INTRODUCTION

The Environmental Protection Agency (“EPA”) released an update to its assessment of the human health risks posed by chlorpyrifos that confirms all uses of chlorpyrifos are unsafe and must be banned. These comments are submitted on behalf of Earthjustice, United Farm Workers, Natural Resources Defense Council, Pesticide Action Network, Farmworker Justice, California Rural Legal Assistance Foundation, National Hispanic Medical Association, Pineros y Campesinos Unidos del Noroeste, GreenLatinos, Migrant Clinicians Network, League of United Latin American Citizens, Labor Council for Latin American Advancement, and Farmworker Association of Florida.

EPA had previously found, based on studies from Columbia University and other academic institutions, that prenatal exposures to chlorpyrifos are correlated with lower IQ, loss of working memory, attention deficit disorders, and developmental delays. EPA, the academic researchers, and EPA’s Scientific Advisory Panel (“SAP”) all found that these brain damage impacts occur at far lower exposures than those associated with acute poisoning. Nonetheless, in its December 2014 chlorpyrifos revised human health risk assessment, EPA continued to use 10% cholinesterase inhibition as its regulatory endpoint. Our previous comments, as well as comments submitted by scientists and health professionals, explained why that endpoint was not protective and left people, and children in particular, vulnerable to extremely unsafe chlorpyrifos exposures.

EPA has now changed its regulatory endpoint. It has lowered what it deems to be allowable exposures to chlorpyrifos in an attempt to prevent brain damage from in utero exposures. Using this updated endpoint, EPA found the following alarming risks:

- All food exposures exceed safe levels, with the most exposed population being children between 1-2 years of age. This vulnerable age group is on average exposed to 140 times what EPA deems safe.

- There is no safe level of chlorpyrifos in drinking water.

- Drift of pesticides from the fields expose children to unsafe levels of chlorpyrifos within 300 or more feet of the fields where the pesticide is sprayed. Children could be exposed to toxic drift at schools and day cares, in their homes, or at playgrounds.
• All workers who mix and apply chlorpyrifos pesticides are exposed to levels greater than what EPA considers safe. That is the case even with the maximum possible protective clothing, equipment, and engineering controls.

• Field workers are currently allowed to re-enter fields within 1-5 days after pesticide spraying, but unsafe exposures continue on average for 18 days after applications.

Chlorpyrifos is contaminating our food and water and exposing workers and their families to poisonings, learning disabilities, and other needless harm.

EPA has proposed to revoke all food tolerances for chlorpyrifos and has found that all uses, including non-food uses, lead to drinking water contamination and dangerous exposures for workers and children. Since EPA proposed revoking chlorpyrifos tolerances, the European Union agreed upon new endpoints following an updated toxicological review of chlorpyrifos. Based on these new endpoints, the United Kingdom banned all but one use of chlorpyrifos on an expedited timeline. EPA should act with similar haste to ban all chlorpyrifos uses with an effective date not more than six months from the date of the revocation determination.

In addition to the sources cited within, these comments rely upon and incorporate the following attached documents: Petition for Emergency and Ordinary Suspension of Chlorpyrifos Uses that Pose Unacceptable Risks to Workers and Petition to Cancel All Uses of Chlorpyrifos, September 21, 2016 (Attachment 1); and Declaration of Philip J. Landrigan, M.D., M.Sc. in Support of Petition to Suspend and Cancel Chlorpyrifos Uses (Attachment 2).

I. EPA HAS APPROPRIATELY ESTABLISHED A REGULATORY ENDPOINT DESIGNED TO GUARD AGAINST BRAIN DAMAGE FROM PRENATAL EXPOSURES

EPA’s use of a regulatory endpoint based on neurodevelopmental effects in the 2016 Chlorpyrifos Revised Human Health Risk Assessment (“RHHRA”) comports with best science and ensures reasonable certainty of no harm as required by the Food Quality Protection Act (“FQPA”). See infra, section II. Historically, EPA has used 10% cholinesterase inhibition as the endpoint for chlorpyrifos and other organophosphate pesticides. However, in reconstructing the chlorpyrifos doses experienced by pregnant women that were associated with serious adverse neurodevelopmental impacts in their children, EPA found that the pregnant mothers would have had less than 1% cholinesterase inhibition. RHHRA at 13. In other words, EPA determined that the neurodevelopmental harm occurred when the mothers were exposed to far lower doses of chlorpyrifos than what produces 10% cholinesterase inhibition. EPA considered both epidemiological studies and toxicological studies conducted on animals in making its determination. Based on these findings and the FQPA safety standard, EPA needed to either update its regulatory end point or add safety factors to account for these risks. EPA’s approach is appropriate, scientifically defensible, and serves to adequately protect pregnant women and children.
A. **EPA and Multiple SAPs, Including the 2016 SAP, Recognized That Using 10% Cholinesterase Inhibition Does Not Protect Kids**

EPA, the 2012 SAP and the 2016 SAP all agree that the point of departure used in the 2014 chlorpyrifos human health risk assessment based on 10% cholinesterase inhibition does not account for neurodevelopmental effects and, therefore, is not sufficiently protective:

In summary, these lines of evidence suggest that chlorpyrifos can affect neurodevelopment at levels lower than those associated with AChE inhibition, and that the use of AChE inhibition data may not be the most appropriate for dose-response modeling and derivation of a point of departure for assessment of the neurodevelopmental risks of chlorpyrifos.¹

The agency agrees with the 2016 FIFRA SAP (and previous SAPs) that there is a potential for neurodevelopmental effects associated with chlorpyrifos exposure to occur at levels below 10% RBC AChE inhibition, and that EPA’s existing point of departure (which is based on 10% AChE inhibition) is therefore not sufficiently health protective. 81 Fed. Reg. 81,049, 81,050 (Nov. 17, 2016).

