

Statement of
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in Support of
SB 300: Pesticides – Use of Chlorpyrifos - Prohibition
before the
House Environment and Transportation Committee
Maryland House of Delegates
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Honorable Chair and members of the Committee. My name is J. Routt Reigart, Professor Emeritus of Pediatrics at Medical University of South Carolina. I have been involved in protecting children from the adverse effects of pesticides for over 40 years. Among my activities, I founded the National Pesticide Telecommunication Network, a dedicated pesticide information center, staffed 24 hours per day 7 days per week to assist health professionals deal with pesticide exposures. This Network is now the National Pesticide Information Center at Oregon State University. I was the founding Chair of the Board of the Children's Environmental Health Network, a nationally recognized and effective NGO dedicated to improving the health of children in relationship to their environment. I was the first Chair of the USEPA's Children's Health Protection Advisory Committee. I chaired the American Academy of Pediatrics Committee on Environmental Health. I am the Co-Editor of the 5th and 6th editions of the USEPA's *Recognition and Management of Pesticide Poisoning*.

I am submitting this testimony in support of Maryland SB 300 because of my concerns regarding continued chlorpyrifos use in Maryland and its demonstrated adverse effects on children. I was disheartened to learn that EPA's Administrator Pruitt rescinded the agency's 2015 proposal to revoke the food tolerances of chlorpyrifos. Researchers, medical professionals, and even EPA's own scientists have reviewed years of data, which all point to chlorpyrifos's long-term adverse impacts on the developing brains of children.

A revocation of its food tolerances would have removed chlorpyrifos from agricultural production and eliminated dietary exposure to the public. It would also end exposure to farmworkers and farmworker families. EPA, in 2000, removed all residential uses from the market because of the chemical's risk to children. EPA continued to review a significant body of research for almost two decades. However, instead of acting on the overwhelming evidence of harm to children that continue to be exposed from ongoing agricultural use of chlorpyrifos, EPA indicated the agency will continue to study the chemical, and would not take any action until 2022. Given my work with EPA on the USEPA Children's Health Protection Advisory Committee and my knowledge of the intensive risk assessment EPA conducted, there is absolutely no reason to delay action for at least four years, other than providing Dow Chemical with permission to grow its market.

Chlorpyrifos Neurotoxicity Can Occur at Very Low Doses

The scientific evidence of neurotoxic dangers associated with chlorpyrifos exposure is extensive and consistent. Chlorpyrifos inhibits the enzyme acetylcholinesterase (AChE), necessary to the transmission to normal nerve impulses and the full functioning of the nervous system. Studies document that exposure to low levels of chlorpyrifos during pregnancy can impair learning, change brain function, and alter thyroid levels of offspring into adulthood.^{1,2,3,4}

Although the acute toxicity of organophosphates (OPs) such as chlorpyrifos has been attributed to inhibition of acetylcholinesterase (AChE), there is growing evidence that this may not account for all the long-term neurotoxic effects of OPs. Studies show that OPs can induce additional neurotoxic effects at very low levels concentrations below those demonstrated to inhibit AChE.⁵ Some studies find that OPs interfere with proper neuronal development and function.⁶ Others find that OP pesticides may influence the nervous system by disrupting the lipid profile of the nervous tissue; disrupting axonal transport (movement of mitochondria, lipids, synaptic vesicles, proteins, and other cell parts to and from neuron cells), and decreasing the number of nerve cells.⁷

EPA in its assessment reviewed selected points of departure and their use in the quantitative risk assessment. (A point of departure (PoD) is a data point used as the basis for low dose extrapolation). In particular, EPA uses the dose causing 10% reduction in red blood cell AChE as a PoD for estimating low dose effects of AChE inhibitors.⁸ Data have shown that alteration of neuron function by chlorpyrifos that is not related to AChE inhibition.^{9,10,11} The agency determined that evidence supports chlorpyrifos's effects at concentrations below AChE inhibition PoD. However, regardless of the potential for multiple pathways of toxicity, the agency noted there remains high confidence in the current available and quantifiable evidence of neurological impact. EPA also stated that its revised analysis indicates "expected residues of chlorpyrifos on most individual food crops exceed the health-based 'reasonable certainty of no harm' safety standard under the Federal Food, Drug,

¹ Haviland et al. 2009. Long-term sex selective hormonal and behavior alterations in mice exposed to low doses of chlorpyrifos in utero. *Reproduc. Tox.* 29(1):74-9.

