

# Farmworker and Conservation Comments on Chlorpyrifos Revised Human Health Risk Assessment

Earthjustice

Farmworker Justice

Natural Resources Defense Council

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Farm Labor Organizing Committee

Pineros y Campesinos Unidos del Noroeste

United Farm Workers

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## INTRODUCTION

On December 29, 2014, the Environmental Protection Agency (EPA) released its revised human health risk assessment (RHRA) for public comment. The assessment marks the culmination of the agency's review of the risks posed to people from chlorpyrifos. EPA will rely on this assessment when it decides whether to ban chlorpyrifos or modify its registrations and tolerances to protect people, particularly workers, communities, and children. These comments are submitted on behalf of Earthjustice, Natural Resources Defense Council (NRDC), Pesticide Action Network (PAN), Farmworker Justice, United Farm Workers (UFW), Pineros y Campesinos Unidos del Noroeste (PCUN), Farm Labor Organizing Committee, and California Rural Legal Assistance Foundation.

## EXECUTIVE SUMMARY

Chlorpyrifos is a widely used pesticide first registered by EPA in 1965. Chlorpyrifos is an organophosphate pesticide, a class of pesticides developed as nerve agents in World War II and adapted for use as insecticides after the war. It should come as no surprise that a chemical developed as a nerve agent would have deleterious effects on people who come into contact with it when it is used as an insecticide.

Indeed, chlorpyrifos is one of the pesticides most often identified as the culprit when workers and bystanders suffer acute pesticide poisonings. Year after year, chlorpyrifos has been associated with an alarming number of pesticide poisonings, and in many states, it is regularly identified among the five pesticides linked to the highest number of pesticide poisoning incidents. This trend is particularly significant given widespread under-reporting of pesticide poisonings due to such factors as inadequate reporting systems, fear of retaliation from employers, and reluctance to seek medical treatment.

Chlorpyrifos is used on an extensive variety of crops, including fruit and nut trees, vegetables, wheat, alfalfa, and corn. In 2006-2012, chlorpyrifos was applied to more than half of the country's apple and broccoli crops, 45% of onion, 46% of walnut, and 41% of cauliflower crops.<sup>1</sup> Eight million pounds are used annually in agriculture, including one million pounds on both corn and soybeans.<sup>2</sup> It is also used on golf courses, for adult mosquito abatement, in greenhouses, and for seed treatments. Its widespread use has led to exposures to people through the air, in water, and through the foods they eat. Workers are exposed when they handle pesticides and when they re-enter treated fields. Workers, as well as children and other bystanders, are exposed to chlorpyrifos through drift and volatilization.

When EPA purported to bring chlorpyrifos into compliance with the Food Quality Protection Act (FQPA) in 2002 and 2006, it identified a level of 10% cholinesterase inhibition as the endpoint it would use to determine whether chlorpyrifos exposures violate the regulatory standards. Chlorpyrifos and other organophosphates suppress the activity of an enzyme called cholinesterase, which degrades a substance that regulates nerve impulses throughout the body. When cholinesterase activity is inhibited, nerves are over-stimulated, causing people to

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<sup>1</sup> EPA, Chlorpyrifos Evaluation of the Potential Risks from Spray Drift and the Impact of Potential Risk Reduction Measures at 7 & Appendix C (July 13, 2012).

<sup>2</sup> *Id.*

experience symptoms analogous to a serious flu: headaches, nausea, dizziness, tremors, difficulty breathing, vomiting, diarrhea, and sometimes convulsions, respiratory paralysis and even death in extreme cases. Cholinesterase activity can be measured through blood tests.

In assessing risks from chlorpyrifos under the FQPA, which required aggregating all exposures to the pesticide, EPA determined that home uses had to be cancelled. Children crawling on treated carpets and hugging pets after flea treatments were exposed to dangerous levels. Seeing the writing on the wall, the chemical makers agreed to cancel homeowner uses of chlorpyrifos in 2000. EPA, however, neglected children in farmworker communities, who are primarily Latino and poor, creating a double standard and ignoring substantial environmental justice concerns. EPA never assessed the extent to which children in agricultural communities are exposed to chlorpyrifos through drift and residues parents take home on their clothes.

EPA also did not account for a growing body of peer-reviewed scientific studies of real-world exposures to pregnant women that have associated chlorpyrifos with loss of IQ, developmental delays, and other neurodevelopmental impacts in children exposed *in utero* to doses much lower than those that cause 10% cholinesterase inhibition in adults. Regulating chlorpyrifos based on exposures that trigger 10% cholinesterase inhibition in adults would not protect children from these neurodevelopmental impairments.

PAN, NRDC, and others commented on EPA's 2001 chlorpyrifos re-registration, urging EPA to address pesticide drift and the mounting evidence of brain and other neurodevelopmental impacts. When EPA finalized its re-registration of chlorpyrifos and other organophosphates in 2006 without protecting children from drift or neurodevelopmental impairments, PAN and NRDC filed a petition presenting extensive evidence of harmful exposures from drift and volatilization, and peer-reviewed studies showing neurodevelopmental impacts. EPA's chlorpyrifos RHHRA will guide EPA's response to the petition to ban chlorpyrifos and its decision with respect to chlorpyrifos in registration review, a statutorily mandated process for ensuring that pesticides already in use comply with regulatory standards based on the most current scientific evidence and legal standards.

### **EPA Finds Chlorpyrifos CAUSES Brain and Other Neurodevelopmental Impairments at Lower Doses Than EPA's Regulatory Limit.**

EPA has finally acknowledged overwhelming evidence from experimental toxicology studies, mechanistic and epidemiological studies that reveal brain and other neurodevelopmental impacts to kids from *in utero* exposures. And EPA has found that the neurodevelopmental impacts occur at a lower dose to adult pregnant women than the dose that causes 10% cholinesterase inhibition. These findings are fully supported by the scientific record and extensive independent review of the scientific studies.

Based on its findings, EPA has retained a tenfold FQPA safety factor for children. When EPA identifies a dose that will avoid causing adverse effects, it adds safety factors to prevent exposing people to adverse effects. The FQPA directed EPA to add an additional safety factor to protect children from harm in the face of data deficiencies or evidence of prenatal toxicity. Despite lobbying from Dow AgroSciences to eliminate the FQPA safety factor, EPA appropriately retained an FQPA safety factor due to the gaps in data and understanding of the mechanism and precise doses at which the neurodevelopmental impacts to children occur.

Despite finding that children experiences neurodevelopmental damage from *in utero* doses smaller than those that cause 10% cholinesterase inhibition, EPA persists in using 10% cholinesterase inhibition as its regulatory endpoint in the RHHRA. In doing so, EPA is violating its policy of choosing the most sensitive endpoint in assessing risk and making registration and tolerance decisions. Given EPA's findings that the peer-reviewed scientific evidence shows that children may suffer serious neurodevelopmental damage at lower doses, this is indefensible.

### **EPA Has Improperly Relied on a Model Developed By Dow, Based on Unethical and Scientifically Flawed Human Testing, to Eliminate Standard Safety Precautions**

EPA used a model developed by Dow AgroSciences to try to pinpoint the exposures that will produce 10% cholinesterase inhibition in people. The rationale for the model is that 10% cholinesterase inhibition from chlorpyrifos can be predicted with precision. Dow has developed a model that tries to predict when cholinesterase activity will be suppressed based on body weight, metabolism, and other factors. For years, Dow has been urging EPA to use its model as a basis for eliminating standard safety factors routinely used in risk assessment to account for uncertainty. A tenfold safety factor is used to reflect the uncertainty in extrapolating from animal studies (the inter-species safety factor), and another tenfold safety factor is used to account for variations among human populations because people have different susceptibilities based on their age, developmental life stage, genetics, health conditions, diet, and exposures to other chemicals or hazards (the intra-species safety factor). These safety factors have been standard practice for decades, and the FQPA reinforced and built on them when it established an additional default safety factor to protect children.

In the RHHRA, EPA eliminated the inter-species safety factor altogether, and it shrunk the intra-species safety factor from 10X to 4X-5X for children, although it retained a 10X for women since the Dow model lacks data reflecting how a pregnant woman's body processes chlorpyrifos. What EPA gave with one hand by retaining the 10X FQPA safety factor, it took away with the other, and then some. The result – EPA will allow chlorpyrifos exposures to be an order of magnitude higher for pregnant women and even higher still for children.

It is unconscionable for EPA to use Dow's model to eliminate or reduce the safety factors in light of the neurodevelopmental effects that occur at lower doses than those used in the model. This approach violates EPA's policy of setting regulatory limits based on the most sensitive endpoint at which adverse effects occur and disregards EPA's mandates to protect children from prenatal toxicity.

EPA relied on the model despite its numerous scientific flaws. In February 2011, EPA's Scientific Advisory Panel found numerous flaws in the model, using terms like "problematic," " cursory," "overstated," "inadequate," "inaccurate," "imprecise," and "incomplete." Dow made some changes in the model, but EPA did not obtain another review by its Scientific Advisory Panel.

EPA also erred in relying on the model because the model is based on ethically and scientifically deficient studies. Congress has required that human testing must meet minimal ethical and scientific standards before EPA can rely on such tests. An EPA ethics advisor found that the key Dow human study fell short of meeting informed consent requirements, and EPA's Human Studies Review Board found the study scientifically deficient in two respects that have

not been corrected. EPA has since strengthened its regulatory standards governing use of intentional human dosing studies, yet EPA failed to resubmit the study to the Human Studies Review Board. EPA has provided no credible basis for relying on human testing without subjecting it to such scrutiny and without confronting the earlier findings of ethical and scientific shortcomings.

### **EPA Fails to Protect People, Including Children, from Pesticide Drift and Volatilization.**

Under the FQPA, EPA must protect children and other bystanders from all aggregate exposures, including drift. It did not do so when it re-registered chlorpyrifos and all of the other old pesticides by the FQPA's 2006 deadline. It is now acknowledging its legal obligation.

EPA made findings that drift exposes children to harmful chlorpyrifos exposures, and the chemical companies agreed to no-spray buffers around school grounds, play fields, residential yards, homes, daycares, nursing homes, hospitals, and pedestrian sidewalks. Creating no-spray buffers around these sensitive sites is an important step forward.

For chlorpyrifos, however, the no-spray buffers are far too small. For ground and airblast spraying, the buffers are only ten feet wide, except for very high application rates using airblast spraying where the buffers are 25-50 feet wide. For aerial spraying, the buffers range from 10-100 feet depending on the application rate and droplet size. By way of comparison, the chlorpyrifos buffers established for water bodies range from 25 feet for ground boom, 50 feet for airblast, and 150 feet for aerial spraying.

These buffers fail to protect children and other bystanders:

- **The Buffers Do Not Guard Against Neurodevelopmental Harm**  
EPA is using 10% cholinesterase inhibition in assessing whether children and other bystanders will be harmed, not the lower doses that cause neurodevelopmental impairments.
- **EPA Under-estimates Drift Exposures.**  
Children and other bystanders are exposed to pesticides when droplets, particles or vapors move offsite. EPA focused primarily on children's exposures to residues that have deposited on the ground. While EPA considered inhalation exposures from aerial applications, it did not aggregate deposited residues and inhalation exposures, and it failed entirely to account for inhalation exposures from air blast or ground boom applications. It assumed that children and other bystanders would only be exposed for 2 hours even though many people, such as the home bound and children who are exposed at school and at home, will be exposed for far longer. EPA also made assumptions the unduly minimize the amount of pesticide residues children will encounter.
- **EPA Ignored Children's Exposure to Chlorpyrifos Through Dust.**
- **EPA is Allowing Volatilization Exposures Based on Flawed Industry Studies.**  
Chlorpyrifos has a propensity to volatilize after applications and move large distances as vapors. When EPA started to look at exposures to chlorpyrifos

through volatilization, it determined that buffers as large as 4000 feet may be necessary to prevent harm exposures to chlorpyrifos vapors. Dow then submitted two rat studies that purport to show that it is impossible to inhale enough chlorpyrifos to produce an adverse effect. The studies have significant flaws because they fail to reflect temperature and soil moisture impacts on volatilization, individual variation, and biomonitoring and incident data showing harmful exposures at distances as large as ½ mile from application sites. And continuing the pattern, EPA used the 10% cholinesterase inhibition limit as an endpoint, rather than the lower doses that cause brain impairments in children.

### **EPA Found Infants and Others Are Exposed to Dangerous Levels of Chlorpyrifos in Drinking Water, But Fails to Spell Out Meaningful Actions to Prevent Those Exposures.**

For drinking water, even using the reduced safety factors, EPA estimated that drinking water concentrations would exceed levels of concern for many uses of chlorpyrifos. Given the amount of water that infants drink, they are particularly at risk. EPA tries to minimize the risks by indicating that it may not be uniform across the country, but may be more pronounced in small watersheds where a high percentage of the crops are treated with chlorpyrifos. EPA has proposed no measures to prevent these exposures. EPA should cancel uses of chlorpyrifos that contaminate drinking water and put children at risk.

### **EPA Needs to Cancel the Many Uses That it Finds Expose Workers to Unsafe Levels of Chlorpyrifos, but These Uses Are Only the Tip of the Iceberg Because EPA Has Grossly Under-Estimated the Risks to Workers.**

For workers, EPA finds that many uses of chlorpyrifos will expose workers to harmful levels of chlorpyrifos, even using reduced safety factors based on the Dow model, ignoring direct drift and volatilization exposure studies, and accounting only for cholinesterase inhibition, not neurodevelopmental damage to children of female workers. Even with maximum protective clothing, EPA finds that unacceptable risks would remain for 126 exposure scenarios, 32 seed treatment scenarios, and greenhouse workers. EPA should immediately eliminate these uses of chlorpyrifos. EPA also finds that many re-entry intervals may need to be doubled to prevent unsafe exposures to field workers performing tasks like hand harvesting and thinning. EPA's findings demonstrate that chlorpyrifos cannot be used in a way that is safe for workers, and support our call for a full ban on the pesticide.

EPA's worker risk assessment grossly under-estimates the risks because it ignores direct drift. Every year, workers are poisoned by chlorpyrifos when it moves offsite onto neighboring fields. While pesticide poisonings are notoriously under-reported, chlorpyrifos is consistently one of the pesticides associated with a large number of pesticide poisonings. A substantial portion of these poisonings occur when people inhale chlorpyrifos droplets, particles or vapors that have drifted offsite. Because pesticide labels already prohibit spraying pesticides directly on people, EPA ignores these documented poisonings, simply assuming they don't happen despite the evidence to the contrary. Instead, EPA looks only at drift that deposits in a field and exposures to the pesticide residues from touching the treated crop at a later time. This approach captures only a small fraction of the harm from pesticide drift. EPA cannot turn a blind eye to the reality that the general label statement prohibiting drift onto people is inadequate to prevent drift-induced poisonings and neurodevelopmental effects.

EPA also under-estimates risks to workers because it makes assumptions that are at odds with the real-life circumstances of the workers, such as how many hours workers are exposed in a day and the average body weight of female workers. EPA also over-estimates the efficacy of protective clothing and engineering controls in the face of evidence that significant exposures remain. EPA must re-assess worker risks and protect workers against the real risks they face in the fields.

**EPA Has Failed to Protect Workers and their Families Against Disproportionate Harm from Pesticide Use In Violation of Environmental Justice Directives.**

Pesticides disproportionately cause harm to farmworkers and their families, who are predominantly poor and majority Latino. Farmworkers are exposed to far greater risks of poisonings on the job than industrial workers, and they and their families bear the brunt of poisonings from pesticide drift and volatilization. In addition, farmworker and poor communities may be more likely to obtain their drinking water from systems that have been contaminated by chlorpyrifos. The Environmental Justice Executive Order requires EPA to identify and take steps to prevent such disproportionate pollution burdens, but EPA failed to comply with these obligations in the RHHRA.

## I. LEGAL AND REGULATORY BACKGROUND

Before addressing each of these types of exposures and adverse effects, these comments summarize the legal obligations and structure underlying the registration review and EPA's obligations; how EPA addressed the bystander exposures and scientific evidence of neurodevelopment impacts in its earlier re-registration determinations for chlorpyrifos; and the petitions and lawsuits that have been trying to correct EPA's past failures to protect children and adults from harmful chlorpyrifos exposures.

### A. The Overlapping Statutes Regulating Pesticide Use

EPA regulates pesticides under two, overlapping statutes, the Federal Food, Drug and Cosmetic Act (FFDCA) and Federal Insecticide, Rodenticide and Fungicide Act (FIFRA). EPA issues tolerances under the FFDCA, which establish the maximum residue of a pesticide allowed on food. 21 U.S.C. § 346a(b) & (c). EPA may “establish or leave in effect a tolerance for a pesticide chemical residue in or on a food only if the Administrator determines that the tolerance is safe.” *Id.* § 346a(b)(2)(A)(i). EPA has the authority to revoke a tolerance if it finds a pesticide residue would not be safe. *Id.* § 346a(b)(2)(A)(i).

Under FIFRA, EPA must register a pesticide (with rare exceptions) before it may be sold or used in the United States. 7 U.S.C. § 136a(a). To register or re-register a pesticide, EPA must determine that its use “will not generally cause unreasonable adverse effects on the environment,” which includes risks to human health. *Id.* § 136a(c)(5)(D); *see id.* § 136(bb) (definition of “unreasonable adverse effects”). EPA has the authority to cancel a pesticide registration if the pesticide use “causes unreasonable adverse effects on the environment,” including human health. *Id.* § 136d(b).

Congress overhauled our food safety laws in 1996 when it unanimously passed the Food Quality Protection Act (FQPA, amending both the FFDCA and FIFRA). The overhaul responded to a seminal 1993 National Academy of Sciences (NAS) report criticizing EPA for treating children like “little adults” by failing to address the unique susceptibility of children to pesticide exposures based on the foods they eat, their play, metabolism, and sensitive stages of their development. National Research Council, *Pesticides: Diets of Infants and Children* (1993).

The NAS recommended that EPA revamp and strengthen its pesticide regulations to account for children's vulnerabilities, consumption patterns, and exposures. Because it would take time to fill gaps in knowledge, safeguards and methodologies, the NAS recommended that additional protection be afforded in the form of “uncertainty” or “safety factors.” The NAS first described how EPA has regularly used uncertainty factors and then proposed an additional uncertainty factor for fetal developmental toxicity and where data are incomplete:

In the absence of data to the contrary, there should be a presumption of greater toxicity to infants and children. To validate this presumption, the sensitivity of mature and immature individuals should be studied systematically to expand the current limited data base on relative sensitivity.

Heeding the NAS recommendations, the FQPA directs EPA to afford added protection to children based on their exposure patterns, their special sensitivities such as during early or

adolescent development, and gaps in available data to assess such risks. 21 U.S.C. § 346a(b)(2)(C)-(D). The statute explicitly requires EPA to assess the risk that a pesticide poses particularly to infants and children. 21 U.S.C. § 346a(b)(2)(C). Before EPA can establish a tolerance, the agency shall “ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure” to the pesticide, and shall “publish a specific determination regarding the safety of the pesticide chemical residue for infants and children.” *Id.* §§ 346a(b)(2)(C)(ii)(I) & (II). In ensuring that the statutory safety standard is met, EPA must consider available information concerning “the special susceptibility of infants and children,” including “neurological differences between infants and children and adults, and effects of in utero exposure to pesticide chemicals.” *Id.* § 346a(b)(2)(C)(i)(II). EPA must also base its tolerance decision on available information about “food consumption patterns unique to infants and children.” *Id.* §§ 346a(b)(2)(C)(i)(I) & (III).

One of the FQPA’s key provisions – incorporated into the FFDCA – is the requirement that EPA use an additional margin of safety to protect infants and children when establishing tolerances. The statute requires that: “an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure shall be applied for infants and children to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and toxicity to infants and children.” 21 U.S.C. § 346a(b)(2)(C). EPA can depart from this requirement and use a different margin of safety “only if, on the basis of reliable data, such margin will be safe for infants and children.” *Id.* (emphasis added).

In addition, because “[e]xposure to pesticide residues from ambient air sources is generally higher in areas close to agricultural lands,” and “[b]ecause infants and children are subject to nondietary sources of exposure to pesticides,” the NAS found that “it is important to consider total exposures to pesticides from all sources combined.” *Id.* at 307, 309, 319. The FQPA requires EPA to “ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure” to pesticides. 21 U.S.C. § 346a(b)(2)(C)(ii)(I), (II) (emphasis added). “Aggregate exposure” includes “all anticipated dietary exposures and all other exposures for which there is reliable information,” including pesticide drift exposures. 21 U.S.C. § 346a(b)(2)(A)(ii); *see also id.* § 346a(b)(2)(C)(vi). The FQPA, therefore, requires an assessment based on aggregation of all exposures to a pesticide whether from eating foods, drinking water with residues of the pesticide, or uses of the pesticide in and around the home or other places where people can be exposed. 21 U.S.C. § 346a(b)(2)(A)(ii), (C)(i)(I). The FQPA also requires EPA to assess and protect against unsafe risks posed by cumulative exposures to pesticides that share a “common mechanism of toxicity,” as is the case with pesticides in the organophosphate family. *See* 21 U.S.C. § 346a(b)(2)(C)-(D).

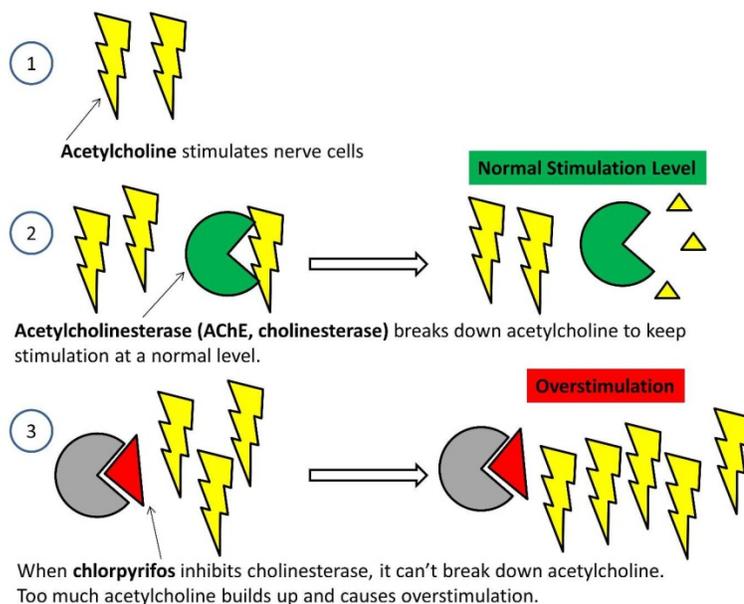
The FQPA also amended FIFRA’s “unreasonable adverse effects” definition to include “a human dietary risk from residues that result from a use of a pesticide in or on any food inconsistent with the [FQPA] standard.” 7 U.S.C. § 136(bb)(2). Accordingly, EPA can register or re-register a pesticide only if there is a reasonable certainty of no harm from aggregate and cumulative exposures to the pesticide under the FQPA standard.

Congress gave EPA a ten-year deadline, which ended in August 2006, to bring all food-use pesticides into compliance with these protective mandates. 21 U.S.C. § 346a(q)(1). The August 2006 deadline applied to both tolerances established under the FFDCA as amended by the FQPA and re-registration decisions under FIFRA.

B. EPA's 2001 and 2006 Chlorpyrifos Determinations Failed to Address Serious Health Impacts to Children and Other Bystanders.

In setting priorities for reviewing old pesticides under the FQPA, EPA gave priority to organophosphates because they are among the pesticides that “pose the greatest risk to public health.” 62 Fed. Reg. 42,020, 42,021 (Aug. 4, 1997). For chlorpyrifos, EPA issued an interim re-registration determination in 2001, and, based on a cumulative risk assessment for organophosphates, finalized its re-registrations for all organophosphates, including chlorpyrifos in 2006.

In addressing human health risks, it is EPA policy to select the most sensitive end point for use in risk assessments and to develop regulatory protections.<sup>3</sup> For chlorpyrifos and other organophosphates, EPA focused on cholinesterase inhibition, more specifically inhibition of the acetyl cholinesterase enzyme (AChE), which is the mechanism by which organophosphates cause acute poisonings.<sup>4</sup> The acetyl cholinesterase enzyme normally functions to break down the neurotransmitter acetyl choline (ACh), thus bringing to a healthy end its stimulatory effects. When the enzyme is inhibited or impaired from functioning – such as by interacting with chlorpyrifos – then ACh will over-stimulate its target nerve or muscle, causing symptoms that range from mild to severe, including severe respiratory distress, nausea and vomiting, abdominal pain, diarrhea, blurred vision, muscle cramping, weakness, tremors, confusion, seizures, and coma or death in extreme poisoning.



In its risk assessments for the re-registration of organophosphates, EPA established 10% red blood cell cholinesterase inhibition as its regulatory limit. It believed exposures that produce 10% cholinesterase inhibition would not cause acute poisonings or harmful chronic exposures to people.

<sup>3</sup> EPA OPP, Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment at 8 (2002).

<sup>4</sup> EPA OPP, Science Policy of the Use of Data on Cholinesterase Inhibition for Risk Assessments of Organophosphate and Carbamate Pesticides (Aug. 18, 2000); Preliminary HHRA at 7 (June 2011).

To comply with the FQPA, EPA conducted an aggregate exposure assessment for chlorpyrifos to add together all of the ways people, and particularly children, are exposed to the pesticide. EPA developed a “risk cup” approach that compares all of the exposures for specific population groups, such as fetuses, infants, and children in different age ranges, to what it finds to be unsafe exposure levels. If aggregate exposures to the pesticide “overflow” the risk cup for a particular subpopulation, the pesticide does not meet the FQPA safety standard. EPA must then reduce exposures to levels that no longer exceed what it has deemed to be safe levels by, for example, banning uses.

For chlorpyrifos, EPA found alarmingly high exposures to children from uses of chlorpyrifos in the home and on pets.<sup>5</sup> EPA modeled the exposures to children after homes or pets had been treated with chlorpyrifos – for example, from crawling on carpets or hugging their pets and found that children would be exposed to levels of chlorpyrifos that could harm their health. In 2000, EPA reached an agreement with the registrants to cancel home and garden uses of chlorpyrifos after determining that residential uses of these pesticides cause the child risk cup to overflow. Administrator Carol Browner heralded this agreement as “particularly good news for children, who are among the most vulnerable to the risks posed by pesticides.”<sup>6</sup>

Inexplicably, EPA failed to assess children’s exposures from chlorpyrifos spray drift and volatilization from agricultural sites to homes, schools, daycares, and playfields. By failing to assess the risks to children who are exposed to agricultural pesticide drift and volatilization, EPA maintained a double-standard: protecting kids from pesticides used in urban and residential settings, while leaving kids who live near agricultural sites—often in low-income and minority communities—unprotected and vulnerable to pesticide poisonings. This failure to protect farmworker and rural children fell short of the FQPA’s requirements and the direction in the federal environmental justice executive order to address disproportionate health risks to people of color and low-income populations. Exec. Order No. 12,898, §§ 1-101(b), 2-202(b), 59 Fed. Reg. 7,629 (Feb. 11, 1994) (requiring each federal agency to “ensure that its policies, programs, activities, and standards address disproportionate risks to children that result from environmental health or safety risks . . . that are attributable to products or substances that the child is likely to come in contact with or ingest (such as the air we breath [sic], the food we eat, the water we drink or use for recreation, the soil we live on, and the products we use or are exposed to).”). EPA also failed to give credence to a growing body of published scientific research linking exposure to chlorpyrifos *in utero* with long-term harmful human health effects, including neurodevelopmental disorders, hyperactivity, and reduced IQ.

In 2001, after negotiating the phase-out of residential uses, EPA issued an interim re-registration determination (IREDD) for chlorpyrifos, which allowed chlorpyrifos uses and exposures to continue, although some at reduced levels.<sup>7</sup> EPA also allowed uses to continue that expose workers to risks of concern from air blast and ground boom applications in open cabs, greenhouse applications, and entry of workers into the fields post-application to perform various tasks.

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<sup>5</sup> EPA, Occupational/Residential Handler and Post Application Residential Risk Assessment for Chlorpyrifos, at 5-7 (Oct. 1999).

<sup>6</sup> EPA Administrator Carol M. Browner, Dursban Announcement, Remarks Prepared for Delivery, at 1 (June 8, 2000).

<sup>7</sup> EPA, Interim Reregistration Eligibility Decision for Chlorpyrifos at 64-68 (Sept. 2001).

C. Petitions and Litigation to Obtain EPA Action on Evidence of Chlorpyrifos Health Risks.

PAN and NRDC commented on the 2001 IRED, but EPA never responded to these public comments. Others weighed in as well. For example, three government unions urged EPA to consider neurotoxic effects and drift exposures, to retain the full suite of safety factors, and to cancel uses that pose risks of concern to workers.<sup>8</sup> NRDC and PAN hoped that EPA would address the concerns raised in its IRED comments when it completed a cumulative risk assessment for all of the organophosphates. However, EPA made no such changes when it finalized that cumulative risk assessment in 2006, even though by that time, additional scientific studies and air monitoring confirmed the drift exposures and neurodevelopmental risks posed by chlorpyrifos.

Farmworker and health advocates then pursued three legal avenues challenging EPA's failure to protect children from the hazards posed by chlorpyrifos. First, UFW, PAN, PCUN, Sea Mar Community Health Center; Beyond Pesticides, Frente Indigena de Organizaciones Binacionales, and Farm Labor Organizing Committee filed a federal district court challenge to the 2001 chlorpyrifos interim re-registration decision, in part, for failing to protect children and other bystanders from pesticide drift and failing to cancel uses that expose workers to admittedly excessive poisoning risks.<sup>9</sup> The parties negotiated principles on which the case could be settled with an EPA commitment to make a new regulatory decision for chlorpyrifos by 2010 that would address drift exposures to children and other bystanders. However, after the Ninth Circuit ruled in a case of first impression that challenges to FIFRA registration determinations must be brought in the courts of appeals within 60 days of the decision, the settlement fell apart.<sup>10</sup>

Second, PAN, UFW, PCUN, Earthjustice, and Farmworker Justice joined other farmworker advocates in petitioning EPA to address pesticide drift as mandated by the FQPA.<sup>11</sup> The Kids' Petition highlighted EPA's violation of its legal duty to protect children from all aggregate exposures to each pesticide in tolerance and re-registration determinations and asked EPA to expedite adoption of mitigation for airborne routes of exposure to organophosphates and n-methyl carbamates, another nerve poisoning pesticide, because of the heightened poisoning risks posed by those classes of pesticides. In March 2014, EPA responded to the petition, acknowledging its legal obligation to address pesticide drift under the FQPA and FIFRA. However, EPA indicated it would not protect children from drift until it reviewed pesticide registrations and tolerance decisions as a matter of course in registration review, and it refused to impose interim protections.<sup>12</sup> The petitioners have filed administrative objections under the FQPA and a challenge in the Ninth Circuit to EPA's refusal to put any protections in place under

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<sup>8</sup> Letter from Nat'l Transp. Emps. Union et al. to Stephen Johnson, EPA Administrator (May 24, 2006) (Ex. 25).

<sup>9</sup> *UFW v. Administrator, EPA*, No. 07-3950-JF (N.D. Cal. filed Aug. 1, 2007).

<sup>10</sup> *UFW v. Administrator*, Stipulation of Voluntary Dismissal, Dkt. 98, No. 07-3950-JF (N.D. Cal. filed April 27, 2010); see *UFW v. Administrator, EPA*, 592 F.3d 1080 (9th Cir. 2010) (challenges to registration decisions must be brought in courts of appeals within 60 days, rather than in district court under a six-year statute of limitations as had previously been the case).

<sup>11</sup> See Pesticides In The Air – Kids At Risk: Petition to EPA to Protect Children From Pesticide Drift (October 13, 2009) (the "Kids' Petition").

<sup>12</sup> Agency Response to Pesticides In The Air – Kids At Risk: Petition to EPA to Protect Children From Pesticide Drift (2009), at 2, 32-33 (March 31, 2014) (EPA-HQ-OPP-2009-0825).

FIFRA, both of which are pending.<sup>13</sup>

Third, on September 12, 2007, PAN and NRDC submitted a petition asking EPA to ban chlorpyrifos based on the mounting evidence of risks from chlorpyrifos that were left unaddressed in EPA's 2001 and 2006 regulatory decisions. At its heart, the 2007 Petition raised two issues. The 2007 Petition raised EPA's failure to account for risks to children and bystanders from chlorpyrifos drift and volatilization, as required by the FQPA. In support of this obligation, the petition presented the California Air Resources Board's air monitoring reports and data, which documented concentrations above EPA's levels of concern near fields and in schoolyards, and community air monitoring, which showed widespread contamination in multiple locations and over a period of years, including in schoolyards.<sup>14</sup>

Moreover, the 2007 Chlorpyrifos Petition (at 4-16) compiled the mounting evidence documenting serious cognitive and behavioral effects from low-dose prenatal chlorpyrifos exposures. Peer-reviewed scientific studies have shown that children and infants exposed to chlorpyrifos can exhibit long-term neurological and neurodevelopmental difficulties from early life exposure. 2007 Chlorpyrifos Petition at 6-14. To investigate how pregnant women and their children are affected by chemicals, the federal government began funding a network of Centers for Children's Environmental Health and Disease Prevention Research at Columbia University (referred to as "CCCEH"), Mount Sinai School of Medicine (referred to as "Mt. Sinai"), and UC-Berkeley Center for Health Assessment of Mothers and Children (referred to as "CHAMACOS"). These centers have been conducting long-term birth-cohort studies that measure environmental exposures *in utero* and track the children throughout their childhood. Chlorpyrifos has been the subject of these studies with alarming results. For example, Columbia University scientists have documented decreases in birth weight, increased mental and motor delays, and increased problems related to attention and attention deficit hyperactivity disorder, reduced IQ, a decline in working memory, and delayed development in children exposed to chlorpyrifos *in utero*. *Id.* at 6-7. Scientists with Mount Sinai School of Medicine have correlated *in utero* exposure to chlorpyrifos with impaired cognitive ability. *Id.* at 7-8. These and other published studies provide strong evidence that prenatal exposure to chlorpyrifos is associated with long-lasting, and possibly permanent, impaired cognitive and behavioral development. *Id.* at 6-9, 11-13. Members of EPA's Scientific Advisory Panel expressed concern that EPA had failed to account for scientific evidence showing brain impacts from early life exposures to chlorpyrifos at lower doses than those used by EPA in its regulatory decisions. *Id.* at 13, 22-23.

In response to the petition and as part of the chlorpyrifos registration review, EPA has conducted various analyses, developed or refined models, solicited reviews from its Scientific Advisory Panel, and conducted a series of risk assessments. EPA has now compiled that work into its Revised Human Health Risk Assessment. EPA has represented that it will complete all health aspects of the chlorpyrifos registration review by the summer of 2015, although that timeline appears to be slipping. Given the severity of the risks posed by chlorpyrifos, its widespread use, and the lengthy delays in acting on the 2007 petition, adhering to this timeline is

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<sup>13</sup> UFW, *et al.*, Written Objections to EPA's Response to Pesticides in the Air – Kids at Risk: Petition to EPA to Protect Children From Pesticide Drift (May 28, 2014); *PAN v. U.S.E.P.A.*, No. 14-71514 (9th Cir.).

<sup>14</sup> Petition to Revoke All Tolerances and Cancel All Registrations for the Pesticide Chlorpyrifos at 17-21 (September 12, 2007), EPA-HQ-OPP-2007-1005.

imperative.

II. THE RHHRA FINDS THAT CHLORPYRIFOS CAUSES NEURODEVELOPMENTAL DAMAGE TO CHILDREN EXPOSED *IN UTERO* AND RETAINS A TENFOLD FQPA SAFETY FACTOR, BUT FAILED TO CONSIDER WHETHER A LARGER SAFETY FACTOR IS WARRANTED DUE TO THE DEMONSTRATED PRENATAL TOXICITY FROM EXPOSURES LOWER THAN EPA'S REGULATORY ENDPOINT.

The RHHRA reviews the extensive body of peer-reviewed scientific literature linking neurodevelopmental effects in children to chlorpyrifos exposures *in utero*. Based on the literature, two SAP reviews, and its own analysis, EPA finds that prenatal exposures result in adverse neurodevelopmental effects, including mental delays, attention problems, pervasive developmental disorders in early childhood, and intelligence decrements in school age children who were exposed prenatally. RHHRA at 42-43. This finding is long overdue and strongly supported by three lines of evidence: the three children's health studies; laboratory studies from whole animals; and, biological plausibility from cellular and *in vitro* assays. Heeding its SAP's advice, EPA determined that the effects occur at exposures that were too low to suppress the mothers' cholinesterase by anywhere near 10%. In other words, the harmful effects to children occurred at doses lower than those that would cause 10% cholinesterase inhibition, EPA's risk assessment and regulatory endpoint. This means that 10% cholinesterase inhibition is not the most sensitive endpoint. While EPA retained a 10X FQPA safety factor due to gaps in the toxicological database regarding adverse neurodevelopmental effects to children, it persisted in basing its risk assessment on the less sensitive endpoint of 10% cholinesterase inhibition. Doing so runs counter to EPA's policy of ensuring that its risk assessments and pesticide regulation are based on the most sensitive endpoint and to the precautionary approach established by Congress in the FQPA for protecting against prenatal toxicity.