The Panel agrees that 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).²

B. **EPA’s Revised Approach is Consistent with the Science on Neurodevelopmental Impacts and Its Proposal to Cancel All Food Tolerances will Protect Kids**

In order to address the neurodevelopmental effects and protect kids, EPA needed to change its approach to the point of departure, which is exactly what the agency has done in the 2016 assessment:

The 2014 revised human health risk assessment used dose-response data on acetylcholinesterase inhibition (AChI) [sic] in laboratory animals to derive a point of departure. However, the EPA believes that evidence from epidemiology studies indicates effects may occur at lower exposures than indicated by the toxicity database. The 2016 revised human health risk assessment uses neurodevelopmental effects as the critical effect, taking into account

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recommendations from the 2016 chlorpyrifos SAP on deriving a point of departure for risk assessment.³

EPA appropriately retained the FQPA safety factor. The agency chose a total value of 10X, but as discussed at greater length in our 2015 comments, the total FQPA safety factor should be greater than 10X due to uncertainties and concerns about prenatal toxicity.⁴

Briefly, EPA has previously set FQPA safety factors at greater than 10X to account for incomplete data and prenatal toxicity. Table 1 provides examples from past assessments that set FQPA safety factors greater than 10X when there are both data deficiencies and concerns for prenatal toxicity. Through these assessments, EPA has established a practice of setting the FQPA safety factor at more than 10X when appropriate based on its consideration of both data completeness and special FQPA concerns.

Table 1. Uncertainty and safety factors used by EPA in past pesticide assessments.

<table>
<thead>
<tr>
<th>Pesticide</th>
<th>Intra-species Factor</th>
<th>Inter-species Factor</th>
<th>Data Completeness Factor (specific data deficiency)</th>
<th>Special FQPA concerns (factors contributing to degree of concern)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbendazim (MBC)⁵</td>
<td>10X</td>
<td>10X</td>
<td>3X (extrapolation from LOAEL)</td>
<td>10X (increased prenatal susceptibility in rat and rabbit studies)</td>
</tr>
<tr>
<td>Molinate⁶</td>
<td>10X</td>
<td>10X</td>
<td>3X (extrapolation from LOAEL)</td>
<td>10X (prenatal toxicity in rodent studies; uncertainties in drinking water exposure)</td>
</tr>
<tr>
<td>Pirimiphos-methyl⁷</td>
<td>10X</td>
<td>10X</td>
<td>10X (extrapolation from LOAEL, severity of effects at LOAEL, data gaps for long term studies)</td>
<td>3X (lack of complete toxicity database for assessing potential for susceptibility)</td>
</tr>
</tbody>
</table>

One of the most common situations in which EPA has established a higher safety factor is when the animal studies lack a no observable adverse effect level (“NOAEL”) and the agency selects a lowest observed adverse effect level (“LOAEL”) as the point of departure. In the 2016 assessment, EPA wrote, “The [time weighted average] blood level resulting from chlorpyrifos exposure from the crack and crevice scenario is considered a [LOAEL], since this is the exposure

⁶ EPA OPP, Health Effects Division, Human Health Risk Assessment: Molinate at 6, 14 (November 6, 2002).
⁷ EPA OPP, Health Effects Division, Interim Reregistration Decision for Pirimiphos-Methyl: Case No. (2535) at 7 (July 31, 2006).
level likely to be associated with neurodevelopmental effects reported in the [Columbia Center for Children’s Environmental Health] study.” RHHRA at 4. Accordingly, EPA should have considered whether the FQPA safety factor should be greater than 10X to account for the additional uncertainty when extrapolating from a LOAEL in addition to the increased vulnerability of infants and children.

These safety factor considerations apply regardless of the endpoint used to determine the point of departure, as we noted in our 2015 comments. The data deficiencies and prenatal toxicity concerns for chlorpyrifos warrant an FQPA factor greater than 10X whether EPA was using an endpoint of 10% cholinesterase inhibition (as in their 2014 assessment) or an endpoint of neurodevelopmental impacts, as in the current assessment.

C. Following the Advice of the 2016 SAP, EPA Used the PBPK Model and Standard Exposure Assessment Protocols to Derive the Time Weighted Average Blood Concentration of Chlorpyrifos

As noted by EPA, the 2016 SAP was supportive of using the physiologically based pharmacokinetic (“PBPK”) model as a tool to analyze exposure data: “Multiple Panel members noted that PBPK modeling is a valuable tool to interpret the biomonitoring data in circumstances where multiple routes of exposure occur and when based on best available information as inputs.” 2016 SAP at 18. Further, the 2016 SAP recommended using the PBPK model to predict a time weighted average blood concentration predicted for women in the Columbia Center for Children’s Environmental Health (“CCCEH”) cohort: “…the estimated peak blood concentration or time weighted average (TWA) blood concentration within the prenatal period should be designated as the point of departure (PoD) for risk assessment…” Id. at 42.

EPA determined that the CCCEH cohort women most likely experienced exposure from crack-and-crevice application of chlorpyrifos based on information from professional pest control applicators, and the fact that other common residential uses were phased out in 1997. RHHRA at 14-15. EPA followed the SAP’s advice and estimated exposures from the crack-and-crevice chlorpyrifos application using standard, peer-reviewed methods and inputs, including the following:

- 2012 Standard Operating Procedures for Residential Pesticide Exposure Assessment (Residential SOPs);

- Amount of chlorpyrifos residue that dissipates daily: based on all available chlorpyrifos-specific floor residue data;

- Post-application exposure durations: from EPA Exposure Factors Handbook 2011; and

- Female bodyweight: from EPA Exposure Factors Handbook 2011.

RHHRA at 16-17. The predicted time weighted average blood concentration, 4 pg/g (0.004 ug/L), is reasonable in comparison to the measurements from the CCCEH study women, which
ranged from 0.8-19.3 pg/g in 1999. EPA 2016 Chlorpyrifos Issue Paper: Evaluation of Biomonitoring Data from Epidemiology Studies at 14. Furthermore, EPA’s application of the time weighted average blood concentration to young children is supported by data from animal studies showing that the post-natal period is a window of susceptibility.8

D. The 2016 Chlorpyrifos Assessment is an Appropriate and Scientifically Defensible Use of Epidemiologic and Biomonitoring Information

While both the 2014 and 2016 chlorpyrifos risk assessments use the PBPK model sponsored by Dow AgroSciences for deriving internal dosimetry measures,9 the 2016 RHHRA has several important improvements over the earlier 2014 assessment. Whereas the 2014 assessment used 10% cholinesterase inhibition as a Point of Departure (“PoD”), in the 2016 assessment EPA followed the recommendations of its SAP to address the risks below 10% cholinesterase inhibition because, “epidemiology and toxicology studies suggest there is evidence for adverse health outcomes associated with chlorpyrifos exposures below levels that result in 10% [cholinesterase inhibition].” RHHRA at 10 (quoting 2016 SAP). By using the CCCEH epidemiologic data to inform the PoD, the new 2016 risk assessment better addresses the elevated risks to vulnerable and sensitive populations from real-world exposures, including levels below those that trigger a 10% cholinesterase inhibition.