² Abou-Donia MB, et al. 2006. In utero exposure to nicotine and chlorpyrifos alone, and in combination produces persistent sensorimotor deficits and Purkinje neuron loss in the cerebellum of adult offspring rats. *Arch Toxicol.*;80(9):620-31.

³ Abdel-Rahman A, et al. 2003. Increased expression of glial fibrillary acidic protein in cerebellum and hippocampus: differential effects on neonatal brain regional acetylcholinesterase following maternal exposure to combined chlorpyrifos and nicotine. *J Toxicol Environ Health A.*;66(21):2047-66.

⁴ Icenogle LM, et al. 2004. Behavioral alterations in adolescent and adult rats caused by a brief subtoxic exposure to chlorpyrifos during neurulation. *Neurotoxicol Teratol*;26(1):95-101.

⁵ Androutsopoulos VP, Hernandez AF, Liesivuori J, Tsatsakis AM. 2013. A mechanistic overview of health associated effects of low levels of organochlorine and organophosphorous pesticides. *Toxicology.* 307:89-94.

⁶ Meijer M, Hamers T, Westerink RH. 2014. Acute disturbance of calcium homeostasis in PC12 cells as a novel mechanism of action for (sub)micromolar concentrations of organophosphate insecticides. *Neurotoxicology.* 43:110-6.

⁷ Roszczenko A, Rogalska J, et al. 2013. The effect of exposure to chlorfenvinphos on lipid metabolism and apoptotic and necrotic cells death in the brain of rats. *Exp Toxicol Pathol.* 65(5):531-9.

⁸ Transmittal of Meeting Minutes of the April 19-21, 2016 FIFRA SAP Meeting Held to Consider and Review Scientific Issues Associated with "Chlorpyrifos: Analysis of Biomonitoring Data." July 20, 2016.

⁹ Lee I, Eriksson P, Fredriksson A, et al. 2015. Developmental neurotoxic effects of two pesticides: Behavior and biomolecular studies on chlorpyrifos and carbaryl. *Toxicol Appl Pharmacol.* 288(3):429-38.

¹⁰ Androutsopoulos VP, Hernandez AF, Liesivuori J, Tsatsakis AM. 2013. A mechanistic overview of health associated effects of low levels of organochlorine and organophosphorous pesticides. *Toxicology.* 307:89-94.

¹¹ Meijer M, Hamers T, Westerink RH. 2014. Acute disturbance of calcium homeostasis in PC12 cells as a novel mechanism of action for (sub)micromolar concentrations of organophosphate insecticides. *Neurotoxicology.* 43:110-6.

and Cosmetic Act (FFDCA).” Additionally, the agency also points out that “risk from the potential aggregate exposure does not meet the FFDCA safety standard.”

Overwhelming Scientific Evidence Demonstrates Chlorpyrifos’s Danger to Children

Columbia University Studies Link Chlorpyrifos Exposure to Brain Changes and Developmental Disorders.

Researchers at Columbia University measured chlorpyrifos in umbilical cord blood of pregnant mothers and conducted intelligence tests for the children of these mothers later in childhood as part of a series of ongoing prospective cohort studies in inner-city minority populations. One study from this research group compared motor and mental development to levels of exposure to the pesticide at birth in 266 children born between 1998 and 2002 living in low-income neighborhoods of the South Bronx and northern Manhattan in New York City. The study found that concentrations of chlorpyrifos in umbilical cord blood correspond to a decrease in the psychomotor development and a decrease in the mental development in three year olds.¹² A follow-up study based on brain imaging by Magnetic Resonance Imaging found that children with high exposure levels of chlorpyrifos appear be related to changes in brain anatomy.¹³

Data from this research group was rigorously reviewed by EPA scientists who concurred that children exposed to high levels of chlorpyrifos had mental development delays, attention problems, attention-deficit/hyperactivity disorder problems, and pervasive developmental disorder problems.^{14,15} The results of these cohort studies have consistently found that depressed cognitive development, birth weights and other neurodevelopmental endpoints are adversely impacted by chlorpyrifos and other pesticide exposures.¹⁶