This section offers the following comments on the RHHRA's assessment of neurodevelopmental effects:

- (1) EPA accurately finds that the extensive scientific record establishes that prenatal chlorpyrifos exposures result in neurodevelopmental impairments.
- (2) EPA accurately finds that these neurodevelopmental effects are the most sensitive endpoint related to chlorpyrifos toxicity.
- (3) EPA appropriately retained a 10X FQPA safety factor for data completeness based on critical gaps in the toxicity database related to neurodevelopmental effects in children, but it erroneously failed to consider whether a larger FQPA safety factor is warranted based on the demonstrated prenatal toxicity, the severity of the adverse effects and the uncertainty as to the exposure levels at which they occur.
- (4) EPA acted arbitrarily and contrary to its own findings and the scientific evidence by continuing to use 10% cholinesterase inhibition as the regulatory endpoint in the RHHRA even though the neurodevelopmental effects occur at lower doses and therefore are the most sensitive endpoint.

A. EPA Accurately Finds That The Extensive Scientific Record Establishes That Prenatal Chlorpyrifos Exposures Result In Neurodevelopmental Impairments.

EPA performed a comprehensive weight of evidence analysis evaluating data on neurodevelopmental effects from experimental toxicology studies, mechanistic studies, and epidemiologic studies. EPA found that, together, all this evidence strongly supports the conclusion that prenatal exposures to chlorpyrifos result in adverse neurodevelopmental effects in children.

Experimental toxicology studies: EPA finds that chlorpyrifos exposures caused neurodevelopmental impacts in rodent studies after reviewing the published literature. “These studies report a range of neurobehavioral changes in rats and mice following developmental exposure to chlorpyrifos... Given the wide array of testing that has been conducted, some variability is not unexpected and in fact, **the consistency of finding neurological effects is striking.** At both the 2008 and 2012 SAP meetings, the Panel agreed that exposure to doses of 1 mg/kg/d and greater, during some developmental period, **produced significant and long-term effects on animal behavior.**” RHHRA at 26 (emphasis added).

Mechanistic studies: EPA states that no definitive mode of action (MOA) has been established for neurodevelopmental outcomes. It notes that the mechanistic studies speak to biological plausibility: “This growing body of literature does demonstrate, however, that chlorpyrifos and/or its oxon are biologically active on a number of processes that affect the developing brain.” Furthermore, the Agency emphasizes that “...lack of mechanistic data, however, is not a reason to reject causality” and “does not undermine or reduce the confidence in the findings of the epidemiology studies.” RHHRA at 48.

Epidemiological studies: EPA has conducted an exhaustive and comprehensive evaluation of the human epidemiologic data on chlorpyrifos’ neurodevelopmental effects. The body of literature referenced comprises 19 peer-reviewed journal articles, a supplemental analysis suggested by the 2008 FIFRA SAP, and a federal panel review of one of the journal articles. EPA’s analysis of the epidemiological data has been through two reviews by scientific experts (the 2008 and 2012 FIFRA SAPs) and one public comment, the 2011 preliminary human health risk assessment. The analysis included validation of the study designs, research methods, health effects found, and supporting epidemiological evidence. EPA made the following findings:

Study design: “Each study includes several hundred (approximately 100-400) mother-infant pairs; these sample sizes are sufficient to perform statistically valid analyses.” RHHRA at 33.

Study design and research methods: “Investigators from each study cohort utilized a similarly strong study design (prospective birth cohort); measured pesticide exposure using several different methods including environmental indicators as well as specific and non-specific biomarkers of chlorpyrifos; ascertained developmental outcomes using validated

assessment tools well-established in both clinical and research settings; and, measured, analyzed, selected and statistically adjusted for potentially confounding variables including socio-economic status and other environmental exposures using reasonable and appropriate methods.” RHHRA at 34.

Research methods: “Across these cohort studies, investigators collected relevant information concerning demographic characteristics and other environmental exposures, and were, to the extent possible with the existing information, able to effectively hold constant the influence of these other variables when estimating the association between prenatal chlorpyrifos and adverse neurodevelopmental outcomes. Control of these variables is important to reduce the chances of a false positive study result. Overall, statistical analyses were judged to be appropriate and reasonable (not overly large number of statistical model variables) to the research question by EPA and expert Panel reviews (FIFRA SAP 2008 and 2012).” RHHRA at 35.

Research methods: “In addition, these investigators performed several methodological investigations which reduce key uncertainties in these data, specifically with regard to exposure measurement error and potential confounding bias. This work reduces the probability that positive associations reported above are inaccurate, i.e., they are false positive results.” RHHRA at 38.

Supporting epidemiological studies: “Results from the Mt. Sinai and CHAMACOS [UC Berkeley] cohort studies support the results of the CCCEH [Columbia University] study in several ways... Taken together, these results bolster the findings reported by CCCEH’s study authors.” RHHRA at 40.

Given the extensive body of literature and the strength of the studies, EPA and the two Scientific Advisory Panel reviews found that prenatal chlorpyrifos exposures likely played a role in causing the adverse neurodevelopmental outcomes observed in the human epidemiological studies: “**EPA believes these are strong studies which support a conclusion that chlorpyrifos likely played a role in these outcomes.**” RHHRA at 33 (emphasis added).

Considering evidence from the toxicology, mechanistic, and epidemiological studies as a whole, “Qualitatively, the Agency concludes that **these lines of evidence together support a conclusion that exposure to chlorpyrifos results in adverse neurodevelopmental outcomes in humans**, at least under some conditions.” RHHRA at 49 (emphasis added).

The RHHRA firmly establishes through extensive and careful analysis that prenatal chlorpyrifos exposures cause adverse neurodevelopmental impacts. Dow continues to try to refute this extensive body of peer-reviewed literature, but the science, as EPA found, is consistent, statistically reliable, and sound. EPA’s findings are the only credible conclusion that can be drawn from the science.

B. EPA Accurately Finds That The Neurodevelopmental Effects Are The Most Sensitive Endpoint Related To Chlorpyrifos Toxicity.

The next question is whether the neurodevelopmental effects are the most sensitive endpoint. This is important, because EPA policy requires selection of the most sensitive endpoint for quantitative evaluation in risk assessments in order to ensure adequate health protections for people. If the Agency protects against the most sensitive effects, it will also protect against any other effects that occur at higher levels of exposure<sup>15</sup>.

The RHHRA describes two relevant endpoints under consideration: 10% cholinesterase inhibition, which is represented as AChEi for acetylcholinesterase inhibition, and neurodevelopmental effects. RHHRA at 6. In 2012, the SAP questioned whether cholinesterase inhibition is the most sensitive endpoint: “The Panel suggested that while there are no data on AChE inhibition in either the Columbia study participants (*e.g.*, Rauh, *et al.*, 2006; Whyatt, *et al.* 2007; 2009; Rauh, *et al.*, 2011) or the NHANES participants (CDC, 2009), the measured levels of chlorpyrifos exposure are not anticipated to produce AChE inhibition.”<sup>16</sup> [NHANES is the National Health and Nutrition Examination Survey, a biomonitoring study conducted by the Centers for Disease Control and Prevention.] The SAP recommended that EPA do a dose-reconstruction analysis to determine whether or not the participants in the epidemiological studies would have experienced 10% cholinesterase inhibition. EPA carried out the dose reconstruction analysis and found that the participants would have been extremely unlikely to have experienced 10% cholinesterase inhibition:

“Overall, the dose reconstruction results support a conclusion that indoor application of chlorpyrifos, when used as allowed prior to cancellation from the residential marketplace in 2000, **likely would not have resulted in RBC AChE inhibition greater than 10% in pregnant women or young children.**” RHHRA at 41 (emphasis added).

“Comparing cord blood concentrations with the concentrations in which AChE inhibition was observed in adult volunteers indicates AChE inhibition would likely not have occurred at levels observed in the epidemiology studies (6.17 pg/g). Therefore, while uncertainty exists as to actual chlorpyrifos exposure at (unknown) critical windows of exposure, **EPA believes it is unlikely mothers enrolled in the birth cohort studies experienced RBC AChE inhibition.**” RHHRA at 45-46 (emphasis added).

“It is noteworthy that **all estimates of exposure based on conservative assumptions lead to predicted AChE inhibition levels < 10%.**” RHHRA at 46 (emphasis added).

“Moreover, exposure levels in the range measured in the epidemiology studies

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<sup>15</sup> EPA OPP, Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment at 8 (2002). *See, e.g.*, [http://www.epa.gov/pesticides/reregistration/atrazine/atrazine\\_update.htm](http://www.epa.gov/pesticides/reregistration/atrazine/atrazine_update.htm), “Reproductive effects are the most sensitive effects observed in atrazine toxicity tests and, as such, our efforts to regulate the pesticide to protect against these effects through drinking water exposure will protect against all other effects that occur at higher levels.”

<sup>16</sup> FIFRA SAP Meeting: A Set of Scientific Issues Being Considered by the EPA Regarding Chlorpyrifos Health Effects, at 51 (April 2012).

(pg/g) are likely low enough that [it] is **unlikely to result in AChE inhibition, as supported by the dose reconstruction analysis** of residential use prior to 2000...” RHHRA at 49 (emphasis added).

Through this analysis, EPA confirms that neurodevelopmental effects occur at lower exposure levels compared to 10% cholinesterase inhibition, and thus that neurodevelopmental effects are the most sensitive endpoint. Under EPA policies and practices, neurodevelopmental effects are the endpoint that should be used for quantitative evaluation in the risk assessment. If the Agency establishes limits that protect against the neurodevelopmental effects, these limits would also protect against 10% cholinesterase inhibition, and any other effects that occur at higher exposure levels. Conversely, if EPA continues to use 10% cholinesterase inhibition as its risk assessment target, it will not protect children against neurodevelopmental harm. EPA continues to use 10% cholinesterase inhibition as the target in the RHHRA.

- C. EPA Appropriately Retains a 10X FQPA Safety Factor For Data Completeness Based on Critical Gaps in the Toxicity Database Related to Neurodevelopmental Effects in Children, But it Erroneously Failed to Consider Whether the Demonstrated Prenatal Toxicity Warrants a Larger FQPA Safety Factor Because of The Severity of the Adverse Effects And The Uncertainty as to the Exposure Levels at Which They Occur.

EPA next addressed whether it should retain an FQPA Safety Factor, which the FQPA mandates “to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and toxicity to infants and children.”<sup>17</sup> In the preliminary HHRA, EPA considered eliminating the FQPA safety factor, but in the revised HHRA, it retains a 10X FQPA safety factor due to gaps in the data.

In deciding to retain a 10X FQPA safety factor, EPA considered “data completeness” and noted that there remain significant uncertainties in the mechanism of action, dose-response, critical duration of exposure, and window(s) of susceptibility for the neurodevelopmental effects. RHHRA at 49. Therefore, EPA appropriately concluded that “...**there is sufficient uncertainty in the human dose-response relationship for neurodevelopmental effects** which prevents the Agency from reducing or removing the statutory 10X FQPA Safety Factor.” RHHRA at 7, 49 (emphasis added).

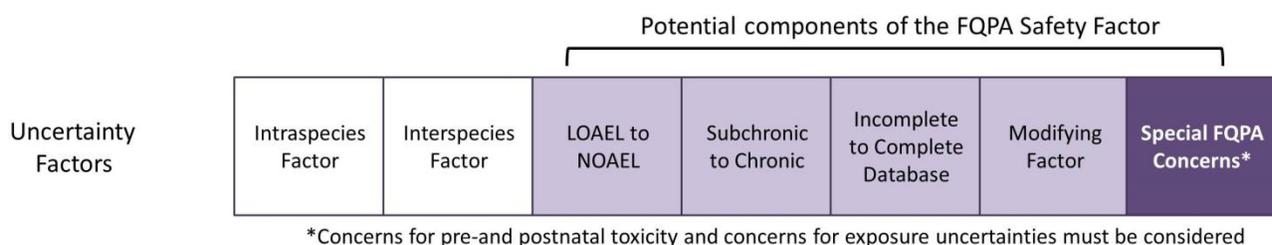
Having decided to retain a 10X FQPA safety factor, EPA ended its analysis. In doing so, it failed to consider the factors laid out in its guidance for deciding the magnitude of an FQPA safety factor that is warranted. In particular, EPA failed to consider, as mandated by FQPA, “potential pre- and post-natal toxicity” in setting the FQPA safety factor. The RHHRA discusses at length the compelling, multiple lines of evidence that chlorpyrifos causes prenatal neurodevelopmental toxicity, resulting in significant, persistent and devastating impacts in children including reduced measures of intelligence and increased likelihood for pervasive developmental disorder.” RHHRA at 38. However, while the RHHRA describes the weight-of-evidence on chlorpyrifos prenatal toxicity, EPA does not use this evidence to set the magnitude of the “special” FQPA safety factor. By ignoring prenatal toxicity in setting the level of the

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<sup>17</sup> 21 U.S.C. § 346a(b) (2)(C)(i).

FQPA safety factor, EPA failed to follow the FQPA's mandates.

EPA also failed to follow its own guidance, which lays out the potential components that can contribute to the FQPA Safety Factor, as shown in Figure A. These components encompass both data deficiency uncertainty factors and "special" FQPA concerns. The data deficiency uncertainty factors derive from the FQPA's "completeness of the data" clause and are employed when there is: extrapolation from a Lowest Observed Adverse Effects Level (LOAEL) to a No Observed Adverse Effects Level (NOAEL), extrapolation from a subchronic study to a chronic outcome, or key data missing from a chemical's database (such as dose-response). "Special" FQPA concerns refer to potential pre- or post-natal toxicity and exposure uncertainties with respect to infants and children.



**Figure A. The FQPA Safety Factor encompasses data completeness, toxicity and exposure considerations.** Adapted from EPA 2002.<sup>18</sup> Light shaded boxes show the data deficiency uncertainty factors and the dark shaded box embodies the FQPA requirement to consider potential pre- and post-natal toxicity and exposure uncertainties.

EPA guidance makes it clear that EPA must conduct a full review of all of these factors and set the FQPA safety factor at a level that will protect children, even if that means a factor of more than 10X:

“In evaluating the size of any factor different from the 10X default safety factor, OPP does not believe that Congress intended that the default 10X factor be split up using some mathematical formula between pre- and postnatal toxicity and the completeness of the toxicology and exposure databases. Rather, **OPP thinks that its focus should be on what factor is needed to protect infants and children. That analysis should concentrate on what the existing data show with regard to the pesticide in question.** When data are missing or otherwise incomplete, the analysis will be concerned with how the results from the missing data could affect the risk assessment. **This analysis may result in a finding that a factor either greater or less than 10X should be added** to the traditional inter- and intraspecies factors or that no factor in addition to these traditional factors is needed.”<sup>19</sup>

<sup>18</sup> EPA OPP, Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment, at 16, Figure 3 (Feb. 28, 2002).

<sup>19</sup> *Id.* at A-5 (emphasis added).

The guidance goes on to say:

**“In fact, the final safety factor could be greater than 10X.** OPP continues to adhere to the core principle that the FQPA establishes an additional 10X safety factor as a default. In this document the phrase ‘consider an FQPA safety factor’ should be interpreted to mean to retain the presumptive 10X FQPA safety factor or to establish a different safety factor that is less than, equal to, or greater than the default value.”<sup>20</sup>

In keeping with this policy, EPA has set FQPA safety factors at greater than 10X to account for incomplete data and prenatal toxicity. Table A 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 at more than 10X when appropriate based on its consideration of both data completeness and special FQPA concerns.

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

<b>Pesticide</b>	<b>Intraspecies Factor</b>	<b>Interspecies Factor</b>	<b>Data Completeness Factor</b> <i>(specific data deficiency)</i>	<b>Special FQPA concerns</b> <i>(factors contributing to degree of concern)</i>
Carbendazim (MBC) <sup>21</sup>	10X	10X	3X (extrapolation from LOAEL)	10X (increased prenatal susceptibility in rat and rabbit studies)
Molinate <sup>22</sup>	10X	10X	3X (extrapolation from LOAEL)	10X (prenatal toxicity in rodent studies; uncertainties in drinking water exposure)
Pirimiphos-methyl <sup>23</sup>	10X	10X	10X (extrapolation from LOAEL, severity of effects at LOAEL, data gaps for long term studies)	3X (lack of complete toxicity database for assessing potential for susceptibility)

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. Chlorpyrifos’ neurodevelopmental effects are analogous since neither a LOAEL nor NOAEL has been established. Accordingly, EPA should have considered whether the FQPA safety factor should be greater than 10X just to account for the additional uncertainty with respect to impacts on infants and children:

“...if missing data are considered to be critical to understanding the potency of a

<sup>20</sup> *Id.* at A-6 (emphasis added).

<sup>21</sup> EPA, Revised Preliminary Human Health Risk Assessment: Thiophanate-methyl. Health Effects Division, Office of Pesticide Programs, at 8-9 (April 25, 2002).

<sup>22</sup> EPA OPP, Health Effects Division, Human Health Risk Assessment: Molinate at 6, 14 (November 6, 2002).

<sup>23</sup> EPA OPP, Health Effects Division, Interim Reregistration Decision for Pirimiphos-Methyl: Case No. (2535), at 7 (July 31, 2006).

chemical and have a good possibility of revealing an especially sensitive subgroup, the size of the uncertainty factor likely would be 10X. There may be situations where a factor greater than 10X is justified based on the data missing and a considerable amount of uncertainty in the weight-of-evidence evaluation.”<sup>24</sup>

In addition to dose-response, significant uncertainties remain about the level, duration and critical windows of exposure that result in adverse neurodevelopmental impacts. As stated in the RHHRA, “With respect to effects on the developing brain, very little is known about the duration of chlorpyrifos exposure needed to precipitate adverse effects in the developing brain... **As such, it is impossible at this time to rule out even a single day of high exposure to chlorpyrifos having a potential adverse neurodevelopmental effect in humans.**” RHHRA at 50 (emphasis added). Additionally, EPA’s analysis of the epidemiological studies concludes that the most likely bias would have been to underestimate the size of the documented effects: “EPA notes that given the nature of the study design, **the most likely effect of any exposure measurement error would be to under-estimate rather than over-estimate risk...**” RHHRA at 43 (emphasis added).

Apart from the risks due to data gaps, EPA also must consider prenatal toxicity in setting the FQPA safety factor. In addition to mandating that the safety factor take prenatal toxicity into account, the FQPA specifically requires EPA to consider available information about the special susceptibility of infants and children, including neurological differences between infants and children and adults, and effects of in utero exposure to pesticide chemicals. 21 U.S.C. § 346a(b)(2)(C)(i). EPA failed to do so. The table below lists the factors laid out in EPA’s guidance for evaluating the magnitude of prenatal toxicity concerns along with our analysis of how each applies to chlorpyrifos based on EPA’s findings in the RHHRA.

**Table B. Factors for evaluating degree of concern for prenatal toxicity.** (Two left columns from EPA 2002.<sup>25</sup>) The evidence on chlorpyrifos from the RHHRA indicates the highest degree of concern for prenatal toxicity.

Factor	Factors that increase concern	Evidence on chlorpyrifos from RHHRA
Pre- and postnatal toxicity	<ul style="list-style-type: none"> <li>• Effects found in humans related to exposure</li> </ul>	✓ “EPA concludes that chlorpyrifos likely played a role in the neurodevelopmental outcomes observed in these epidemiology studies.” RHHRA at 43.
	<ul style="list-style-type: none"> <li>• Same types of effects seen in more than one species</li> </ul>	✓ “Taken together, these <b>studies in rats and mice show altered cognitive function</b> using well accepted tests of spatial learning and memory (radial arm maze, Morris water maze).” RHHRA at 164 (emphasis added).
	<ul style="list-style-type: none"> <li>• Effects of a different type with greater potential consequences in young compared to adults</li> </ul>	✓ “There is evidence of <b>delays in mental development in infants (24-36 months), attention problems and pervasive developmental disorder in early childhood, and intelligence decrements in</b>

<sup>24</sup> EPA, Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment, at 16, Figure 3 (Feb. 28, 2002).

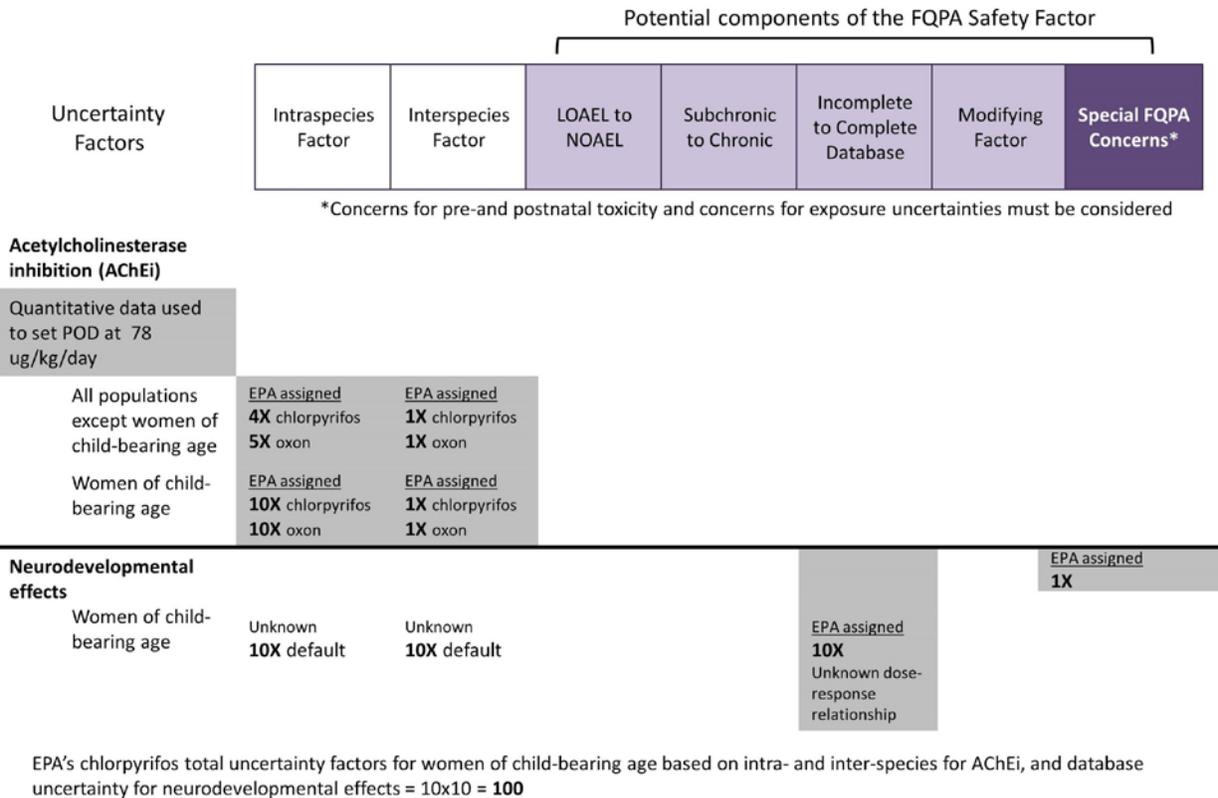
<sup>25</sup> *Id.* at 31.

		school age children who were exposed to chlorpyrifos or OP during gestation.” RHHRA at 42. <b>No adverse effects on the adult mothers were reported in any of the epidemiological studies.</b>
	<ul style="list-style-type: none"> <li>● Persistence or relatively longer recovery of effects in young compared to adults</li> </ul>	<ul style="list-style-type: none"> <li>✓ “Although uncertainties remain as articulated above, these uncertainties are diminished in the context of the qualitative similarity between the databases, and <b>the concern for long-term neurodevelopmental effects</b> as a result of prenatal, perinatal and possibly early life exposure.” RHHRA at 48 (emphasis added). <b>No adverse effects on the adult mothers were reported in any of the epidemiological studies.</b></li> </ul>
Dose response	<ul style="list-style-type: none"> <li>● Effects observed at a lower dose in young compared to adults</li> </ul>	<ul style="list-style-type: none"> <li>✓ The levels of chlorpyrifos exposures that resulted in neurodevelopmental outcomes for children had no discernible adverse effects on the adult mothers in epidemiologic studies. Animal studies also found neurodevelopmental effects at doses (1 mg/kg/day or less) that are not expected to result in cholinergic toxicity to the adult. RHHRA at 25.</li> </ul>
	<ul style="list-style-type: none"> <li>● NOAEL not identified</li> </ul>	<ul style="list-style-type: none"> <li>✓ EPA identified no NOAEL for neurodevelopmental effects.</li> </ul>
	<ul style="list-style-type: none"> <li>● Poor data on dose-response</li> </ul>	<ul style="list-style-type: none"> <li>✓ “As noted above, the lack of an established MOA/AOP [mode of action/adverse outcome pathways] <b>makes quantitative use of the epidemiology study in risk assessment challenging, particularly with respect to dose response</b>, critical duration of exposure, and window(s) of susceptibility.” RHHRA at 49 (emphasis added).</li> </ul>
Toxicokinetics	<ul style="list-style-type: none"> <li>● Metabolic profile indicates higher internal dose of active moiety in young compared to adult, or in humans compared to animals</li> </ul>	<ul style="list-style-type: none"> <li>✓ Though the active moiety for neurodevelopmental effects has not been identified, data indicates that young children have less activity of the enzyme PON1, which metabolizes chlorpyrifos, and that PON1 activity may play a significant role in determining susceptibility to neurodevelopmental effects.<sup>26</sup></li> <li>✓ Also, there is a key difference in dose response when comparing the human and animal studies, with <b>greater sensitivity seen in humans:</b> “...in animals, the doses most often used in the behavior studies (1 and 5 mg/kg/day) are sufficient to elicit approximately ≥10% brain inhibition and ≥30% in RBC inhibition...in the epidemiology studies, based on the comparisons with biomonitoring data and the results of the dose reconstruction analysis, it is unlikely that</li> </ul>

<sup>26</sup> Eskenazi, Brenda, Karen Huen, Amy Marks, Kim G. Harley, Asa Bradman, Dana Boyd Barr, and Nina Holland. “PON1 and Neurodevelopment in Children from the CHAMACOS Study Exposed to Organophosphate Pesticides in Utero.” *Environmental Health Perspectives* 118, no. 12 (December 2010): 1775–81. doi:10.1289/ehp.1002234.

		RBC AChE would have been inhibited by any meaningful or measurable amount, if any at all, and most likely none in the brain..." RHHRA at 46-47.
Mode of action	<ul style="list-style-type: none"> <li>Mode of action supports relevance to humans and concern for animal findings</li> </ul>	<ul style="list-style-type: none"> <li>✓ Though the mode of action is unknown, there is consistency in the types of neurodevelopmental effects seen in animal and human studies: "Given the difference across laboratory animal and human studies, the <b>qualitative similarity in research findings is striking.</b>" RHHRA at 46.</li> </ul>
	<ul style="list-style-type: none"> <li>Mode of action may lead to several adverse consequences for the offspring</li> </ul>	<ul style="list-style-type: none"> <li>✓ Though the mode of action is unknown, four serious adverse neurodevelopmental impacts associated with prenatal chlorpyrifos exposure are documented: <b>developmental delays, decreases in psychomotor abilities, attentional abilities, and intelligence.</b></li> </ul>

As shown in the table, there is evidence from the RHHRA which indicates higher degrees of concern for every factor which EPA is supposed to consider in their weight of evidence evaluation of prenatal toxicity. Moreover, as discussed below, EPA lacks sufficient data to estimate children's exposure to chlorpyrifos and its oxon in drinking water. Its preliminary HHRA found all infants would be at risk from drinking water contamination. In the absence of complete data allowing EPA to identify which watersheds pose such risks, it must account for the gaps in its knowledge through the FQPA uncertainty factor. EPA acted in disregard of its own guidance by failing to consider prenatal toxicity and all of the concerns its guidance identifies as affecting the magnitude of the FQPA safety factor. In doing so, it failed to ensure that children will be afforded the protection prescribed by the FQPA and the evidence of harm from chlorpyrifos. EPA needs to complete this evaluation as mandated by FQPA.



**Figure B. Uncertainty factors and FQPA considerations in the chlorpyrifos RHHRA.** Top portion adapted from EPA 2002.<sup>27</sup> Gray shaded boxes show the uncertainty factors proposed by EPA in the chlorpyrifos RHHRA. EPA used uncertainty factors from two different endpoints (AChEi and neurodevelopmental effects) and failed to assign a safety factor to account for prenatal toxicity as required under FQPA.

**D. EPA Acted Arbitrarily And Contrary to its Own Findings and the Scientific Evidence by Continuing to Use 10% Cholinesterase Inhibition as The Limit in the RHHRA Even Though Neurodevelopmental Effects Occur at Lower Doses and Therefore are the Most Sensitive Endpoint.**

In order to ensure that people are protected from adverse health impacts, it is EPA's policy to use the most sensitive endpoint to calculate regulatory limits for risk assessment.<sup>28</sup> But in the RHHRA, EPA finds that the neurodevelopmental effects are the most sensitive endpoint because they occur at exposures too low to cause 10% cholinesterase inhibition, but inexplicably, EPA nevertheless continues to use 10% cholinesterase inhibition as its risk assessment endpoint. By not using the most sensitive endpoint, neurodevelopmental effects, to set regulatory limits, it is acting contrary to its findings and the evidence and falls far short of its obligation to protect

<sup>27</sup> EPA OPP, Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment, at 16, Figure 3 (2002).

<sup>28</sup> *Id.* at 8; *see, e.g.*, [http://www.epa.gov/pesticides/reregistration/atrazine/atrazine\\_update.htm](http://www.epa.gov/pesticides/reregistration/atrazine/atrazine_update.htm): "Reproductive effects are the most sensitive effects observed in atrazine toxicity tests and, as such, our efforts to regulate the pesticide to protect against these effects through drinking water exposure will protect against all other effects that occur at higher levels."

children from prenatal toxicity.

The SAP has repeatedly cautioned that using the dose-response data for 10% cholinesterase inhibition could put pregnant women at risk because of the significant uncertainties about neurodevelopmental effects:

**“Additional Panel concerns about the use of AChE inhibition dose-response data to protect against neurodevelopmental effects** is based on the potential for AChE inhibition and adverse neurodevelopmental effects to be two separate events. AChE inhibition is the result of an acute exposure scenario and neurodevelopmental effects likely being caused by chronic low level exposure to chlorpyrifos in utero.”<sup>29</sup>

**“The Panel expresses concern over the Agency’s focus on a 10% AChE activity reduction.** They point out that to their knowledge there is no proposed mechanism whereby a 10% AChE activity reduction in pregnant women would be responsible for a cognitive defect or developmental delay in their offspring.”<sup>30</sup>

**“As advised by the Panel, options for dose response analysis for acute effects should be considered independent from those based on long term exposures,** i.e., measures representing acute adverse neurological outcomes (ChE inhibition) commonly associated with occupational exposure versus those potentially related to lower level long term exposure in the general population, such as neurobehavioral disorders.”<sup>31</sup>

**“Furthermore, since the neurodevelopmental effects may be independent of AChE inhibition, it needs to be considered whether AChE inhibition represents a critical marker for derivation of points of departure when considering chronic studies.”**<sup>32</sup>

**“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.”**<sup>33</sup>

The SAP notes that if EPA chooses to proceed with setting regulatory limits based on cholinesterase inhibition, despite the SAP’s cautions, it will be absolutely critical for EPA to validate that the results will protect infants and children from the neurodevelopmental effects: **“Given that AChE inhibition results from acute exposure and adverse neurodevelopmental effects are likely to be caused by chronic low levels of chlorpyrifos, it is important to verify whether or not maintaining long-term exposure to levels below those likely to cause AChE**

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<sup>29</sup> FIFRA SAP Meeting: A Set of Scientific Issues Being Considered by the EPA Regarding Chlorpyrifos Health Effects, at 20 (2012) (emphasis added).

<sup>30</sup> *Id.* at 25 (emphasis added).

<sup>31</sup> *Id.* at 26 (emphasis added).

<sup>32</sup> *Id.* at 39 (emphasis added).

<sup>33</sup> *Id.* at 53 (emphasis added).

**inhibition is likely to be sufficiently protective** to prevent neurodevelopmental effects.”<sup>34</sup>

In order to do this, the SAP repeatedly recommends that EPA should use the epidemiologic data to “bound” the AChEi reference dose (RfD)/Population Adjusted Dose (PAD). By “bounding”, the SAP means that a simple comparison needs to be done to ascertain how the RfD or PAD established based on cholinesterase inhibition compares to the estimated chlorpyrifos doses associated with neurodevelopmental effects in the epidemiological studies.

“In reference to the epidemiology data being used to support the quantitative uncertainty characterization and analysis, the Panel agrees with the 2008 SAP suggestion that **at a minimum the Agency should use available data from these studies to at least ‘bound’ reference doses** developed on the basis of animal data.”<sup>35</sup>

“The Panel also recommends that the Agency **maximizes its use of available data on dose response from the epidemiology studies as a basis to at least ‘bound’ reference doses** developed on the basis of points of departure from animal data.”<sup>36</sup>

“As a minimum, then, it seems important to **maximally utilize available data on dose response from these studies to at least ‘bound’ reference doses** developed on the basis of animal data (Given that this was also recommended by the 2008 SAP, prioritization of this work seems critical).”<sup>37</sup>

“As indicated, in response to previous questions, **the maximal use of the available dose response data from the epidemiological studies is recommended as a basis to at least, ‘bound’ reference doses** developed on the basis of points of departure from animal data.”<sup>38</sup>

“As noted by the Panel, if one assumes that cord blood measurements reflect exposure levels during the critical prenatal period for induction of neurodevelopmental effects, then in theory, these would be the ideal data from which to derive the POD for chlorpyrifos in humans. Specific Panel suggestions included using the Columbia data ‘as an exercise’ to derive a POD for neurodevelopmental effects in infants, and **analyzing the data from each of the cohorts to put some bounds on the range of chlorpyrifos doses associated with the observed neurodevelopmental effects.**”<sup>39</sup>

The SAP called for this “bounding” comparison to avoid an under-protective risk evaluation. More specifically, if the regulatory limit – a reference dose (RfD or Population Adjusted Dose (PAD) – set based on 10% cholinesterase inhibition for women of child-bearing age is greater than or equal to the chlorpyrifos doses associated with the neurodevelopmental effects in the

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<sup>34</sup> *Id.* at 58 (emphasis added).

<sup>35</sup> *Id.* at 21 (emphasis added).

<sup>36</sup> *Id.* at 26 (emphasis added).

<sup>37</sup> *Id.* at 57 (emphasis added).

<sup>38</sup> *Id.* at 74 (emphasis added).

<sup>39</sup> *Id.* at 50 (emphasis added).

epidemiological studies, pregnant women could continue to be exposed to chlorpyrifos at levels that likely harmed the fetuses of women in the Columbia study. This, of course, would be unacceptable.

EPA never conducted the validation sought by the SAP. Nowhere within the 531 pages of the RHHRA does EPA attempt to verify that the regulatory limit – the PAD that it calculated based on 10% cholinesterase inhibition – will protect infants and children from permanent neurological damage. It is possible that EPA failed to conduct the validation because it had already found that chlorpyrifos can cause neurodevelopmental effects at doses lower than those that cause 10% cholinesterase inhibition.

EPA also may have believed, as the RHHRA suggests, that it is not possible to derive quantitative information on the neurodevelopmental effects from the available animal or human studies. RHHRA at 49. However, EPA’s Office of Research and Development has funded research that used data from the Columbia study to derive an exposure level linked to adverse neurodevelopmental effects. Dr. Dale Hattis led a team that associated observed blood levels of chlorpyrifos in the published epidemiologic studies of Whyatt, *et al.* and the impaired cognition that was observed in the children that had been exposed during fetal development to chlorpyrifos and other pesticides. That project is now complete and being submitted for publication.<sup>40</sup>

The Hattis research assessed loss of working memory from chlorpyrifos based on the Columbia studies. Working memory is a standard measurement of brain function. It is a component of IQ and refers to the ability to memorize new information, retain it in the short-term, and manipulate it. There is no identified clear amount of loss of working memory that is considered acceptable on a population basis. Using working memory as a measurement of neurological deficits is well-established from scientific literature on lead poisoning, for which no safe level of prenatal and early life lead exposure has been established.

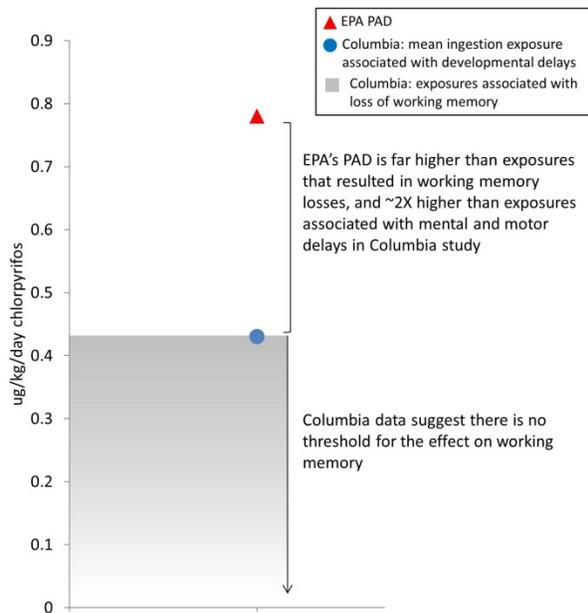
EPA’s steady-state point of departure (POD) or endpoint for women of child-bearing age, based on 10% cholinesterase inhibition, is 78 ug/kg/day. When EPA’s selected uncertainty factors totaling 100X are taken into account (10X intra-species x 10X FQPA), the resulting PAD is 0.78 ug/kg/day for women of child-bearing age, viewed by EPA to be a level of exposure without deleterious effects over a lifetime, considering vulnerable populations. RHHRA at 76. In contrast, Hattis, *et al.* (2015) used the real-world data from the Columbia University study to calculate determined that 0.6 Standard Deviations of IQ would be lost in the offspring if woman were exposed during late pregnancy to EPA’s proposed chronic Population Adjusted Dose (cPAD (0.78 ug/kg-day, Hattis *et al.* 2015 at 65). The table below illustrates the comparison.