1. Epidemiologic data and biomonitoring from unintentional human exposures provide valuable information used across EPA programs to calculate risk estimates and support regulations.

To generate accurate and relevant risk assessments, EPA should use all available information relevant to hazard, exposure, use, manufacturing process, disposal, and other aspects of the life cycle of chemicals. In particular, occupational or environmental epidemiologic studies – cohort, case-control, ecological, and others – can provide very valuable information to inform risk evaluation because such studies capture real-world exposure conditions that do not exist in laboratory settings. As noted in EPA’s Draft Framework for Incorporating Human Epidemiologic & Incident Data in Health Risk Assessment:

Specifically, these types of human information provide insight into the effects caused by actual chemical exposures in humans and thus can contribute to problem formulation and hazard/risk characterization. In addition, epidemiologic and human incident data can guide additional analyses or data generations (e.g., dose and endpoint selection for use in in vitro and targeted in vivo experimental

8 Animal studies are reviewed in EPA’s 2014 Revised Human Health Risk Assessment for Chlorpyrifos, pg. 25-26. Specifically, EPA finds that, “There is a considerable and growing body of literature on the effects of chlorpyrifos on the developing brain of laboratory animals (rats and mice) indicating that gestational and/or postnatal exposure may cause persistent behavioral effects into adulthood. These data provide support for the susceptibility of the developing mammalian brain to chlorpyrifos exposure.”

studies), identify potentially susceptible populations, identify new health effects or confirm the existing toxicological observations.¹⁰

The EPA IRIS program has effectively and appropriately used epidemiologic and human biomonitoring data from unintentional exposure studies to calculate risk estimates and support regulatory decisions. For example:

- Mercury (IRIS 2012). Epidemiologic data (the Faroe Islands analysis) was used quantitatively in EPA’s evaluation of risk for methylmercury, as recommended by the National Academies.¹¹ EPA based the oral RfD on lasting neurological effects in children exposed during early life (Grandjean et al., 1977; Budtz-Jorgensen et al., 1999).¹²

- Tetrachlroethylene (IRIS 2012). EPA IRIS risk assessment of tetrachloroethylene (perchloroethylene), which was reviewed and approved by the National Academies in 2010, used both epidemiologic and animal study data, along with a pharmacokinetic model,¹³, ¹⁴ similar to the data-integration approach used by EPA in this 2016 chlorpyrifos assessment.

- 1,3-Butadiene (IRIS 2002). Generated the cancer risk from inhalation exposure based on the epidemiologic study of styrene-butadiene rubber production workers (Delzell et al., 1995). Health Canada used the same data and same approach for its cancer risk estimate.¹⁵

- Benzene (IRIS 2003). The oral RfD was based on decreased lymphocyte count in a workplace epidemiologic study (Rothman et al., 1996). The RfD is based on route-to-route extrapolation of the results of benchmark dose (BMD) modeling of the absolute lymphocyte count (ALC) data from the occupational epidemiologic study by Rothman et al. (1996), in which workers were exposed to benzene by inhalation. Rothman et al. (1996) conducted a cross-sectional study of 44 workers exposed to benzene and 44 age- and gender-matched unexposed controls.

Twenty-one of the 44 subjects in the exposed and control groups were female. Mean (standard deviation) years of occupational exposure to benzene were 6.3 (4.4), with a range of 0.7-16 years. Benzene exposure was monitored by organic vapor passive dosimetry badges worn by each worker for a full workshift on 5 days within a 1-2 week period prior to collection of blood samples.\(^{16}\)

- Arsenic carcinogenicity (IRIS 1991). EPA classified arsenic as a human carcinogen based on sufficient evidence of lung cancer deaths in multiple epidemiologic studies of inhalation exposures, and organ cancers (liver, kidney, lung, bladder) and skin cancers in populations consuming inorganic arsenic-contaminated drinking water. The animal data were considered inadequate. EPA calculated the cancer risk estimate based on the oral dose-response data from a study of a Taiwan population exposed through drinking water (Tseng et al., 1968; Tseng 1977). The inhalation cancer risk estimate was based on epidemiologic evidence of lung cancer in men exposed occupationally (Brown and Chu 1983, Lee-Feldstein 1983; Higgins 1982; Enterline and Marsh, 1982).\(^{17}\)

IRIS risk assessments are used by states and local governments, federal agencies, and countries worldwide, to support regulatory decisions such as air quality and water quality standards to protect public health and to set cleanup standards for hazardous waste sites.

Epidemiological studies have a long history as the basis for regulatory decision-making and standard setting to reduce exposures to lead, another developmental neurotoxicant where low-level exposure has been tied to significant and permanent harm to children. Both EPA’s air quality standard (National Ambient Air Quality Standard – NAAQS) and soil clean-up level are based on concentration-response functions derived from epidemiological studies comparing levels of lead measured in blood with neurodevelopmental outcomes in children at different ages. In both cases, EPA determined that IQ point loss was the most sensitive and robust outcome variable on which to derive the concentration-response function.\(^{18,19}\) In 2007, California’s Office of Environmental Health Hazard Assessment relied on epidemiological studies to derive a critical effect level which has formed the basis for re-evaluating health-based standards for lead in residential soil and drinking water.\(^{20}\) This analysis was triggered by findings in epidemiologic studies that neurobehavioral deficits were recorded at levels below the existing regulatory thresholds and concluded that the loss of one IQ point was an appropriate point of departure on

which to derive a blood-lead level of concern.\textsuperscript{21} In 2012, the Centers for Disease Control and Prevention relied on epidemiologic data to replace a previous blood lead level of concern of 10 \(\mu g/dL\) with a blood lead action level of 5 \(\mu g/dL\).\textsuperscript{22}