You may hear from bill opponents that the Columbia study was not considered a valid study by the EPA’s Science Advisory Panel (SAP). This is untrue. Although the SAP disagreed with the use of a data point defined by concentrations of chlorpyrifos in umbilical cord blood as a point of departure for a quantitative risk assessment following agency protocol, it did agree that there is *an association between chlorpyrifos prenatal exposure and neurodevelopmental outcomes in children as noted in the Columbia study*. Specifically, “both epidemiology and toxicology studies suggest there is evidence for adverse health outcomes associated with chlorpyrifos exposures below levels that result in 10% red blood cell (RBC) acetylcholinesterase (AChE) inhibition (i.e., toxicity at lower doses).”¹⁷

¹² Lovasi, GS, et al. 2011. Chlorpyrifos Exposure and Urban Residential Environment Characteristics as Determinants of Early Childhood Neurodevelopment. *Am J Public Health*;101(1):63-70.

¹³ Rauh VA, Perera FP, Horton MK, et al. 2012. Brain anomalies in children exposed prenatally to a common organophosphate pesticide. *Proc Natl Acad Sci U S A*. 109(20):7871-6.

¹⁴ Rauh VA. 2006. Impact of prenatal chlorpyrifos exposure on neurodevelopment in the first 3 years of life among inner-city children. *Pediatrics*;118(6):e1845-59.

¹⁵ Rauh V, Arunajadai S, Horton M, Perera F, Hoepner L, Barr DB, et al. 2011. Seven-Year Neurodevelopmental Scores and Prenatal Exposure to Chlorpyrifos, a Common Agricultural Pesticide. *Environ Health Perspect* 119:1196-1201.

¹⁶ Perera FP, et al. 2005. A summary of recent findings on birth outcomes and developmental effects of prenatal ETS, PAH, and pesticide exposures. *Neurotoxicology*;26(4):573-87.

¹⁷ Transmittal of Meeting Minutes of the April 19-21, 2016 FIFRA SAP Meeting Held to Consider and Review Scientific Issues Associated with “Chlorpyrifos: Analysis of Biomonitoring Data.” July 20, 2016. <https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0062-0140>.

University of California Research Finds OP Exposure is linked to IQ Deficit.

Researchers from the University of California, Berkeley, examining families in the intensive agricultural region of Salinas Valley, California, found that IQ levels for children with the highest OP exposure were a full seven IQ points lower than those with the lowest exposure levels. This team also found that every tenfold increase in measures of OPs detected during a mother's pregnancy corresponded to a 5.5 point drop in overall IQ scores in seven-year-olds.¹⁸

Mount Sinai Research Finds Prenatal Exposure Affects Cognitive Development.

Researchers from Mount Sinai School of Medicine also found that prenatal exposure to organophosphates is negatively associated with cognitive development, particularly perceptual reasoning, with evidence of effects beginning at 12 months and continuing through early childhood.¹⁹ These data sets and others accumulated over years of study support a need to protect children from this pesticide.

Chlorpyrifos Has Disproportionate Impacts on Children Living Near Treated Fields.

Research on chlorpyrifos also underscores that certain subpopulations are disproportionately affected by chlorpyrifos exposures. Low-income African-American and Latino families, including farmworker families, continue to suffer the most, and this disproportionate impact creates an environmental justice issue that continues. For farmworkers and their families, threats from chlorpyrifos are dire. Farmworker studies routinely show high exposure from pesticide drift in these communities.^{20,21} Pregnant women in these communities are especially at risk. Research from the University of California, Davis, Childhood Autism Risks from Genetics and the Environment (CHARGE) finds that pregnant women who live within a mile of agricultural fields treated with insecticides like chlorpyrifos are more likely to have their child develop autism.²² For women who lived less than one mile from crops sprayed with OP insecticides during their pregnancy, the chance their child being diagnosed with autism increases by 60%. Women in the second trimester living near chlorpyrifos-treated fields are 3.3 times more likely to have their children diagnosed with autism.²³

Federal Regulation Fails to Protect Children.