<b>Preliminary Human Health Risk Assessment (EPA 2011)</b>	<b>RHHRA (EPA 2014)</b>	<b>Hattis <i>et al.</i> (2015)</b>

<sup>40</sup> Hattis, *et al.*, Chlorpyrifos Doses to Women of the Columbia University Cohort and Neurodevelopmental Impairment—A Bayesian-Inspired Uncertainty Analysis and Risk Projection Reflecting Inputs from Different Sources of Information (2015) (Ex. 1).

POD=30 ug/kg-day based on 10% AChEi in pregnant rats	Based on animal and in vitro data in a PBPK-PD model, 78 ug/kg-day (at 65, Steady state POD)	Based on blood samples drawn within a few days of birthing from 335 women in the Columbia study, in a PBPK model
UF=100	UF=100	
Oral RfD = 0.3 ug/kg-day or 300 ng/kg-day	PAD=0.78 ug/kg-day 780 ng/kg-day	Women in Columbia study exposure (daily dose, primarily via inhalation): Mean=28 ng/kg-day 90 <sup>th</sup> = 190 ng/kg-day

The RHHRA's exposure endpoints are higher than the exposures for women in the Columbia study estimated by Hattis, *et al.* Maternal exposures correlated with mental and motor delays in offspring range from 0.35 ug/kg/day (inhalation) to 0.43 ug/kg/day (ingestion). The exposures correlated with loss of working memory are even lower and the Columbia data suggest there may be no threshold for this effect. As shown in Figure C below, EPA's PAD at 0.78 ug/kg/day is four times higher (less protective) than the 90<sup>th</sup> percentile of exposures associated with neurodevelopmental effects in the Columbia study, and 28 times higher than the average (mean) exposures. This shows that prenatal exposure at the PAD is associated with neurodevelopmental effects in the Columbia study, and therefore that EPA's current PAD is not likely to afford effective protection against neurodevelopmental effects.



**Figure C. EPA’s PAD for women of child-bearing age is far higher than the chlorpyrifos doses that resulted in harm to children in the Columbia study.** The estimated range of chlorpyrifos doses associated with neurodevelopmental effects in the Columbia study is taken from Hattis, *et al.* (2015).

By continuing to use 10% cholinesterase inhibition as the risk assessment endpoint, while not increasing the FQPA safety factor and shrinking the traditional safety factors as discussed in the next section, EPA’s assessment does not ensure “a reasonable certainty of no harm” to infants and children as mandated by the FQPA. EPA could have selected a more protective endpoint that would reflect neurodevelopmental effects, or it could have increased the FQPA safety factor to account for prenatal toxicity and the lack of a LOAEL or NOAEL. It cannot use an endpoint that admittedly is not the most sensitive one and fail to take steps to guard against the unacceptable risks that approach presents to children.

### III. EPA CANNOT REDUCE THE TRADITIONAL SAFETY FACTORS BASED ON A DOW MODEL THAT IS DESIGNED TO PREDICT 10% CHOLINESTERASE INHIBITION.

The FQPA safety factor is “an additional” safety factor added by the FQPA with the intent that it afford additional protection to children beyond that afforded by traditional safety factors in place for many decades. While risk assessments strive to quantify risks, EPA has long recognized that, “[e]ven at its best, risk assessment does not estimate risk with absolute certainty,” and that “it is important that the risk assessment process handle uncertainties in a predictable way that is scientifically defensible, consistent with the Agency’s statutory mission, and responsive to the needs of decision makers.”<sup>41</sup>

EPA uses safety or uncertainty factors because they enable EPA “to adhere to its goal of

<sup>41</sup> EPA Office of the Science Advisor, Risk Assessment Principles and Practices, at 30 (March 2004).

protecting public health and the environment by ensuring that it does not underestimate the risk that certain chemicals pose.”<sup>42</sup> Two uncertainty factors have been longstanding features in pesticide risk assessment. The interspecies factor accounts for the uncertainty in extrapolating data from animals to humans. It is used because “[t]here are major uncertainties in extrapolating both from animals to humans and from high to low doses. There are important species differences in uptake, metabolism, and organ distribution of carcinogens, as well as species and strain differences in target-site susceptibility.”<sup>43</sup> The intra-species uncertainty factor accounts for the uncertainty in extrapolating data across the human population and accounts for “variations in susceptibility within the human population (inter-human variability) and the possibility (given a lack of relevant data) that the database available is not representative of the dose/exposure-response relationship in the groups of the human population that are most sensitive to the health hazards of the chemical being assessed.”<sup>44</sup> It can account for the inherent differences from person to person in the human population due to such factors as genetic predisposition, other illnesses, exposure to other toxicants, and susceptibility due to poverty or poor access to health care. Each of these traditional uncertainty factors has a default value of 10X for a total of 100X together.<sup>45</sup>

It is standard practice for EPA to incorporate this combined 100X safety factor into its risk assessments. As EPA explained in its 2002 guidance, *Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment*:

For almost 30 years, EPA, as well as others in the scientific and regulatory community, has routinely been using at least two tenfold safety or uncertainty factors when relying on animal testing to assess the potential for human hazard posed by exposure to chemicals. The two tenfold factors used most often are designed to address both the extrapolation of the results of animal studies to humans (i.e., the interspecies uncertainty factor) and variability and sensitivity within humans (i.e., intraspecies uncertainty factor) and to serve as the starting point for defining an acceptable exposure level for a chemical. Furthermore, it is also well-established regulatory practice to apply, on a case-by-case basis, additional safety, uncertainty, or modifying factors along with the baseline inter- and intraspecies factors where the circumstances warrant such factors. These uncertainty factors have been used principally to address gaps in the toxicology database or inadequacies in the key existing toxicology studies.<sup>46</sup>

In fact, when Congress enacted the FQPA, it created an additional safety factor to account for data gaps and toxicity to children. It intended for the FQPA safety factor to be added to these traditional ones and specified that the FQPA safety factor could be increased or decreased only when reliable information demonstrated that the resulting safety factor would be safe for infants and children. 21 U.S.C. § 346a(b)(2)(C).

EPA may adjust uncertainty factors, but only when the scientific record supports doing

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<sup>42</sup> *Id.*

<sup>43</sup> *Id.*

<sup>44</sup> *Id.*

<sup>45</sup> The National Academy of Sciences has endorsed the use of default uncertainty factors to address uncertainties in risk assessments in pivotal studies. See, e.g., NAS, *Science and Decisions* at 7-8, 192 (2009).

<sup>46</sup> EPA OPP, *Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment* at A-3 (2002).

so. Whether EPA has a basis for adjusting the 10X uncertainty factor “will depend on the quality of the studies available, the extent of the database, and scientific judgment.”<sup>47</sup> To reduce the default 10X safety factor, EPA must undertake “a critical analysis of the entire body of available data for consistency and biological plausibility,” must examine all relevant studies, and must give high quality studies more weight than low quality studies.<sup>48</sup> While the NAS also recognizes that defaults can be adjusted, it has stated that a “default uncertainty factor is needed for ‘the steps in risk assessment that require inferences beyond those that can be clearly drawn from the available data or to otherwise fill common data gaps.’”<sup>49</sup> It also has concluded that, “[w]hen EPA elects to depart from a default assumption, it should quantify the implications of using an alternative assumption, including describing how use of the default and the selected alternative influences the risk estimate for risk-management options under consideration.”<sup>50</sup> In the end, a guiding principle is that EPA must “adhere to its goal of protecting public health and the environment by insuring that it does not underestimate the risk that certain chemicals pose.”<sup>51</sup> EPA has appropriately exercised its discretion “to decide that in the wake of uncertainty, it would be better to give the values a conservative bent rather than err on the other side.”<sup>52</sup>

In the RHHRA, EPA reduced the traditional safety factors based on a model Dow developed to predict when exposures will result in 10% cholinesterase inhibition. EPA eliminated the inter-species factor altogether. For all groups except women of child-bearing age, including children, it reduced the intra-species safety factor to 4X for chlorpyrifos and 5X for the chlorpyrifos oxon. EPA retained the 10X safety factor for intra-species variation for women of child-bearing years since the model lacked sufficient data to be used for pregnant women. It made this downward adjustment in the traditional safety factors based on the model outputs, but without applying the factors EPA has deemed relevant and necessary to address before reducing public health protection. EPA acted arbitrarily, contrary to the evidence before it, and in violation of its statutory mandates to protect public health in three ways. First, the Dow model is tailored to 10% cholinesterase inhibition, while EPA has found that the human health endpoint of greatest concern is early life exposures leading to neurodevelopmental effects, which occur at lower doses. Second, the Dow model has serious scientific limitations, lacks proper validation, and was met with significant criticisms by EPA’s SAP, which have not been addressed through a subsequent SAP review. Third, it improperly relies on human dosing studies that EPA’s advisors have criticized on both scientific and ethical grounds and that EPA have not been fully reviewed in accordance with its recently strengthened regulations.

A. The Dow Model is Tailored to 10% Cholinesterase Inhibition While EPA Has Found That the Human Health Endpoint of Greatest Concern is Early Life Exposures Leading To Neurodevelopmental Effects, Which Occur at Lower Doses.

EPA has relied heavily on a PBPK-PD model supplied by Dow Agrochemical Sciences to estimate the internal dose metrics associated with cholinergic toxicity as measured by

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<sup>47</sup> EPA, Office of the Science Advisor, Guidance for Applying Quantitative Data to Develop Data-Device Extrapolation Factors for Interspecies and Intraspecies Extrapolation at 1 (Sept. 2014).

<sup>48</sup> *Id.*

<sup>49</sup> NAS, *Science and Decisions*, at 192.

<sup>50</sup> *Id.* at 208.

<sup>51</sup> Office of the Science Advisor, Risk Assessment Principles and Practices, at 52.

<sup>52</sup> *Am. Iron & Steel Inst. v. E.P.A.*, 115 F.3d 979, 993 (D.C. Cir. 1997).

inhibition of the acetyl cholinesterase enzyme (AChE). As explained above, when the acetyl cholinesterase enzyme is inhibited or impaired from functioning, it will over-stimulate its target nerve or muscle, causing symptoms that acute poisonings and other effects. The acute poisoning symptoms associated with cholinesterase inhibition are well described in the medical literature, the same cannot be said with respect to the precise exposures or mechanisms that cause neurodevelopmental effects of fetal chlorpyrifos exposures.

The most obvious problem with EPA's use of the Dow PBPK-PD model is that it does not model the endpoint of concern – neurodevelopmental impacts from fetal exposures. Rather, the model is used to “predict critical dose metrics associated with cholinergic toxicity following chlorpyrifos exposure: red blood cell and brain cholinesterase inhibition.” RHHRA at 51. However, the Columbia University data clearly show that neurodevelopmental effects occur following fetal exposures to chlorpyrifos at levels below those that are expected to result in the arbitrary 10% inhibition of red blood cell cholinesterase in pregnant mothers. RHHRA at 41. In fact, according to EPA's calculations, the pregnant women in the Columbia Cohort would be expected to have had no more than 0.45% red blood cell cholinesterase inhibition from indoor residential applications allowed prior to the 2000 cancellation. RHHRA at 42. Nonetheless, prenatally exposed children born to these women show measurable, persistent, and significant mental deficits associated with exposure to chlorpyrifos and other pesticides.

In relying on the Dow model, EPA never expressly addresses the fact that it measures a different, less sensitive endpoint than the demonstrated neurodevelopmental effects of chlorpyrifos. EPA has found no correlation between cholinesterase inhibition and the neurodevelopmental damage to children. The Agency acknowledges that, “the SAP concurred with the Agency in 2008 and 2012 about the lack of definable key events in a MOA/AOP leading to neurobehavioral effects. The Agency has considered the new literature since the 2012 SAP related to mechanistic hypotheses (Appendix 11), and note that such a MOA/AOP still cannot be established.” RHHRA at 27. This introduces an incalculable level of uncertainty in the current application of the Dow model, given that the 10% red blood cell cholinesterase inhibition criterion is not predictive for the endpoints of greatest concern.<sup>53</sup>

The Dow model incorporates age-specific parameters from infant through adulthood, RHHRA at 51, but misses completely the fetal stage that is critically associated with the neurodevelopmental effects seen in the epidemiologic studies. It fails to capture the physiological changes associated with pregnancy that would impact the chemical dose and toxicity during critical early life-stages. RHHRA at 62. The model does not include any descriptions of physiological, anatomical and biochemical changes associated with pregnancy. Due to the uncertainty in extrapolating the current model predictions among women who may be pregnant, the agency appropriately retains the standard 10X intra-species extrapolation factor for women of child bearing age. However, this is inadequate. EPA must consider the quality of the science/studies, the extent of the database, and scientific judgment in reducing safety factors. Pregnancy is associated with many physiological and biological changes. The major hemodynamic alterations during pregnancy include increased cardiac output, sodium and water retention leading to blood volume expansion, reduction in systemic vascular resistance, and

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<sup>53</sup> See Comments Submitted by Drs. R. Whyatt, D. Hattis, T. Slotkin at 2 (April 2015); EPA OPP, Status of Cumulative Risk Assessment for Organophosphate Pesticides, at 24, 37 (Jan. 15, 2002), *available at* [http://www.epa.gov/oppsrrd1/cumulative/files/guidefinal\\_4-new.pdf](http://www.epa.gov/oppsrrd1/cumulative/files/guidefinal_4-new.pdf).

reduction in systemic blood pressure. During pregnancy total blood volume increases by about 30%, or 1.5 L more than normal, to supply new vascular demands and compensate for blood loss at delivery.<sup>54</sup> Pregnancy is associated with an increase in red blood cell mass (driven by an increase in material erythropoietin production), and a drop in hemoglobin and oxygen binding capacity.<sup>55</sup> Pregnancy increases oxidative metabolism. An increase in platelets during pregnancy is known as “gestational thrombocytopenia.” Plasma volume increases 30-50% during pregnancy.<sup>56</sup> Cardiac hypertrophy occurs during pregnancy as a result of blood volume increases and hormonal changes, making pregnancy similar to exercise-induced cardiac hypertrophy and cardiac stress.<sup>57</sup> Cardiac output is a major parameter in Dow’s PBPK model, along with blood flow for many different organs, and the ratio of tissue to blood flow. RHHRA at 55. Finally, pregnancy causes major changes in xenobiotic metabolism; there are no studies of the effects of pregnancy on the catabolism of chlorpyrifos, its conversion to the oxon, clearance of the oxon, or clearance of either compound from the fetal compartment, which would permit appropriate PBPK modeling of chlorpyrifos exposure in pregnancy.

The SAP that reviewed the model in 2011 identified several concerns related to the lack of age-dependent metabolism in the model, and particularly the lack of data on very young individuals. SAP 2011 at 11, 17. At that meeting, Dr. Dale Hattis presented comments in which he emphasized the need for the Dow model to be calibrated with available human data from known *in vivo* exposures.<sup>58</sup> EPA has obtained no peer review of the model’s use for the neurodevelopmental endpoints of most concern, occurring at exposures too low to trigger the modeled amount of cholinesterase inhibition.

In summary, the Dow model is designed to predict what exposures will produce 10% cholinesterase inhibition in people of various ages and sizes. However, chlorpyrifos causes neurodevelopmental impairments at doses lower than those that produce 10% cholinesterase inhibition. In light of these effects, it is inappropriate to use a model that predicts only 10% cholinesterase inhibition and to reduce safety factors on this basis. Relying on the Dow model is inconsistent with EPA’s finding that neurodevelopmental effects result from lower doses than what produces 10% cholinesterase inhibition. It is contrary to EPA’s own findings and good scientific judgment for EPA to rely on such a model and particularly where it is reducing safety factors based on it.

- B. The Dow Model has Serious Scientific Limitations, Lacks Proper Validation, and was met With Serious Criticisms by EPA’s SAP, Which Have Not Been Addressed Through a Subsequent Peer Review.

To adjust default safety factors, the FQPA requires EPA to have reliable information that

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<sup>54</sup> Chandra S, Tripathi AK, Mishra S, Amzarul M, Vaish AK. Physiological Changes in Hematological Parameters During Pregnancy. *Indian Journal of Hematology & Blood Transfusion*. 2012;28(3):144-146.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3422383/>.

<sup>55</sup> *Id.*

<sup>56</sup> Foley, MR. Maternal cardiovascular and hemodynamic adaptations to pregnancy.

<http://www.uptodate.com/contents/maternal-cardiovascular-and-hemodynamic-adaptations-to-pregnancy>.

<sup>57</sup> Chung E, Leinwand LA. Pregnancy as a cardiac stress model. *Cardiovascular Research*. 2014;101(4):561-570.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3941597/>.

<sup>58</sup> Dale Hattis, Chlorpyrifos Pharmacokinetics – Need and Opportunity for Calibration with Available Human Data from Known *In Vivo* Exposures (Feb. 16, 2011) (EPA-HQ-OPP-2010-0588-0024) (Ex. 3).

the margin of safety will protect infants and children. EPA's policy and best scientific practice require that EPA assess the quality of the science, the extent of the available data, and scientific judgment. It failed to conduct such an assessment and instead subjected Dow's model to far less external peer review and scrutiny than what it imposed on the cohort epidemiologic studies.

The EPA SAP held a meeting in early 2011 to review the Dow PBPK-PD model linked to the Cumulative and Aggregate Risk Evaluation System (CARES model) for dietary exposure.<sup>59</sup> The SAP was considering how the two models, when coupled together, would relate pesticide crop residues to the probability of cholinesterase inhibition in people. SAP 2011 at 8.

The 2011 SAP Report is extremely critical of the Dow PBPK-PD model, raising scientific concerns that are significant enough to prevent EPA from using the model at all, or at least not without addressing the SAP concerns and then having the model re-reviewed by the SAP. This was not done. We summarize the SAP's most serious concerns below.

The Dow model does not handle age-dependent metabolism very well, and is lacking age-specific inputs, particularly for the early life stages that are relevant to the neurodevelopmental health outcomes of interest. "Limited data from younger individuals is an important source of uncertainty." SAP 2011 at 17. In particular, the Panel highlighted the problem of having small numbers of human tissue samples for deriving the estimates of human-specific metabolic rates, and even less information on "very young individuals where the greatest differences from older age groups may exist." SAP 2011 at 17. These data gaps raise the uncertainty with the model, and lower confidence in its ability to model the population of greatest interest.

The dietary assessment is incomplete. The SAP recommended that it include dietary intake over many days, and not just the five days that were in the model at the time of this assessment and that it should include various, correlated, eating behaviors. SAP 2011 at 13. It seems that this concern was ignored, since all the references to the dietary the RHHRA exposure are from 2011 or earlier, and there is no discussion in the RHHRA of this concern.

The Dow model fails to comport with the NHANES government biomonitoring data, which the SAP found to be very "problematic." SAP 2011 at 14. The model authors inaccurately conclude that the model predictions were consistent with the observed exposure values, but the SAP panel disagreed. SAP 2011 at 14. The panel also noted that comparison of the model predictions to occupational exposures in the Curwin et al (2007) data set was inappropriate since exposures other than dietary were included. The panel stated that, "the inability of the CARES dietary exposure model to generate representative input data significantly reduces confidence in the predictability of the [Dow] PBPK-PD model." SAP 2011 at 15. This further puts into question the reliability and accuracy of the model, and there is no evidence EPA has addressed this serious concern that the model does not comport with real-world biomonitoring data.

The Dow model is improperly validated, and uncertainties in the model have not been captured (discussed in more detail above and in the SAP 2011 Report at 13, 14). "The Panel

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<sup>59</sup> Meeting minutes, report, and background material is available in Docket EPA-HQ-OPP-2010-0588 and on the SAP meetings website at: <http://www.epa.gov/scipoly/sap/meetings/2011/021511meeting.html>.

concluded that the sensitivity, variability, and uncertainty analysis were incomplete as outlined in the DAS [Dow AgroSciences] report”, and “strongly recommended” implementing Bayesian analytical methods. SAP 2011 at 16. There is no indication that this was ever done.

The Dow model fails to address human variability. Examples noted by the SAP include the lack of data from younger individuals, small number of human tissue samples to estimate human-specific metabolic rates, and the considerable uncertainty from using in vitro conditions to estimate biotransformation rates in intact human tissue. SAP 2011 at 17. The Dow model uses non-physiological conditions to estimate metabolic rates, especially regarding PON1, which is relevant to chlorpyrifos metabolism and highly variable across individuals. SAP 2011 at 11. This introduces significant uncertainty and unreliability into the model. Yet EPA has not addressed the concern that the model is unable to represent the full range of variability in the population.

The Dow model fails to capture the most vulnerable and highly exposed populations. The SAP recommended that the Dow model be “expanded to accurately predict all pertinent human exposures for target populations of interest (e.g., early life stages), group with specific exposure characteristics (e.g., farm workers and their families), and multiple routes of exposure (e.g., inhalation, dermal, ingestion from food, water, hand-to-mouth activities). These are all important factors for fully and adequately addressing the inter-individual variability and uncertainty. DAS [Dow] employed only two immature age-groups (six months and three years) in their modeling exercise.” SAP 2011 at 17. In her comments at the SAP 2011 meeting, Carol Dansereau of Farm Worker Pesticide Project emphasized that the model failed to capture the real-world experiences of farmworkers and their families exposed to chlorpyrifos and other pesticides, including acute and chronic adverse health effects, impacts on lung function and health, and reduced cognitive ability. Ms. Dansereau also cited research identifying other mechanisms of toxicity besides cholinesterase inhibition. Importantly, she noted the lack of reliable and comprehensive exposure data to support the chlorpyrifos human health risk assessment, whether modeled or not, in the absence of national pesticide use reporting, air monitoring, and health tracking associated with pesticide use.<sup>60</sup> The lack of data weakens both the PBPK-PD model and the RHHRA, and biases towards the null because data gaps in both exposure and harm are *de facto* treated as non-existent, yet EPA never addressed the significant concerns raised by the SAP and in public comments.

The Dow model fails to identify, analyze and disclose sensitivity, variability, and uncertainty within the model. The SAP recommended “a larger systematic effort to simplify the [Dow] model using known assumptions, processes, and interactions in both the exposure and the biological (pharmacokinetic and pharmacodynamic) models.” SAP 2011 at 16. It also recommended that the potential sources of uncertainties, including problems with parameterization and validation, “be captured for assessing the overall model uncertainties if the model is used for risk assessment.” SAP 2011 at 14. This was never done, to our knowledge.

As indicated above, in comments to the SAP, Dr. Dale Hattis stressed the need to calibrate the Dow model using available human data from the cohort studies. Dr. Hattis performed such an analysis using the Columbia data. Hattis, et al. (2015) is far more rigorous than the Dow model in that it uses human-derived data, accounts for the early life stages and

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<sup>60</sup> EPA-HQ-OPP-2010-0588-0023.

neurodevelopmental endpoints of greatest interest, and applies a Bayesian-inspired uncertainty analysis. It illustrates the type of rigorous scrutiny that is imperative before EPA can rely on studies to reduce safety factors, particularly in the face of demonstrated fetal toxicity.

Dow claims to have validated its model using data from the Kisicki study, but this is inappropriate both for the reasons discussed below and because the same data were used to calibrate and validate the model. “The panel commented that since some of the model parameters were calibrated to the Kisicki data, it puts into question the independence of the model-to-data validation.” SAP 2011 at 13. The 2011 SAP instead recommended that a different study should be used for calibration of the Dow model. However, it does not appear that this was ever done, therefore calling into question the validity of the model.

At a minimum, EPA should require a series of direct comparisons of the blood levels predicted by the model and those observed in both the unpublished, industry-sponsored human testing studies, Kisicki, et al. (1999) and Nolan, et al. (1984). This would help illuminate the degree of uncertainty in the model predictions with the aid of these two sets of disparate observations.

The SAP noted that the Dow model lacks transparency, making it difficult for the SAP to assess the validity of the structure and parameters of the model components. SAP 2011 at 11. Given that the SAP determined that the model is improperly validated, and uncertainties in the model have not been captured, EPA lacks a credible scientific basis to rely on the model to reduce or eliminate the traditional safety factors.

In addition to the flaws identified by the SAP, the Dow PBPK-PD model, despite its lofty name, is missing pharmacodynamic (PD) inputs, which reflect what the chemical does at the target organ. For the model, PD really only means the cholinesterase inhibition at the target organ, which is red blood cells, but the epidemiologic evidence shows that this is not a relevant endpoint to account for the neurodevelopmental effects. The model is built primarily of metabolism and pharmacokinetic (PK) data (ADME, absorption, distribution, metabolism, and excretion of the chemical of interest) from in vitro and in vivo (animal) studies. RHHRA at 21. But, the PD inputs are largely absent. Further, there is no adjustment for the uncertainty in the implicit animal to human extrapolation of PD information! This is a very significant gap. This application of the model simply cannot be used if there are no data to inform the PD component. EPA has overlooked this flaw so completely that it has even removed the standard 3-fold pharmacodynamic component of the 10-fold interspecies factor that is normally used to adjust for uncertainty in extrapolations from animal data to human risk. RHHRA at 70.

Given all these scientific questions about the validity and soundness of Dow’s model, EPA lacks an adequate basis for using it to depart from the traditional safety factors. It cannot make the findings compelled by its own policy and the best science that the model is of sufficient quality, is comprehensive and devoid of data gaps, and comports with rigorous scientific standards, such as SAP review and validation. For these reasons, by relying on the model to reduce safety factors, EPA acted arbitrarily, contrary to best scientific practice and the evidence before it, and in disregard of its mandates to select a margin of safety that will protect infants and children.

C. Dow's Model Relies on Deliberate Human Dosing Studies That EPA's Advisors Have Criticized on Scientific and Ethical Grounds, and EPA Has Failed to Subject to Thorough Review Under the Controlling Regulations.

Dow's model uses data from two deliberate human dosing studies: Kisicki, et al., *A Rising Dose Toxicology Study to Determine the No-Observable Effect-Levels (NOEL) for Erythrocytes Acetylcholinesterase (AChE) Inhibition and Cholinergic Signs and Symptoms of Chlorpyrifos at Three Dose Levels* (1999) ("Kisicki") and Nolan et al., *Chlorpyrifos: Pharmacokinetics in Human Volunteers Following Single Oral and Dermal Doses* (1982) ("Nolan"). By relying on the Dow model to eliminate or shrink safety factors, EPA relied on data from human testing, which it cannot do without ensuring the tests meet rigorous ethical and scientific standards. The RHHRA contains no analysis of the ethical flaws in the industry-performed human studies and their lack of scientific rigor. See RHHRA at 52 n.21, 56, 61, 136 n.61. It is arbitrary and capricious and contrary to the governing law for EPA to rely on the Kisicki and Nolan studies because they are ethically deficient and scientifically unsound. At the very least, before EPA may rely on human testing, EPA must resubmit the studies to its review board and explain how they meet ethical and scientific standards that are currently in effect. It cannot rely on stale reviews conducted under inadequate and outdated review regulations. EPA has taken no such actions, and instead, the RHHRA uses the Dow model, which is based on the Kisicki and Nolan studies in violation of FIFRA and EPA's own regulations.

1. Legal Background.

In December 2001, reacting to public controversy over human toxicity studies, EPA asked the National Academy of Sciences to provide guidance on whether "EPA should consider, accept, and rely on data from third-party studies that deliberately expose humans to toxicants." National Academy of Sciences, *Intentional Human Dosing Studies for EPA Regulatory Purposes: Scientific and Ethical Issues* (2004) (the "NAS Report"). The Academy's Report set out to address "the vexing question of whether and, if so, under what circumstances EPA should accept and consider intentional human dosing studies conducted by companies or other sources outside the agency . . . to gather evidence relating to the risks of a chemical . . ." *Id.* at 1. After an extensive review, the Academy concluded that the standards then in place were both too "general" and also too "unclear, indeterminate, inconsistent, and even contradictory" to ensure that intentional human dosing experiments for EPA regulatory purposes would be ethical and scientifically valid. *Id.* at 112. The Academy also concluded that, to ensure such experiments were conducted and used in a scientifically valid manner, EPA must "introduce much greater scientific care and rigor into its process." *Id.* at 66.

Not long after the NAS completed its investigation, EPA announced it would "generally accept" third-party human studies that the Agency deemed scientifically valid "unless there is clear evidence that the conduct of these studies was fundamentally unethical . . . or was significantly deficient relative to the ethical standards prevailing at the time the study was conducted." 70 Fed. Reg. 6661, 6666 (2005). EPA stated it would "consider" the Academy's Report, but made no commitment to follow the Report's seventeen recommendations. *Id.* In June 2005, EPA issued a review draft of an EPA human testing rule, which proposed to adopt many but not all of the NAS recommendations. For example, the draft rule would not have provided criteria or guidelines for determining whether an experiment included "representative study populations" or had "adequate statistical power." The draft rule also would not have

prohibited all third-party intentional dosing toxicity studies for pesticides on pregnant women and children, but would instead have restricted such experiments only if the research had been conducted with an intention to submit the results to EPA under FIFRA or FFDCA § 408.

Congress barred EPA from using appropriated funds to rely on intentional human studies for pesticides until EPA issued a final rule on the subject.<sup>61</sup> In particular, Congress required EPA's rule to ensure that human tests complied with the Nuremberg Code's ethical safeguards for intentionally exposing people to toxic chemicals and the NAS Report's recommendations with respect to scientific credibility and validity.<sup>62</sup>

EPA adopted its final Human Testing Rule on February 6, 2006. A group of environmental and health advocacy organizations challenged the EPA rule's failure to prohibit intentional human dosing pesticide experiments on children and pregnant women; failure to ensure consistency with the Nuremberg Code's and FIFRA's informed consent requirements; and failure to ensure consistency with the NAS recommendations, including ensuring that human dosing studies meet rigorous scientific standards, not pose risks to human subjects absent overriding health or environmental benefits, and comport with ethical standards prevailing when the studies were conducted. The environmental and public health advocates also challenged EPA's addition of the critical word "significantly" to the rule allowing EPA to consider a study that was ethically deficient when conducted, so long as the study was not "*significantly* deficient" under then-prevailing standards.<sup>63</sup> After briefing and oral argument, the case settled, and EPA agreed to promulgate revised human testing regulations that would remove the word "significantly" from the standard, would prohibit testing on children and pregnant women, and would add a number of other protections.<sup>64</sup>

## 2. The Strengthening of the Regulations Since the HSRB Review of the Dow Human Dosing Studies.

In 2013, EPA issued regulations consistent with the settlement agreement that require a stronger showing for EPA to rely on old human testing studies. The new regulations maintained the same structure as before, providing for a rigorous review process for new study designs that rely on human subjects and a separate set of standards governing EPA's use of studies conducted prior to the issuance of the regulations. *See* 40 C.F.R. §§ 26.1101, .1701. For old studies, the 2013 regulations have both an ethics and a science component, and studies must pass both tests for their use to be acceptable. 40 C.F.R. § 26.1703, .1704. Under the 2013 regulations, "EPA must not rely on data from any research subject to this section if there is clear and convincing evidence that . . . [t]he conduct of the research was deficient relative to the ethical standards prevailing at the time the research was conducted." 40 C.F.R. § 26.1704. That rule prohibits EPA from using studies that are merely ethically deficient, i.e. no further finding is required. *Id.* The word "significantly" was dropped in the 2013 promulgation of the regulations, meaning that any ethical deficiency can be sufficient to block EPA's use of a human study. EPA also is prohibited by FIFRA from using the results of tests that "use any pesticide in tests on human

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<sup>61</sup> Department of Interior, Environment, and Related Agencies Appropriations Act, Pub. L. No. 109-54, § 201 (2006).

<sup>62</sup> *Id.*

<sup>63</sup> *See NRDC v. E.P.A.*, 06-0820-ag, Opening Br. at 5, 41 (Oct. 4, 2006) (emphasis in original).

<sup>64</sup> *See NRDC*, Joint Mot. Stay Implement Settlement Agreement (Dec. 18, 2011).

beings unless such human beings (i) are fully informed of the nature and purposes of the test and of any physical and mental health consequences which are reasonably foreseeable therefrom.” 7 U.S.C. § 136j(a)(2)(P).

Likewise, in assessing the scientific validity and reliability of human testing studies, EPA must evaluate whether “the research was designed and conducted in accordance with appropriate scientific standards,” the representativeness of the study subjects, the study’s statistical power, and “whether a dose level in the study gave rise to a biological effect.” *Id.* at § 26.1703(1)-(4). These requirements are designed to ensure that a study is scientifically valid for EPA to rely on it. *Id.*

All human studies must be submitted to EPA’s Human Studies Review Board (“HSRB”), which considers the “the scientific merits and ethical aspects of the completed research, and must apply the appropriate standards” in EPA’s human testing regulations. *Id.* at §§ 26.1604(b)(2), 26.1607. EPA established the HSRB in 2006 to advise EPA on the ethics and scientific soundness of studies using human subjects. 71 Fed. Reg. 6071, 6072 (2006).

### 3. The Kisicki and Nolan Studies

Both the Kisicki and the Nolan studies are intentional human dosing studies that involved exposing human beings to chlorpyrifos for the purpose of reducing public health protections with respect to chlorpyrifos. The Kisicki study, conducted in 1999, was sponsored by Dow AgroSciences to determine the no-observable effect level (“NOEL”) for cholinesterase inhibition in human volunteers. It exposed through oral dosing a total of sixty people, “college students and members of the community at large,” to chlorpyrifos.<sup>65</sup>

John M. Carley, EPA’s Human Research Ethics Review Officer, twice found the Kisicki study significantly deficient on ethical grounds, even under the earlier, less-rigorous standard. The First Carley Report contained a detailed analysis of informed consent lapses, which spanned 11 pages, and concluded that “EPA is forbidden by 40 CFR § 26.1704 to rely on the Kisicki *et al.* study,” and under FIFRA.<sup>66</sup> After Dow submitted additional information about the informed consent failures that led Carley to the adverse ethics finding, Carley reevaluated the study in a six-page report, but reached the same conclusion as before.<sup>67</sup>

The HSRB, however, disagreed with Carley’s conclusions. It acknowledged substantial and unresolved ethical questions, but opined that the Kisicki study was not significantly flawed.<sup>68</sup> The HSRB went on to find four scientific deficiencies in the study, two of which remain unresolved.<sup>69</sup>

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<sup>65</sup> John M. Carley, Human Research Ethics Review Officer, *Ethics Review of Chlorpyrifos Human Toxicity Study* at 2-3 (May 29, 2009) (“First Carley Kisicki Report”).

<sup>66</sup> *Id.* at 1, 10.

<sup>67</sup> John M. Carley, Human Research Ethics Review Officer, Supplemental Ethics Review of Chlorpyrifos Human Toxicity Study by Kisicki, et al. (Aug. 7, 2009) (“Second Carley Kisicki Report”).

<sup>68</sup> EPA Human Studies Review Board, *June 24-25, 2009 EPA Human Studies Review Board Meeting Report* at 23-25 (“HSRB Report”).

<sup>69</sup> *Id.*; EPA Human Studies Review Board, *April 13-14, 2011 EPA Human Studies Review Board Meeting Report*, App’x 1 (“HSRB Report App’x 1”).

In 2014, a different EPA ethics advisor, Kelly Sherman, evaluated the Kisicki study under the new ethics regulations. The Sherman report provides only a summary analysis, relies on the HSRB's conclusions, and never discussed the Second Carley Report in which Carley continued to find informed consent shortcomings in the Kisicki study even after reviewing the additional information provided by Dow.<sup>70</sup> Additionally, EPA has not sent the Kisicki study back to the HSRB for evaluation under the new regulations.

After the HSRB questioned the scientific validity of the Kisicki Report, the SAP evaluated the Kisicki study as part of its review of the PBPK model and reiterated a number of unresolved scientific concerns.<sup>71</sup> The SAP identified uncertainties in the Kisicki study and the model. Dow responded to two of the issues raised by the HSRB, which satisfied the concerns of both the SAP and the HSRB.<sup>72</sup> Neither Dow nor EPA took any steps to address the HSRB's remaining two concerns about the reliability and utility of the blood and urine Kisicki measurements of chlorpyrifos and/or its principal metabolite 3,5,6-trichloropyridinol (TCP), for risk assessment purposes. These concerns are highly relevant because the Kisicki study was used to calibrate the PBPK/PD model for blood measurements. In other words, it was used for precisely the purpose the HSRB said it should not be used.<sup>73</sup>

The Nolan study, conducted from 1981 to 1982, was designed to evaluate the fate of orally and dermally administered chlorpyrifos. It involved only six adult male volunteers, all employees of Dow AgroSciences. EPA's ethics advisor evaluated the Nolan study in 2009, and the HSRB also evaluated it that same year.<sup>74</sup> It does not appear that the Nolan study has been evaluated under the currently applicable review standard for intentional human dosing studies. Instead, EPA and the HSRB reviewed the Nolan study only under the more-lenient "significantly deficient" standard.<sup>75</sup>

#### 4. EPA Is Prohibited from Relying on Both Human Studies to Reduce Safety Factors.

In order to rely on human testing studies, EPA must submit the studies to the HSRB for review and determine that the studies can be used under the applicable regulatory criteria and FIFRA's standard. EPA has not taken those mandatory steps that apply to its use of human testing in risk assessments, like the RHHRA. The HSRB has reviewed the Kisicki and Nolan studies only under the old regulations governing EPA's reliance on intentional human dosing studies. EPA's ethics advisor evaluated the Kisicki study under the new standard, but that report fails to discuss Carley's contrary findings when Carley reviewed the study based on the supplemental information submitted by Dow. EPA must address the conflicting conclusions

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<sup>70</sup> Kelly Sherman, Human Studies Ethics Review Officer, Supplemental Ethics Review of Chlorpyrifos Human Toxicity Study by Kisicki, *et al.*, at 4-6 (Dec. 12, 2014) ("Sherman Report").