It should be noted that the above examples of use of epidemiologic and biomonitoring data by EPA programs and others are very different from the intentional human dosing studies that have been conducted by pesticide registrants and sometimes used by EPA. An expert workshop of ethicists, physicians, toxicologists, and policy experts hosted by Mount Sinai School of Medicine (2002) reported on several of these intentional-dosing pesticide studies, including this example: “In 1998, after signing a seven-page consent form, dozens of college-age Nebraskans were paid $450 to swallow a pill containing chlorpyrifos. Chlorpyrifos is the active ingredient in Raid roach spray, manufactured by the Dow Chemical Company (Midland, MI). The students learned about this study after reading school newspaper ads urging students to call (402) 474-PAYS to ‘earn extra money.’”\textsuperscript{23} Indeed, Dow’s PBPK model, which is used in the chlorpyrifos assessments, relies on data from deliberate human dosing studies.\textsuperscript{24} Prominent scientists and physicians have condemned these pesticide-dosing studies – and EPA’s use of them for regulatory decisions - as unethical and bad science.\textsuperscript{25}

However, the same experts agree that the use of well-conducted epidemiologic and human biomonitoring studies, from unintentional exposures, can provide useful and important information for risk assessments and regulations. The 2002 Mount Sinai workshop participants recommended that:

Public health scientists and practitioners use biomonitoring information for


\textsuperscript{24} More detailed comments on the use of human dosing studies are available at Farmworker and Conservation Comments on Chlorpyrifos Revised Human Health Risk Assessment (Apr. 30, 2015) at 36-42, Docket No. EPA-HQ-OPP-2008-0850; see also supra note 8.


tracking, control, and treatment. Biomonitoring data can also play a critical role in identifying novel hazards and high-risk populations, tracking trends in human exposure, and characterizing exposure levels that pose health hazards. Many workshop participants suggested that biomonitoring provides important and useful information for risk assessment, particularly for determining patterns of exposure and the risks that pesticides pose to children’s health. Workshop participants agreed that human biomonitoring should be conducted for every pesticide that is currently in use or present in the environment and posing human exposure risks. They also recommended that special consideration be given to assessing the body burdens of pesticides in children.  

EPA’s use of CCCEH cohort biomonitoring data in its 2016 chlorpyrifos assessment is very much consistent with this recommendation.

2. The CCCEH findings are consistent with a robust body of scientific evidence

The following section is excerpted from comments submitted to the public docket from environmental health scientists and healthcare professionals in support of EPA’s 2016 Revised Human Health Risk Assessment and EPA’s 2015 proposed tolerance revocation for chlorpyrifos (Sass, Whyatt et al., 2017, 2016):

- Extensive published science from diverse populations correlates pre-natal chlorpyrifos exposure to reduced birth weights, delayed mental and motor development in preschoolers, and reduced IQ and delays in working memory in elementary school children (Rauh et al., 2006, 2011, Whyatt et al., 2005). These persistent neurocognitive findings are especially troubling. In addition, in a pilot study of 6-11 year olds, chlorpyrifos concentrations in umbilical cord blood were associated with changes in brain structure measured by magnetic resonance imaging, including cortical thinning and regional specific cortical deformations (Rauh et al., 2012).

- A 2015 study of inner city minority children reported a link between prenatal exposure and mild to moderate arm tremors measured when the children were middle-school aged, suggesting an even broader scope of effects on the nervous system from early life exposures, and potentially latent or long term neurological damage manifesting a decade later or beyond (Rauh et al., 2015).

- Application of chlorpyrifos to agricultural fields within 1.5 km of the home during pregnancy has also been associated with an increased incidence of autism spectrum disorders in a recent study (Shelton et al., 2014). A recently published study of Costa Rican children living near banana and plantain farms showed a dose-dependent adverse impairment of working memory in boys, oppositional

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disorders, ADHD, decreased ability to discriminate colors, and an increased prevalence of cognitive problems in the parents (van Wendel de Joode et al., 2016).

- These epidemiologic results are consistent with data from toxicological studies which found disruption in neuronal development, neurotransmitter systems and synaptic formation as well as behavioral and cognitive impairments in test animals following low-dose perinatal chlorpyrifos exposure (Slotkin 2004; Aldridge et al., 2004, 2005; Slotkin and Seidler, 2005, Levin et al 2001; Roy et al., 2004; Garcia et al., 2002).

- Associations in newborns also were seen between prenatal exposures to organophosphate pesticides generally and abnormalities in primitive reflexes, suggesting an impact on the development of the central nervous system (Engel et al., 2007; Young et al, 2005) and in children with reduction in motor function (Eskenazi et al., 2007; Rauh et al., 2006; Grandjean et al., 2006; Handal et al., 2008; Harari et al., 2010, Rauh et al., 2015), decreases in working and visual memory, processing speed, verbal comprehension, perceptual reasoning, and full scale IQ (Bouchard et al., 2011, Engel et al., 2011, Rauh et al., 2011; Handal et al., 2008) and increases in neuropsychological problems including ADHD, pervasive developmental disorder and behaviors typical of the autism spectrum (Rauh et al., 2006, Marks et al., 2010, Furlong et al., 2014). Certain subpopulations demonstrate greater susceptibility including children of farmworkers (Castorina et al., 2010; Engel et al., 2015) and those who have reduced capacity to detoxify the OPs (Engel et al., 2015).

II. EPA HAS FOUND UNSAFE EXPOSURES FROM FOOD, DRINKING WATER, TOXIC DRIFT, AND WORKER ACTIVITIES, COMPELLING AN IMMEDIATE BAN ON ALL CHLORPYRIFOS USES

In its 2016 Chlorpyrifos Revised Human Health Risk Assessment (“RHHRA”), EPA found that chlorpyrifos presents unacceptable safety risks through exposures from food, drinking water, spray drift, and occupational activities. The risks were found to be particularly alarming for children and farm workers. Under the Federal Food, Drug and Cosmetics Act (“FFDCA”), EPA may not “establish or leave in effect a tolerance for a pesticide chemical residue in or on a food” unless the Administrator determines that the tolerance is safe. 21 U.S.C. § 346a(b)(2)(A)(i). The Food Quality Protection Act (“FQPA”), a 1996 amendment to the FFDCA, requires that EPA make an affirmative determination that there is reasonable certainty of no harm from use of a pesticide in accordance with its label, and it must make this finding considering aggregate and cumulative exposures to infants and children. Id. § 346a(b)(2)(C)(ii)(I), (II). EPA must revoke a tolerance if it finds a pesticide residue would not be safe. Id. § 346a(b)(2)(A)(i).