Despite several extensive risk assessments over almost two decades showing unacceptable risks, EPA had continued to attempt to mitigate exposures by imposing no-spray buffer zones around public spaces including recreational areas, schools, and homes to reduce bystander exposure risks over the years. In 2000, the agency even banned indoor use of chlorpyrifos in light of indoor risks to children. In 2015, EPA announced it would revoke all food tolerances for chlorpyrifos. This decision to revoke tolerances therefore came at a time when EPA could no longer continue to mitigate risks, given the overwhelming data unquestionably showing risks to human health. EPA's

¹⁸ Bouchard MF, Chevrier J, Harley KG, Kogut K, Vedar M, Calderon N, et al. 2011. Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children. *Environ Health Perspect.* 119:1189-1195.

¹⁹ Engel, S. et al. 2011. Prenatal Exposure to Organophosphates, Paraoxonase 1, and Cognitive Development in Childhood. *Environ Health Perspect.* 119:1182-1188.

²⁰ Das R, Steege A, Baron S, et al. 2001. Pesticide-related illness among migrant farm workers in the United States. *Int J Occup Environ Health.* 7(4):303-12.

²¹ Reeves M, Schafer KS. 2003. Greater risks, fewer rights: U.S. farmworkers and pesticides. *Int J Occup Environ Health.* 9(1):30-9.

²² Shelton, J, Geraghty, EM, Tancredi, DJ, et al. 2014. Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study. *Environ Health Perspect.* 122:1103-1109.

²³ Ibid.

assessments have continually found ‘significant risks’ to children and farmworkers as a result of the chemical’s use.

In 2016, EPA convened a Scientific Advisory Panel (SAP) meeting to discuss its proposal. Overall, the SAP agreed with EPA’s conclusions and those of independent study – that there is an association between chlorpyrifos prenatal exposure and neurodevelopmental outcomes in children.

²⁴ Finding in accordance with the recommendations from the SAP, EPA concluded again that there is “sufficient evidence that there are neurodevelopmental effects occurring at chlorpyrifos exposure levels below that required for AChE inhibition,”²⁵ and that EPA’s current approach for evaluating chlorpyrifos’s neurological impact is “not sufficiently health protective.”

It is clear that chlorpyrifos fails to meet the health standards of “no unreasonable adverse effect” and “reasonable certainty of no harm” set forth in law. As a pediatrician, I am concerned about children’s unique susceptibilities to pesticide toxicity that cause prenatal and early life exposures to be associated with decreased cognitive function, and behavioral problems. The American Academy of Pediatrics (AAP) published a clinical finding in October 2012 that states that reducing pesticide residues in food is beneficial for children’s health. The Academy’s policy statement on pesticides identifies the current shortfalls in medical training, public health tracking, and U.S. regulatory action on pesticides. These should serve as a sobering wake-up call for government agencies and elected officials to protect our children and environment from toxic compounds.

EPA’s Previous Decision to Remove Chlorpyrifos Has Been a Long Deliberative Process

When EPA negotiated the removal of all chlorpyrifos for residential use in 2000, it was keenly aware of the elevated neurotoxic effects to children. That sweeping action by EPA and Dow was not taken lightly. However, it was done knowing that there were still risk factors in the market due to retaining most agricultural uses and drastically reducing allowable residues. But, as more data came in over the last almost two decades, it is impossible to justify retaining the agricultural uses any longer.

The time to act is now. As a pediatrician, I am dedicated to the concept that all children have the right to reach their full potential. They should not be exposed to factors in fetal development and early life that will impair them throughout life and not allow them to reach their full potential. Exposure to chlorpyrifos is clearly one of those factors. It can be removed quite –simply improving the potential of thousands of children in Maryland. Its continued use is of no benefit to these children, but suspension of use will benefit all of them. It has often been said that children are about 20% of our population but represent 100% of our future. Lets work for the best future for them. I urge the adoption of SB 300 so the children in Maryland are free from the toxic dangers posed by chlorpyrifos and are afforded the opportunity to develop in a more healthy environment.

Thank you for your consideration.

²⁴ Gunier, RB, Bradman A, Harley K, et al. 2016. Prenatal Residential Proximity to Agricultural Pesticide Use and IQ in 7-Year-Old Children. Environ Health Perspect DOI: 10.1289/EHP504.

²⁵ USEPA. 2016. Chlorpyrifos: Revised Human Health Risk Assessment for Registration Review. Office of Chemical Safety and Pollution Prevention. Washington DC.