<sup>71</sup> See FIFRA SAP Meeting Held February 15-17, 2011 on the Scientific Issues Associated with Chlorpyrifos Physiologically Based Pharmacokinetic and Pharmacodynamic (PBPK/PD) Modeling Linked to Cumulative and Aggregate Risk Evaluation System (CARES), at 13-14 (May 12, 2011) ("SAP PBPK Minutes").

<sup>72</sup> *Id.* at 35; April 13-14, 2011 EPA HSRB Meeting Report, Appendix 1 (May 26, 2011), available at <http://www.epa.gov/hsrb/files/meeting-materials/apr-13-14-2011/appendix1.pdf>.

<sup>73</sup> See HSRB Report App'x 1.

<sup>74</sup> John M. Carley, Human Research Ethics Review Officer, *Ethics Review of Chlorpyrifos Pharmacokinetics Study* at 1 (May 29, 2009) ("Carley Nolan Report").

<sup>75</sup> See HSRB Report at 12-14; Carley Nolan Report at 5-6.

reached by its ethics advisors with respect to the Kisicki study's ethical lapses. And the HSRB found scientific deficiencies that have not been addressed and that preclude use of the Kisicki study for the very purpose for which the RHHRA uses it. Using the Kisicki study in the face of the unresolved scientific deficiencies and without reconciling the conflicting ethical conclusions is arbitrary and unconscionable in light of the health risks at stake.

The ethical flaws that led Carley to find that EPA was forbidden from using the Kisicki study are very troubling and warrant EPA's scrutiny. First, the consent form suggested that one side effect of being intentionally dosed with chlorpyrifos could be "improved performance on numerous tests of mental function."<sup>76</sup> That statement is a flat-out falsehood. There is absolutely no basis for representing that intentionally dosing humans with chlorpyrifos might improve mental function. Similarly, Carley found "incomplete and potentially confusing passages concerning the risks of ingesting chlorpyrifos."<sup>77</sup> Carley also found it unacceptable that the subjects did not give informed consent prior to being screened and that subjects may not have had "sufficient opportunity to consider whether or not to participate."<sup>78</sup> Because of the weight of these ethical failings, Carley found the study's conduct was "significantly deficient relative to the FDA regulations cited as governing its ethical conduct" along with FIFRA.<sup>79</sup>

Also troubling—and unexplained—is what happened with one study participant who showed a significant reduction in RBC AChE. That subject withdrew from the study, but there is apparently no documentation as to why that subject withdrew, whether her or his monitoring continued, and whether she or he experienced medical problems resulting from the intentional dosing.<sup>80</sup> There is also no indication that Dow reported that deviation to the HSRB. This is even more important because that subject appears to have been the only one to have shown cholinesterase inhibition, leading one reviewer to characterize the study as of "poor design."<sup>81</sup>

The HSRB, in its brief consideration of the ethics of the Kisicki study, disagreed with Carley's conclusions but nonetheless highlighted several ethical problems with the study.<sup>82</sup> The HSRB did not explain why it reached the opposite conclusion reached by Carley, and instead merely offered several possible (but unsubstantiated) reasons why the study may not have been ethically deficient. For example, the HSRB Report found that "[i]t is possible, for example, that the deficiencies noted in the logs are the result of sloppy record keeping rather than a serious impaired participant recruitment, consent and screening process."<sup>83</sup> Yet the HSRB did not describe its basis for that suggestion. The HSRB also found that some documents submitted to EPA

raise questions as to whether all potential study participants or alternates underwent voluntary informed consent prior to screening. Although the failure to

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<sup>76</sup> First Carley Kisicki Report at 4.

<sup>77</sup> *Id.* at 5.

<sup>78</sup> *Id.* at 6-7.

<sup>79</sup> *Id.* at 9.

<sup>80</sup> *See id.* at 7.

<sup>81</sup> Doherty, Reviews of the Coulston (1972), (Nolan (1982) and Kisicki (1999) studies with human volunteers including comments from the Human Studies Review Board (HSRB) meeting (June 2009) (Jan. 2010), available at <http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0242>.

<sup>82</sup> HSRB Report at 24-25.

<sup>83</sup> *Id.* at 24.

have potential participants and alternates sign an informed consent form prior to screening could be a violation of applicable FDA regulations, given these circumstances it does not appear that the informed consent process was serious [sic] impaired.<sup>84</sup>

The HSRB went on to find that “various sentences in the informed consent form . . . appear inappropriate, such as characterizing the study compound as one that improves performance on tests of mental functioning.”<sup>85</sup> The HSRB, remarkably, then found that the informed consent document “provided prospective subjects with an accurate and understandable picture.”<sup>86</sup> The HSRB did not provide any basis for its conclusory determination that despite the many informed consent concerns, there were no ethical violations, and it is likely that these ethical deficiencies would prohibit use of the study under the revised regulations. The Sherman report provided an even shorter analysis and did not discuss Carley’s second report.<sup>87</sup>

Before relying on the Kisicki study or a model based on it, EPA must resubmit the Kisicki study to the HSRB for a review under the stronger regulatory standard now in effect. EPA also must address the serious ethical concerns raised in both Carley reports, which contain far more analysis than the HSRB’s and Sherman’s cursory dismissals, before it can use a human testing study in which significant ethical problems have been identified by EPA’s own ethics advisor even under the prior, more lenient standard.

It is also arbitrary for EPA to use the Kisicki study in light of the two outstanding scientific problems uncovered by the HSRB. As stated above, the scientific concerns focused on the reliability and utility of the blood and urine measurements for risk assessment purposes. Yet that is precisely how Dow’s model and the RHHRA use the Kisicki study. The entire purpose of Dow’s model is to predict 10% cholinesterase inhibition in red-blood cells. EPA cannot credibly justify using the Kisicki study for this purpose when the HSRB questioned the reliability of the study’s blood measurements.

EPA is also prohibited from relying on the Nolan study. Before EPA can use the Nolan study, EPA and the HSRB must reevaluate it under the current regulatory standard. When EPA’s ethic advisor reviewed the Nolan study, he raised concerns even though he ultimately found that EPA could rely on the study under the earlier, more lenient standard. Most troubling, Carley noted that five of the six human subjects in the study were employees of the study’s sponsor (Dow) and therefore may have been vulnerable to undue influence in their decisions to participate in the research.<sup>88</sup> Likewise, the informed consent forms given to subjects did not characterize the nature and purpose of the research or its risks.<sup>89</sup> Carley concluded that there were remaining “significant gaps in the documentation of the ethical conduct of this research,” despite concluding that reliance on that study was not prohibited under the earlier standard.<sup>90</sup> The HSRB reached the same conclusion.<sup>91</sup> In light of these concerns, before EPA can use the

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<sup>84</sup> *Id.* at 25.

<sup>85</sup> *Id.*

<sup>86</sup> *Id.*

<sup>87</sup> See Sherman Kisicki Report.

<sup>88</sup> Carley Nolan Report at 5-6.

<sup>89</sup> *Id.* at 5.

<sup>90</sup> *Id.*

<sup>91</sup> HSRB Report at 13-14.

Nolan study, it must resubmit its ethics advisory and the HSRB for evaluation under the 2013 promulgation of EPA's human testing regulations.

Intentionally dosing people to reduce public health protection raises serious moral issues. Indeed, the use of a human testing study (with a dubious informed consent process) to reduce protections to humans is on its face contrary to long-established EPA ethical guidance.<sup>92</sup> Congress has insisted on informed consent in any study used by EPA, and EPA's human testing rule likewise calls for review of studies in accordance with ethical standards in place at the time of the study and minimal scientific validity and reliability before EPA can rely on such studies. EPA has fallen short of these standards. It has not subjected the Kisicki and Nolan studies to adequate review under the stronger regulatory standards now in place. Nor has it made any findings as to whether these studies pass muster under those standards with a full consideration of all past findings and in light of the changed regulations. It has received conflicting reviews from its ethics advisors and the HSRB on ethical deficiencies in the Kisicki study, which it has not addressed. And the HSRB found scientific deficiencies in the Kisicki study that are precisely relevant to how EPA is using that study and yet EPA is absolutely silent as to the HSRB's reliability concerns. In doing so, EPA has violated its own regulation, which provides that: "EPA must not rely on data from research subject to this subpart unless EPA determines that the data are relevant to a scientific or policy question important for EPA decisionmaking, that the data were derived in a manner that makes them scientifically valid and reliable." 40 C.F.R. § 26.1703. Likewise, EPA has failed to fulfill its obligation to submit all studies to the HSRB for review under the appropriate review standard. 40 C.F.R. §§ 26.1604(b)(2), 26.1607. For all of these reasons, EPA is prohibited from using the human testing studies in the RHHRA, which precludes it from relying on Dow's model to reduce the traditional safety factors.

#### IV. EPA IS FAILING TO PROTECT CHILDREN AND OTHER BYSTANDERS FROM CHLORPYRIFOS.

Through petitions and lawsuits, farmworker advocates have pressed EPA to protect children and other bystanders from spray drift. Ever since EPA determined home uses had to be cancelled to meet its obligation to protect children from aggregate exposures to chlorpyrifos, advocates have sought to extend the same types of protections to the children of farmworkers and rural communities that bear the brunt of pesticide drift. In response to the Kids Petition, EPA acknowledged its legal obligation under the FQPA to protect children from pesticide drift. Similarly, in its preliminary human health risk assessment for chlorpyrifos, EPA acknowledged that it has a legal obligation to protect children from drift and that monitoring both by communities and California regulators has revealed chlorpyrifos exposures at schools and other places children congregate. EPA has finally and appropriately assessed drift exposures to children and other bystanders and has established safeguards in the form of use reduction, buffers, and other constraints on pesticide applications to prevent such exposures. This approach sets a precedent that should be followed for all drift-prone pesticides and particularly for organophosphates and carbamates, which cause acute pesticide poisonings and other adverse health effects.

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<sup>92</sup> See Science Advisory Board and FIFRA Scientific Advisory Panel, Comments on the Use of Data from the Testing of Human Subjects at 2 (Sept. 11, 2000), available at <http://www.epa.gov/scipoly/sap/meetings/1999/november/ec0017.pdf>.

The way EPA has addressed drift in the RHHRA, however, is fatally flawed in four respects. First, EPA has failed to account for all drift exposures and has afforded insufficient protection for inhalation exposures. Second, the no-spray buffers imposed around schools, homes, playgrounds, and other places children and other bystanders congregate are far too small, in most instances only 10-feet. Third, EPA has ignored exposures from chlorpyrifos-contaminated dust. Fourth, EPA has dismissed volatilization exposures altogether based on two recent Dow studies, again evincing a willingness to embrace Dow studies that have not undergone peer review and that have significant flaws.

A. Chlorpyrifos Causes Poisonings of Children and Other Bystanders Every Year.

Before turning to each of these flaws, EPA's approach is at odds with the extensive empirical evidence that chlorpyrifos causes pesticide poisonings when it drifts through the air or volatilizes. Not only is chlorpyrifos one of the most widely used pesticides, but chlorpyrifos has repeatedly been among the top pesticides causing acute poisonings of workers, their families, and others who live near places where it is applied. Exposure incidents are well-documented. A study of 11 states found 2,945 documented cases of pesticide drift exposure from 1998-2006.<sup>93</sup> Among these cases, the majority were non-occupational exposures.<sup>94</sup> Chlorpyrifos was the third-most common active ingredient in high-severity drift cases.<sup>95</sup> One seven-year study of agricultural worker exposure incidents in California found that drift was the most common factor in exposure cases, occurring in 63% of all exposures.<sup>96</sup> California's pesticide exposure incident database contains 356 chlorpyrifos exposure incidents from 1992 through 2009.<sup>97</sup> Spray drift is responsible for 56% of the chlorpyrifos incidents reported to the California Pesticide Illness Surveillance Program from 1999 to 2008.<sup>98</sup> Pesticide incident data in Washington State indicates drift as the source of the greatest number of application complaints and exposure events from 2007-2011.<sup>99</sup>

Several air monitoring studies near application sites have found chlorpyrifos present in levels exceeding the level of concern for children. Air monitoring in Lindsay, California, found chlorpyrifos levels over six times the level of concern for children in 2004 and 2005.<sup>100</sup> A 2006 air monitoring study in Washington's Yakima Valley found chlorpyrifos present in the air for all 40 days studied and above acceptable levels for 14 days.<sup>101</sup> A study in California's San Joaquin

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<sup>93</sup> Soo-Jeong Lee et. al., *Acute Pesticide Illnesses Associated with Off-Target Pesticide Drift from Agricultural Applications — 11 States*, 119 *Envtl. Health Persp.* 1162 (2011) (Ex. 4).

<sup>94</sup> *Id.* at 9.

<sup>95</sup> *Id.* at 17.

<sup>96</sup> Geoffrey M. Calvert et. al., *Acute Pesticide Poisoning Among Agricultural Workers in the United States, 1998–2005*, 51 *Am. J. Indus. Med.* 883 (2008) (Ex. 5).

<sup>97</sup> <http://www.cdpr.ca.gov/docs/whs/pisp.htm>.

<sup>98</sup> *Chlorpyrifos Evaluation of the Potential Risks from Spray Drift and the Impact of Potential Risk Reduction Measures* at 11 (July 2012).

<sup>99</sup> Washington State Department of Health, *Pesticide Data Report* (June 2013), available at <http://www.doh.wa.gov/Portals/1/Documents/Pubs/334-319.pdf> (Ex. 6).

<sup>100</sup> Katherine Mills and Susan Kegley, *Pesticide Action Network North America, Air Monitoring for Chlorpyrifos in Lindsay, California* (July 14, 2006) (Ex. 7).

<sup>101</sup> Farm Worker Pesticide Project & Pesticide Action Network North America, *Poisons on the Wind: Community Air Monitoring for Chlorpyrifos in the Yakima Valley* (Dec. 2006).

Valley found chlorpyrifos present in one-third of all ambient air samples collected.<sup>102</sup> The California Air Resources Board found chlorpyrifos present in 74% of air samples taken in a study done at elementary schools and other sites in Tulare County, California.<sup>103</sup>

Peer-reviewed studies support the conclusion that chlorpyrifos carried through drift results in exposure to vulnerable populations, including children. A 2011 peer-reviewed study found that urine samples of children ages 6 to 24 months were more likely to have six organophosphate metabolites the closer the child lived to application sites.<sup>104</sup> A similar proximity effect has been observed in household dust, which suggests that pesticides remain in the areas to which they have drifted.<sup>105</sup> This result also suggests that agricultural workers, who live near their worksites, take home pesticide residue on their skin and clothing.<sup>106</sup>

While the evidence confirms that chlorpyrifos drift causes acute poisonings resulting from off-site drift, the evidence represents just the tip of the iceberg due to under-reporting of pesticide incidents. In its proposed worker protection standard revisions, EPA rightly acknowledges that “[u]nderreporting of pesticide incidents is a challenge,” and assumes that only 25% of acute pesticide incidents are reported.<sup>107</sup> Farmworkers are deterred from reporting pesticide illnesses due to fear of retaliation, health care workers often lack the training to diagnose illnesses from pesticide exposures, and there is no national pesticide incident reporting system that could be utilized by clinicians and others who work with farmworkers.<sup>108</sup> Other factors contributing to underreporting include language barriers, lack of access to medical care, lack of information for workers about hazards they face, workers’ lack of awareness of poisoning symptoms, and lack of health care professionals trained in diagnosis of pesticide illness.<sup>109</sup>

EPA’s RHHRA calls for the most meager safeguards in the face of pervasive pesticide poisonings from chlorpyrifos, not to mention the neurodevelopmental effects. It is hard to imagine how 10-foot buffers, which are all that is being done in connection with ground spraying, will make a difference for the children and other bystanders in harm’s way.

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<sup>102</sup> Sean Gray et al., Environmental Working Group, *Every Breath You Take: Airborne Pesticides in the San Joaquin Valley* (Jan. 2001) (Ex. 8).

<sup>103</sup> California Air Resources Board, *Final Report for the 1996 Chlorpyrifos Monitoring in Tulare County* (Apr. 13, 1998) (Ex. 9).

<sup>104</sup> Asa Bradman et al., *Determinants of Organophosphorus Pesticide Urinary Metabolite Levels in Young Children Living in an Agricultural Community*, 8 *Int. J. Env'tl. Res. Public Health* 1061 (2011) (Ex. 10).

<sup>105</sup> Robert B. Gunier et al., *Determinants of Agricultural Pesticide Concentrations in Carpet Dust*, 119 *Env'tl. Health Persp.* 970 (2011) (Ex. 11).

<sup>106</sup> *Id.*

<sup>107</sup> *Worker Protection Standard Revisions*, 79 *Fed. Reg.* 15,444, 15,453, 15,459 (Mar. 19, 2014).

<sup>108</sup> U.S. Gen. Accounting Office, *Pesticides on Farms: Limited Capability Exists to Monitor Occupational Illnesses and Injuries* 9 (1993), available at <http://archive.gao.gov/t2pbat4/150612.pdf>; see also Geoffrey M. Calvert et al., *Acute Pesticide Poisoning Among Agricultural Workers in the United States*, 199802995, 51 *AM.J. INDUS. MED.* 883, 894-95 (2008) (discussing reasons why agricultural workers are deterred from seeking health care and why health care professionals misdiagnose acute pesticide poisonings).

<sup>109</sup> *Id.*; see also Earthjustice, *Annotated Bibliography: Underreporting of Agricultural Pesticide Illness and Injury* (Dec. 12, 2013), which was submitted to EPA as part of the comments on the Worker Protection Standard Revisions by Earthjustice, Farmworker Justice, California Legal Assistance Foundation, PAN, PCUN, UFW, and others.

B. EPA Fails to Account for all Drift Exposures and Affords Insufficient Protection From Inhalation Exposures.

EPA conducted a residential exposure assessment to provide a basis for protecting children and other bystanders from spray drift. This assessment is grossly under-protective because it is based on 10% cholinesterase inhibition, not the neurodevelopmental impairments that occur at a lower dose, and it is based on the shrunken safety factors adopted based on the Dow model.

To reduce concerns for children and other bystanders from spray drift, Dow proposed reduced application rates for aerial spraying and no-spray buffers around sensitive sites, such as school grounds, playfields, and residential buildings, frequented by children and other non-occupational bystanders. EPA then conducted an assessment of whether the mitigation package would eliminate the risks of concern. Its analysis, however, suffers from several shortcomings that lead it to under-estimate exposure.

First, it is based primarily on dermal exposure to residues that deposit on the ground and fails to account fully for inhalation exposures. EPA does model inhalation exposures, but only for aerial spraying for agricultural applications. It fails to account for any inhalation exposures from air blast and ground boom applications.<sup>110</sup> Even as to aerial applications for which it modeled inhalation risk, EPA did not aggregate the dermal exposure from lawn deposition and the inhalation exposures because it states that the two types of exposure have different attributes and uncertainties.<sup>111</sup> It simply ignores one of the pathways through which children may be exposed, rather than assume and account for the exposure that its model shows will occur. And the RHHRA does not consider exposures from aerosol inhalation in its re-assessment of the mitigation package, although that omission is not explained.<sup>112</sup>

Second, in assessing inhalation exposures, the preliminary HHRA used a 24-hour exposure duration. Without any explanation, EPA's spray drift mitigation evaluation uses only a two-hour exposure duration, despite identifying a 14-hour half-life for chlorpyrifos.<sup>113</sup> If the timeframe is designed to reflect how long people will be at exposed sites, it is a gross under-estimation. Infants, young children, and elderly with limited mobility are likely to remain at their homes or in their yards where they can be exposed for far longer than 2-hour intervals, and children may move from their schools or day cares to their homes, all of which may be near fields where pesticides are being sprayed.

Third, in modeling dermal exposures, EPA assumes that pesticide residues spread evenly across a 50-foot wide lawn, even though its AgDRIFT model shows that the amount of pesticide residues diminishes as distances from the application site increase. This assumption leads EPA to under-estimate exposures to children playing closest to the field where the pesticide has been applied.

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<sup>110</sup> Chlorpyrifos Evaluation of the Potential Risks from Spray Drift and the Impact of Potential Risk Reduction Measures at 17-19, 31 (July 2012).

<sup>111</sup> *Id.* at 31.

<sup>112</sup> Appendix D of the updated Occupational and Residential Exposure Assessment presents only dermal MOEs.

<sup>113</sup> *Id.* at 30-33.

Fourth, the RHHRA appropriately has an aggregate exposure assessment to comply with its FQPA obligation to consider all exposures to a pesticide whether from food, water, or activities like playing on a treated field. That assessment considers food, drinking water, and residential exposures from golf courses and mosquito abatement, but it contains no assessment of bystander exposures and yet children and other bystanders in agricultural communities face some of the greatest exposures to pesticides. It is possible that EPA omitted bystander exposures to spray drift based on the spray drift mitigation put in place in 2012. However, there is no evidence that EPA has validated the mitigation measures to ensure they are, in fact, protecting children from pesticide exposures, even though its 2012 evaluation of the mitigation measures (at 43) stated that “[d]ata to confirm the efficacy of any [drift reduction] measures implemented to reduce risk estimates associated with spray drift from chlorpyrifos should be developed.”

Fifth and most troubling, the way EPA is treating inhalation risk appears to condone applications of pesticides directly onto people. EPA models the pesticide aerosols that will be 3-5 feet off the ground where people will inhale them in the first two hours after aerial applications. Its modeling shows that the pesticide aerosols will travel to sensitive sites where people congregate, and EPA has required no safeguards to prevent applications when people are actually in the schools, playfields, parks, backyards and other sensitive sites in the impact zone at the time of application. While pesticide labels prohibit applying the pesticide in a manner that will directly contact people, EPA must require more prescriptive label instructions when it knows an application is otherwise likely to result in direct drift, as discussed below in connection with worker risks. EPA’s evaluation of the spray drift mitigation package focuses instead on whether people will be exposed to chlorpyrifos at levels above EPA’s permissible exposure levels. EPA must re-evaluate bystander risks and the mitigation package to ensure that spraying chlorpyrifos will not result in direct drift onto children and others.

C. The Buffers Are Far Too Small to Prevent Harmful Pesticide Drift Exposure.

By December 2012, labels included agreed-upon measures that reduced application rates for aerial applications and established no-spray buffers around sensitive sites, which are defined as areas frequented by non-occupational bystanders, especially children.<sup>114</sup> The labels indicate that such sites include “residential lawns, pedestrian sidewalks, outdoor recreational areas such as school grounds, athletic fields, parks and all property associated with buildings occupied by humans for residential or commercial purposes. Sensitive sites include homes, farmworker housing, or other residential buildings, schools, day care centers, nursing homes, and hospitals.”<sup>115</sup> We endorse no-spray buffers as a proven and effective safeguard to prevent harmful pesticide exposures.

However, the no-spray buffers established for chlorpyrifos are far too small. The table below lays out the buffers put into place in 2012:

**Table C: Buffer Distances from Sensitive Sites**

Application rate (lb ai/A)	Nozzle Droplet Type	Required Setback (Buffer Zones) (feet)		
		Aerial	Airblast	Ground
>0.5 – 1	coarse or very coarse	10	10	10

<sup>114</sup> Spray Drift Mitigation Decision for Chlorpyrifos (059101) (July 2012).

<sup>115</sup> *Id.* at 3.

>0.5 - 10	medium	25	10	10
>1 - 2	coarse or very coarse	50	10	10
>1 - 2	medium	80	10	10
>2 - 3	coarse or very coarse	80 <sup>1</sup>	10	10
>2 - 3	medium	100 <sup>1</sup>	10	10
>3 - 4	medium or coarse	NA <sup>2</sup>	25	10
>4	medium or coarse	NA	50	10

<sup>1</sup>Aerial application of greater than 2 lb ai/A is only permitted for Asian Citrus Psylla control, up to 2.3 lb ai/A.

<sup>2</sup>NA is not allowed.

A distance of only 10 feet is required for all ground boom applications, most airblast spraying (the 25- and 50-foot buffers apply only to the highest application rates used in airblast spraying), and even some aerial spraying.

These buffers are inadequate for four reasons. First, they are premised on EPA’s assumption that direct drift does not happen. As discussed above, EPA assumes that it has done all that it needs to do to prevent direct drift onto people by prohibiting such drift on the pesticide label. But direct drift does happen with alarming frequency, and EPA’s modeling shows that it will happen in the absence of larger buffers. By way of example, the Centers for Disease Control and Prevention found that the poisoning of 20 Latino workers in a cherry orchard was caused by off-target drift from an air blast application of a pesticide mixture at a neighboring pear orchard. The workers were dispersed with their distance from the edge of the pear orchard ranging from 30 to more than 350 feet.<sup>116</sup> And California’s poisoning incident reports contain numerous incidents from chlorpyrifos alone at distances from 80-feet up to ½ mile from the field where the application occurred.<sup>117</sup> In the face of evidence and findings like those in the CDC report, EPA must impose sufficient safeguards to ensure that spray drift will not continue to reach schools, playgrounds, homes, and other places people may be located.

Second, EPA conducted its drift assessment and established buffer sizes without taking into account the demonstrated neurodevelopmental damage to children from chlorpyrifos. It set buffers to guard against cholinesterase inhibition, even though it has found that damage to children’s brains occurs at lower doses. It did this by reducing longstanding safety factors based on the PBPK model and the flawed human studies on which that model is based. As shown above, EPA should have retained the traditional safety factors for variations among people and differences between people and animals, and it should have not only retained the FQPA safety factor, but should have expanded it to account for the prenatal toxicity to children and the uncertainties surrounding at what exposures those neurodevelopmental impacts occur. Had it done so, it would have determined that larger buffers are needed to protect children. Instead, it asserts that smaller buffers than those put in place in 2012 might be sufficient. RHHRA at 82.

Third, EPA did not validate that the buffers are working. Its 2012 evaluation of the mitigation measures (at 43) stated that “[d]ata to confirm the efficacy of any [drift reduction] measures implemented to reduce risk estimates associated with spray drift from chlorpyrifos

<sup>116</sup> Centers for Disease Control and Prevention, *Worker Illness Related to Newly Marketed Pesticides – Douglas County, Washington*, 2014, 64 Morbidity and Mortality Wkly. Rep. 42 (Jan. 23, 2015) .

<sup>117</sup> App’x 1.

should be developed.”<sup>118</sup> When proposing mitigation measures, agencies must evaluate their effectiveness.<sup>119</sup>

Fourth, the buffers are smaller than buffers put in place by EPA or other regulators without any rational basis. EPA’s 2001 IRED for chlorpyrifos is instructive. In that decision (at 108, 113), EPA required the following label statements:

Do not apply this product in a way that will contact workers or other persons, directly or through drift.

Do not allow spray to drift from the application site and contact people, structures people occupy at any time and the associated property, parks and recreation areas, nontarget crops, aquatic and wetland areas, woodlands, pastures, rangelands, or animals.

The first statement pertains to direct drift, while the second statement goes further to prohibit drift that impacts people, waterbodies, natural areas, and crops. Interestingly, EPA required no buffers to prevent drift exposures to people, but it did establish no-spray buffers around “rivers, natural ponds, lakes, streams, reservoirs, marshes, estuaries, and commercial fish ponds.” The buffers range in size from 25 feet for ground boom applications, 50 feet for airblast applications, and 150 feet for aerial spraying. In addition to the buffers, EPA imposed other restrictions like wind speeds, spray heights, and spray size (fine, medium or coarse).

**Table D. Proposed No-Spray Buffer Zones around Water Bodies**<sup>120</sup>

Application Method	Required Setback (No-spray Zone)
Ground Boom	25 feet
Chemigation	25 feet
Orchard Airblast	50 feet
Aerial (fixed-wing or helicopter)	150 feet

In response to a Freedom of Information Act request, we obtained EPA’s review of Dow’s proposed label changes to reduce water contamination from chlorpyrifos, which EPA had found exceeded its levels of concern for aquatic life. For some uses, Dow was proposing to increase intervals between applications, delete some application methods, and decrease application rates. For all uses, Dow agreed to require the no-spray buffers that made their way onto the labels. EPA quantified the reduction in risk that would occur with the buffers, limiting its analysis to spray drift; it assumed that runoff would continue to occur at the same levels. It analyzed five scenarios and found for each that the buffers would reduce but not eliminate amounts of chlorpyrifos in surface waters that exceeded EPA’s levels of concern for fish and other aquatic life. The reductions in risk ranged from 1% with 50 foot buffers for citrus to 80% for 50-foot buffers for cotton. The risk reductions generally increased with the size of the buffers. For example for corn, a 25-foot buffers resulted in a 44% risk reduction while a 150-

<sup>118</sup> Chlorpyrifos Evaluation of the Potential Risks from Spray Drift and the Impact of Potential Risk Reduction Measures at 43 (July 2012).

<sup>119</sup> See *S. Fork Band Council of W. Shoshone of Nevada v. U.S. Dep’t of Interior*, 588 F.3d 718, 727 (9th Cir. 2009) (citing *Robertson v. Methow Valley Citizens Council*, 490 U.S. 332, 352 (1989)).

<sup>120</sup> Chlorpyrifos IRED at 208.

foot buffer for aerial spraying led to a 70% reduction.<sup>121</sup> EPA's analysis demonstrates the efficacy of buffers and that the efficacy increases with the size of the buffers, even though it cannot eliminate all risks of concern.

Chlorpyrifos has also been the subject of an ESA consultation in which NOAA Fisheries found that uses of chlorpyrifos are likely to jeopardize the survival and recovery of all threatened and endangered West Coast salmon populations and adversely modify their critical habitat. NOAA Fisheries proposed a mitigation package to avoid this prohibited result, which include buffers. The mitigation included 500-foot buffers for ground applications and 1000-foot buffers for aerial applications.<sup>122</sup> These buffers are supported by robust scientific analysis by a team of scientists, including some who have studied the impacts of chlorpyrifos on both salmon and their prey base. While the biological opinion is being revised to consider whether, *inter alia*, larger water bodies need buffers as large as the tributaries and small streams where salmon spawn and rear, that revision is unlikely to retreat from substantial no-spray buffers, as NOAA Fisheries has continued to impose buffers for pesticides less toxic to salmon than chlorpyrifos.

Buffers to protect salmon and water bodies admittedly are designed to reduce toxic runoff as well as prevent spray drift. The aerial buffers, however, are set at larger distances because of the greater propensity for spray drift. And EPA's assessment of the buffers put in place in 2001 to reduce drift into water focused exclusively on the ability of the buffers to reduce spray drift, not runoff. There is no basis for treating people or waters differently in assessing and mitigating for spray drift. The properties and movement of spray drift remain the same no matter who or what is in harm's way. And yet EPA has set buffer zones at larger sizes to reduce water contamination than what it is requiring to protect people with no discernible reason. It would be arbitrary and inexcusable for EPA to afford less protection to people than waterbodies and wildlife.

As if to prove this point, EPA recently included buffers in its proposed Worker Protection Standard Revision. For the first time, EPA proposes a buffer area around the field or forest area being sprayed. Applicators must cease spraying if other workers or other people enter the buffer zone and no workers other than applicators may enter the buffer zone after spraying for a designated re-entry interval. These restrictions apply only to adjacent fields owned by the same employer. They, therefore, leave other workers and residential areas unprotected. The sizes of the buffers are illuminating. The buffer is 100 feet for aerial spraying, upwardly applied pesticides, or applications using a high-spray pressure, and it is 25 feet for downwardly applied pesticides. 79 Fed. Reg. at 15,490, 15,521. Inexplicably, EPA proposed larger generic buffers than what it has deemed sufficient for chlorpyrifos, and those larger proposed WPS buffers are still inadequate to protect bystanders and workers in other fields.<sup>123</sup>

#### D. EPA Fails To Account For Exposure To Dust.

EPA's risk assessments both for residential bystander exposure, as well as workers, fail to consider exposures from indoor dust. Numerous studies document that levels of chlorpyrifos

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<sup>121</sup> Rice *et al.*, OPP Environmental Fate & Effects Division Revised Labels and Mitigation Review for Lorsban (July 31, 2001).

<sup>122</sup> [http://www.nmfs.noaa.gov/pr/pdfs/pesticide\\_biop.pdf](http://www.nmfs.noaa.gov/pr/pdfs/pesticide_biop.pdf).

<sup>123</sup> Centers for Disease Control and Prevention, *Worker Illness Related to Newly Marketed Pesticides – Douglas County, Washington*, 2014, 64 Morbidity and Mortality Wkly. Rep. 42 (Jan. 23, 2015).

in indoor dust are correlated to nearby agricultural use.<sup>124</sup> The data indicate that off-target movement of chlorpyrifos and deposition of indoor dust is significant.

Studies find significant associations between proximity to agricultural fields, levels of pesticides in indoor dust, and pesticide urinary metabolite levels for adults and children, both for farmworker and non-farmworker households.<sup>125</sup> Chlorpyrifos is ubiquitous in indoor dust and found in close to 100% of the homes tested.<sup>126</sup> Because they crawl, play on the floor, and constantly put their hands in their mouths, young children have greater exposures to indoor dust.<sup>127</sup> Since chlorpyrifos degrades slowly in the indoor environment and is very persistent in indoor dust, the duration of exposures to pregnant women and young children are far longer than a transient drift event, and may be almost continuous.

A study of Hispanic farmworkers in Washington State's Lower Yakima Valley found significant evidence that farmworkers take home chlorpyrifos and other organophosphate residues in dust. During periods of maximum use of pesticides, farmworkers had consistently higher levels of chlorpyrifos in vehicle and house dust that correlated with elevated urinary metabolites for adults and children.<sup>128</sup> Based on exposure models for children three to five years of age, dust ingestion was the primary route of exposure to chlorpyrifos among farmworkers' children from an agricultural community in California.<sup>129</sup>

Children come into contact with pesticides through residues from their parents' skin and clothing, soil and dust tracked into their homes, contaminated soil and other surfaces where they play. The omission of indoor dust in the exposure assessment under-estimates risks to residential bystander children, farmworker children, and workers.

#### E. The RHHRA Erroneously Ignores Volatilization Exposures.

In the preliminary HHRA, EPA found that risks of concern are exceeded for bystanders.<sup>130</sup> Indeed, many of the actual air samples from air monitoring posed bystander risks. Specifically, EPA's assessment of volatilization risks showed that one-quarter of the acute ambient air concentrations resulted in risks of concern to residential bystanders, as did over half

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<sup>124</sup> Martha Harnly et al., *Pesticides in Dust from Homes in an Agricultural Area*, 43 *Envtl. Sci. and Tech.* 8767 (2009) (Ex. 24); Robert B. Gunier et al., *Determinants of Agricultural Pesticide Concentrations in Carpet Dust*, 119 *Envtl. Health Persp.* 970 (2011) (Ex. 25).

<sup>125</sup> Gloria D. Coronado et al., *Organophosphate Pesticide Exposure and Residential Proximity to Nearby Fields: Evidence for the Drift Pathway*, 53 *J. Occup. and Env'tl. Med.* 884 (2011) (Ex. 26).

<sup>126</sup> Lesliam Quirós-Alcalá et al., *Pesticides in House Dust from Urban and Farmworker Households in California: An Observational Measurement Study*, 10 *Envtl. Health* 19 (2011) (Ex. 27), available at <http://www.ehjournal.net/content/10/1/19>; Asa Bradman et al., *Pesticides and Their Metabolites in the Homes and Urine of Farmworker Children Living in the Salinas Valley, CA*, 17 *J. Exposure Sci. and Env'tl. Epidemiology* 331 (2006) (Ex. 28).

<sup>127</sup> Jacqueline Moya and Linda Phillips, *A Review of Soil and Dust Ingestion Studies for Children*, 6 *J. Exposure Sci. and Env'tl. Epidemiology* 545 (2014) (Ex. 29).

<sup>128</sup> Beti Thompson et al., *Variability in the Take-Home Pathway: Farmworkers and Non-Farmworkers and Their Children*, 24 *J. Exposure Sci. and Env'tl. Epidemiology* 522 (2014) (Ex. 30).

<sup>129</sup> Beamer, Paloma I., Robert A. Canales, Alesia C. Ferguson, James O. Leckie, and Asa Bradman, "Relative Pesticide and Exposure Route Contribution to Aggregate and Cumulative Dose in Young Farmworker Children." *International Journal of Environmental Research and Public Health* 9, no. 1 (January 3, 2012): 73–96. doi:10.3390/ijerph9010073.)

<sup>130</sup> Preliminary HHRA at 55.

of the acute application site concentrations and most of the short- and intermediate-term application site concentration assessments.

In its 2013, EPA evaluated volatilization risks and identified buffer distances necessary to reduce off-site concentrations below risk levels of concern. It presented its findings as maximum buffers measured from the edge of the field and whole buffers measured from the whole perimeter around the field. For some low-application rates for alfalfa, the buffer zones needed to protect against 95% of the bystander exposures were 16 feet for maximum buffers and 148 feet for whole buffers and 98 and 377 feet respectively in Florida. For other crops at higher application rates like oranges, the buffer zones would need to be much larger: 1476-4724 feet or larger maximum buffers and 623-2838 feet whole buffers.<sup>131</sup>

In the RHHRA, EPA reverses course and ignores all volatilization exposures based on two Dow studies, which purport to show that people will not experience adverse effects from volatilization exposures. Before turning to these flawed studies, it is important to note that EPA evaluated the studies against the wrong endpoint. It focused on 10% cholinesterase inhibition, not the serious neurodevelopmental damage that occurs at lower doses.