Additionally, under the Federal Insecticide, Fungicide and Rodenticide Act (“FIFRA”), a pesticide may not be registered for a food use unless a food tolerance is in place, and whenever a food tolerance is revoked, the registration for use of the pesticide on that food crop must be cancelled. See 7 U.S.C. § 136a(c)(5)(D); see also id. § 136(bb). Because of this
interdependence, the FQPA directs EPA to coordinate FQPA actions to revoke tolerances with any related, necessary FIFRA action. 21 U.S.C. § 346a(l). Chlorpyrifos fails to meet the FQPA “reasonable certainty of no harm” safety standard, so EPA must revoke all food tolerances and cancel all food uses.

A. EPA Must Revoke All Food Tolerances For Chlorpyrifos Because Dietary Exposures Exceed Safe Levels

Food exposures for chlorpyrifos were found to be unsafe for all population subgroups analyzed, with young children having the highest risks of concern. RHHRA at 23. While the adult subgroup had an alarming risk estimate at 62 times the safe level of exposure, the risk estimate for children ages 1-2 was more than double that of adults at 140 times what EPA deems safe. Id.

Additionally, EPA’s 2014 Acute and Steady State Dietary (Food Only) Exposure Analysis to Support Registration Review identified extensive use of chlorpyrifos on food crops and widespread contamination in the form of detectable residues.27 Of particular concern is the frequent detection of residues on fruits consumed regularly by children. Fruits that are a typical part of children’s diets – like apples, peaches, oranges and strawberries28 – are widely grown using chlorpyrifos. Chlorpyrifos residues are found on these fruits, according to the results of USDA Pesticide Data Program (USDA PDP) testing, even after they are washed and peeled (in the case of citrus, bananas, and melons). Residues are routinely found on fruits that are not heavily treated with chlorpyrifos in the U.S., due to high consumption of frequently imported fruits, like peaches, grapes, and melons.

### Table 2: Chlorpyrifos use and residues on fruit consumed by children

<table>
<thead>
<tr>
<th>Fruit</th>
<th>Percent of whole fruit (not juice) in kids diet*</th>
<th>Chlorpyrifos residue detected**</th>
<th>Percent of US crop treated with chlorpyrifos**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apples</td>
<td>36%</td>
<td>Yes</td>
<td>55%</td>
</tr>
<tr>
<td>Bananas</td>
<td>13%</td>
<td>Yes</td>
<td>N/A</td>
</tr>
<tr>
<td>Melons</td>
<td>11%</td>
<td>Yes</td>
<td>&lt;2.5%</td>
</tr>
<tr>
<td>Other fruit/fruit salads</td>
<td>10%</td>
<td>No data</td>
<td>N/A</td>
</tr>
<tr>
<td>Citrus</td>
<td>9%</td>
<td>Yes</td>
<td>Oranges - 20%</td>
</tr>
<tr>
<td>Berries</td>
<td>8%</td>
<td>Yes</td>
<td>Strawberries - 20%</td>
</tr>
<tr>
<td>Peaches/nectarines</td>
<td>7%</td>
<td>Yes</td>
<td>25%/10%</td>
</tr>
<tr>
<td>Grapes</td>
<td>5%</td>
<td>Yes</td>
<td>10%</td>
</tr>
</tbody>
</table>

27 EPA’s 2014 Acute and Steady State Dietary (Food Only) Exposure Analysis to Support Registration Review can be found under docket number EPA-HQ-OPP-2008-0850-0197.

USDA’s PDP testing prioritizes regular monitoring of pesticide levels on foods with high consumption rates and a focus on foods consumed by children and infants. USDA Pesticide Data Program, Annual Summary – Calendar Year 2015. 29 For this reason, the program regularly tests apples, and other fruit, for residues. EPA’s dietary exposure assessment cited USDA PDP residue detections for apples from 2009-2010. The most recent data available from USDA (calendar year 2015) confirms that chlorpyrifos is regularly detected on apples. Id. In 2015, residues were also detected on cherries (fresh and frozen), cucumbers, grapes, nectarines, oranges, peaches, pears, potatoes, spinach, strawberries, and tomatoes – nearly 1 in 10 of the peaches sampled were found to have chlorpyrifos residues. Id. USDA PDP data confirms the widespread presence of chlorpyrifos residues on fruits and vegetables regularly consumed by children and pregnant women.

Surveys show that apples are regularly consumed by children on a daily basis and EPA’s CALENDEx_FCID Profile for children appropriately considers dietary risk from consumption of approximately 1 apple per day. 30, 31 In USDA’s PDP testing, the limit of detection for chlorpyrifos on apples ranges from 0.001 – 0.005 ppm. At any level above the detection limit, a child’s daily exposure to chlorpyrifos would exceed safe levels.32 Since USDA PDP data regularly finds detections of chlorpyrifos on apples, and any detection would exceed the steady state Population Adjusted Dose (“ssPAD”) due to exposure to apples alone, chlorpyrifos residues on apples present a clear risk to children in the United States. Therefore, EPA cannot set a tolerance that would protect children from the neurodevelopmental risks posed by chlorpyrifos exposures and the tolerances must be revoked immediately.

Apples are not the only commodity of concern. In the 2015 USDA PDP data, the highest chlorpyrifos residue detected was on peaches at 0.38 ppm. Using EPA’s standard assumptions for consumption frequency and body weight, exposures to pregnant women (Adult Females 13-49) at this residue level are 413 times safe levels.33 The frequency and magnitude of chlorpyrifos residues found on highly consumed commodities demonstrates the threat to the safety of the food supply.

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30 Herrick et al., supra note 2.
32 Calculation = 0.001 ug chlorpyrifos/g apples X 182 g apples/day = 0.182 ug chlorpyrifos / 15 kg = 0.012 ug/kg-day which is 700% of the ssPAD only considering exposure from apples.
33 Calculation = 0.38 ug chlorpyrifos/g peaches X 95.16 g peaches/day = 36.16 ug chlorpyrifos /72.9 kg = 0.496 ug/kg-day which is 41,300% of the ssPAD for Adult Females.
B. Drinking Water Exposures Present Risks of Concern

In its 2014 drinking water assessment, EPA found that many label uses of chlorpyrifos resulted in drinking water contamination levels that exceeded EPA’s levels of concern. 80 Fed. Reg. 69,079, 69,083 (Nov. 6, 2015). Total dietary exposure to a pesticide is usually assessed by taking into account combined exposures through food and water. Because in the 2016 RHHRA food exposure alone exceeded target risk levels, any presence of chlorpyrifos in water is unsafe.

