and performance on the Brazelton Neonatal Behavioral Assessment Scale in a multiethnic pregnancy cohort. *Am J Epidemiol* 2007;265 (12):1397–404.

¹⁵ Rohlman DS, Arcury TA, Quandt SA, Lasarev M, Rothlein J, Travers R, Tamulinas A, Scherer J, Early J, Marín A, Phillips J, McCauley L. Neurobehavioral performance in preschool children from agricultural and non-agricultural communities in Oregon and North Carolina. *Neurotoxicology* 2005 Aug;26(4):589-98.

deficit/hyperactivity disorder (ADHD) in children 8 to 15 years of age and found that children with higher levels of the metabolite had higher odds of having ADHD.¹⁶

Pesticides, such as DDT, methoxychlor and chlordecone, have displayed the ability to disrupt endocrine function in animal studies.¹⁷ There have been very few studies looking at the effects of pesticides on pubertal development in children, however.¹⁸ The findings of the studies have also been mixed, highlighting the importance of further examination of the role of pesticides in pubertal development.^{19,20} Rogan et al stated that the “inconsistency seen in the current literature is less evidence of no effect than a consequence of the broadness of the topic and the difficulty in studying it, especially with available epidemiologic tools. Of course, by the time epidemiology can demonstrate effects, people have been exposed and affected, so it is in some sense too late.”²¹

In summary, research studies have shown a wide range of negative health consequences for children and adolescents from their exposure to pesticides (such as neurologic impairment as well as increased risk of cancer and potential reproductive damage). With knowledge of this scientific evidence, we need to identify and implement policy strategies to reduce children’s exposure to these toxic chemicals. We know that legislative bans are the single most effective intervention in reducing human exposure to chemical toxins. The classic example is the removal of lead from gasoline resulting in a subsequent significant decline in blood lead levels in US children.^{22, 23} Similarly, the EPA ban of the pesticide chlorpyrifos resulted in a 10 fold decline in blood levels of chlorpyrifos detected in mother and babies.²⁴ In essence, what is toxic to the

¹⁶ Bouchard MF, Bellinger DC, Wright RO, Weisskopf MG 2010. Attention-deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides. *Pediatrics* 125:e1270–e1277

¹⁷ Rogan WJ, Ragan NB. Some evidence of effects of environmental chemicals on the endocrine system in children. *International Journal of Hygiene and Environmental Health* 2007; 210(5): 659-667

¹⁸ Wigle, Donald T. , Arbuckle, Tye E. , Walker, Mark , Wade, Michael G. , Liu, Shiliang and Krewski, Daniel 'Environmental Hazards: Evidence for Effects on Child Health', *Journal of Toxicology and Environmental Health, Part B*, 10:1, 3 - 39

¹⁹ Gladen, B. C. , Ragan, N. B. and Rogan, W. J. Pubertal growth and development and prenatal and lactational exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene. *J. Pediatr* 2000; 136: 490-496

²⁰ Saiyed, H. , Dewan, A. , Bhatnagar, V. , Shenoy, U. , Shenoy, R. , Rajmohan, H. , Patel, K. , Kashyap, R. , Kulkarni, P. , Rajan, B. and Lakkad, B. Effect of endosulfan on male reproductive development. *Environ. Health Perspect* 2003; 111: 1958-1962.

²¹ Rogan WJ, Ragan NB. Some evidence of effects of environmental chemicals on the endocrine system in children. *International Journal of Hygiene and Environmental Health* 2007; 210(5): 659-667

²² CDC. Children’s blood lead levels in the United States. Atlanta, GA: US Department of Health and Human Services, CDC; 2007. Available at <http://web.archive.org/web/20080526204553/http://www.cdc.gov/nceh/lead/research/kidsBLL.htm>.

²³ Jones RL, Homa DM, Meyer PA, et al. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988-2004. *Pediatrics* 2009;123:e376--85.

²⁴ Whyatt RM, Barr DB, Camann DE, Kinney PL, Barr JR, Andrews HF, Hoepner LA, Garfinkel R, Hazi Y, Reyes A, Ramirez J, Cosme Y, Perera FP. Contemporary-use pesticides in personal air samples during pregnancy and blood samples at delivery among urban minority mothers and newborns. *Environ Health Perspect* 2003;111:749.

pests is also toxic for our children and we need to take steps to reduce children's and adolescents' exposure to pesticides.

Reducing children's and adolescents' exposure to pesticides

An effective, less toxic approach to controlling pests is the implementation of Integrated Pest Management (IPM) programs that reduce children's exposure to pesticides. IPM is an approach that controls pests by preventing their access to food, water and shelter. IPM programs can be more cost-efficient than traditional pest control techniques using pesticides and as effective as these methods.²⁵ Resources for integrated pest management in schools can be found in the following EPA websites: <http://www.epa.gov/pesticides/ipm/> and <http://www.epa.gov/pesticides/ipm/schoolipm/>.

The Child Safe Playing Fields Act is a critical legislative approach that builds upon IPM programs by further defining and requiring only the safest methods for playground and ball field maintenance. Similar legislation was passed in New York, Massachusetts and Connecticut. By banning synthetic chemical lawn pesticides on playgrounds, ball fields, day cares, schools, and public parks, New Jersey will be taking an essential step forward in protecting children's health and ensuring children learn and grow in a safe environment.

²⁵ National Institute for Occupational Safety and Health's website at: <http://www.cdc.gov/niosh/docs/2007-150/>.