Not only did EPA use an insufficiently protective regulatory framework, but it failed to submit the new Dow studies for review by the SAP or obtain other peer review. It accepted the studies with far less scrutiny than what it has applied to independent scientific research by academic institutions, like Columbia, Mt. Sinai and UC-Berkeley. For example, it appears from the presentation given by Dow to EPA that the studies did not conform to one of the most basic principles of good experimental design: there was no positive control used to verify that the chemicals used in the study were capable of producing cholinesterase inhibition and that the experimental set up was capable of detecting cholinesterase inhibition.<sup>132</sup> In light of the serious health effects from chlorpyrifos, and the fact that Dow pursued the studies in order to reduce public health protection, it is critical that EPA ensure the studies reflect the real-world risks. This type of scrutiny is imperative given Dow's self-interest in designing the studies and the substantial flaws that call into question whether they are appropriate as the basis for ignoring all volatilization exposures from chlorpyrifos.

1. Cholinesterase Activity Levels Between Individuals are Inherently Variable and Do Not Provide a Valid Measure of Effect

In the inhalation, nose-only studies, a reduction in cholinesterase levels between a control group of rats and the test animals was used as the measure of effect. This is inadequate, because of the wide variability in individual cholinesterase activity levels. A review of chemical warfare agent effects on a variety of laboratory animals, including Crl:CD Sprague-Dawley rats<sup>133</sup> provides an interesting point of comparison for the chlorpyrifos nose-only inhalation studies. Yet there is no evidence EPA considered these studies or reconciled its findings of no effect for chlorpyrifos with their approach and experimental design. In particular, the use of a more reliable measure of cholinesterase inhibition, miosis, is now recommended by the CDC because of high variability in individual cholinesterase activity levels.

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<sup>131</sup> EPA Chlorpyrifos Preliminary Evaluation of Potential Risks from Volatilization at 32-46 (Jan. 31, 2013).

<sup>132</sup> See Slides in docket at <http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0189>.

<sup>133</sup> EPA 2002. NERVE AGENTS GA, GB, GD, GF, AND VX. <http://www.epa.gov/oppt/aegl/pubs/tsd21a.pdf>.

*The selection of miosis induction as the basis for deriving final AEGL- 1 values is supported by the evaluation of a U.S. Surgeon General’s review panel on agent exposure limits convened by the Chemical Demilitarization Branch of the National Center for Environmental Health of the Centers for Disease Control and Prevention (CDC) (67 Fed. Reg. 894 [2002]; DHHS 2002). Although the CDC has not yet finalized its position, the review panel generally concluded that cholinesterase activity depression is too variable for application as a critical effect in the estimation of nerve agent exposure limits and that miosis is an appropriate and readily quantified critical effect.*

## 2. The Effect of Temperature on Vapor Pressure Is Significant, but Was Not Considered in the Nose-Only Inhalation Studies

In its evaluation of the 2014 nose-only inhalation study for chlorpyrifos,<sup>134</sup> EPA noted that Dow’s studies measured no statistically significant inhibition cholinesterase, although they did find a statistically significant increase over a six-hour exposure period.<sup>135</sup>

One important fact was omitted from the DER evaluation of the results of the 2014 inhalation study: the effect of temperature on vapor pressure and consideration of the field conditions in effect when chlorpyrifos is applied. It is well known that the saturation vapor pressure of a chemical increases with temperature. The Clausius-Clapyron equation describes this phenomenon quantitatively and allows prediction of vapor pressure as a function of temperature.<sup>136</sup>

$$\ln(VP_1) - \ln(VP_2) = \frac{\Delta H_{vap}}{R} \left( \frac{1}{T_2} - \frac{1}{T_1} \right)$$

where

$VP_1$  = saturation vapor pressure at temperature  $T_1$  (in K)

$VP_2$  = saturation vapor pressure at temperature  $T_2$  (in K)

$\Delta H_{vap}$  = the enthalpy of vaporization of the chemical

For chlorpyrifos, the enthalpy of vaporization is 124 kJ/mol.<sup>137</sup>

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<sup>134</sup> W. Irwin. Review of Nose-Only Inhalation of Chlorpyrifos Vapor: Limited Toxicokinetics and Determination of Time-Dependent Effects on Plasma, Red Blood Cell, Brain and Lung Cholinesterase Activity in Femal CD(SD): Crl Rats. EPA Office of Chemical Safety and Pollution Prevention. 6/25/14. D411959. TXR# 0056694. EPA MRID# 49119501.

<sup>135</sup> The Dow studies monitor exposures over a 6-hour period, even though air monitoring data (see Figure 2) indicate that chlorpyrifos exposure from a single application persists at measureable levels for several days after the application. The preliminary HHRA used a 24-hour exposure duration, but the 2013 preliminary evaluation of the potential risks from volatilization (at 21) used a six-hour exposure duration. People can be expected to spend more than six hours in a stretch in their homes, hospitals and nursing homes, and children regularly move from their homes to schools to playfields and may be exposed at all of these locations. EPA must consider studies and use regulatory assumptions that reflect the full duration people will be exposed.

<sup>136</sup> Atkins P, de Paula J, Physical Chemistry: Thermodynamics, Structure, and Change (W.H. Freeman 10<sup>th</sup> ed. 2014).

<sup>137</sup> Calculated from data provided in *Review Report for the Active Substance Chlorpyrifos*, SANCO/3059/99 - rev. 1.5; European Commission Health and Consumer Protection Directorate-General Directorate D - Food Safety:

Because the nose-only inhalation studies were conducted at the temperature at which the animals were housed—22°C—the concentrations evaluated in the nose-only inhalation study do not represent field-realistic values that people living, playing, or working near chlorpyrifos applications are likely to experience. The saturation vapor pressure of chlorpyrifos at 20°C is 0.0014 Pa. Applications of chlorpyrifos in the Central Valley of California peak in July, when the average daytime highs are 35°C and often over 40°C. The saturation vapor pressure of chlorpyrifos at 35°C is 0.017 Pa, 12 times higher than the saturation vapor pressure at 20°C. At 40°C, the saturation vapor pressure is 0.037 Pa, 26 times higher than at 20°C. Thus the exposure experienced by the test animals is at least an order of magnitude lower than that experienced by people living near chlorpyrifos application sites. Similarly, for chlorpyrifos oxon, when the temperature increases from 20 to 40°C, the saturation vapor pressure increases by nearly an order of magnitude (0.0036 Pa to 0.028 Pa).<sup>138</sup>

### 3. The Studies Did Not Account for an Increase in Volatilization with Increased Soil Moisture

Chlorpyrifos volatilization rates also depend on soil and plant surface moisture. Concentrations of chlorpyrifos in excess of saturation in dry air are likely when substrates are wet. Majewski, *et al.*, determined the change in flux of chlorpyrifos after an application.<sup>139</sup> The authors found that volatilization was enhanced by soil moisture, as could be predicted by chlorpyrifos's high Henry's law constant,  $K_H$ .

*Irrigation water was not added to the field during the course of the experiment, and consequently, the top few millimeters became very dry during daylight hours. This surface drying apparently inhibited the volatilization process almost completely within the first few hours after sunrise. Heavy dew formed during the early morning hours of 12 September and light dew formed in the morning of 14 September. Dew formation and the consequent wetting of the soil surface coincided with high early morning flux values (Table 111). In contrast, flux rates were very low on the morning of 13 September, when no dew formed. The increased flux during the third period (1430 h) on 13 September coincided with a very light rainfall lasting approximately 20 min. This rainfall did not thoroughly wet the soil.*<sup>139</sup>

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Production and Distribution Chain. Unit D.3 - Chemicals, Contaminants and Pesticides: Brussels, 2005, 1–70.  
ec.europa.eu/food/plant/.../existactive/list\_chlorpyrifos.pdf

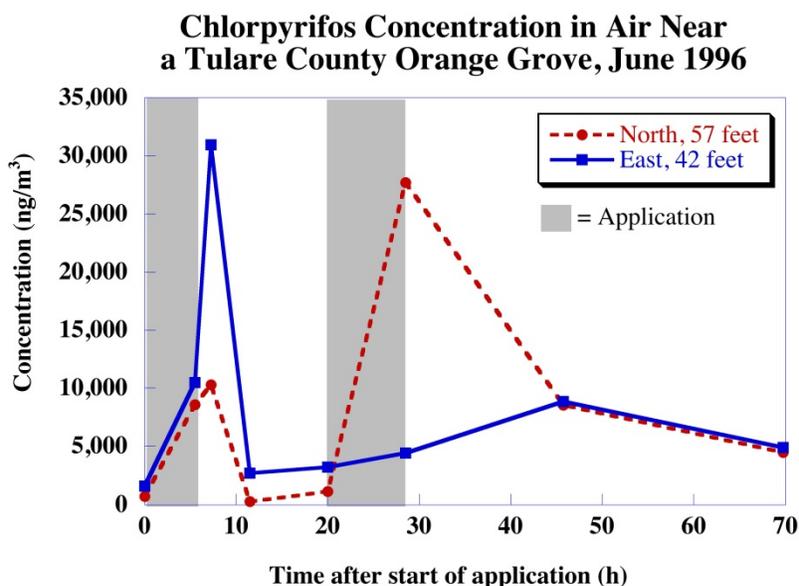
<sup>138</sup> Goel A, McConnell LL, Torrents A. 2007. Determination of vapor pressure-temperature relationships of current-use pesticides and transformation products. *Journal of Environmental Science and Health, Part B* 42:343–349; doi:10.1080/03601230701309494.

<sup>139</sup> Majewski MS, Glotfelty DE, U KTP, Seiber JN. 1990. A field comparison of several methods for measuring pesticide evaporation rates from soil. *Environmental Science & Technology* 24: 1490–1497.



would not have predicted these events. Thus, before the rat data can be used to estimate a toxicological endpoint, it must be validated against incident data and likely concentrations in air that caused those incidents.

An application site monitoring study of chlorpyrifos conducted by the California Air Resources Board (CARB) provides a reference point for estimated concentrations near an application site. In June 1996, CARB conducted an air monitoring study for a chlorpyrifos application to a Tulare County orange grove.<sup>141</sup> The study monitored both chlorpyrifos and its breakdown product chlorpyrifos oxon. The Figures below show monitoring results in terms of measured air concentrations of chlorpyrifos over time for sampling sites approximately downwind of the grove (*see* Appendix 1 for the full data set). Measurements of chlorpyrifos concentrations in air adjacent to application sites indicate maximum measured concentrations of 0.043 mg/m<sup>3</sup>.



**Figure E:** CARB monitoring of a chlorpyrifos application shows high concentrations during the application period.<sup>141</sup> This particular application was interrupted by high winds, but resumed 10 hours later. The similarity of the peak concentrations for the two different application periods suggests that the measured concentrations are typical for chlorpyrifos.

##### 5. Study Validation Must Account for Biomonitoring Data Showing Elevated Levels of Chlorpyrifos Metabolites in People Living in an Area of High Chlorpyrifos Use

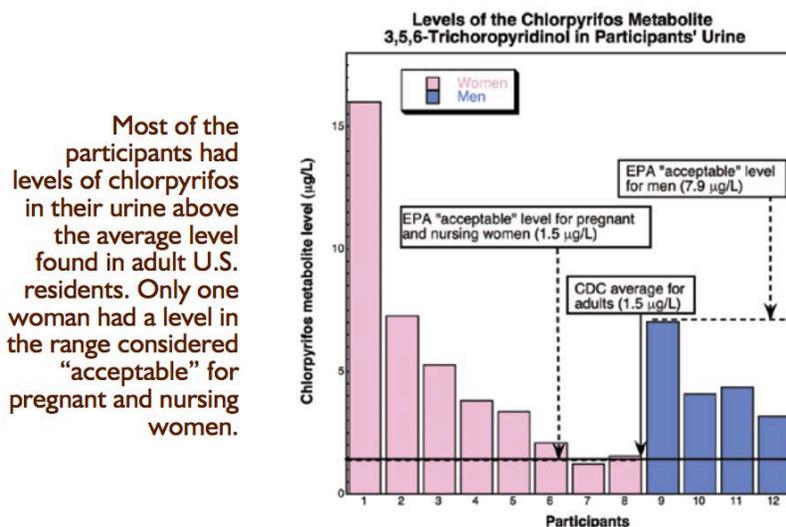
The nose-only inhalation studies provide data on the concentration of chlorpyrifos and trichloropyridinol (TCPy) in the blood of rats after exposure. A 2006 study<sup>142</sup> of chlorpyrifos in

<sup>141</sup> CARB 1998. Report for the Application and Ambient Air Monitoring of Chlorpyrifos (and the Oxon Analogue) in Tulare County during Spring/Summer 1996, California Air Resources Board, Test Report #C96-040 and # C96-041 (April 7, 1998), <http://www.cdpr.ca.gov/docs/emon/pubs/tac/chlrpfs.htm>.

<sup>142</sup> PAN, Airborne Poisons: Pesticides in Our Air and in Our Bodies (2006). <http://www.panna.org/sites/default/files/Biodrift-Summary-Eng-1.pdf>.

air and levels of the chlorpyrifos metabolite trichloropyridinol (TCPy) in the urine of people living in Lindsay, CA (an area of high chlorpyrifos use in a season of high use) indicates that inhalation exposure to chlorpyrifos leads to statistically significantly higher levels of TCPy in urine compared to a national average of 1994 subjects in the 2005 NHANES data<sup>143</sup> ( $p < 0.001$ , based on Mann-Whitney Rank Sum test).

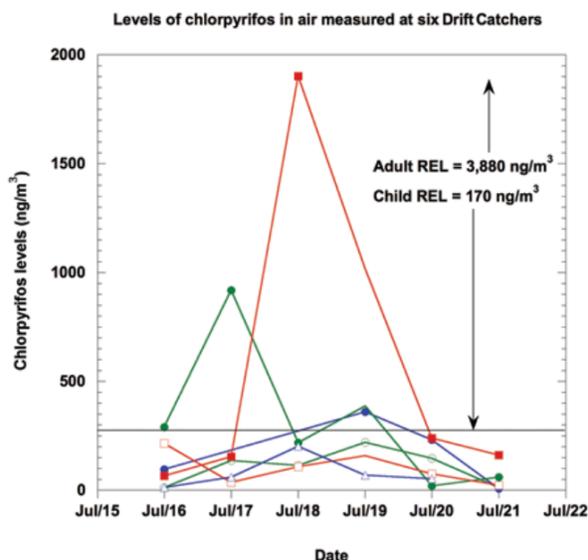
For most of the women in the study, exposure to chlorpyrifos exceeded the “acceptable” level for pregnant and nursing women, based on the US EPA Population Adjusted Dose in use at the time.<sup>144</sup> Participants did not work in agriculture, packing or shipping of produce or other tasks where they would have been exposed to chlorpyrifos, thus all exposures could be attributed to inhalation exposure or dermal exposure from residues from spray or volatilization drift.



**Figure F:** Chlorpyrifos exposure was measured via biomonitoring in the town of Lindsay during July 2006, a time of high chlorpyrifos use shows that participant exposure significantly exceeds levels of chronic concern based on US EPA’s cPAD.

<sup>143</sup> The geometric mean level of chlorpyrifos metabolite TCPy was 1.5 µg/L for adults 20–85 years old as reported in a large U.S. government study of chemicals in the bodies of U.S. residents. Eleven of the 12 participants had levels above that level (numbers ranged from 0.9 to 16.0 µg/L). If levels are shown per gram of the urine protein creatinine (which controls for dilution in individuals who drink relatively more water), then participants’ levels in study participants ranged from 2.4 µg/g to 13.3 µg/g and were all above the U.S. average levels of 1.8 µg/g for women (of all ages) and 1.7 µg/g for men (of all ages). Source: Department of Health and Human Services and Centers for Disease Control and Prevention. *Third National Report on Human Exposure to Environmental Chemicals*, National Center for Environmental Health, Division of Laboratory Sciences (NCEH Pub. No. 05-0570, July 2005).

<sup>144</sup> EPA’s “acceptable” level of TCPy for pregnant or nursing women is based on the chronic Population Adjusted Dose for chlorpyrifos and was calculated according to the method outlined in Appendix B: Calculating Pesticide Exposure from Metabolites in Urine in K Schafer, M Reeves, S Spitzer and S Kegley, *Chemical Trespass: Pesticides in our bodies and corporate accountability* (2004), available at <http://www.panna.org/campaigns/docsTrespass/chemicalTrespass2004.dv.html>.



On the days when urine samples were collected, three of the six Drift Catchers detected chlorpyrifos levels above the “acceptable” level of 170 ng/m<sup>3</sup> for short term (acute and sub-chronic) exposure for children. The data not shown (extending from July 11 to July 30) indicated that every house had at least one day when measured levels were above the reference level. Of the 116 air samples taken over a 21-day period, 33 (or 28%) were above this children’s reference level. At one house, nearly surrounded by orange groves, levels reached 1902 and 1017 ng/m<sup>3</sup> on two consecutive days. (Symbols on this graph correspond to the Drift Catcher locations shown on the map on the next page.)

**Figure G:** Chlorpyrifos levels in air were measured during the same period of time the biomonitoring experiment was being conducted. No statistically significant correlation was found between air concentrations and levels of TCPy in urine; however, the Drift Catcher monitoring devices were stationary, while the participants did not stay in the same location at all times.

**Wilcoxon-Mann-Whitney Rank Sum Test**

**Group 1:** Lindsay TCPy conc (ug/g creatinine)

**Group 2:** CDC NHANES TCPy (ug/g creatinine)

	<b>Group 1</b>	<b>Group 2</b>
Count	16	1994
Median	4.467	1.49
Median Difference		2.977
Sum of Group 1 ranks		27949
Sum of Group 2 ranks		1.99E+06
Group1 U		27812
Group2 U		4091.5
P Value		< .0001

In its 2013 volatilization evaluation, EPA reviewed human incident reports from several governmental reporting databases. It limited its review to the over 500 incidents in which chlorpyrifos was the only pesticide to which the person was exposed. EPA found “an association between chlorpyrifos incidents and respiratory effects.”<sup>145</sup> While EPA could not determine whether a particular incident resulted from drift or volatilization, many occurred further from the application site than the minimal spray drift buffers added to chlorpyrifos labels in 2012. In other words, children and other bystanders will continue to suffer from respiratory effects from chlorpyrifos with the mitigation being put into place to address bystander exposures and risks. As an agency charged with protecting children and public health, EPA must look beyond models and industry-sponsored studies that try to show such poisoning events will not occur. They have

<sup>145</sup> EPA Chlorpyrifos Volatilization Evaluation at 18.

occurred and will continue to occur unless EPA takes steps to prevent exposures that cause poisoning incidents.

V. THE RHHRA REVEALS UNACCEPTABLE RISKS TO WORKERS THAT MUST BE PREVENTED AND UNDERESTIMATES THE EXTENT OF THE RISKS THAT ARE UNACCEPTABLE.

As part of the RHHRA, EPA conducted an assessment of occupational risks. As its standard approach, EPA first identifies risks of concern to workers and whether workers will be exposed to levels of chlorpyrifos that exceed the triggers for those risks. If it finds risks of concern, EPA then tries to ascertain whether the risks can be reduced or eliminated through the use of protective clothing and gear, engineering controls, like closed mixing systems, or re-entry intervals to keep field workers out of the fields until exposures would be lessened.

EPA's occupational risk assessment identified many dozens of worker scenarios that put workers at extreme risk even with the maximum protective gear and engineering controls EPA regularly employs. These uses of chlorpyrifos must be cancelled to protect workers.

However, EPA's risk assessment grossly underestimates the number of such unacceptable worker scenarios. First, EPA underestimates the full exposures to workers by making a series of unsupportable assumptions. Second, EPA cannot ignore direct drift onto workers. Third, EPA overestimates the efficacy of the protective clothing and gear and engineering controls in the face of evidence demonstrating they offer less protection than EPA has assumed.

A. EPA Must Cancel The Uses That Admittedly Expose Workers To Unacceptable Risks.

In its occupational risk assessment, EPA used the Dow model to estimate exposure levels resulting in 10% RBC AChE inhibition following occupational exposures. RHHRA at 64 (Table 4.8.4). EPA classifies a risk as a risk of concern if the margin of exposure (MOE) is less than 100, which incorporates the a 10X inter-species safety factor and a 10X FQPA safety factor, and asks whether the worker would be exposed to a dose that is less than 100 times the no-adverse effect level. For chlorpyrifos, EPA used 10% red-blood cell cholinesterase inhibition as the no adverse effect level.

The RHHRA explains: "In order to determine what level of personal protection is required to alleviate risk concerns and to ascertain if label modifications are needed, steady state exposure and risk estimates were calculated for occupational handlers of chlorpyrifos for a variety of scenarios at differing levels of personal protection including engineering controls." RHHRA at 102. Estimates of dermal and inhalation exposure were calculated for various levels of personal protective equipment (PPE). RHHRA at 100. The lowest tier is designated as the baseline exposure scenario (i.e., long-sleeve shirt, long pants, shoes, socks, and no respirator). If risks of concern occur with baseline attire, then increasing levels of personal protective equipment or PPE are evaluated. If risks remain a concern with maximum PPE, then engineering controls are considered.

EPA calculated that of 285 handler scenarios reviewed, 126 remain a concern (MOE

<100) regardless of the levels of personal protection (e.g. long sleeved shirt and pants, shoes and socks, coveralls, chemical resistant gloves, and dust/mist respirators) and engineering controls considered (e.g. enclosed cabs or cockpits, water-soluble packaging, and closed mixing/loading systems). RHHRA at 12, 100. It also found that 32 seed treatment scenarios pose risks of concern, and greenhouse workers may also be over-exposed. RHHRA at 103.<sup>146</sup>

EPA's occupational risk assessment grossly under-estimates the risks to workers by using 10% cholinesterase inhibition as the targeted effects level when it has found that neurodevelopmental impairments to children occur *in utero* at far lower doses. It also underestimates worker risks for the reasons set out below. In other words, the risks to workers are far greater than EPA's assessment reflects.

EPA has represented that it "has already begun discussions with the chlorpyrifos registrants regarding mitigation of worker risks."<sup>147</sup> EPA's findings that risks of concern persist even with maximum PPE and engineering controls means EPA must cancel all uses that expose workers to such risks. EPA acknowledges in the RHHRA that workers could suffer from heat stress or respiratory harm with more protective clothing in the hot climates where chlorpyrifos is often used. RHHRA at 100. EPA cannot in good conscience settle for more PPE or engineering controls that will fall short of preventing the risks of concern.

While EPA regulates worker risks under FIFRA, which allows balancing of risks and benefits in determining whether an adverse effect is unreasonable, it would be indefensible for EPA to find these risks reasonable. Industrial workers are afforded far greater protection under the Occupational Safety and Health Act, and EPA purports to provide equivalent protection under FIFRA.<sup>148</sup> It is inconceivable that OSHA would allow workers to be subjected to the magnitude of worker poisoning risks that led EPA to find risks of concern for chlorpyrifos, especially when the chemical also causes neurodevelopmental damage to children exposed *in utero*. Moreover, when EPA balances the benefits of continued use of chlorpyrifos against its risks, it must consider all risks, including the neurodevelopmental harm to children, including reduced IQ and autism, drinking water contamination, drift exposures to bystanders, and harm to endangered species. These risks unquestionably outweigh the benefits to growers from continuing to use chlorpyrifos.

The evidence that chlorpyrifos poses risks of concern to workers is not new. When EPA assessed worker risks as part of the 2001 re-registration, it identified risks of concern to workers from air blast and ground boom applications in open cabs, some greenhouse applications, and some post-application field worker activities that were not mitigated, and EPA did not cancel those uses. Indeed, EPA's failure to cancel those uses was one of the grounds for the legal challenge to the chlorpyrifos re-registration decision.<sup>149</sup> It is time for EPA to stop allowing workers to be exposed to these admittedly unacceptable risks.

EPA also assessed the risks posed to field workers who enter the fields to perform tasks

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<sup>146</sup> Chlorpyrifos Occupational Risk Assessment at 10.

<sup>147</sup> Letter to A. Colangelo from J. Housenger, Director OPP, at 4 (March 26, 2015).

<sup>148</sup> 29 U.S.C. § 655(b)(5) requires that standards adequately assure, to the extent feasible, that no employee will suffer material impairment of health from workplace exposures. It does not allow risk-benefit balancing in deciding whether such protection is required.

<sup>149</sup> *UFW v. EPA*, No. 07-3950-JF (N.D. Cal. Filed Aug. 1, 2007).

like weeding, pruning and harvesting. EPA typically establishes re-entry intervals (REIs) that prevent field workers from entering the fields for a set period of time to prevent unsafe exposures to pesticide residues. For some activities and formulations EPA calculates that REIs will need to increase to 5 or 10 days. RHHRA at 105.<sup>150</sup> For others (e.g. microencapsulated liquid formulation), the estimated post-application REIs range up to 35 days depending on activity. This 35-day calculation reflects only the end of the observation time, rather than the time after which the risk fell below levels of concern. RHHRA at 13. Thus, an adequately protective REI would presumably be much greater. It is hard to imagine a growing operation could reasonably be expected to function without worker contact with the crop for more than 35 days.

For the reasons discussed above with respect to handler risks, EPA must expand the REIs to eliminate the risks of concern until EPA has finally and fully banned chlorpyrifos. It cannot balance away worker health. For those scenarios where it is infeasible to keep workers out of the fields for the needed REI, the use must be cancelled immediately.

#### B. EPA Cannot Lawfully Ignore Direct Spray Drift.

The chlorpyrifos RHHRA uses the 2012 Standard Operating Procedures for Residential Pesticide Exposure Assessment to assess spray drift. RHHRA at 76. The Operating Procedures limit EPA's drift analysis to dermal exposures from contact with residues of a pesticide that has drifted onto crops, soil, lawns or ground.<sup>151</sup> It "focuses only on evaluating spray drift in application situations that are considered compliant with label statements that prohibit applications sprays from contacting people directly through drift." SOPs at 3.

EPA excludes direct drift from consideration in human health risk assessments because it views direct drift onto people as an enforcement issue, rather than part of its assessment of unreasonable adverse effects and registration determinations. "Direct contact with sprays is considered a violation of standard label language, and as applicable, EPA's Worker Protection Standard. This means that direct contact is not evaluated in risk assessment but is addressed through enforcement action against persons not complying with label prohibitions/directions, through applicator education, and through other means." 79 Fed. Reg. 4691, 4692 (Jan. 29, 2014). "Situations involving direct human contact from spray drift are addressed by the Agency using other types of already existing requirements such as enforcement actions, basic requirements for worker protection, or modifying use instructions (e.g. add drift minimization language to all pertinent labels)." SOPs at 3.

Under this approach, EPA ignores direct exposures to people from drift. It is ignoring inhalation exposures altogether and is limiting its consideration to dermal exposures to surfaces where residues have landed. SOPs at 4-5. EPA's drift assessment uses an adaptation of a model that predicts the amount of a pesticide that will deposit on turf. The model predicts the amount of pesticide residues deposited at various distances from the edge of a treated field. "Exposures considered for risk assessment purposes occur solely as a result of contact with a surface that has been previously impacted by spray drift such as nearby lawns." SOPs at 5.

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<sup>150</sup> Chlorpyrifos Occupational Risk Assessment at 11.

<sup>151</sup> As explained above, EPA assessed inhalation exposures to bystanders from aerial spraying, which equates to direct drift, but it then did not aggregate those exposures and dermal exposures to deposited residues.

EPA identifies a suite of regulatory steps that can be taken to reduce drift and lower risks. SOPs at 12, 19. Its guidance identifies potential mitigation measures, including prohibiting aerial applications, establishing no-spray buffers, reducing application rates, and prescribing application measures, such as larger droplet sizes, lower release heights, and technologies. *Id.* However, EPA has required none of these measures to reduce or eliminate direct drift onto workers in neighboring fields.

Ignoring direct drift onto people is indefensible in the face of extensive evidence that direct drift is poisoning significant numbers of workers and bystanders every year and is exposing people to acute poisoning and other risks from chlorpyrifos. The recent CDC investigation of the poisoning of 20 Latino workers from off-target drift from an air blast application at the neighboring orchard is instructive. The workers who were poisoned were 30 to over 350 feet from the edge of the neighboring orchard. The CDC found the regulatory prohibition on applying pesticides in a manner that results in contact with workers or other persons inadequate to prevent off-target drift incidents and noted that 31% of all acute pesticide-related illnesses identified among farmworkers during 2005-2012 involved exposure to off-target drift from pesticides applied to a neighboring farm. The CDC recommended additional safeguards, such as notifications to workers on neighboring farms, better communication between the managers of adjacent farms, and amending regulations to require that applications must cease if workers or bystanders are in neighboring areas where drift may reach them.<sup>152</sup>

Under FIFRA's mandates, EPA must guard against unreasonable adverse effects. And under the FQPA, EPA must ensure a reasonable certainty of no harm to children and other bystanders from aggregate exposures to chlorpyrifos, including from drift. If the current label directions are not up to the task, EPA must impose more stringent safeguards to prevent such effects, as agencies and courts have required in other contexts.

1. EPA Must Consider Direct Drift in order to Discharge its Legal Obligations.

In order to register or re-register a pesticide, EPA must determine that “when used in accordance with widespread and commonly recognized practice it will not generally cause unreasonable adverse effects on the environment,” which includes harm to people. 7 U.S.C. § 136a(c) (5); *see id.* § 136(bb) (definition of “unreasonable adverse effects”). EPA must ensure the pesticide labeling complies with FIFRA's requirements, meaning the labeling must guard against unreasonable adverse effects. *Id.* § 136a(c)(5); *see also id.* (EPA must also determine that the pesticide will perform its intended function without unreasonable adverse effects).

This is a preventive scheme. Through risk assessments and registration determinations, EPA identifies adverse effects from a pesticide and either prohibits uses or requires mitigation to prevent those it deems unreasonable. EPA undertakes a balancing of risks and benefits to determine if harmful effects should nevertheless be allowed (are not unreasonable) due to overriding benefits from a pesticide use. However, if the risks outweigh the benefits, they are unreasonable and EPA must require safeguards that will prevent the adverse effects.

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<sup>152</sup> CDC, *Worker Illness Related to Newly Marketed Pesticides – Douglas County, Washington, 2014*, Morbidity and Mortality Weekly Report, 64(02); 42-44 (Jan. 23, 2015).

EPA decided long ago that direct drift onto people in an unreasonable adverse effect. Such a chemical trespass is prohibited in the worker protection standard. This prohibition is also prescribed in many pesticide labels. The fact that EPA has already made this unreasonable adverse effects determination does not mean it can ignore evidence that such adverse effects nonetheless are happening. As part of its unreasonable adverse effects determination, EPA must consider real-world pesticide uses and practices. It must review both the pesticide's intended function and widespread and commonly recognized practice. If the pesticide cannot perform its intended function when used in accordance with common practices without unreasonable adverse effects, the use must be prohibited or safeguards imposed. And the labeling is the mechanism to change those standard practices or intended uses to prevent the harm.

Here, the label and the worker protection standard prohibit direct contact of a pesticide with people. Yet that general label admonition has proven ineffective. Widespread and commonly used pesticide practices are resulting in drift directly onto people. The labels are therefore not in compliance with FIFRA because they are inadequate to prevent unreasonable adverse effects. To be compliant, the labels must specify application practices that will avoid direct contact with people. EPA has stated that drift minimization techniques may be necessary to prevent direct drift onto people. SOPs at 3, 19. Where that is the case, those drift minimization techniques must be required on the label. It is through the registration and label approval that EPA discharges its obligations to ensure an approved pesticide use will not cause unreasonable adverse effects. If EPA knows that broad prohibitions on a label are insufficient to prevent direct drift, then it must require more prescriptive label directives to achieve that result.

Drift must be taken into account not only under FIFRA, but also under the FQPA for food use pesticides and non-occupational exposures. Under the FQPA, EPA must ensure there is a reasonable certainty of no harm from aggregate exposures to the pesticide, including through drift. 21 U.S.C. § 346a(b)(2)(A)(ii). EPA's response to the Kids Petition and the RHHRRA acknowledge its FQPA legal obligation to protect children and other bystanders from pesticide drift.

Under neither of these statutes is EPA allowed to turn a blind eye to exposures that frequently occur despite general label or regulatory language prohibiting them. EPA cannot ignore a known route of exposure that regularly causes pesticide poisonings.

It is well settled under other statutes that agencies may not ignore violations of the law that it knows occur regularly. For example, in the context of evaluating the impacts of off-road vehicles, the Forest Service was required to consider the effects of known illegal vehicular use. Failure to quantify known, unauthorized use was a "glaring shortcoming."<sup>153</sup> Similarly, when proposing mitigation measures in an environmental impact statement, the effectiveness of those measures must be evaluated, including the measures' ability to curb unauthorized behavior. Indeed, it is "useless" to discuss mitigation without evaluating effectiveness.<sup>154</sup> An agency cannot adequately consider the environmental significance of its proposed action if it omits a

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<sup>153</sup> See *Found. for N. Am. Wild Sheep v. U.S. Dep't of Agric.*, 681 F.2d 1172, 1178 (9th Cir. 1982).

<sup>154</sup> See *S. Fork Band Council of W. Shoshone of Nevada v. U.S. Dep't of Interior*, 588 F.3d 718, 727 (9th Cir. 2009) (citing *Robertson v. Methow Valley Citizens Council*, 490 U.S. 332, 352 (1989)).

discussion of known, illegal use and, thus, does not permit the public assessment.<sup>155</sup>

## 2. EPA Cannot Leave Direct Drift to Enforcement, But Instead Must Take Steps to Prevent Pesticide Poisonings.

EPA takes the position that direct drift is solely an enforcement matter. That position is not in furtherance of the statutory scheme. FIFRA and FFDCA, like many environmental statutes, are fluid, establishing a standard of protection that necessitates additional restrictions and greater protection over time when there is evidence that the existing registrations and tolerance are falling short of meeting that standard. EPA can and must cancel or modify registrations and revoke tolerances where necessary to avoid unreasonable adverse effects or unsafe exposures.

EPA's position also runs counter to the reality that enforcement of the worker protection standard and pesticide label prohibitions has failed to prevent harm from direct drift onto people. Incident reports show that drift complaints make up the majority of reported poisoning events in both agricultural and non-agricultural settings. Moreover, many states report that the most common complaint of a WPS violation occurs as pesticide drift.<sup>156</sup> Many state agencies define and classify "pesticide drift" as drift that moves through the air and is not deposited on the target area at the time of application.<sup>157</sup> In other words, the incidents in such states occur from the very type of exposure that EPA is ignoring – direct drift of the pesticide onto people – rather than contact with deposited residues in the field.

Not only is direct drift associated with a disturbing number of reported pesticide incidents, but enforcement of the WPS and pesticide labels has proven inadequate. In 2011, the U.S. Office of the Inspector General found that the EPA "does not administer a consistent national program . . . As a result, state performance (of enforcement) remains inconsistent across the country, providing unequal environmental benefits to the public and an uneven playing field for regulated industries."<sup>158</sup> The consequences of violating the WPS or pesticide labels are generally insufficient to deter violations. Summary enforcement statistics show that the number of safety violations far exceeds that of warning letters, notices and fines.<sup>159</sup> Fines are issued less than 20% of the time that violations are found.<sup>160</sup> Equally troubling is the fact that many of these fines, even when issued, are reduced or unenforced without explanation. In 2013, the United

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<sup>155</sup> See *Nat'l Audubon Soc'y v. Hoffman*, 132 F.3d 7, 17 (2d Cir. 1997).

<sup>156</sup> See e.g., Margaret Reeves, Anne Katten, Martha Guzman, *Fields of Poison: California Farmworkers and Pesticides* at 17 (2002) ("Fields of Poison") (Ex. 12); Bud Hover, Director, Washington State Dept. of Agriculture Pesticide Management Division, *2013 Annual Report to the Legislature* (Feb. 2014, Revised March 2014) (Ex. 13), Executive Summary p. 1; California Department of Pesticide Regulation Pesticide Illness Surveillance Program, *Illness and Injuries Reported in California Associated with Pesticide Exposure Summarized by the Type of Activity and Type of Exposure* (2012) (Ex. 14); California Environmental Protection Agency Department of Pesticide Regulation, *Summary of Results from the California Pesticide Illness Surveillance Program 2012* at 8, HS-1896 (Jan. 13, 2015) (Ex. 15).

<sup>157</sup> California Environmental Protection Agency, *Department of Pesticide Regulation Overview, Pesticide Drift, Overview* (Oct. 30, 2000), available at <http://www.cdpr.ca.gov/docs/enforce/drftinit/drftover.pdf>.

<sup>158</sup> OIG, *EPA Must Improve Oversight of State Enforcement*, Report No. 12-P-0113, December 9, 2011, (By establishing stronger organizational structures, EPA can directly implement a national enforcement strategy that ensures all citizens have, and industries adhere to, a baseline level of environmental protection.)

<sup>159</sup> *Fields of Poison* at 18-22.

<sup>160</sup> *Id.*

States Office of the Inspector General found that “EPA regions generally did not consistently determine and document reductions in proposed penalties based on good faith of the violators, and in some regions reductions appeared automatic and without adequate justification.”<sup>161</sup> And in times of stressed budgets, fewer resources are available for inspections and enforcement.

FIFRA and FQPA are regulatory schemes that call for enforceable mitigation measures to prevent unreasonable adverse effects or harm to children and bystanders. When general admonitions fall short, clearer prescriptions are required to prevent the harm.