EPA finalized a refined drinking water assessment for chlorpyrifos in April 2016, which served to “combine, update and complete the work presented in the 2011 and 2014 drinking water assessments...” 2016 Chlorpyrifos Refined Drinking Water Risk Assessment for Registration Review (“2016 DWA”) at 6.34 The 2016 drinking water assessment results were consistent with the previous assessments and suggested “potential exposure to chlorpyrifos or chlorpyrifos-oxon in finished drinking [sic] based on currently labeled uses.” Id. Unsurprisingly, higher concentrations of chlorpyrifos and the more potent chlorpyrifos-oxon are likely to be found in areas with higher chlorpyrifos use and areas that are more vulnerable to runoff. Id. at 7. Thus, agricultural communities, including farmworkers and their families, are more likely to have their drinking water contaminated by chlorpyrifos. EPA’s revised assessment did not result in any changes to its finding that “the majority of estimated drinking water exposures from currently registered uses, including water exposures from non-food uses, continue to exceed safe levels even taking into account more refined drinking water exposures.” 81 Fed. Reg. 81,049, 81,050 (Nov. 17, 2016).

It was not possible for EPA to calculate a drinking water level of concern because food exposures alone exceeded risks of concern. However, if one assumed that there are no food exposures to chlorpyrifos, the “no food” drinking water level of concern for infants would be 0.014 ppb (ug/L). RHHRA at 24. In the 2016 Refined Drinking Water Assessment, EPA performed additional analysis to assess potential chlorpyrifos drinking water exposures based on national modeling, regional modeling and monitoring data. All three analyses showed that drinking water concentrations across the country exceed the “no food” drinking water level of concern.

The national-level assessment included both agricultural and non-agricultural (golf course) scenarios. As shown in Table 3 below, surface water sourced estimated drinking water concentrations of chlorpyrifos far exceed the “no food” drinking water level of concern for both the low-end and high-end scenarios by 50 to 12,000-fold.

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34 The 2016 Chlorpyrifos Refined Drinking Water Risk Assessment for Registration Review can be found under docket number EPA-HQ-OPP-2015-0653-0437.
Table 3. Comparison of EPA’s national-level estimated chlorpyrifos drinking water concentrations\textsuperscript{35} to the “no food” drinking water level of concern.

<table>
<thead>
<tr>
<th></th>
<th>1-in-10-year concentration (ug/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absolute Peak (ug/L)</td>
</tr>
<tr>
<td>High end scenario (Michigan tart cherries)</td>
<td>172</td>
</tr>
<tr>
<td>Exceedance of “no food” drinking water level of concern</td>
<td>12,286</td>
</tr>
<tr>
<td>Low end scenario (Georgia bulb onions)</td>
<td>8.5</td>
</tr>
<tr>
<td>Exceedance of “no food” drinking water level of concern</td>
<td>607</td>
</tr>
</tbody>
</table>

EPA also completed a regional analysis of all 21 HUC-02 regions in the United States. EPA considers this analysis highly refined and included scenarios to represent agricultural (food and non-food such as Christmas trees), non-agricultural (i.e., golf courses), impervious surface and urban uses. EPA used regionally-specific model inputs, including representative meteorological data from weather stations and application scenarios appropriate to each region.

The regional analysis indicates that all 24 hour and 21-day average estimated concentrations exceed the “no food” drinking water level of concern for all scenarios by 15 - 87,000 fold.\textsuperscript{36} EPA’s sensitivity analysis indicated that varying standard model inputs would not be expected to change these conclusions. EPA also considered all available water monitoring data. As shown in Table 4 below, bias-factor adjusted chlorpyrifos water concentrations exceed the “no food” drinking water level of concern by 7- 10,500 fold.

\textsuperscript{35} From Table 1 of the 2016 Chlorpyrifos Refined Drinking Water Risk Assessment for Registration Review (“2016 DWA”) at 7.

\textsuperscript{36} Comparison of “food only” drinking water level of concern (0.014 ppb (ug/L) to values in Table 2 of the 2016 DWA at 8-9.
Table 4. Comparison of EPA’s bias-factor adjusted estimated chlorpyrifos water concentrations\textsuperscript{37} to the “no food” drinking water level of concern.

<table>
<thead>
<tr>
<th></th>
<th>Highest measured concentration (ug/L)</th>
<th>Most frequently detected concentrations (ug/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unfiltered</td>
<td>Filtered</td>
</tr>
<tr>
<td>Exceedance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>of “no food”</td>
<td></td>
<td></td>
</tr>
<tr>
<td>drinking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>water level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>of concern</td>
<td>10,500</td>
<td>4,007</td>
</tr>
</tbody>
</table>

EPA found that the concentrations of chlorpyrifos in water obtained from their modeling analysis corresponded to monitoring data within an order of magnitude, indicating that the models are not overly conservative. In summary, EPA’s modeling and monitoring data analysis found that chlorpyrifos drinking water contamination is likely and that such contamination is unsafe.

C. EPA Must Protect Agricultural Communities From Toxic Drift and Other Bystander Exposures

People living in agricultural communities are at particular risk from chlorpyrifos spray drift, especially children who are exposed to drift near their schools and day cares, in their homes, and at playgrounds. Spray buffers are currently in place for chlorpyrifos, but those buffers are far too small to protect people from drift. See RHHRA at 30-1. EPA found unsafe levels of chlorpyrifos from the field’s edge to distances of more than 300 feet from where the pesticide is sprayed. Id. at 31. As with drinking water contamination, farmworkers and their families are disproportionately exposed to toxic chlorpyrifos drift – they are, quite literally, getting hit from all sides. The risks presented by spray drift weigh in favor of a ban on chlorpyrifos, as all uses lead to risks of concern and necessitate buffers in excess of 300 feet.\textsuperscript{38}

1. 300 feet buffers do not protect children and pregnant women from unsafe exposures.

EPA analyzed spray drift exposures for adults (dermal only) and children (dermal and incidental oral) resulting from different application methods, on different crop types, and differing application rates at the edge of the field and up to 300 feet away. At the farthest distance evaluated (300 feet from the field), almost all application scenarios resulted in significant risk. When aggregate exposures are considered factoring in inhalation and dietary

\textsuperscript{37}From Table 3 of the 2016 DWA at 10.