### 3. To Meet Its FIFRA and FQPA Obligations, EPA Must Prescribe Label Requirements That Will Prevent Drift.

By leaving direct drift to after-the-fact enforcement, EPA is abdicating its statutory obligations. In its SOP for spray drift and in Pesticide Registration Notices addressing spray drift, EPA has identified a suite of drift mitigation measures that can be included on pesticide labels, including the droplet size, nozzle types, application rates, and no-spray buffers.<sup>162</sup>

In addressing exposures to chlorpyrifos spray drift for residential bystanders, EPA attempted to follow this approach. Though the mitigations that resulted are inadequate as explained elsewhere in these comments, EPA worked with Dow and the other registrants to develop a mitigation package that is now on the chlorpyrifos labels. That mitigation package includes reduced application rates for aerial applications and no-spray buffers around sites that may be occupied by people. The buffer sizes vary depending on the application rate and the droplet size with larger buffers for higher application rates and finer droplets that are more prone to drift longer distances.<sup>163</sup> EPA did not allow the registrants to respond to drift exposures that pose risks of concern by including a statement on the label saying “chlorpyrifos should not be applied in a manner that would leave residues on the ground offsite where children, bystanders or workers neighboring fields may come in contact with the residue.” Such an admonition would be ineffective and contrary to evidence of drift incidents; EPA should have done no less where direct drift is concerned.

Similarly, in its 2001 chlorpyrifos re-registration, EPA identified drift of chlorpyrifos into water as a concern for aquatic life. It did not address this risk by requiring a statement on the label saying “do not use chlorpyrifos in a manner that will allow residues to drift into water.” Instead, it approved labels that imposed use restrictions such as reduced application rates, limitations on the number of applications, and no-spray buffers around waterbodies. This use-restriction approach is in keeping with the advice of the Pesticide Program Dialogue Committee’s Spray Drift Workgroup, which highlighted problems with unenforceable and confusing labels and preferred performance standards such as droplet size requirements and design standards such as maximum wind speeds.<sup>164</sup>

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<sup>161</sup> OIG, EPA Needs to Update Its Pesticide and Chemical Enforcement Penalty Policies and Practices, Report No. 13-P-0431, September 26, 2013, (“The lack of adequate guidance for determining good faith reductions and supporting documentation for good faith reductions creates a risk that violators may not be treated equitably. In addition, EPA may be losing opportunities to fully collect all penalties due.”).

<sup>162</sup> SOP at 10-11; 2009X Draft Pesticide Registration Notice – Drift Labeling.

<sup>163</sup> Spray Drift Mitigation Decision for Chlorpyrifos (059101) (July 2012).

<sup>164</sup> Spray Drift Workgroup, Final Report to PPDC (Apr. 27, 2007) (Ex. 16).

Since EPA and the states enforce pesticide labels as the principal mechanism for ensuring pesticides are not used in a manner that causes harm, it makes sense that the labels need to convey clearly what actions are prohibited, rather than articulate a lofty goal, devoid of the steps that can achieve it. General label statements would fall short of creating enforceable standards and complying with the FIFRA and FQPA mandates. Pesticide labels need to say more than “do not allow this pesticide to contaminate drinking water” or “do not use this flea treatment in a manner that will allow children to become sick from playing with their pets.”

How EPA’s sister agency – National Marine Fisheries Service (NMFS) – has addressed risks that chlorpyrifos and other organophosphates pose to threatened and endangered salmon is instructive. As the fisheries wildlife agency, NMFS conducted an Endangered Species Act (ESA) consultation and determined in November 2008 that EPA’s registration of chlorpyrifos and other pesticides allows uses that are likely to jeopardize the survival and recovery of 27 of the 28 listed salmon and steelhead populations and to adversely modify critical habitat designated for 25 of them.<sup>165</sup> To prevent that outcome, which would violate the ESA, NMFS proposed a reasonable and prudent alternative with reduced use and no-spray buffers to ensure the pesticides would not migrate into salmon streams. NMFS did not rely on a general admonition to avoid using the pesticides in a manner that will allow the pesticides to get into salmon streams. Instead, it recommended binding label requirements that would prescribe mitigation measures, including no-spray buffers and reduced application rates.<sup>166</sup>

In other contexts where federal agencies have a statutory obligation to prevent harm to the environment or public health, the agencies or courts have often found it necessary to impose prescriptive requirements, rather than leave compliance to more general admonitions. The Clean Water Act is also instructive.

Pursuant to the Clean Water Act (“CWA”), states must adopt, and EPA must approve, water quality standards preventing pollution that will impair bodies of water and keep them from meeting water quality goals. Many states initially adopted broad narrative standards governing nitrogen and phosphorous pollution. Florida, for example, implemented a standard that provided: “In no case shall nutrient concentration of a body of water be altered so as to cause an imbalance in natural populations of aquatic flora or fauna.” Fla. Admin. Code r. 62-302.530(47)(b). After water tests showed that 40% of assessed waters nationwide did not meet water-quality goals, EPA concluded that many states had to adopt numerical limitations.<sup>167</sup> In addition, President Clinton announced the Clean Water Action Plan to restore and protect the nation’s waters, which “found numeric nutrient water criteria to be necessary and directed the

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<sup>165</sup> [http://www.nmfs.noaa.gov/pr/pdfs/pesticide\\_biop.pdf](http://www.nmfs.noaa.gov/pr/pdfs/pesticide_biop.pdf).

<sup>166</sup> In litigation challenge EPA’s failure to comply with the ESA with respect to salmon, a district court imposed interim measures to protect salmon from these pesticides during the consultation process. Upon finding that pesticide-free buffer zones are “a common, simple, and effective strategy” that will “substantially contribute to the prevention of jeopardy,” the court imposed 20-yard no-use buffers and 100-yard no aerial spray buffers for the pesticides at issue, unless they had received a “no effect” or “not likely to adversely affect” determination. *Washington Toxics Coalition v. EPA*, No. C01-132C, Order at 16, 18 (W.D. Wash. Aug. 8, 2003); Injunction at 16, 18 (Jan. 22, 2004), *aff’d*, 413 F.3d 1024, 1031 (9th Cir. 2005).

<sup>167</sup> See Carol Browner, EPA Administrator, and Dan Glickman, Secretary USDA to Albert Gore, Jr., Vice President. (Feb. 14, 1998) (AR000069); Notice of National Strategy for the Implementation of Regional Nutrient Criteria, 63 Fed. Reg. 34,648-34,650 (Jun. 25, 1998); see also Geoffrey Grubs, EPA to Water Dir. Reg. I-X, Development and Adoption of Nutrient Criteria into Water Quality Standards (Nov. 14, 2001).

EPA to develop, within two years, ‘numerical ranges for acceptable levels of nutrients for all water body types and ecoregions throughout the country.’<sup>168</sup> After Florida failed to comply, EPA stepped in and established numerical nutrient limits necessary in order to bring Florida’s waters into compliance with the CWA. A federal district court upheld the EPA Administrator’s determination that numeric nutrient criteria were necessary for Florida waters to meet the Clean Water Act’s requirements because the narrative criteria had proved insufficient to control Florida’s widespread nutrient pollution.<sup>169</sup>

Professor Oliver Houck has commented on the case, observing that “the importance of numbers magnifies in environmental decision-making because without, it devolves to case-by-case applications that are extremely resource-intensive, riddled with variables, endlessly arguable, and close to unenforceable until there is a sufficient quantity of dead organisms . . . .”<sup>170</sup>

The difference between numerical and narrative standards is quite simple: one works. A speed limit of 65 mph is obvious to any driver, state trooper, and court of law; 70 mph breaks the law. A limit of “unreasonable speed” is obvious to no one and, depending on the circumstances and the resources of the arrested driver, begs an argument to come. How could it be unreasonable if there was no one else on the highway? Or if the driver were Mario Andretti? Or if his congressman calls up and asks for a little understanding? This is why we have speed limits, blood alcohol thresholds, and ambient air quality standards, all expressed in numbers. For things we care about protecting--highway safety, clean air--we assign numerical limits.<sup>171</sup>

Under the ESA, fish and wildlife agencies must impose limits on the take of threatened and endangered species to ensure the species’ populations will not be put in jeopardy of extinction.<sup>172</sup> Courts have recognized that Congress expressed a clear “preference for expressing take in numerical form.”<sup>173</sup> A numerical limit allows both the actors and the regulators to know when the allowable level of take has been exceeded and it is necessary to reinitiate ESA consultation and develop a more effective mitigation plan.

Under another CWA program that requires cities to obtain permits to control stormwater pollution, the permits “shall require controls to reduce the discharge of pollutants to the maximum extent practicable . . . .”<sup>174</sup> A court of appeals in Maryland recently found Montgomery County’s stormwater permit inadequate under this standard because it consisted of

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<sup>168</sup> EPA Press Release, EPA Clean Water Action Plan, *available at* <http://www2.epa.gov/aboutepa/clean-water-action-plan> (Feb. 19, 1998); *see generally* EPA & USDA, Clean Water Action Plan: Restoring and Protecting America’s Waters 58-59 (1998) (AR000142-43).

<sup>169</sup> *Florida Wildlife Federation v. Jackson*, 853 F.Supp.2d 1138 (N.D. Fl. 2012), *appeal dismissed*, 737 F.3d 689 (11th Cir. 2013).

<sup>170</sup> *See* Oliver Houck, *Cooperative Federalism, Nutrients, and the Clean Water Act: Three Cases Revisited*, 44 *Envl. L. Rep. News & Analysis* 10426, 10431 (May 2014).

<sup>171</sup> *Id.*

<sup>172</sup> 16 U.S.C. § 1536(b)(4); 50 C.F.R. § 402.14(i).

<sup>173</sup> *Oregon Natural Resources Council v. Allen*, 476 F.3d 1031, 1037 (9<sup>th</sup> Cir. 2007).

<sup>174</sup> 33 U.S.C. § 1342(p)(3)(B).

aspirational goals instead of particularized objectives, such as numeric limits.<sup>175</sup> The court found fault because the permit did “not contain ascertainable metrics that define how the County must comply, or whether at some point it has complied.”<sup>176</sup> Only if the permit articulates what the County must do, would the County know what is required of it.<sup>177</sup> Not only do clear permit requirements foster compliance, but they enable the public to monitor and enforce permits.

Pesticide labels are analogous. If they contain general admonitions or aspirational goals, it will be difficult for applicators and growers to know what is required of them. More prescriptive label directions enable pesticide users to avoid the prohibited result by following the detailed directions. In addition, it is far easier for the states and EPA to ascertain whether users have run afoul of clear label directions as opposed to general admonitions like avoid direct drift onto people. To discharge its obligations to prevent unreasonable adverse effects and ensure reasonable certainty of no harm, EPA cannot leave direct drift to the general prohibition in the WPS and on pesticide labels and must instead require mitigation measures that will ensure that result will not occur.

### C. EPA Underestimates Worker Risks By Making Unsupported Assumptions.

EPA’s occupational risk assessment underestimates the risks to workers by ignoring demonstrated neurodevelopmental effects, ignoring common routes of exposure, and making assumptions that are not reflective of real-world exposures.

#### 1. EPA Improperly Ignores Neurodevelopmental Effects.

EPA’s occupational risk assessment suffers from the same fundamental flaw that permeates the entire RHHRA by continuing to use 10% cholinesterase inhibition as the target endpoint in the face of demonstrated evidence and its own finding that neurodevelopmental damage to children occurs from *in utero* exposures. It is standard policy for EPA and OSHA to protect pregnant workers from such harm, and yet EPA ignores this risk altogether in its occupational risk assessment.

This fundamental error is reflected in EPA’s use of DOW’s PBPK-PD model to estimate worker risk, as well as its use of a 100X safety factor. RHHRA at 64. Under EPA’s 2009 policy, it is now incorporating the science that has developed under the FQPA and become standard risk assessment methodology when it assesses worker risks. Specifically, it will incorporate an additional safety factor to protect against fetal toxicity or incomplete data about such toxicity in its worker risk assessments.<sup>178</sup> While EPA purports to have done so in its chlorpyrifos worker risk assessment, it concurrently eliminated the traditional safety factor for uncertainties in extrapolating from animal studies. As discussed above, it is arbitrary for EPA to eliminate this safety factor based on ethical, scientifically flawed human studies used to design a model targeted to an endpoint that is not the most sensitive critical effect from chlorpyrifos.

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<sup>175</sup> See *Maryland Dep’t of the Env’t v. Riverkeeper*, No. 2199 Sept. Term 2013, 2015 WL 1510556, at \*15-16 (Md. Ct. Spec. App. Apr. 2, 2015).

<sup>176</sup> *Id.* at \*11.

<sup>177</sup> *Id.* at \*13-16.

<sup>178</sup> EPA, Revised Risk Assessment Methods for Workers, Children of Workers in Agricultural Fields, and Pesticides with no Food Uses at 4-5, 7 (Dec. 7, 2009).

2. EPA Makes Assumptions in its Occupational Risk Assessment that Do Not Reflect Real-World Exposures.

EPA made other inappropriate assumptions such that exposure levels under real conditions or uses would be even greater than those calculated by EPA. Those assumptions, related to exposure estimates, include (a) an unrealistically high body weight that is not representative of farmworker youth; (b) an eight-hour work day; (c) adequate cleaning, laundry, and changing facilities; and (d) ignoring exposure to the oxon form in most cases.

- a. EPA uses an unrealistically high body weight that does not protect farmworker youth.

In the RHHRA, EPA has calculated inhalation or skin exposure assuming a worker body weight of 69 kg (152 lbs) for an adult female between the ages of 13 to 49. RHHRA at 64. In its 2009 policy, EPA acknowledges that it must protect farmworker children.<sup>179</sup> In its residential exposure assessment, EPA uses 57 kg as the standard body weight for youth 11-16 years old, which conforms to its Exposure Factors Handbook.<sup>180</sup> EPA is using a body weight in its exposure assessment that is not protective of farmworker youth.

Several sources estimate that hundreds of thousands of youth under the age of 18 years work in U.S. agriculture. In 2012, farm operators reported directly hiring 130,232 children under age 18 to work on crop and livestock farms, and an additional 388,084 children worked on the farms on which they resided.<sup>181</sup> A 2014 report by Human Rights Watch suggested that this was an underestimate of total number of children working in agriculture as it excluded children hired by farm labor contractors or employed informally.<sup>182</sup> A USDA 2008 report indicated that between 1989 and 2006, on average, children under age 18 made up 5.5 percent of the hired crop farmworker labor force,<sup>183</sup> while a Department of Labor estimate in 2000 suggested that approximately 126,000 children aged 14 to 17 work on America's farms each year.<sup>184</sup>

- b. EPA assumes an 8-hour workday, which underestimates the duration and extent of exposure.

EPA's occupational risk assessment assumes an 8-hour workday. However, in 2008, the USDA reported that from 68-81% of hired farmworkers and 78-82% of wage and salary workers worked more than 40 hours per week. In both groups, more than 80% of the non-citizen workers

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<sup>179</sup> EPA, Revised Risk Assessment Methods for Workers, Children of Workers in Agricultural Fields, and Pesticides with no Food Uses at 7 (Dec. 7, 2009).

<sup>180</sup> Occupational and Residential Exposure Assessment at 19; EPA, Exposure Factors Handbook, Chapter 8 – Body Weight Studies, Table 8-1, at 8-2 (2011), available at <http://www.epa.gov/ncea/efh/pdfs/efh-chapter08.pdf>. (Ex. 2).

<sup>181</sup> Human Rights Watch, *Tobacco's Hidden Children* (2014), available at [http://www.hrw.org/sites/default/files/reports/us0514\\_UploadNew.pdf](http://www.hrw.org/sites/default/files/reports/us0514_UploadNew.pdf) (Ex. 17) (citing Email from Kitty Hendricks, Research Health Scientist, Division of Safety Research, National Institute for Occupational Safety and Health (NIOSH), to Human Rights Watch, December 31, 2013).

<sup>182</sup> Human Rights Watch at 30 (citing telephone conversation with Kitty Hendricks, Research Health Scientist, Division of Safety Research, NIOSH, February 7, 2014).

<sup>183</sup> William Kandel, USDA, *A Profile of Hired Farmworkers, A 2008 Update* (July 2008), available at <http://www.ers.usda.gov/publications/err-economic-research-report/err60.aspx> (Ex. 18).

<sup>184</sup> Department of Labor, Report on Children in the Labor Force at 53 (2000), available at <http://www.bls.gov/opub/rylf/pdf/chapter5.pdf> (Ex. 19).

worked more than 40 hours per week.<sup>185</sup> Recent findings from the U.S. Department of Labor's National Agricultural Workers Survey indicate that 54% of workers interviewed worked more than 40 hours per week.<sup>186</sup> By assuming an 8-hour workday, EPA is underestimating exposure.

c. Assumption of adequate cleaning facilities underestimates exposure

Exposure continues as long as workers cannot adequately change out of contaminated clothing and into clean clothing. It is unrealistic to assume daily showers following exposure.

A 2007 survey of farmworker camps in North Carolina concluded that, "at any point in the 2007 agricultural season, between 11% and 44% of camps had inadequate bathing, laundry, or storage facilities."<sup>187</sup> Other studies of North Carolina farmworker camps showed that both bathroom and laundry room violations were common (at 4.5 and 1.2 violations per camp respectively),<sup>188</sup> and that bathroom violations were predictive of increased exposure to chlorpyrifos (as measured in urinary metabolites).<sup>189</sup>

A complementary study demonstrated that farmworkers provided with adequate washing facilities (water for bathing and laundry, soap for hand washing, and water for hand washing at work) were more likely to use pesticide safety practices or PPE.<sup>190</sup>

d. EPA Fails To Account For Exposure To Dust.

As discussed above, chlorpyrifos is ubiquitous in household dust. Not only do workers bring pesticide dust home to their families, but they continue to be exposed to pesticide residues through dust, particularly in the absence of adequate showering and laundering facilities. Because chlorpyrifos degrades slowly and is very persistent in the indoor environment, EPA must account for the fact that worker exposures continue for long periods of time after they leave the fields.

e. Oxon exposures should not be ignored

EPA also excluded consideration of occupational exposure to the oxon form of chlorpyrifos except "under very limited circumstances in greenhouses for post-application workers." RHHRA at 12. According to EPA, the oxon form is between 11.9 (acute) and 18

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<sup>185</sup> Kandel, 2008, at 16.

<sup>186</sup> U.S. Department of Labor. This analysis utilizes the National Agricultural Workers Survey public access data from fiscal years 2011-2012. The data can be retrieved from <http://www.doleta.gov/agworker/naws.cfm>.

<sup>187</sup> Quirina M. Vallejos *et al.*, *Migrant Farmworkers' Housing Conditions Across an Agricultural Season in North Carolina*, 54 *Am. J. Indus. Med.* 533 (2011) (Ex. 20); *see also id.* at 15, Table II (providing more specific information showing variability in access to facilities throughout the growing season).

<sup>188</sup> Thomas A. Arcury *et al.*, *Migrant Farmworker Housing Regulation Violations in North Carolina*, 55 *Am. J. Indus. Med.* 191 (2012), available at <http://www.ncbi.nlm.nih.gov/pubmed/22237961> (Ex. 21).

<sup>189</sup> J. H. Raymer *et al.*, *Pesticide exposures to migrant farmworkers in eastern NC: Detection of metabolites in farmworker urine associated with housing violations and camp characteristics*, 57 *Am. J. Indus. Med.* 323 (2014) (Ex. 22).

<sup>190</sup> D. L. Levesque *et al.*, *Association between workplace and housing conditions and use of pesticide safety practices and personal protective equipment among North Carolina farmworkers in 2010*, 3 *Int'l J. Occup. and Env'tl. Med.* 53 (2012).

(chronic) times more toxic than the parent and inhibits brain acetylcholinesterase at 1,000 times the rate of chlorpyrifos. RHHRA at 106.<sup>191</sup> The oxon form has been detected outdoors, though EPA argues at very low levels compared to the parent compound in an attempt to justify ignoring chlorpyrifos oxon exposure outside greenhouses. Although trace levels of oxon metabolite that are formed are generally rapidly hydrolyzed,<sup>192</sup> the presence of the oxon and potential exposure among workers, should not be discounted.

Agricultural applications of organophosphates and their oxon products may have substantial volatilization and off-field movement representing a probable source of exposure to both parent and oxon form. Cole et al. interpreted results from a mouse study to suggest that degradation in the field of chlorpyrifos to its oxon form may represent a significant contaminant in exposures among both adults and children (with particular concerns for young children with reduced levels of the enzyme detoxifying paraoxonase (PON1) involved in the detoxification of both chlorpyrifos and its oxon).<sup>193</sup>

California Air Resources Board collected air samples in agricultural communities and reported that agricultural use of chlorpyrifos within a three-mile radius on the monitoring day, and use on the two to four prior days, were significantly associated with air concentrations of chlorpyrifos ( $p < 0.01$ ). Among analytes tested, chlorpyrifos oxon showed the strongest association ( $p < 0.0001$ ).<sup>194</sup> Environmental oxons were also previously identified in environmental samples from air and surfaces following agricultural spray applications in California (in the range of  $0.07 \text{ ug/m}^3$  to  $3.01 \text{ ug/m}^3$ )<sup>195</sup> and Washington State.<sup>196</sup> Air monitoring data from the California Department of Pesticide Regulation consistently detects the oxon at a similar frequency as chlorpyrifos, suggesting that the oxon is common outdoors (for example, in air monitoring data from 2013, chlorpyrifos had a detection frequency of 33%, while the oxon had a detection frequency of 26%).<sup>197</sup>

Uncertainty about presence of the oxon form is not a justification for its dismissal. In fact, dismissal of presence and importance of oxon metabolites is ill-advised both due to greater known toxicity of the oxon form compared to the parent and because there remain concerns that oxon metabolites may act in ways other than on cholinergic receptors. The oxon metabolites of several OP pesticides, including chlorpyrifos, have been shown to damage mature sperm

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<sup>191</sup> Huff, R. A., A. W. Abu-Qare and M. B. Abou-Donia (2001). Effects of sub-chronic in vivo chlorpyrifos exposure on muscarinic receptors and adenylate cyclase of rat striatum. *Arch Toxicol* 75(8): 480-6. Cited in: [http://oehha.ca.gov/prop65/hazard\\_ident/pdf\\_zip/ChlorpyrifosHID0908.pdf](http://oehha.ca.gov/prop65/hazard_ident/pdf_zip/ChlorpyrifosHID0908.pdf).

<sup>192</sup> 2012 FIFRA SAP at 60.

<sup>193</sup> Toby B. Cole et al., *Neurobehavioral assessment of mice following repeated postnatal exposure to chlorpyrifos-oxon*, 34 *Neurotoxicol Teratol.* 311 (2012) (Ex. 31).

<sup>194</sup> Martha Harnly et al., Correlating agricultural use of organophosphates with outdoor air concentrations: a particular concern for children CA DPH, 113 *Envtl. Health Persp.* 1184 (2005), available at [http://www.ehib.org/paper.jsp?paper\\_key=AIR\\_OP\\_PESTS](http://www.ehib.org/paper.jsp?paper_key=AIR_OP_PESTS) (Ex. 32).

<sup>195</sup> California Air Resources Board, Final Report for the 1996 Chlorpyrifos Monitoring in Tulare County (Apr. 13, 1998), available at <http://www.cdpr.ca.gov/docs/emon/pubs/tac/tacpdfs/chlrpfs.pdf> (Ex. 33).

<sup>196</sup> Jenna L. Armstrong et al., *Presence of organophosphorus pesticide oxygen analogs in air samples*, 66 *Atmos. Evt.* 145 (2013) (Ex. 34).

<sup>197</sup> Vidrio, Edgar, Pamela Wofford, Randy Segawa, and Jay Schreider. "Air Monitoring Network Results for 2013: Volume 3." California Department of Pesticide Regulation, December 2014.

chromatin, particularly the DNA, being more toxic than their parent compounds.<sup>198</sup>

D. EPA Overestimates the efficacy of Protective Clothing and Engineering Controls.

In addition to concerns regarding exposure estimates, use of Personal Protective Equipment (PPE) often provides inadequate protection. As EPA notes, engineering controls provide much better protection than PPE. Therefore, the threshold for switching from PPE to engineering controls should, in general, be lowered. However, EPA also identified several scenarios in which even the use of engineering controls provided inadequate protection. These uses should immediately be disallowed altogether in the absence of a complete ban of chlorpyrifos. Furthermore, additional uses should be disallowed when the use of engineering controls presents additional hazards such as heat stress when using closed cabs without adequate air conditioning. As a result of these considerations, an even greater number of scenarios (product, application methods, PPE, engineering controls) would fall below an MOE of 100 and/or increase the degree to which the associated MOEs fall below 100. If this is the case, then many scenarios would need to be immediately prohibited altogether.

1. Personal protective equipment provides inadequate protection and increases certain risks

Use of PPE for protection during pesticide loading and mixing of pesticides is inadequate. For example, although the WPS permits the use of safety glasses to satisfy the requirements for eye protections, a Washington State study found that safety glasses “were not effective in protecting against splashes or wind-blown spray mist.”<sup>199</sup> “Black light and fluorescent tracers dramatically demonstrate the extent to which pesticide exposure may occur, even with the use of PPE.”<sup>200</sup> In addition, it is well recognized that a full set of protective clothing is “cumbersome and can be very uncomfortable in hot weather, causing workers to shed their protective gear.”<sup>201</sup> Indeed, an analysis performed by EPA scientists concluded that wearing a full body Tyvek coverall over a shirt and pants would likely produce an internal body temperature of 38.3 degrees centigrade (or 100.94 degrees Fahrenheit), at the cusp of the body temperature that is considered a sign of heat stress.<sup>202</sup> Thus, if a pesticide handler wore full PPE while mixing and loading pesticides, there would be a real risk that heat stress symptoms would

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<sup>198</sup> Elsa Salazar-Arredondo et al., Sperm chromatin alteration and DNA damage by methyl-parathion, chlorpyrifos and diazinon and their oxon metabolites in human spermatozoa, 25 *Reproductive Toxicology* 455 (2008) (Ex. 35).

<sup>199</sup> Washington State Department of Health Pesticide Incident Reporting & Tracking Panel, 2000-2001 Annual Report (2002), available at <http://www.doh.wa.gov/Portals/1/Documents/Pubs/334-293.pdf> (Ex. 36); see also Washington State Department of Health., Pesticide Incident Reporting & Tracking Panel, 2009, Annual Report 61-64 (2009).

<sup>200</sup> Frederick M. Fishel, *Exposing Pesticide Exposure Using Fluorescent Tracer Dyes* (2014), available at <http://edis.ifas.ufl.edu/pdf/PI/PI19900.pdf> (Ex. 37).

<sup>201</sup> Jacobs, WW. 1982. Closed Mixing and Loading Systems and Pesticide Containers, in *PESTICIDE TANK MIX APPLICATIONS: FIRST CONFERENCE* 58, 61 (John F. Wright et al. eds., 1982); Rutz, R. 1987. Closed System Acceptance and Use in California, in *Pesticide Formulations and Application Systems Vol. 7*, at 28-34 (G.B. Beestman & D.I.B.Vander Hooven eds., 1987).

<sup>202</sup> Lunchick, C et al. 1988. Engineering Controls and Protective Clothing in the Reduction of Pesticide Exposure to Tractor Drivers, in *PERFORMANCE OF PROTECTIVE CLOTHING: SECOND SYMPOSIUM* 605, 608 (Seymour Zack Mansdorf et al. eds., 1988).

reduce his/her alertness, creating a potential hazard.<sup>203</sup>

Moreover, many employers do not provide adequate PPE to their employees. Among the Washington State pesticide handlers who suffered an acute pesticide related illness in 2008, 56% were missing at least one piece of required PPE; and the most common reason was that the employer did not provide it.<sup>204</sup> In this circumstance, some farmworkers may be reluctant to request the missing PPE for fear of retaliation; other farmworkers may not make this request because they do not know they are entitled to PPE, or that it is needed. In other instances, the PPE provided by the employer was in poor repair or did not fit well – problems that were especially prevalent with respirators and goggles.<sup>205</sup>

One reason that workers are unaware of the PPE requirements is that they cannot read them on the pesticide label. Pesticides are often applied by individuals who read only Spanish, and EPA does not require that label language be printed in Spanish.

## 2. Engineering controls offer improved, yet still inadequate protections

Numerous studies have confirmed that engineering controls (e.g., enclosed cabs or cockpits, water-soluble packaging, and closed mixing/loading systems) can significantly reduce workers' exposures compared to properly used PPE. EPA's own Pesticide Handler Unit Exposure Data dramatically illustrates this point.

The most recent Occupational Pesticide Handler Unit Exposure Surrogate Reference Table assumes that a pesticide handler mixing and loading wettable powder pesticides would have a dermal exposure of between 130 and 3700 micrograms per pound of active ingredient if the handler relied on PPE, but only 9.8 micrograms per pound of active ingredient using water-soluble packaging.<sup>206</sup> In other words, workers who do not use engineered protections are potentially exposed to over 375 times as much of the pesticide active ingredient compared to the exposures if engineered systems are in place.

Similarly, a pesticide handler mixing and loading dry flowable pesticides would have a dermal exposure of between 41.2 and 227 micrograms per pound of active ingredient if s/he relied on PPE, but only 9.8 micrograms per pound of active ingredient using the closed system of water-soluble packaging. A pesticide handler mixing and loading dry flowable pesticides would have a dermal exposure of between 41.2 and 227 micrograms per pound of active ingredient if s/he relied on PPE, but only 9.8 micrograms per pound of active ingredient using the closed system of water-soluble packaging. In both cases, use of closed systems would reduce exposures by over twenty-fold compared to the most basic PPE level.<sup>207</sup> Older studies too showed dramatic safety benefits to using closed systems rather than relying exclusively on PPE.<sup>208</sup>

California's Department of Pesticide Regulation—DPR is currently in the process of

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<sup>203</sup> *Id.*

<sup>204</sup> Washington PIRT 2010 Report, at 61.

<sup>205</sup> Washington PIRT 2010 Report, at 62.

<sup>206</sup> EPA OPP, Occupational Pesticide Handler Unit Exposure Surrogate Reference Table 1 (2013).

<sup>207</sup> *Id.*

<sup>208</sup> Rutz, *Closed System Acceptance and Use in California*, in *Pesticide Formulations and Application Systems*, at 28-34

reviewing and modifying state regulations regarding closed systems.<sup>209</sup> DPR's analysis suggests that use of closed systems may serve to reduce the chance for dermal exposure from spills which can contribute to both worker dermal and take home exposure.

## VI. EPA MUST PREVENT HARMFUL DRINKING WATER CONTAMINATION.

The drinking water risk estimates in EPA's preliminary HHRA indicate that all infants (children under 1 year old) are exposed to chlorpyrifos in drinking water at levels that exceed EPA's levels of concern for all scenarios. The RHHRA uses different modeling, but EPA continues to find that a substantial amount of chlorpyrifos uses will result in exceedances of EPA's drinking water levels of concern.<sup>210</sup> This is a very serious finding, particularly since both assessments use 10% cholinesterase inhibition as the regulatory endpoint and fail to protect children from brain damage. EPA's assessment under-estimates the risks because it is based on assumptions at odds with the scientific evidence and EPA's own findings. Therefore, the risks are likely to be even greater. This section makes the following points:

- (1) EPA appropriately finds that chlorpyrifos-oxon would be present in finished drinking water.
- (2) The drinking water level of concern (DWLOC) was calculated based on 10% cholinesterase inhibition as the regulatory endpoint, which suffers from serious flaws as described above, including the failure to protect infants and children from neurodevelopmental impacts.
- (3) Using the flawed DWLOC, EPA finds that existing label regulations are inadequate to prevent unsafe chlorpyrifos contamination of drinking water. Numerous chlorpyrifos application scenarios cause contamination of drinking water at levels exceeding the DWLOC.
- (4) EPA appropriately determined that the identification of chlorpyrifos drinking water exceedances is likely to be conservative because EPA's modeling is validated by empirical monitoring data.
- (5) EPA cannot ignore or refuse to mitigate risks simply because best-case scenarios in some locations may lessen those risks. EPA must cancel all uses that produce exceedances of drinking water levels of concern.

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<sup>209</sup> California Department of Pesticide Regulation. Initial Statement Of Reasons And Public Report. Title3. California Code of Regulations Amend Sections 6000, 6188, 6742, 6746, and 6793 Pertaining to Closed Mixing Systems [http://www.cdpr.ca.gov/docs/legbills/rulepkgs/14-004/initial\\_reasons.pdf](http://www.cdpr.ca.gov/docs/legbills/rulepkgs/14-004/initial_reasons.pdf)

<sup>210</sup> EPA, Office of Chemical Safety and Pollution Prevention, Chlorpyrifos: Updated Drinking Water Assessment for Registration Review (Dec. 23, 2014). The RHHRA uses a combined 50X safety factor based on the Dow model and a 10X FQPA safety factor, while the preliminary HHRA used a 100X combined safety factor since it assumed an FQPA safety factor of 0 and 100X for the traditional safety factors.

- A. EPA appropriately finds that chlorpyrifos-oxon would be present in finished drinking water.

Chlorination, the most common drinking water treatment, results in chlorpyrifos being converted to chlorpyrifos-oxon. Drinking water systems are not equipped to remove pesticide residues, including chlorpyrifos or chlorpyrifos-oxon, from finished drinking water. Therefore, EPA's evaluation reasonably and appropriately proceeds with the assumption that chlorpyrifos entering a drinking water system would be converted to chlorpyrifos-oxon, and that the chlorpyrifos-oxon would be present in the finished drinking water.<sup>211</sup>

1. The drinking water level of concern (DWLOC) was calculated based on 10% cholinesterase inhibition, which suffers from serious flaws, including failure to protect infants and children from neurodevelopmental impacts.

The Health Effects Division calculated the DWLOC by determining the average 21-day concentration of chlorpyrifos-oxon necessary to cause **10% Cholinesterase Inhibition** and then dividing by a safety factor of 50X (10X FQPA data deficiency for infants by 5X intraspecies for infants for chlorpyrifos-oxon).<sup>212</sup> As described above, neurodevelopmental effects, and not 10% cholinesterase inhibition, are the most sensitive endpoint related to chlorpyrifos toxicity. Protecting against 10% cholinesterase inhibition will not protect against the neurodevelopmental impacts, because these impacts occur at much lower exposure levels as described above. The DWLOC needed to protect the most sensitive population, which may be infants or the fetuses of pregnant women, from neurological harm would likely be significantly lower than the current value that EPA used, 3.9 ug/ L (ppb).

2. Even using the flawed DWLOC, EPA finds that existing label regulations are inadequate to prevent unsafe chlorpyrifos contamination of drinking water.

EPA first completed a national screen, estimating drinking water contamination from a single chlorpyrifos application at a rate of 1 or 4 pounds per acre for the screen. Chlorpyrifos can be used at lower and higher application rates, and in more than a single application per year.

For the low application rate, exceedances are expected at the 99<sup>th</sup> percentile for 51 of the 124 scenarios (41%) evaluated. For the high application rate, exceedances are expected at the 99<sup>th</sup> percentile for all but one of the uses evaluated: 20 of the 21 scenarios (**95%**). Indeed, the drinking water assessment notes that "The current maximum single application rates for a wide range of chlorpyrifos use scenarios may result in a 21 day average concentration that exceed the DWLOC."<sup>213</sup> In total for the low and high application rates, exceedances are expected for about half (49%) of all scenarios evaluated.

EPA then completed a regional screen, looking at the Pacific Northwest and the South Atlantic Gulf regions, again for a single chlorpyrifos application at a rate of 1 or 4 pounds per acre. In the Pacific Northwest, no exceedances were expected for the 15 scenarios evaluated at

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<sup>211</sup> *Id.* at 2-3.

<sup>212</sup> *Id.* at 4.

<sup>213</sup> *Id.* at 23.

the low application rate. At the high application rate, again all but one, 4 of the 5 scenarios (80%) had exceedances at the 99<sup>th</sup> percentile.

For the South Atlantic Gulf, at the low application rate, exceedances were expected for 9 of the 26 scenarios (35%) at the 99<sup>th</sup> percentile. EPA did not evaluate the higher application rate but notes that "**more exceedances are expected for higher application rates.**"<sup>214</sup> Indeed, the other evaluations above of the high application rate showed that almost all resulted in exceedances.

All of EPA's national and regional modeling was completed based on a single chlorpyrifos application. EPA notes that "For those scenarios where exceedances are already expected, **more exceedances would be expected for multiple applications.**"<sup>215</sup>

EPA's national and regional evaluations found that existing label regulations do not adequately protect drinking water from harmful chlorpyrifos contamination. Even for a single application of chlorpyrifos at a low application rate (1 lb/ acre), exceedances of the DWLOC are expected for many scenarios. Almost all scenarios at the higher application rate (4 lb/ acre) result in exceedances. The highest application rates (for example, 6 lb/ acre on citrus) or multiple applications a year would increase the level of the exceedance, as well as the number of scenarios expected to result in exceedances.

3. EPA accurately determined that the identification of chlorpyrifos drinking water exceedances is likely to be conservative because EPA's modeling is validated by empirical monitoring data.

In order to validate that its model provides an accurate estimate of chlorpyrifos drinking water concentrations, EPA compared model outputs to water monitoring data from a number of sources across the country, including the National Water Quality Assessment Program, California, and Washington State. Based on the comparison results, EPA concludes that "This analysis demonstrates that the model estimated concentration reasonably compare to measured concentrations."<sup>216</sup> The concordance with monitoring data bolsters confidence that EPA's predictions of exceedances are realistic and accurate.

4. EPA cannot ignore the risks, but must immediately cancel uses that result in unsafe levels of chlorpyrifos in drinking water.

In order to better understand which water systems may be affected by exceedances, EPA completed a preliminary analysis looking at corn cropland overlaid with drinking water intake watersheds for the South Atlantic Gulf. This analysis considered only one crop, corn, with a single chlorpyrifos application made at the low application rate 1 lb/ acre. Even for this minimal scenario, exceedances are expected for 3% of the watersheds, each serving multiple community water systems. This analysis did not consider the other main crops which led to exceedances in the regional screen, such as cotton, soybeans, vegetable and ground fruit, fruit and nut trees. Furthermore, higher application rates and multiple applications were also not considered. If

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<sup>214</sup> *Id.* at 25.

<sup>215</sup> *Id.* at 25.

<sup>216</sup> *Id.* at 35.

these were also modeled, many more watersheds with harmful chlorpyrifos contamination would be identified.

EPA notes that variations are expected in the chlorpyrifos water contamination based on the particulars of an area, such as the specific crops, chlorpyrifos use patterns, watershed size and how much area is covered with crops. Some areas with larger watersheds or not as much area covered with crops might be in the fortunate situation of not having dangerous chlorpyrifos drinking water contamination. However, for the remaining watersheds and drinking water systems, the fact remains that many registered chlorpyrifos uses would result in drinking water contamination that is unsafe for infants, according to EPA estimates. Under the FQPA, it is not acceptable for infants to be sickened by chlorpyrifos in the unlucky communities with intensive agriculture and small watersheds.

Furthermore, EPA estimated drinking water exposures for infants in the 2011 PRA, and found that all infants would be ingesting chlorpyrifos-oxon at levels that exceed the level of concern for all crop scenarios. Introducing regional variation cannot be a basis for delaying or taking no action to protect children. Especially given that EPA is using the wrong and least sensitive regulatory endpoint and a reduction in the traditional safety factors, it must consider whether the FQPA safety factor must be enlarged to account for the incomplete data on drinking water contamination and exposures. Such an enlarged safety factor would inevitably mean that EPA must cancel all uses of chlorpyrifos to protect children from neurodevelopmental damage.

## VII. THE RHHRA FAILS TO ANALYZE AND PROTECT AGAINST ENVIRONMENTAL JUSTICE IMPACTS.

The discussion of environmental justice in the RHHRA is limited to a scant one page of analysis that leaves unaddressed and unmitigated myriad environmental justice concerns. RHHRA at 17-18. Instead of engaging in a robust analysis of environmental justice impacts, the RHHRA does no more than flag that an Executive Order requires EPA to consider the disproportionate burdens of its actions on people of color and low-income communities. EPA undertook no analysis of those burdens. EPA must go back to the drawing board and analyze the disproportionate impacts of chlorpyrifos—there are many, particularly to farmworkers and their families—and take steps through a ban and mitigation measures such as buffers while a ban is being instituted to protect against chlorpyrifos exposures.

### A. Background

The 1994 Environmental Justice Executive Order requires EPA to ensure that its actions do not have disproportionate impacts on low-income and/or minority populations. *Exec. Order No. 12,898*, 59 Fed. Reg. 7,629 (Feb. 11, 1994). Specifically, EPA and other executive agencies must, to the maximum extent practicable, “identify[] and address[] . . . disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low income populations.” *Id.* at § 1-101. In furtherance of this mandate, EPA is required to “collect, maintain, and analyze information assessing and comparing environmental and human health risks borne by populations identified by race, national origin, or income” and “use this information to determine whether their programs, policies, and activities have disproportionately high and adverse human health or environmental effects on minority populations and low-income populations . . . .” *Id.* at § 3-302(a).

Likewise, the 1997 Executive Order on Children’s Health requires EPA to protect children from environmental health and safety risks. *Exec. Order No. 13,045*, 62 Fed. Reg. 19,885 (Apr. 23, 1997). Specifically, EPA is required to “ensure that its policies, programs, activities, and standards address disproportionate risks to children that result from environmental health or safety risks . . . that are attributable to products or substances that the child is likely to come in contact with or ingest (such as the air we breath [sic], the food we eat, the water we drink or use for recreation, the soil we live on, and the products we use or are exposed to).” *Id.* at §§ 1-101(b), 2-202(b). Viewed together, these two executive orders require EPA, in making pesticide registration and tolerance decisions such as in the case of chlorpyrifos, to assess pesticide drift exposures and all other pesticide exposures to ensure that chlorpyrifos exposures do not disproportionately impact children, low income populations, and/or minority populations.

Chlorpyrifos has a long history of causing significant impacts to people, and since at least 2000 those impacts have largely been confined to rural children and adults, often farmworkers and their families. The unacceptable impacts of chlorpyrifos on children through home uses—exposures in the home itself and on lawns—led EPA to negotiate a phase out of home uses in 2000. However, at that time and continuing to today, EPA has failed to protect rural children from similar harms, despite acknowledging its obligation to protect children and other bystanders from chlorpyrifos drift and volatilization. Because the children most often exposed to chlorpyrifos are the children of farmworkers, this harm falls disproportionately on children in low-income families and communities of color.

#### 1. EPA’s Treatment of Environmental Justice in the RHHRA Is Inadequate.

The Environmental Justice Executive Order requires EPA to address disproportionate impacts of pesticide use on minority and low income populations, and the Child Health Executive Order requires EPA to address risks to children from pesticides. Contrary to these obligations, EPA has ignored the broad impacts chlorpyrifos has directly and pervasively on low income minority children who live near the fields. Indeed, EPA maintains a double-standard by protecting children from urban and residential uses, but ignoring exposures to children who live, play, and go to school near fields. These failures not only violate EPA’s statutory obligations, they also violate EPA’s obligations to address disproportionate impacts to children, minority, and low-income populations when it authorizes pesticide uses.

EPA does no more than pay lip service to environmental justice impacts in the RHHRA, and by failing to include a robust analysis it is in violation of these two executive orders and has acted arbitrarily. EPA’s environmental justice section includes less than one page of text, spanning two pages of the document. *See RHHRA* at 17-18. In that section, EPA considered “food and water consumption, and activities in and around the home that involve pesticide use in a residential setting.” *Id.* at 17. EPA goes on to mention NHANES/WWEIA data that analyze pesticide risk from food use, though EPA fails to discuss how that data is useful here and what it says about chlorpyrifos. Rather than engage in the required, robust analysis, EPA listed without discussion two areas of potential environmental justice impacts, while ignoring at least two other major environmental justice issue areas.

2. EPA Failed to Analyze and Mitigate Environmental Justice Impacts from Spray Drift and Volatilization.

Chlorpyrifos is found in air and water across the United States. In California, for example, the California Department of Pesticide Regulation showed chlorpyrifos as having the highest number of detections in its 2011, 2012 and 2013 air monitoring.<sup>217</sup> At the same time, water monitoring showed chlorpyrifos in 17.7% of samples and exceeding the concentration limit in 9.9% of samples. *Id.* In California, the counties with the highest use of chlorpyrifos are the counties with the highest levels of poverty and Latino/a populations. *Id.* at 2-3. Many residents of the areas most affected suffer exposures to multiple chemicals and many are monolingual Spanish speakers who are underserved by state and federal decision makers.

Likewise, in April of 2014, the California Department of Public Health issued a report showing that thousands of children, disproportionately people of color, attend school in close proximity to pesticide use.<sup>218</sup> It also found that chlorpyrifos was the eighth most common highly hazardous pesticide used within a quarter mile of public schools in the counties it studied. Latino/a children made up 54.1% of the population for all public schools in the counties studied but made up 67.7% of the population for schools in the highest quartile of pesticide use.<sup>219</sup> Latino children were 46% more likely than white children to attend schools with any use of pesticides within a quarter mile and 91% more likely to attend a school in the top quartile of pesticide use.<sup>220</sup>

Despite this overwhelming evidence of impact, EPA mentions only in passing that it would evaluate potential exposures from spray drift and volatilization in another section of the RHHRA. That inadequate analysis,<sup>221</sup> however, is no substitute for a full and robust assessment of environmental justice impacts. EPA is required to “identify[] and address[] . . . disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low income populations.” *Exec. Order No. 12,898* § 1-101, 59 Fed. Reg. 7,629 (Feb. 11, 1994).

EPA has long known about the risks pesticide drift causes to children, and especially rural children and the children of farmworkers. In 1993, the NAS published a pivotal study documenting the ways pesticides pose severe risks to infants and children. NAS found that pesticides pose heightened risks to children because “[i]nfants and children are growing and developing,” “[t]heir metabolic rates are more rapid than adults,” and “[t]here are differences in their ability to activate, detoxify, and excrete xenobiotic compounds.”<sup>222</sup> Children are also at

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<sup>217</sup> See Letter from Tracey Brieger, Californians for Pesticide Reform et al., to Arsenio Mataka, Assistant Secretary for Environmental Justice and Tribal Affairs, Cal. Env'tl. Prot. Agency at 1 (Aug. 26, 2014) (“Coalition Letter”).

<sup>218</sup> California Environmental Health Tracking Program, *Agricultural Pesticide Use Near Public Schools in California* (“Schools Report”), available at [http://cehtp.org/projects/ehss01/pesticides\\_and\\_schools/Pesticides\\_Schools\\_Report\\_April2014](http://cehtp.org/projects/ehss01/pesticides_and_schools/Pesticides_Schools_Report_April2014) (Ex. 39).

<sup>219</sup> See Coalition Letter at 5.

<sup>220</sup> *Id.* at 5-6. The map attached as Appendix 2, prepared by NRDC, shows counties with high chlorpyrifos use and outlines in black those that have a higher than 50% population of color. While definitive conclusions cannot be reached from this map, it is clear that EPA should have evaluated environmental justice impacts and disproportionality.

<sup>221</sup> Additionally, EPA’s treatment of pesticide drift is riddled with flaws of its own, including a failure to consider direct spray drift onto people. See *infra/supra* at Section IV.B.

<sup>222</sup> NAS, *Pesticides in the Diets of Infants and Children* 3-7 (1993).

heightened vulnerability because they eat and drink more than adults in proportion to their body weight, consume large quantities of certain fruits and vegetables, and engage in behaviors that expose them to pesticides such as playing on floors or lawns or putting objects in their mouths.<sup>223</sup>

One of the many routes through which children are exposed to pesticides is through pesticide drift—the airborne movement of pesticides off the target application site. The NAS observed that “[e]xposure to pesticide residues from ambient air sources is generally higher in areas close to agricultural lands and in communities surrounding pesticide manufacturing factories.” NAS Report at 309. To guard against harms associated with pesticide exposures, NAS recommended “exposure from all sources—not just ingestion—must be considered when estimating total [pesticide] exposure and risk to children.” *Id.* at 307.

On October 13, 2009, a group of health, environmental, and farmworker advocates jointly petitioned EPA to address the problem of pesticide drift, in particular to protect children from pesticide drift exposures. The Kids’ Petition called on EPA to correct its earlier failure to address exposure to pesticides drift in its pesticide re-registration decisions, and requested that as EPA undertakes the process to correct that legal error, EPA impose interim spray buffer zones around homes, schools, playgrounds, and any other areas where children play or congregate in order to protect children from health risks associated with drift.<sup>224</sup>

The record for the Kids’ Petition is replete with evidence of poisoning incidents, air monitoring reports, and statements of members of the Kids’ Coalition, all of which repeatedly show pesticide drift poses an ongoing risk to people, particularly children. The California Department of Pesticide Regulation (“CDPR”) documented 3,997 reported pesticide drift incidents in California between 1992 and 2007. Cal. Dep’t of Pesticide Regulation, California Pesticide Illness Query. In 2006, the Washington State Pesticide Incident Reporting and Tracking Review Panel found that “[e]xposure to pesticide drift is an important cause of documented pesticide-related illness in Washington.”<sup>225</sup> Monitoring and modeling studies confirm pesticide drift poses significant health risks to children who live near fields.

Pesticide drift disproportionately affects rural children, and EPA was obligated to include that in its analysis and to evaluate appropriate mitigating measures. In light of the evidence of exposure and poisoning incidents, the discussions of chlorpyrifos drift and volatilizations do not fulfill that obligation as EPA has failed to identify the disproportionate impacts chlorpyrifos has on minority and low-income populations.

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<sup>223</sup> *Id.* See also EPA, Pesticides and Food: Why Children May be Especially Sensitive to Pesticides (Mar. 2008). EPA-funded research confirmed and strengthened the NAS findings. See Centers for Children’s Environmental Health & Disease Prevention Research, Exposures & Health of Farm Worker Children in California; EPA, Children’s Exposure to Pesticides and Related Health Outcomes (June 21, 2007).

<sup>224</sup> *Pesticides in the Air—Kids at Risk: Petition to EPA to Protect Children from Pesticide Drift* (Oct. 13, 2009) (the “Kids’ Petition”). After the Kids’ Coalition brought a mandamus action to compel a response, *Pesticide Action Network of North America v. U.S. Environmental Protection Agency*, Case No. 13-72616, EPA responded on March 31, 2014, acknowledging—as the Kids’ Petition sought—EPA’s legal obligation to protect children from pesticide drift. However, EPA denied any interim safeguards against harmful exposures to children during the time EPA engages in a lengthy registration review of pesticides over the next eight or more years.

<sup>225</sup> Washington Department of Health, *Pesticide Incident Reporting and Tracking Review Panel, Annual Report: 2005*, at 81 (May 2007); see also Barbara Morrissey, *Washington State Dep’t of Health, Spray Drift and Human Health Incidents* (Ex. 40).

### 3. EPA Failed to Analyze and Mitigate Environmental Justice Impacts to Farmworkers.

Likewise, the RHHRA does not discuss the environmental justice impacts of chlorpyrifos on farmworkers. Farmworker families tend to be poor—on average, a farmworker family earns an annual income ranging from \$17,500- \$19,999.<sup>226</sup> In the top five agricultural counties in Texas (the state with the most acres of agriculture), between 10 to 30 percent of children live below the poverty line.<sup>227</sup> Likewise, in California (the top agricultural state by revenue), between 24 to 32 percent of children under the age of 17 live in poverty in the top three agricultural counties (compared with the state average poverty rate of 12.4%).<sup>228</sup> A 2014 study showed that farmworkers had higher residue concentrations of chlorpyrifos dust.<sup>229</sup>

The vast majority of U.S. farmworkers are of Latin American origin—approximately 76 percent of U.S. farmworkers are of Latin American ancestry.<sup>230</sup> A majority of these farmworkers have children, *id.*, and these children live and go to school near the agricultural sites where their parents work. For example, in California over 73 percent of children attending schools within 1.5 miles of sites where at least 10,000 pounds of pesticides were applied in 1998 were non-white.<sup>231</sup> Similarly, in 2008 approximately 53 percent of students in Washington State’s top five agricultural counties were non-white (the statewide average was 31 percent).<sup>232</sup>

Farmworkers’ persistent exposure to harmful pesticides has resulted in an average of 57.6 out of every 100,000 agricultural workers reporting acute pesticide poisoning, illness or injury each year.<sup>233</sup> These numbers exclude the many workers who suffer chronic health problems as a result of pesticide exposures, and do not factor in the known under-reporting of pesticide poisonings and illnesses. Agricultural workers are in great need of effective workplace protections because they represent some of the most economically and educationally disadvantaged people in the United States.<sup>234</sup>

When recently working on a revised Worker Protection Standard, EPA portrayed the plight of these workers:

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<sup>226</sup> U.S. Department of Labor, *National Agricultural Workers Survey* (2011-2012) (“NAWS”), available at <http://www.doleta.gov/agworker/naws.cfm>. Additionally attached is a fact sheet on the NAWS data prepared by Farmworker Justice (Ex. 41).

<sup>227</sup> U.S. Department of Agriculture, 2007 County-Level Poverty Rates for TX (Dec. 2008).

<sup>228</sup> Alice Larson, *Migrant and Seasonal Farmworker Enumeration Profiles Study: California* (Sept. 2000).

<sup>229</sup> Aug. 26, 2014 Coalition letter at 5 (citing Beti Thompson et al., *Variability in the Take-Home Pathway: Farmworkers and Non-Farmworkers and Their Children*, 24 *J. Exposure Sci. and Env'tl. Epidemiology* 522 (2014) (Ex. 30)).

<sup>230</sup> NAWS (FY 2011-12).

<sup>231</sup> Environmental Working Group, *Every Breath You Take: Airborne Pesticides in the San Joaquin Valley* (Jan. 2001).

<sup>232</sup> School Data Direct, *District-by-District Query*, available at <http://www.schooldatadirect.org/> (select “District” in the brown search box at the top of the screen, enter the district “Name” and “State” in the respective boxes. then click on the hyperlink for the district) (last viewed September 24, 2009).

<sup>233</sup> Geoffrey M. Calvert et al., *Acute Pesticide Poisoning Among Agricultural Workers in the United States, 1998 – 2005*, 51 *AM. J. INDUS. MED.* 883, 890 (2008).

<sup>234</sup> Daniel Carroll et al., *Changing Characteristics of U.S. Farm Workers: 21 Years of Findings from the National Agricultural Workers Survey* (May 12, 2011), available at <https://migrationfiles.ucdavis.edu/uploads/cf/files/2011-may/carroll-changing-characteristics.pdf> (Ex. 42).

According to information published by the Department of Labor's (DOL) NAWS in 2001-2002, 75% of agricultural workers in the United States were born in Mexico and 2% in Central America (Ref. 3 p. 3). A majority (81%) of this group speaks Spanish as a native language, but a growing percentage speaks languages such as Creole, Mixteco, and indigenous languages (Ref. 3 p. 17). Approximately 44% could not speak English at all, and 53% could not read any English (Ref. 3 p. 21).

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Approximately 43% of the survey respondents were classified as migrant, having traveled at least 75 miles in the previous year to find a job in agriculture (Ref. 3 p. 7). Over 20% of respondents lived in housing provided by their employer and 58% rented housing from someone other than their employer (Ref. 3 p. 43). In general, agricultural workers surveyed by NAWS do not use health care facilities. Estimates of agricultural workers lacking health insurance range from 77% to 85% and estimates from the late 1990s indicate only 20% of those surveyed had visited a health care facility in the preceding 2 years (Ref. 5 pp. 12-13). U.S. Department of Agriculture (USDA) research, based on NAWS data, also reports that workers have difficulty entering the health care system to receive treatment. Cost was a significant barrier for two-thirds of farmworkers, while about a third listed language barriers as an impediment to receiving care. The problem is more severe among undocumented workers because they fear seeking treatment will lead to deportation or other adverse legal action (Ref. 6).<sup>235</sup>

Yet EPA's cryptic treatment of environmental justice in the RHHRA did not address the disproportionate burdens on farmworkers and their families from chlorpyrifos exposures. EPA mechanically identifies various types of exposures and hazards throughout the RHHRA. Thus it finds that chlorpyrifos exposures cause brain damage to children exposed *in utero*, but it never assesses the race, ethnicity, or income of the most impacted populations, even though the cohort studies documenting such damage involved poor and multi-ethnic participants. Similarly, in assessing drinking water impacts, EPA believes the exceedances of its drinking water levels of concern will be in small watersheds where a large percentage of the crops are treated. It is highly likely that these impacted areas will be where farmworkers and their families live. Yet EPA failed to explore whether the drinking water contamination will disproportionately be in communities of color and low-income communities. And EPA identifies risks of concern to workers, but it fails to reveal that most of the workers are low-income and Latino and subjected to other burdens from environmental degradation and poor health care. Nor does EPA compare the risks faced by farmworkers compared to industrial workers who are protected under the Occupational Safety and Health Act. While EPA purports to afford analogous protection to workers under FIFRA, it has failed to do so with respect to chlorpyrifos, allowing numerous risks of concern to continue under the 2001 IRED. It must address these disproportionate burdens and be candid about the level of protection it will afford farmworkers as it proceeds to determine what regulatory actions need to be taken to reduce the numerous risks of concern. By failing thus far to engage in such analyses, EPA is in violation of its obligations under the environmental justice executive order.

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<sup>235</sup> 79 Fed. Reg. 15,444, 15,452.

## CONCLUSION

The RHHRA lays the groundwork for EPA to correct the shortcomings in its 2001 re-registration of chlorpyrifos. It has finally found that chlorpyrifos causes prenatal toxicity and neurodevelopmental damage to children, and it is acknowledging its obligation to protect children and bystanders from chlorpyrifos exposures, including through no-spray buffers around schools, homes, playfields and other places people congregate.

While EPA retains a 10X FQPA safety factor because of the large gaps in data related to brain damage to children caused by prenatal exposures, it continues to use 10% cholinesterase inhibition to set its regulatory limit. It does this even though neurodevelopmental damage resulted from doses that did not produce anywhere near 10% cholinesterase inhibition. The result is that EPA would allow pregnant women to be exposed to chlorpyrifos at levels that could cause IQ losses, reduced working memory, attention disorders, and other diminished motor and mental functioning in their children.

To compound its error, EPA relies on Dow's model designed to pinpoint exposures that will produce 10% cholinesterase to eliminate or reduce the traditional safety factors used in risk assessments, a model based on unethical human dosing studies. As a result, it will allow exposures that are an order of magnitude higher for pregnant women and higher still for children, precisely when its findings of brain damage impacts from *in utero* exposures should lead it in the other direction, *i.e.*, to be more protective which would require a reduction or more likely a complete elimination of exposures to chlorpyrifos.

In addressing exposures to children and other bystanders, EPA continues to look at the wrong endpoint – 10% cholinesterase inhibition – and it ignores volatilization, drift and most inhalation exposures and settles for no-spray buffers that are far too small to prevent drift upon children on school grounds or in their yards.

For workers, EPA identifies many dozens of activities that will expose workers to risks of concern. EPA must prevent such exposures. These scenarios are the tip of the iceberg. EPA has ignored direct drift onto workers and has made assumptions that lead it to under-estimate exposures and risks.

In assessing drinking water risks, EPA first found that all infants would be at risk from drinking water contamination. It then conducted additional modeling, which led it to believe the risks would be highest in small watersheds where a large percentage of the crops are treated with chlorpyrifos. EPA must immediately cancel uses that would produce such contamination, particularly since they are likely to be in communities already over-burdened by pesticide and other pollution risks.

The risks that EPA has found to workers, drinking water, and bystanders, as well as from food, are all based on the 10% cholinesterase inhibition endpoint and the shrunken safety factors. Using the appropriate endpoint (neurodevelopmental effects) and the appropriate safety factors will show that all exposures are unsafe under the various scenarios used by EPA in its risk assessments. EPA has a legal obligation to protect people, and especially children, from all aggregate exposures, and the RHHRA fails to provide an objective and fair assessment for it to do so. When EPA does the math right by accounting for the most sensitive endpoint and

appropriate safety factors, the numbers will inevitably show that all uses of chlorpyrifos must be cancelled immediately.

Sincerely,

A handwritten signature in black ink that reads "Patti Goldman". The signature is written in a cursive, flowing style.

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# **APPENDIX 1**

CHLORPYRIFOS-ONLY INCIDENTS FROM CALIFORNIA PESTICIDE ILLNESS  
SURVEILLANCE PROGRAM

The following table contains data from the California Department of Pesticide Regulation's 2015 Pesticide Illness Surveillance Program, <http://www.cdpr.ca.gov/docs/whs/pisp.htm>. For some incidents, there is more than one entry based on the number of people affected. Some, but not all, entries identify the distance from the pesticide application site where the incident occurred. Ten incidents where the distance was 80 feet to 1/2 mile are color-coded in green in column G. For several incidents, the description describes drift from an "adjacent field," "nearby" or "across the road," which suggests that the incident occurred farther from where the pesticide was applied than the no-spray buffers put in place in 2012. Twenty-five such incidents are color-coded in yellow.

Year	Case No.	Pesticide	Application Site	County	Medical Description	Narrative Description	Relationship
2009	91	Adjuvant, Chlorpyrifos	Alfalfa	FRESNO	NAUSEA, VOMITING, SWEATING, AGITATION, DIZZINESS, HEADACHE, VISUAL DEFICIT, LOSS OF CONSCIOUSNESS. THE HEADACHE PERSISTED FOR MORE THAN A WEEK.	ON A STILL NIGHT, AN AERIAL APPLICATOR BEGAN SPRAYING ALFALFA AT LEAST HALF A MILE FROM TOMATO HARVESTERS, UNAWARE OF TOMATO TRAILERS PARKED NEAR THE ALFALFA FIELD. THIS TRACTOR DRIVER FELT SPRAY MIST WHEN HE CAME TO GET A TRAILER. SEE 2009-242.	Probable
2009	242	Adjuvant, Chlorpyrifos	Alfalfa	FRESNO	HEADACHE, SWOLLEN LIPS AND TONGUE. SYMPTOMS SUBSIDED WITHIN HOURS.	SEE 2009-91. A FIELD FINDER CAME TO ASK THE HARVESTERS TO MOVE THE TRAILERS, WHICH SPARKED A CONFRONTATION. THE TOMATOES HARVESTED THAT NIGHT WERE DUMPED, ALTHOUGH TESTS DETECTED NO CONTAMINATION. THIS HARVESTER DRIVER SMELLED AN ODOR BUT FELT NO SPRAY.	Possible
1992	1055	Chlorpyrifos	Ornamental Plan	SAN MATEO	HEADACHE, DIZZINESS, NAUSEA, WATERY EYES.	SPRAY CREW INSTRUCTED PRUNER TO MOVE TO OTHER SIDE OF NURSERY. A GUST OF WIND BLEW THE SPRAY TOWARDS HIM. HE FELT NO DRIFT BUT SMELLED PESTICIDE ODOR. HE WENT HOME SICK, BUT RECOVERED BY THE NEXT DAY WHEN HE WAS SENT FOR A CHECK-UP.	Possible
1992	1103	Chlorpyrifos	Walnuts	YUBA	PAIN AND CLOUDY VISION IN LEFT EYE.	APPLICATOR WAS SPRAYING WALNUTS WITH CHLORPYRIFOS AND GOT SOME FOREIGN MATTER IN HIS LEFT EYE CAUSING IRRITATION AND PAIN. NO DETERMINATION IF SUBSTANCE WAS CHLORPYRIFOS.	Possible
1992	1867	Chlorpyrifos	Lemons	VENTURA	COUGHING, SHORTNESS OF BREATH, NAUSEA, VOMITING AND TIGHTNESS IN CHEST.	FIELD INSPECTOR WAS GOING TO CHECK ON BERRIES WHEN RIGHT AFTER SHE GOT OFF HER TRUCK, SHE SMELLED MATERIAL AND SAW A "PESTICIDE FOG" ON THE GROUND DUE TO INVERSION. SHE DEVELOPED SYMPTOMS AND SAW A DOCTOR	Possible
1993	981	Chlorpyrifos	Citrus	KERN	RASH ON ARMS.	WHILE IRRIGATING CITRUS GROVES, WORKER WAS DRIVING A THREE-WHEELER DOWN A DIRT ROAD AND WAS DRIFTED ON BY A NEIGHBORING APPLICATOR. CONFIRMING DETAILS ARE DIFFICULT TO FIND.	Possible
1993	1144	Chlorpyrifos	Unknown	YUBA	NAUSEA AND DIZZINESS.	A MECHANIC FIXED A SPRAY TANK PREVIOUSLY USED FOR CHLORPYRIFOS APPLICATION. HE FILLED THE TANK WITH WATER TO CHECK FOR LEAKS AND PUMP FUNCTION AND WAS EXPOSED WHEN THE WIND BLEW THE MATERIAL TOWARDS HIM. HE RINSED HIMSELF OFF AFTER THE	Possible

1993	1277	Chlorpyrifos	Apples	MADERA	ITCHING AND BURNING SENSATION TO SKIN ON FACE AND EYES.	WORKER MIXED, LOADED AND APPLIED CHLORPYRIFOS TO APPLES. HE SAID HE WAS NOT SURE WHEN HE MAY HAVE BEEN EXPOSED. HIS SUPERVISOR STATED THAT THE WORKER HAD TOLD HIM THAT MIST HAD BLOWN INTO HIS FACE WHILE LOADING AND THAT HE HAD IMMEDIATELY WASHED UP.	Possible
1993	1852	Chlorpyrifos	Lemons	VENTURA	HEADACHES, WEAKNESS, LEG CRAMPS, SMALL PUPILS.	NURSE TANK DRIVER DROVE THROUGH SPRAY MIST WHILE TAKING A LOAD OF WATER TO THE MIX/LOAD SITE. HE DID NOT TIGHTEN THE BOTTOM STRIP OF HIS RESPIRATOR. HE ALSO FEELS HE WAS EXPOSED WHILE STEAM CLEANING APPLICATION EQUIPMENT AND WELDING	Possible
1994	957	Chlorpyrifos	Oranges	FRESNO	BURNING NASAL PASSAGES, HEADACHE.	A POLICE OFFICER RESPONDED TO A CHEMICAL ODOR COMPLAINT. UPON INVESTIGATION, HE FOUND A STRONG ODOR COMING FROM A CHLORPYRIFOS APPLICATION IN A NEARBY ORANGE GROVE. HE FELT OVERPOWERED BY THE ODOR AND SUFFERED MINOR ILL EFFECTS.	Probable
1994	972	Chlorpyrifos	Oranges	TULARE	NAUSEA, VOMITING, SWEATING, LIGHTHEADEDNESS.	WORKER WAS EXPOSED TO DRIFT FROM HIS OWN APPLICATION WHEN HE GOT OUT OF HIS NELSON ENCLOSED CAB TO PLACE A ROW MARKER. HE MIXED AND LOADED ONE MORE LOAD, AND SPRAYED THREE ROWS BEFORE FEELING SICK. HE DID NOT WASH UP UNTIL AN HOUR AFTER THE EXPOSURE.	Possible
1994	1328	Chlorpyrifos	Cotton	KINGS	WATERY EYES, NAUSEA, RUNNY NOSE, HEADACHE, METALLIC TASTE IN MOUTH.	43-KIN-94. APPLICATION DRIFTED OFF TARGET SITE. 47 EMPLOYEES AND 1 CHILD COMPLAINED OF ODOR AND WERE TAKEN TO A HOSPITAL. SWAB SAMPLES AND LEAF SAMPLES WERE POSITIVE FOR RESIDUE. POSSIBLE AIR INVERSION AT TIME OF APPLICATION. SEE ALSO 94-1329-1375.	Probable
1994	1329	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA.	43-KIN-94. SEE 94-1328.	Probable
1994	1330	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA, CHEST PAIN, SHORTNESS OF BREATH.	43-KIN-94. SEE 94-1328.	Probable
1994	1331	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA, DIZZINESS, SHORTNESS OF BREATH, WATERY EYES, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1332	Chlorpyrifos	Cotton	KINGS	TEMPORARY BRADYCARDIA.	43-KIN-94. SEE 94-1328.	Possible
1994	1334	Chlorpyrifos	Cotton	KINGS	HEADACHE.	43-KIN-94. SEE 94-1328.	Probable

1994	1335	Chlorpyrifos	Cotton	KINGS	HEADACHE, UPSET STOMACH, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1336	Chlorpyrifos	Cotton	KINGS	ASTHMA, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1337	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA.	43-KIN-94. SEE 94-1328.	Probable
1994	1339	Chlorpyrifos	Cotton	KINGS	HEADACHE, MUCOSAL BURNING, SHORTNESS OF BREATH, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1340	Chlorpyrifos	Cotton	KINGS	SHORTNESS OF BREATH.	43-KIN-94. SEE 94-1328.	Probable
1994	1341	Chlorpyrifos	Cotton	KINGS	BURNING EYES, DIFFICULTY BREATHING, LIGHTHEADEDNESS, HEAVINESS IN CHEST.	43-KIN-94. SEE 94-1328.	Probable
1994	1342	Chlorpyrifos	Cotton	KINGS	HEADACHE.	43-KIN-94. SEE 94-1328.	Probable
1994	1345	Chlorpyrifos	Cotton	KINGS	HEADACHE, DIZZINESS.	43-KIN-94. SEE 94-1328.	Probable
1994	1347	Chlorpyrifos	Cotton	KINGS	CHEST TIGHTNESS, SLIGHT TINGLING IN FOREARM, ANXIOUSNESS.	43-KIN-94. SEE 94-1328.	Probable
1994	1348	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA, DIZZINESS, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1349	Chlorpyrifos	Cotton	KINGS	HEADACHE, WATERY EYES, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1350	Chlorpyrifos	Cotton	KINGS	HEADACHE.	43-KIN-94. SEE 94-1328.	Probable
1994	1351	Chlorpyrifos	Cotton	KINGS	HEADACHES, CHEST TIGHTNESS.	43-KIN-94. SEE 94-1328.	Probable
1994	1352	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA, ITCHY ARMS, AND FACE, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1353	Chlorpyrifos	Cotton	KINGS	HEADACHE, UPSET STOMACH.	43-KIN-94. SEE 94-1328.	Probable
1994	1354	Chlorpyrifos	Cotton	KINGS	HEADACHE, BURNING EYES, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1355	Chlorpyrifos	Cotton	KINGS	HEADACHE, DIZZINESS, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1356	Chlorpyrifos	Cotton	KINGS	HEADACHE, ITCHY SKIN, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1357	Chlorpyrifos	Cotton	KINGS	DIZZINESS, HEADACHE.	43-KIN-94. SEE 94-1328.	Probable

1994	1358	Chlorpyrifos	Cotton	KINGS	TEMPORARY ALTERED STATE OF CONSCIOUSNESS, METALLIC TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Possible
1994	1359	Chlorpyrifos	Cotton	KINGS	EYE IRRITATION, BREATHING DIFFICULTY, HEADACHE.	43-KIN-94. SEE 94-1328.	Probable
1994	1360	Chlorpyrifos	Cotton	KINGS	HEADACHE, SCRATCHY THROAT, SNEEZING.	43-KIN-94. SEE 94-1328.	Probable
1994	1361	Chlorpyrifos	Cotton	KINGS	LIGHTHEADEDNESS, SORE THROAT, WATERY EYES.	43-KIN-94. SEE 94-1328.	Probable
1994	1362	Chlorpyrifos	Cotton	KINGS	NAUSEA, NERVOUSNESS.	43-KIN-94. SEE 94-1328.	Probable
1994	1363	Chlorpyrifos	Cotton	KINGS	SEVERE HEADACHE, BAD TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1365	Chlorpyrifos	Cotton	KINGS	HEADACHE, SHORTNESS OF BREATH, EYE	43-KIN-94. SEE 94-1328.	Probable
1994	1366	Chlorpyrifos	Cotton	KINGS	CHEST PAIN, SHORTNESS OF BREATH, HEADACHE.	43-KIN-94. SEE 94-1328.	Probable
1994	1367	Chlorpyrifos	Cotton	KINGS	HEADACHE. DRY THROAT.	43-KIN-94. SEE 94-1328.	Probable
1994	1368	Chlorpyrifos	Cotton	KINGS	UPSET STOMACH.	43-KIN-94. SEE 94-1328.	Possible
1994	1369	Chlorpyrifos	Cotton	KINGS	WATERY EYES, DRY THROAT, HEADACHE.	43-KIN-94. SEE 94-1328.	Probable
1994	1370	Chlorpyrifos	Cotton	KINGS	HEADACHE.	43-KIN-94. SEE 94-1328.	Probable
1994	1371	Chlorpyrifos	Cotton	KINGS	HEADACHE, DIZZINESS, BREATHING DIFFICULTY.	43-KIN-94. SEE 94-1328.	Probable
1994	1372	Chlorpyrifos	Cotton	KINGS	LIGHTHEADEDNESS.	43-KIN-94. SEE 94-1328.	Probable
1994	1373	Chlorpyrifos	Cotton	KINGS	HEADACHE, NAUSEA, BAD TASTE IN MOUTH.	43-KIN-94. SEE 94-1328.	Probable
1994	1374	Chlorpyrifos	Cotton	KINGS	HEADACHE, SHORTNESS OF BREATH, SCRATCHY THROAT.	43-KIN-94. SEE 94-1328.	Probable
1995	1151	Chlorpyrifos	Broccoli	SANTA BARB	HEADACHE, DIZZINESS, NAUSEA, DRY FEELING IN THE MOUTH.	10 YDS. 15-SB-95. FOUR WORKERS IN A GREENHOUSE AND TWO IN AN ADJACENT FIELD BECAME ILL AFTER DETECTING AN ODOR FROM A CHLORPYRIFOS APPLICATION ON A NEARBY FIELD. THE WORKERS REPORTED TO THEIR SUPERVISOR AND WERE SENT TO A DOCTOR. SEE 95-1152 TO 1156.	Possible
1995	1152	Chlorpyrifos	Broccoli	SANTA BARB	NAUSEA, HEADACHE, STOMACH PAIN, VOMITING, BREATHING DIFFICULTY.	10 YDS. 15-SB-95. SEE 95-1151. FOLIAGE AND SWAB SAMPLES INDICATED MINIMAL DRIFT. APPLICATION WAS TERMINATED PROMPTLY WHEN WORKERS APPEARED ON THE NEIGHBORING PROPERTY.	Possible
1995	1153	Chlorpyrifos	Broccoli	SANTA BARB	NAUSEA, HEADACHE.	10 YDS. 15-SB-95. SEE 95-1151.	Possible
1995	1154	Chlorpyrifos	Broccoli	SANTA BARB	DIZZINESS, NAUSEA, VOMITING.	10 YDS. 15-SB-95. SEE 95-1151. WORKER IS 8 MONTHS PREGNANT.	Possible