\textsuperscript{38}Indeed, it is unclear how large buffers would actually need to be to adequately protect children because the spray drift modeling does not go beyond 300 feet.
exposures, none of the application scenarios meet the safety standard for bystander exposures. Even at the lowest application rates, aerial and groundboom applications result in estimated exposures for children at 300 feet from the field that are extremely worrisome with all of the margins of exposure (“MOEs”) at 10 or below.\textsuperscript{39} Given these low MOEs, it would likely require buffer zones much larger than 300 feet to lower exposures to meet the safety standard.

Moreover, these estimates likely do not capture the high-end of the exposure distribution since they are based on an exposure duration of only 1.5 hours per day and do not include inhalation exposures. Given the proximity of homes, schools, parks and playgrounds to fields where chlorpyrifos is applied, there are opportunities for exposure that extend beyond 1.5 hours.

2. Chlorpyrifos levels measured in the air in agricultural communities pose a risk to children and pregnant women.

By evaluating inhalation exposures from chlorpyrifos drift in the 2016 RHHRA, EPA has filled an important exposure gap that was ignored in the 2014 HHRA. Evidence from the multiple air monitoring studies conducted in agricultural communities, summarized in the 2016 RHHRA, show that chlorpyrifos is regularly detected in the ambient air where children and pregnant women are exposed (e.g., in communities and at schools). In addition, research studies have shown that chlorpyrifos is found in the air at considerable distance from where it was applied and persists for multiple days – for example, one study found strong correlations with detections of chlorpyrifos in the air with applications made within 1.5 miles and up to 4 days prior to the sampling event.\textsuperscript{40} This is consistent with previous analysis finding that chlorpyrifos detections and air concentrations are correlated with amount of use within a 5 mile (8 km) area around the monitoring site.\textsuperscript{41} EPA’s evaluation of these studies to consider inhalation exposures is critical to understanding exposures in agricultural communities and should be relied upon.

Even in the absence of comprehensive modeling of volatilization and transport from treated fields under different atmospheric conditions, the ambient monitoring data illustrates that real-world exposures in agricultural communities do not meet the safety standard due to inhalation exposure alone. When aggregate dietary and spray drift exposures are also considered, the risk faced in these communities is staggering. For example, the Shafter Air Monitoring Site is located at a school in close proximity to almond orchards where chlorpyrifos is used. The most recent published data available (2015) from the California Department of Pesticide Regulation (“DPR”) showed that chlorpyrifos was detect in nearly two-thirds (61%) of the samples taken at this site.\textsuperscript{42} In 2014, the closest field application site was 0.3 miles from the

\textsuperscript{39} The margin of exposure in these scenarios must be above 100 to not be of concern.


monitoring site, and a total of 13,837 pounds of chlorpyrifos were used within 5 miles of the monitoring site. EPA’s evaluation of the monitoring data from this air monitoring site found both acute and steady-state risks of concern with MOEs below 10. For children attending this school and living nearby, the inhalation exposures are compounded with the potential for spray drift and dietary exposure. In addition, DPR’s recent review of the Air Monitoring Network found that almost 30 communities in California were at greater risk of organophosphate drift than Shafter due to the quantity of pesticides applied within 5 miles and meteorological conditions. Given that chlorpyrifos is the dominant organophosphate applied in California fields, it is clear that the inhalation risk EPA found for children and pregnant women at the Shafter site is likely much greater for other communities around the state.

The peak values recorded in all 11 air monitoring data sets result in acute inhalation exposure that do not meet the safety standard for children, and the vast majority do not meet the safety standard for pregnant women. It is clear from this analysis that the levels of chlorpyrifos routinely measured in the air in agricultural communities pose a significant threat to public health.

3. **Bystander exposures for children are likely significantly higher than estimates in the 2016 RHHRA due to indoor dust exposures.**

The exposure assessment ignored the substantial evidence that chlorpyrifos in indoor dust represents a potentially significant contributor to the total exposure experienced in agricultural communities. Based on exposure models for children 3-5 years of age, dust ingestion was the primary route of exposure to chlorpyrifos among farmworkers’ children from an agricultural community in California.

This exposure pathway has been identified in numerous studies conducted in California as well as in Washington State. Although chlorpyrifos breaks down readily when exposed to sunlight and moisture in the outdoor environment, it is known to persist in the indoor environment. Therefore, spray drift deposition that is entrained in dust and blows inside, or is

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brought indoors by workers who take it home on their clothes and boots, can represent a critical route of exposure, particularly for young children.

In Washington State, several studies have documented evidence supporting the take-home pathway. In one study, chlorpyrifos house dust concentrations were found to be elevated in agricultural (farmworker) family homes located more than ¼ mile from farmland, and chlorpyrifos residues were detected on parents’ work boots and children’s hands for many of the agricultural families.47 In this study, common practices among workers likely contributed to pesticide concentrations in dust because most workers did not change out of work clothes or boots before leaving the workplace and stored work clothes and boots at home. More than 2/3 of workers did not have laundry facilities in their homes and most wore both work clothes and work boots into their homes. In another study in Washington State, chlorpyrifos residues were found on the hands and toys of children living in agricultural communities, and chlorpyrifos was found in half of the indoor air samples taken.48

D. A Ban on Chlorpyrifos is Necessary to Protect Workers.

Concerning risks to workers, EPA found that even with maximum levels of personal protective equipment or engineering controls, all agricultural occupational handler scenarios, primary seed treatment handler scenarios, and secondary seed treatment scenarios expose workers to unsafe levels of chlorpyrifos. RHHRA at 36-7. Indeed, the harm faced by occupational handlers is perhaps understated by simply referring to the exposures as unsafe given that, in all agricultural scenarios, the level of concern is exceeded by several orders of magnitude. See id., Appendix E, Chlorpyrifos Occupational Handler Risk Estimates. The margin of exposure in these scenarios must be more than 100 to not be of concern, and in the airblast applicator scenario for California and Arizona citrus, for example, the combined (dermal and inhalation) margin of exposure is 0.0092. Moreover, even though current labels allow workers to re-enter the fields within 1-5 days after pesticide spraying, EPA found that, on average, re-entry intervals of at least 18 days were needed to protect workers from risks of concern. RHHRA at 38. Because there are no scenarios in which chlorpyrifos can be safely handled, a ban on the pesticide is the only way to protect workers.

III. THE TOLERANCE REVOCATIONS AND CANCELLATIONS SHOULD BE EFFECTIVE WITHIN MONTHS OF THE DETERMINATION BECAUSE OF THE IRREPARABLE HARM FROM UNSAFE CHLORPYRIFOS EXPOSURES

EPA must act quickly to revoke all tolerances for chlorpyrifos based on its findings of woefully unsafe exposures from food, drinking water, spray drift, and occupational activities. While the 2016 RHHRA more accurately illustrates the risks presented by chlorpyrifos for reasons stated above, it is worth noting that EPA initially proposed revocation of all chlorpyrifos food tolerances based on its conclusions from the 2014 Chlorpyrifos RHHRA, which used the under-protective regulatory endpoint of 10% cholinesterase inhibition. See 80 Fed. Reg. at


69,081 (EPA was “unable to conclude that the risk from aggregate exposure from the use of chlorpyrifos meets the safety standard of the Federal Food, Drug, and Cosmetic Act (FFDCA)”). The 2016 RHHRA serves to reinforce EPA’s previous conclusion that uses of chlorpyrifos do not meet the FFDCA/FQPA safety standard. A ban on all food uses of chlorpyrifos must necessarily follow. Furthermore, the grave risks associated with chlorpyrifos exposure offer strong support for an effective date not more than six months from the date of the revocation determination.

Since October 2015, when EPA proposed revoking all food tolerances for chlorpyrifos, the European Union agreed upon new, more protective endpoints following an updated toxicological review of chlorpyrifos. As a result of these new endpoints, the United Kingdom banned all but one use of chlorpyrifos and took swift action to protect its citizens from the pesticide. The United Kingdom announced its ban in February 2016, and uses of chlorpyrifos, including those of existing stocks, had to end by April 2016. Because no safe uses have been identified since EPA proposed revocation over a year ago, EPA should act with similar haste to ban all uses of chlorpyrifos and protect people, particularly children and farm workers, from irreparable harm.

A. A Ban is Necessary to Protect Children from the Developmental Delays and Learning Disabilities Correlated with Chlorpyrifos Exposure

The 2016 RHHRA appropriately used a regulatory endpoint based on neurodevelopmental harms associated with in utero chlorpyrifos exposure. The types of neurodevelopmental impacts correlated with exposure to chlorpyrifos and other organophosphates are every parent’s nightmare. Every parent watches with wonder as their children start to crawl and walk, yet chlorpyrifos has delayed motor development. Parents marvel as their children start to learn, yet chlorpyrifos reduces working memory and IQ.

Chlorpyrifos is also associated with learning disabilities like attention deficit disorders that seem to be reaching epidemic proportions. These types of learning disabilities frustrate and impair the child’s growth and well-being, and necessitate substantial societal investments in education, accommodations, and behavior management. Individual and societal harms have been well-studied and even quantified in connection with chemicals like lead, and federal agencies, including EPA, have found regulation to prevent exposures to such chemicals cost-effective.

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51 See, e.g., Declaration of Philip J. Landrigan, ¶¶ 33-6 (Attachment 2).
B. EPA Must Take Quick Action to Protect Farmworkers and Their Families Who are Disproportionately Harmed by Chlorpyrifos Exposure

EPA has recognized that pesticides disproportionately cause harm to farmworkers and their families, who are predominantly poor and majority Latino. 79 Fed. Reg. 15,452 (March 19, 2014). Farmworkers frequently experience acute poisoning from chlorpyrifos exposure. While there is no nationwide reporting system for pesticide poisoning incidents, every year the California and Washington incident reporting systems are filled with reports of worker poisonings from chlorpyrifos. Moreover, poisoning incidents are underreported due to fear of retaliation, reluctance to seek medical care, misdiagnoses, and other disincentives to report.52 These poisonings take their toll. Workers describe the onset of severe headaches and body-wrenching flu symptoms that sometimes lead to seizures, blackouts, and worse. Many workers report heightened sensitivities to pesticide illnesses that persist, and some have long-lasting neurological impacts. When workers become sick, there are societal costs as well. Workers often become unproductive, miss work, or need to seek medical care, which may be covered by workers’ compensation, other public health systems, or at the workers’ expense.

Not only are farmworkers exposed to undue risk of chlorpyrifos poisoning on the job, they and their families are more likely to be harmed by toxic pesticide drift and drinking water contamination in the places where they live. For instance, air monitoring conducted in 2004 and 2005 in the agricultural community of Lindsay, California, found chlorpyrifos in the air at levels far exceeding the level of concern for children even when using the prior, less protective endpoint.53 As to drinking water contamination, EPA’s 2016 drinking water assessment noted that higher concentrations of chlorpyrifos and chlorpyrifos-oxon are likely to be found in areas with higher chlorpyrifos use, such as agricultural communities. See DWA at 7. Executive Order 12898 on environmental justice requires EPA to identify and take steps to prevent these kinds of disproportionate pollution burdens. Exec. Order No. 12,898, 59 Fed. Reg. 7629 (Feb. 16, 1994). Thus far, EPA has failed to meet this requirement, leaving farm workers and their families grossly underprotected from a toxic pesticide that causes, among other serious harms, permanent brain damage in children. EPA must act quickly to alleviate this burden, which can only be accomplished by a swift ban on all food uses of chlorpyrifos.

CONCLUSION

EPA must act expeditiously to revoke all food tolerances and cancel all food uses for chlorpyrifos. Even when using the wrong endpoint of 10% cholinesterase inhibition, EPA found that aggregate exposures to chlorpyrifos did not meet the FQPA safety standard and proposed revocation of all food tolerances. Using the appropriate endpoint of neurodevelopmental effects, EPA found that food exposures alone exceed safe levels, especially for young children. A ban on all food uses of chlorpyrifos is the only defensible next step, and an effective date of not more

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52 EPA has acknowledged the underreporting of pesticide poisoning incidents and assumes that only 25% of acute incidents are reported. Worker Protection Standard Revisions, 79 Fed. Reg. 15,444, 15,453, 15,459 (Mar. 19, 2014).

53 Katherine Mills and Susan Kegley, Pesticide Action Network North America, “Air Monitoring for Chlorpyrifos in Lindsay, California” (July 14, 2006).
than six months from the date of the revocation determination is necessary based on the grave risk of harm, particularly to farmworkers and their families.

Respectfully submitted,

[Signature]

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