1995	1155	Chlorpyrifos	Broccoli	SANTA BARB	HEADACHE, DIZZINESS, NAUSEA, VOMITING, BURNING IN THE CHEST AND EYES.	10 YDS. 15-SB-95. SEE 95-1151.	Possible
1995	1156	Chlorpyrifos	Broccoli	SANTA BARB	HEADACHE, DIZZINESS.	10 YDS. 15-SB-95. SEE 95-1151.	Possible
1995	1281	Chlorpyrifos	Sugarbeets	MADERA	DIZZINESS, NAUSEA, VOMITING.	A RESIDENT NOTICED AN ODOR COMING FROM HIS COOLER VENTS. HE WALKED OUTSIDE AND SMELLED A STRONG ODOR FROM AN APPLICATION 2 MILES AWAY. HE BECAME ILL & SOUGHT MEDICAL ATTENTION. RESIDUE SAMPLES WERE NEGATIVE, BUT A NEIGHBOR ALSO	Probable
1995	1944	Chlorpyrifos	Alfalfa	SOLANO	HEADACHE, NAUSEA, CHEST PRESSURE, PAIN IN THE EYES.	A FLAGGER WARNED A JOGGER OF A PESTICIDE APPLICATION. THE JOGGER THOUGHT SHE COULD COMPLETE HER RUN BEFORE THE APPLICATION RESUMED. SHE DID NOT SMELL ANY ODOR, BUT CLAIMED EXPOSURE TO THE SPRAY MIST. SHE DEVELOPED SYMPTOMS AFTER TAKING A	Possible
1996	773	Chlorpyrifos	Oranges	KERN	NAUSEA, CHEST PAIN.	440 YDS. WHILE HE REPAIRED A TRACTOR IN AN INDOOR SHOP, A MECHANIC SMELLED AN ODOR FROM AN APPLICATION ABOUT A QUARTER MILE AWAY. HE DOES WORK ON PESTICIDE APPLICATION EQUIPMENT AND HAD NEVER BEFORE REACTED TO A PESTICIDE ODOR.	Possible
1996	1293	Chlorpyrifos	Oranges	KERN	FATIGUE, WEIGHT LOSS, MILD WEAKNESS.	AN APPLICATOR DEVELOPED SYMPTOMS AFTER SEVERAL WEEKS OF 14-HOUR WORK DAYS. HE SOUGHT MEDICAL ATTENTION 8 DAYS LATER. THE DOCTOR DIAGNOSED CHOLINESTERASE DEPRESSION, BUT DID NOT REPORT THE RESULTS.	Possible
1996	1563	Chlorpyrifos	Alfalfa	KERN	DRY MOUTH, SCRATCHY THROAT.	RESIDENTS WERE BARBECUING IN THE BACK YARD OF A HOME WHEN AN AERIAL APPLICATOR DRIFTED CHLORPYRIFOS ONTO THEIR PROPERTY. FOLIAGE SAMPLES HAD PESTICIDE LEVELS COMPARABLE TO THE TREATED CROP. SHE CONSULTED A DOCTOR BY PHONE WHO CALLED POISON CONTROL.	Probable
1997	231	Chlorpyrifos	Alfalfa	STANISLAUS	ITCHING, BURNING SENSATION TO THE FACE, LIPS AND NOSE, PAIN IN THE LUNGS, RUNNY NOSE, BURNING EYES	A BUS DRIVER DETECTED STRONG ODOR WHILE DRIVING HER ROUTE. WHEN SHE BEGAN DEVELOPING SYMPTOMS, SHE STOPPED AND WASHED HER FACE AND EYES. SHE COMPLETED HER MORNING BUS ROUTE BEFORE SEEKING MEDICAL ATTENTION.	Probable

1997	611	Chlorpyrifos	Oranges	FRESNO	SNEEZING, THROAT IRRITATION, WHEEZING, COUGHING, WATERY EYES, DIZZINESS.	A FIELD CHECKER DEVELOPED SYMPTOMS WHEN SHE SAW MIST FROM A SPRAY RIG PASSING NEARBY. THE RIG HAD ITS FAN ON AND NOZZLES SHUT. SHE SOUGHT MEDICAL ATTENTION 5 HOURS LATER. SHE STATED SHE IS ALLERGIC TO PESTICIDES AND MANY OTHER CHEMICALS.	Possible
1997	944	Chlorpyrifos	Oranges	FRESNO	DIZZINESS.	A MIXER/LOADER AND APPLICATOR TRAINEE APPARENTLY LOOKED INTO THE OPEN SPRAY TANK AND EXPOSED HIMSELF TO THE FUMES. HE DEVELOPED SYMPTOMS AND SOUGHT MEDICAL ATTENTION THAT EVENING. HE HAS LEFT THE AREA AND COULD NOT BE INTERVIEWED.	Possible
1997	1128	Chlorpyrifos	Lettuce	FRESNO	VOMITING, HEADACHE, NAUSEA, ABDOMINAL PAIN, 102 DEGREE FEVER, CHILLS.	AT THE COMPLETION OF A CHLORPYRIFOS APPLICATION, A FLAGGER GOT OUT OF HIS TRUCK. THE WIND BLEW SOME SPRAY MIST ON THE BACK OF HIS COVERALLS. HE DEVELOPED SYMPTOMS 5 HOURS LATER AND SOUGHT MEDICAL ATTENTION THE NEXT	Possible
1997	1769	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, BURNING THROAT, UPSET STOMACH, DIZZINESS.	32-RIV-97. AN AERIAL APPLICATOR DRIFTED CHLORPYRIFOS ONTO 36 MELON HARVESTERS IN AN ADJACENT FIELD. THE PILOT ESTIMATED DISTANCE AS 1/4 MILE, THE HARVESTERS AS 40 METERS. DRIFT CONFIRMED BY POSITIVE RESIDUE IN FOLIAGE SAMPLES. SEE 97-1770 THROUGH	Probable
1997	1770	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, HEADACHE.	32-RIV-97. SEE 97-1769. THE INVESTIGATORS COULD NOT PRACTICALLY PERFORM IN PERSON INTERVIEWS WITH THE AFFECTED WORKERS. THE SUPERVISORS HELPED 34 OF THE 36 WORKERS COMPLETE AND RETURN INTERVIEW FORMS.	Probable
1997	1771	Chlorpyrifos	Alfalfa	RIVERSIDE	DIZZINESS, NAUSEA.	32-RIV-97. SEE 97-1769. 34 OF 36 WORKERS EVENTUALLY WERE PERSUADED TO STRIP IN THE FIELD FOR SHOWERS. THEY WERE GIVEN SHEETS TO WEAR DURING TRANSPORT TO A LOCAL HOSPITAL. THEIR CLOTHING WAS COMINGLED, SO IT WAS NOT ANALYZED FOR RESIDUE.	Probable
1997	1772	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, NAUSEA, EYE PAIN.	32-RIV-97. SEE 97-1769.	Probable
1997	1773	Chlorpyrifos	Alfalfa	RIVERSIDE	IRRITATED & BURNING EYES.	32-RIV-97. SEE 97-1769.	Probable

1997	1774	Chlorpyrifos	Alfalfa	RIVERSIDE	VOMITING, NAUSEA, HEADACHE, SCRATCHY THROAT, SOUR TASTE IN THE MOUTH.	32-RIV-97. SEE 97-1769.	Probable
1997	1775	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, DIZZINESS, THROAT IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1776	Chlorpyrifos	Alfalfa	RIVERSIDE	DIZZINESS, HEADACHE.	32-RIV-97. SEE 97-1769.	Probable
1997	1777	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, SORE THROAT, EYE IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1778	Chlorpyrifos	Alfalfa	RIVERSIDE	DIFFICULTY BREATHING, PAIN ON INHALATION.	32-RIV-97. SEE 97-1769. THIS WORKER DID NOT RETURN THE INTERVIEW QUESTIONNAIRE. HIS MEDICAL RECORDS INDICATE HE COMPLAINED OF DIFFICULTY BREATHING AND PLEURODYNIA. MANY OF THE WORKERS REPORTED SYMPTOMS NOT RECORDED IN THEIR MEDICAL RECORDS.	Possible
1997	1779	Chlorpyrifos	Alfalfa	RIVERSIDE	EYE IRRITATION, ALLERGIC SYMPTOMS.	32-RIV-97. SEE 97-1769.	Probable
1997	1780	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, HEADACHE.	32-RIV-97. SEE 97-1769.	Probable
1997	1782	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, ALLERGY SYMPTOMS.	32-RIV-97. SEE 97-1769.	Probable
1997	1783	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, DIZZINESS, HEADACHE, BREATHING DIFFICULTY, ALLERGY SYMPTOMS.	32-RIV-97. SEE 97-1769.	Probable
1997	1784	Chlorpyrifos	Alfalfa	RIVERSIDE	THROAT IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1785	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, HEADACHE, BITTER TASTE IN THE MOUTH.	32-RIV-97. SEE 97-1769.	Probable
1997	1787	Chlorpyrifos	Alfalfa	RIVERSIDE	STOMACH ACHE, THROAT IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1788	Chlorpyrifos	Alfalfa	RIVERSIDE	DIZZINESS.	32-RIV-97. SEE 97-1769.	Probable
1997	1791	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, HEADACHE, AND STOMACHACHE.	32-RIV-97. SEE 97-1769.	Probable
1997	1792	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, BREATHING DIFFICULTY, EYE IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1793	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, TRANSIENT DIZZINESS.	32-RIV-97. SEE 97-1769.	Probable
1997	1794	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, DIZZINESS, "FEELING LIKE A ROCK IS IN THE BRAIN".	32-RIV-97. SEE 97-1769.	Probable
1997	1795	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, DIZZINESS, HEADACHE, BREATHING DIFFICULTY, BURNING EYES, ALLERGY	32-RIV-97. SEE 97-1769.	Probable

1997	1796	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE, VOMITING, SLIGHT NAUSEA.	32-RIV-97. SEE 97-1769.	Probable
1997	1797	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, DIZZINESS, EYE IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1798	Chlorpyrifos	Alfalfa	RIVERSIDE	NAUSEA, DIZZINESS, STOMACHACHE, EYE IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1799	Chlorpyrifos	Alfalfa	RIVERSIDE	SORE THROAT.	32-RIV-97. SEE 97-1769.	Probable
1997	1801	Chlorpyrifos	Alfalfa	RIVERSIDE	HEADACHE.	32-RIV-97. SEE 97-1769.	Probable
1997	1802	Chlorpyrifos	Alfalfa	RIVERSIDE	BREATHING DIFFICULTY, EYE IRRITATION.	32-RIV-97. SEE 97-1769.	Probable
1997	1803	Chlorpyrifos	Alfalfa	RIVERSIDE	DIZZINESS.	32-RIV-97. SEE 97-1769.	Probable
1997	1804	Chlorpyrifos	Alfalfa	RIVERSIDE	UPSET STOMACH.	32-RIV-97. SEE 97-1769.	Probable
1998	224	Chlorpyrifos	Alfalfa	KERN	LIGHTHEADEDNESS, HEADACHE, DIZZINESS, NAUSEA.	220 YARDS. A WORKER DROVE A TRACTOR BESIDE A FIELD BEING TREATED. HE SMELLED THE PESTICIDE AND BECAME NAUSEATED. HE INFORMED THE PCA OBSERVING THE APPLICATION. THE PCA TOOK HIM TO DECONTAMINATION FACILITIES AND THEN TO THE DOCTOR.	Probable
1999	269	Chlorpyrifos	Alfalfa	TULARE	CONFUSION, BODY ACHES, FATIGUE, NERVOUSNESS.	AS A WORKER BROKE THE SOIL SURFACE AT THE BOTTOM OF TREE HOLES IN A WALNUT ORCHARD, HE SMELLED A PESTICIDE ODOR COMING FROM AN AERIAL APPLICATION TO AN ADJACENT ALFALFA FIELD. THE TREE HOLES WERE FUMIGATED WITH METHYL BROMIDE 3 MONTHS EARLIER.	Possible
1999	1008	Chlorpyrifos	Lemons	VENTURA	NAUSEA, VOMITING, BODY ACHES, JOINT ACHES, LETHARGY.	20 YARDS. A FIELDWORKER OBSERVED A HELICOPTER APPLICATION AND NOTICED AN ODOR, BUT HAD NO PESTICIDE CONTACT. HE DEVELOPED SYMPTOMS 4 HOURS LATER, AND SOUGHT MEDICAL TREATMENT THE NEXT DAY DUE TO WORSENING SYMPTOMS.	Possible
1999	1139	Chlorpyrifos	Oranges	KERN	HEADACHE, NAUSEA, BURNING EYES, TINGLING ALL OVER, FATIGUE.	440 YARDS. A LANDFILL GATE ATTENDANT SMELLED A PESTICIDE ODOR AND DEVELOPED SYMPTOMS. THE APPLICATORS HALTED PROMPTLY UPON REQUEST, AND HER SYMPTOMS RESOLVED QUICKLY. SHE SAW A DOCTOR TWO DAYS LATER AS A	Possible
2000	527	Chlorpyrifos	Lemons	VENTURA	EYE IRRITATION, DIARRHEA, RASH ON THE NECK.	30 YARDS. WHILE WORKING AT A HOUSE, A CARPENTER REPORTED CONTACT WITH A MIST CLOUD FROM AN ADJACENT LEMON GROVE. HE FAILED TO RETURN THE INVESTIGATOR'S TELEPHONE CALLS. THE INVESTIGATOR OBTAINED INFORMATION FROM THE HOMEOWNER AND THE	Possible

2000	542	Chlorpyrifos	Walnuts	STANISLAUS	HEADACHE, NAUSEA, VOMITING.	A CREW FOREMAN DEVELOPED SYMPTOMS AFTER SMELLING THE ODOR FROM A CHLORPYRIFOS APPLICATION TO A NEARBY WALNUT GROVE. HE DENIED ANY CONTACT WITH THE SPRAY. HE RECEIVED APPLICATOR TRAINING AS HIS EMPLOYER IS ALSO A PEST CONTROL OPERATOR.	Possible
2001	75	Chlorpyrifos	Equipment	FRESNO	LIGHTHEADEDNESS, POSTERIOR HEADACHE, GENERALIZED TINGLING SENSATION.	A FARM WORKER THOUGHT HE APPLIED OIL ON TRACTORS TO AVOID CORROSION BY LIME-SULFUR SPRAY. THE OIL CONTAINER PROVED TO HOLD CHLORPYRIFOS CONCENTRATE, BUT NO ONE KNOWS WHO Poured IT IN THERE. THE OIL WAS ALSO A PESTICIDE AND NOT REGISTERED FOR THE	Possible
2001	575	Chlorpyrifos	Alfalfa	STANISLAUS	DIZZINESS, NAUSEA, VOMITING. MANAGEMENT NOTES ALSO MENTION "PERSISTENT ATAXIA", EVALUATED AS UNRELATED TO	100 YARDS. WHILE DRIVING ON A FARM ROAD BETWEEN FIELDS WITH HIS PICK-UP TRUCK WINDOWS OPEN, AN IRRIGATOR SAW AN AERIAL PESTICIDE APPLICATION AND SMELLED AN ODOR, BUT FELT NO DRIFT. HE BECAME ILL ABOUT 30 MINUTES LATER, AFTER EATING LUNCH.	Possible
2002	149	Chlorpyrifos	Pears	LAKE	BURNING EYES, THROAT AND SKIN, LIGHTHEADEDNESS, MOUTH NUMBNESS, RASH ON THE NECK.	11-LAK-02. WHILE SPRAYING A PEAR ORCHARD, AN APPLICATOR DRIFTED THE SPRAY ONTO A TRIBAL SCHOOL FACILITY. THREE SCHOOL EMPLOYEES FELT THE SPRAY ON THEIR SKIN AND DEVELOPED SYMPTOMS. TWO OF THE THREE SOUGHT MEDICAL ATTENTION. SEE 2002-150 & 151.	Probable
2002	150	Chlorpyrifos	Pears	LAKE	NAUSEA, UPSET STOMACH, WATERING AND BURNING EYES, RED SKIN ON THE FACE,	11-LAK-02. SEE 2002-149. LABORATORY ANALYSIS FOUND CHLORPYRIFOS IN CLOTHING, SWAB AND FOLIAGE SAMPLES TAKEN THE DAY OF THE INCIDENT.	Probable
2002	151	Chlorpyrifos	Pears	LAKE	DIZZINESS, BURNING SKIN.	11-LAK-02. SEE 2002-149. THE GROWER AND TRIBAL REPRESENTATIVES MET AND WORKED OUT PLANS TO KEEP EACH OTHER INFORMED OF THEIR ACTIVITIES. THE GROWER AGREED TO APPLY EARLY MORNING OR ON WEEKENDS WHEN THE TRIBAL FACILITY SHOULD BE UNOCCUPIED.	Probable
2003	908	Chlorpyrifos	Walnuts	TEHAMA	CHEST TIGHTNESS, EYE IRRITATION, DIFFICULTY BREATHING, RUNNY NOSE, SNEEZING.	AS 2 WORKERS WEEDED ALONG A RIVER BANK, THEY NOTICED A HELICOPTER APPLYING PESTICIDES TO A WALNUT ORCHARD ABOUT 300 YARDS AWAY. THEY DEVELOPED SYMPTOMS, INFORMED A MANAGER & SOUGHT FOR MEDICAL ATTENTION. NEITHER REMEMBERS SMELLING AN ODOR. SEE 2003-909.	Possible

2003	909	Chlorpyrifos	Walnuts	TEHAMA	COUGHING, SNEEZING, WATERY EYES.	SEE 2003-908.	Possible
2005	480	Chlorpyrifos	Alfalfa	KERN	RED AND BURNING EYES, BURNING NOSE, HEADACHE, COUGHING, DIFFICULTY BREATHING.	AS A WORKER DROVE A TRACTOR ADJACENT TO AN ALFALFA FIELD, HE FELT SPRAY FROM AN AERIAL APPLICATOR HIT HIS FACE. HIS EYES & NOSE IMMEDIATELY BEGAN BURNING. HIS FOREMAN HAD HIM WASH OFF & SHOWER, THEN RETURN TO WORK. OTHER SYMPTOMS DEVELOPED THAT	Probable
2006	134	Chlorpyrifos	Walnuts	TULARE	INITIALLY: TINGLING IN THE ARMS, BREATHING DIFFICULTY, NERVOUSNESS, SKIN FELT "LIKE RUBBER"; LATER: WEAKNESS, DIZZINESS, BURNING EYES, EYE TWITCH, DROOLING, HEART FLUTTERS, HEADACHE; FOLLOWING DAY: NAUSEA AND DIARRHEA.	AS HE BEGAN WORK AT HIS EQUIPMENT YARD, A MAN HEARD SPRAY EQUIPMENT IN THE ADJACENT WALNUT GROVE, SMELLED A STRONG ODOR, SAW A MIST CLOUD FLOAT TOWARDS HIM, AND NOTICED INSECTS DYING. PHOTOS, WITNESSES AND SAMPLES DOCUMENTED DRIFT. SEE 2006-135 & 136.	Probable
2006	135	Chlorpyrifos	Walnuts	TULARE	SORE THROAT (DESCRIBED AS A MILD, DULL ACHE).	SEE 2006-134. ALERTED BY THEIR SON (2006-134), THIS MAN AND HIS WIFE (2006-136) LEFT THEIR HOME TO CHECK THEIR LIVESTOCK. THE WIFE FELT SPRAY MIST. THE INVESTIGATORS IDENTIFIED THREE OTHER RESIDENTS WHO SAW THE DRIFT, BUT DEVELOPED NO SYMPTOMS.	Probable
2006	136	Chlorpyrifos	Walnuts	TULARE	EXACERBATION OF COUGHING, WHEEZING, SINUS IRRITATION, EYE IRRITATION, SORE THROAT, HEADACHE, SENSATION OF SWOLLEN LIPS AND TONGUE, BAD TASTE IN THE MOUTH.	SEE 2006-134. THE GRADIENT OF SAMPLE RESULTS SUGGESTED THAT THE PESTICIDE HAD COME FROM THE DIRECTION OF THE WALNUTS. THE APPLICATORS CONCEDED THEY HAD NOT MEASURED WIND SPEED AS REQUIRED. THE INVESTIGATORS FOUND NO OTHER SOURCE FOR THE DETECTED RESIDUE.	Probable

2007	571	Chlorpyrifos	Oranges	TULARE	SALIVATION, HEADACHE, SEVERE ABDOMINAL PAIN, PULSING STOMACH SENSATION, STOMACH ACHE, NAUSEA, VOMITING, DIARRHEA, FATIGUE, LOSS OF APPETITE, BLOATING, DIFFICULTY BREATHING. ON EXAM, THE DOCTOR NOTED SCALING LESIONS & OPEN SORES OVER HIS	A MIXER/LOADER/APPLICATOR FELT ILL AFTER HANDLING CHLORPYRIFOS FOR 9 DAYS. HE USED A TRACTOR WITH A MISSING WINDOW AND ALTHOUGH HE HAD ALL LABEL-REQUIRED PPE AVAILABLE, HE DID NOT WEAR THEM ALL. HIS DOCTOR IDENTIFIED CONDITIONS BEYOND PESTICIDE EXPOSURE.	Definite
2007	652	Chlorpyrifos	Almonds	FRESNO	RAPID HEART BEAT, LUNG TIGHTNESS, SHORTNESS OF BREATH, NAUSEA, DIZZINESS.	WORKERS FOR TWO GROWERS APPLIED THE SAME PESTICIDES TO ADJACENT ALMOND ORCHARDS ON THE SAME NIGHT. ONE APPLICATOR ACCIDENTALLY DRIFTED THE PESTICIDES ONTO THE MIXER/LOADER FOR THE OTHER APPLICATION. HE DEVELOPED SYMPTOMS AND SOUGHT MEDICAL CARE.	Probable
2007	666	Chlorpyrifos	Almonds	STANISLAUS	COUGHING, CHEST TIGHTNESS, HEADACHES, ITCHING, SORE THROAT, METALLIC TASTE IN THE MOUTH.	33 YARDS. TWO GIRLS AWOKE TO THE NOISE AND ODOR OF AN AIRBLAST SPRAYER APPLYING CHLORPYRIFOS IN AN ADJACENT ALMOND ORCHARD. THEY EXPERIENCED SYMPTOMS & THEIR MOTHER TOOK THEM FOR CARE. GRADIENT SAMPLES DEMONSTRATED DRIFT. SEE 2007-667.	Probable
2007	667	Chlorpyrifos	Almonds	STANISLAUS	COUGHING, CHEST TIGHTNESS, HEADACHE, ITCHING, STOMACH RASH, SORE THROAT, METALLIC TASTE IN THE MOUTH. UPON EXAM, THE DOCTOR NOTED A SLIGHT DECREASE IN VESICULAR BREATH SOUNDS WHICH INCREASED POST NEBULIZATION. SHE HAS A HISTORY OF REACTIVE	SEE 2007-666.	Probable
2007	689	Chlorpyrifos	Almonds	TULARE	THROAT AND MOUTH NUMBNESS, CHEST PAINS ESPECIALLY UPON INSPIRATION.	41-TUL-07. 17 YARDS. THREE WORKERS APPLIED CHLORPYRIFOS TO AN ALMOND ORCHARD WITH AIRBLAST SPRAYERS. THEY DRIFTED THE CHLORPYRIFOS INTO A NEIGHBORING VINEYARD. SEE 2007-690 – 696, 704 – 708, 1389 – 1403.	Probable

2007	690	Chlorpyrifos	Almonds	TULARE	NAUSEA, VOMITING, HEADACHE, DIZZINESS, DIFFICULTY BREATHING, FINGER AND MOUTH NUMBNESS, HOT AND COLD HANDS, MILDLY STINGING & ITCHY EYES, ITCHY FACE AND NECK, SNEEZING. ON EXAM, THE DOCTOR NOTED EPIGASTRIC PAIN, CONGESTED EYES AND RASH ON THE NECK.	41-TUL-07. SEE 2007-689. THE VINEYARD CREWS CONSISTED OF 70 WORKERS, 28 OF WHOM WENT TO THE AREA'S EMERGENCY ROOM TO BE EXAMINED. OF THE 28 WORKERS, 26 WORKERS REPORTED SYMPTOMS.	Probable
2007	691	Chlorpyrifos	Almonds	TULARE	VOMITING, HEADACHE, DIZZINESS, NAUSEA, WEAKNESS, ITCHY FACE AND NECK, ITCHY EYES, NUMB ARMS. ON EXAM, THE DOCTOR NOTED CONGESTED EYES AND A MILDLY TENDER EPIGASTRIC AREA.	41-TUL-07. SEE 2007-689. THE VINEYARD OWNER AND 7 WORKERS REPORTED FEELING THE SPRAY MIST. THE INVESTIGATOR COLLECTED CLOTHING SAMPLES FROM THE VINEYARD OWNER AND 11 WORKERS. ELEVEN OF THE 12 CLOTHING SAMPLES CAME BACK POSITIVE FOR CHLORPYRIFOS.	Probable
2007	692	Chlorpyrifos	Almonds	TULARE	NAUSEA, HEADACHE, VOMITING, MILD UPSET STOMACH, GENERALIZED ITCHING. ON EXAM, THE DOCTOR NOTED A RASH ON THE BODY.	41-TUL-07. SEE 2007-689. A 4-POINT GRADIENT SAMPLE SHOWED A POSITIVE DRIFT PATTERN FROM THE ALMOND ORCHARD TO THE GRAPE VINEYARD.	Probable
2007	693	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, VOMITING, NUMB LIPS AND TONGUE, DIFFICULTY BREATHING, BURNING THROAT AND CHEST, GENERALIZED ITCHING, ITCHY EYES, WEAKNESS, TIREDNESS. ON EXAM, THE DOCTOR NOTED A RASH ON THE BODY AND A TENDER EPIGASTRIC	41-TUL-07. SEE 2007-689. THE AGRICULTURAL COMMISSIONER FINED THE ALMOND GROWER \$28,600 FOR VIOLATIONS MOSTLY RELATED TO CREATING AN ACTUAL HEALTH HAZARD FOR THE VINEYARD WORKERS.	Probable
2007	694	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, MILD UPSET STOMACH, CHEST PAIN, GENERALIZED ITCHING. ON EXAM, THE DOCTOR NOTED A TENDER	41-TUL-07. SEE 2007-689.	Probable

2007	695	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, GENERALIZED ITCHING, MILD SHORTNESS OF BREATH, CHEST TIGHTNESS. ON EXAM, THE DOCTOR NOTED A RASH ON THE ARMS AND	41-TUL-07. SEE 2007-689.	Probable
2007	696	Chlorpyrifos	Almonds	TULARE	HEADACHE, DIZZINESS, NAUSEA, ITCHING AND RASH ON THE NECK AND TRUNK, SORE THROAT, COUGH, CHEST TIGHTNESS, SENSATION OF SWOLLEN TONGUE,	41-TUL-07. SEE 2007-689. THIS WORKER STATED SHE FELT THE MIST HIT HER FACE.	Probable
2007	704	Chlorpyrifos	Almonds	TULARE	HEADACHE, ITCHING, MILD UPSET STOMACH, MOUTH NUMBNESS, SLEEPINESS.	41-TUL-07. SEE 2007-689. THIS WORKER DEVELOPED SYMPTOMS ABOUT 12 HOURS AFTER EXPOSURE. SHE SAW A DOCTOR IN THE MORNING. SHE WENT BACK TO THE HOSPITAL THAT EVENING, BUT LEFT BEFORE SEEING THE DOCTOR. SHE SAW A DIFFERENT DOCTOR 2 DAYS LATER.	Possible
2007	705	Chlorpyrifos	Almonds	TULARE	VOMITING, DIZZINESS, HEADACHE, NAUSEA, GENERALIZED ITCHING ON THE FACE AND BODY, TONGUE NUMBNESS, SHORTNESS OF BREATH, SORE THROAT, TIREDNESS. ON EXAM, THE DOCTOR NOTED A RASH ON THE BODY AND A TENDER EPIGASTRIC	41-TUL-07. SEE 2007-689.	Probable
2007	706	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, GENERALIZED ITCHING, TONGUE NUMBNESS, DIFFICULTY BREATHING, CHEST TIGHTNESS, TIREDNESS. ON EXAM, THE DOCTOR NOTED A RASH ON THE ARMS AND LEGS, AND A TENDER EPIGASTRIC AREA.	41-TUL-07. SEE 2007-689.	Probable

2007	707	Chlorpyrifos	Almonds	TULARE	HEADACHE, ITCHY EYES, NUMB & ITCHY HANDS, CHILLS, BLURRED VISION, DRY MOUTH, SLIGHT SHORTNESS OF BREATH. UPON EXAM, THE DOCTOR NOTED SLIGHT EPIGASTRIC TENDERNESS AND A RASH ON THE	41-TUL-07. SEE 2007-689. THIS WORKER SAW THE EMERGENCY ROOM DOCTOR IN THE MORNING (HOSPITAL COULD NOT LOCATE RECORDS). SHE RETURNED THAT EVENING, BUT LEFT BEFORE SEEING THE DOCTOR. SHE SAW ANOTHER DOCTOR 2 DAYS LATER.	Probable
2007	708	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, DIZZINESS, TONGUE AND MOUTH NUMBNESS, DIFFICULTY BREATHING, THROAT TIGHTNESS, STERNAL PAIN, GENERALIZED ITCHING.	41-TUL-07. SEE 2007-689.	Probable
2007	1389	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, STOMACH ACHE, DIZZINESS, WATERY MOUTH, VOMITING, GENERALIZED ITCHING, WEAKNESS, FATIGUE. UPON EXAMINATION, THE DOCTOR NOTED A TENDER EPIGASTRIC AREA, NECK AND TORSO RASH AND CONGESTED	41-TUL-07. SEE 2007-689.	Probable
2007	1390	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA, DIZZINESS.	41-TUL-07. SEE 2007-689. THIS WORKER WENT HOME, SHOWERED AND CHANGED CLOTHES. SHE DEVELOPED SYMPTOMS ABOUT 4 HOURS AFTER EXPOSURE.	Probable
2007	1391	Chlorpyrifos	Almonds	TULARE	NECK, BACK, CHEST & EPIGASTRIC PAIN, NAUSEA, MILD HEADACHE, WEAKNESS. ON EXAM, THE DOCTOR NOTED MILD ABDOMINAL	41-TUL-07. SEE 2007-689.	Probable
2007	1392	Chlorpyrifos	Almonds	TULARE	HEADACHE, THROAT IRRITATION, MOUTH AND TONGUE NUMBNESS, ITCHING, IRRITATION.	41-TUL-07. SEE 2007-689.	Probable
2007	1393	Chlorpyrifos	Almonds	TULARE	HEADACHE, NAUSEA.	41-TUL-07. SEE 2007-689.	Probable

2007	1394	Chlorpyrifos	Almonds	TULARE	HEADACHE, GENERALIZED ITCHING, IRRITATION, FATIGUE, WEAKNESS. UPON EXAM, THE DOCTOR NOTED A RASH ON THE UPPER AND LOWER QUADRANTS.	41-TUL-07. SEE 2007-689.	Probable
2007	1395	Chlorpyrifos	Almonds	TULARE	VOMITING, NAUSEA.	41-TUL-07. SEE 2007-689.	Probable
2007	1396	Chlorpyrifos	Almonds	TULARE	HEADACHE, DIZZINESS, VOMITING, ITCHING OF THE EXTREMITIES, NUMBNESS AROUND THE MOUTH, NAUSEA. UPON EXAM, THE DOCTOR NOTED A SLIGHT RASH ON THE ABDOMEN.	41-TUL-07. SEE 2007-689.	Probable
2007	1397	Chlorpyrifos	Almonds	TULARE	HEADACHE, DIZZINESS, BLURRED VISION, WATERY MOUTH, ABDOMINAL PAIN, GENERALIZED ITCHING, HEARTBURN. UPON EXAM, THE DOCTOR NOTED A TENDER	41-TUL-07. SEE 2007-689.	Probable
2007	1398	Chlorpyrifos	Almonds	TULARE	NAUSEA, HEADACHE, GENERALIZED ITCHING, NECK PAIN, WEAKNESS, VOMITING. ON EXAM, THE DOCTOR NOTED A SLIGHT BODY RASH.	41-TUL-07. SEE 2007-689.	Probable
2007	1399	Chlorpyrifos	Almonds	TULARE	HEADACHE, ITCHING ON THE ARMS AND NECK.	41-TUL-07. SEE 2007-689. THIS WORKER WENT TO THE HOSPITAL THE AFTERNOON OF THE INCIDENT. A NURSE ASSESSED HIM, BUT HE LEFT THE HOSPITAL BEFORE BEING SEEN BY THE DOCTOR. HIS SYMPTOMS BEGAN LATE IN THE MORNING OF THE NEXT DAY.	Possible
2007	1400	Chlorpyrifos	Almonds	TULARE	ITCHING ON THE ARM AND HANDS, RED RASH ON THE ARMS, NAUSEA.	41-TUL-07. SEE 2007-689. THIS WORKER WENT TO THE HOSPITAL THE AFTERNOON OF THE INCIDENT. A NURSE ASSESSED HIM, BUT HE LEFT THE HOSPITAL BEFORE BEING SEEN BY THE DOCTOR.	Probable

2007	1401	Chlorpyrifos	Almonds	TULARE	NAUSEA, HEADACHE, PUFFY NAPE, GENERALIZED ITCHING, ABDOMINAL PAIN, WEAKNESS. UPON EXAM, THE DOCTOR NOTED SLIGHT ABDOMINAL TENDERNESS AND A	41-TUL-07. SEE 2007-689.	Probable
2008	419	Chlorpyrifos	Walnuts	BUTTE	HEADACHE, DIZZINESS, DIFFICULTY BREATHING, NAUSEA, VOMITING; BY ARRIVAL AT A HOSPITAL HE COMPLAINED ONLY OF CHEST TIGHTNESS. HE FELT WELL AND RETURNED TO WORK TWO DAYS LATER, BUT HAD HEADACHES FOR	A FIELD WORKER APPLIED FERTILIZER WELL AHEAD OF A CHLORPYRIFOS APPLICATION. WHEN HE FINISHED, HE CLEARED ROOTS FROM AN ADJACENT FIELD. THERE, HE SMELLED THE SPRAY BUT DID NOT FEEL IT. WHEN HE VOMITED, HE STOPPED THE APPLICATOR AND WAS TAKEN FOR CARE.	Probable
2008	520	Chlorpyrifos	Walnuts	TEHAMA	WEAKNESS, SORE THROAT, HEADACHE, SHORTNESS OF BREATH. SYMPTOMS RESOLVED WITHIN 20 MINUTES OF BREATHING FRESH AIR.	UPON ARRIVAL TO THEIR WORK SITE, A PACKING CREW NOTICED A STRONG SMELL FROM AN APPLICATION TO AN ADJACENT ORCHARD. BY THE TIME THE COUNTY INSPECTOR ARRIVED, THE SMELL HAD DISSIPATED. MOST OF CREW DEVELOPED SYMPTOMS BUT ONLY THIS WORKER SOUGHT CARE.	Probable
2009	888	Chlorpyrifos	Oranges	KERN	COUGHING, DIZZINESS, NAUSEA. HE WAS STILL NAUSEATED AND HAD LITTLE APPETITE FOUR DAYS LATER.	31-KER-09. SIX LANDFILL WORKERS DEVELOPED SYMPTOMS DURING A PESTICIDE APPLICATION SOUTH OF THE LANDFILL. THEY WERE DECONTAMINATED BY EMERGENCY RESPONDERS. THIS WORKER WAS THE ONLY ONE WHO DID NOT SMELL AN ODOR. HE WAS TAKEN FOR CARE. SEE 2009 889-893.	Possible
2009	889	Chlorpyrifos	Oranges	KERN	COUGHING, LIGHTHEADEDNESS, VOMITING, HEADACHE, ABDOMINAL DISCOMFORT, DIZZINESS.	31-KER-09. SEE 2009-888. THE LANDFILL WAS SHUT DOWN FOR THE REST OF THE DAY. THIS WORKER WORKED IN A GATEHOUSE AND ONLY WENT OUTSIDE ONCE. SHE NOTICED 'A MIST BEING SPRAYED OVER THE TREES' BUT DID NOT FEEL PESTICIDES DRIFT ON HER. SHE WAS TAKEN FOR CARE.	Possible

2009	890	Chlorpyrifos	Oranges	KERN	<p>NAUSEA, DIZZINESS, LIGHTEADEDNESS AND DISORIENTATION, DIFFICULTY BREATHING, WEAKNESS, CHILLS, HEADACHE, 'SHAKINESS', 'A LITTLE INCOHERENT', BODY FELT LIKE IT WAS TINGLING. SHE STILL HAD SOME SYMPTOMS 2 DAYS LATER.</p>	<p>31-KER-09. SEE 2009-888. SHE WORKED IN A GATEHOUSE &amp; SAID SHE WENT OUTSIDE BRIEFLY. SHE SAW A 'PLUME OF MIST' OVER THE ORCHARD &amp; WHILE SHE DID NOT FEEL PESTICIDE DRIFT ON HER, SHE SAID SHE FELT 'A FILM' ON HER. SHE WASHED HER FACE BEFORE HAZMAT ARRIVED.</p>	Possible
2009	891	Chlorpyrifos	Oranges	KERN	<p>SORE AND IRRITATED THROAT, DIFFICULTY BREATHING, MILD NAUSEA, LIGHTEADEDNESS, DIZZINESS. HE SAID HE HAD A 'FILM INSIDE HIS MOUTH.' HE STILL HAD</p>	<p>31-KER-09. SEE 2009-888. HE WAS FILLING A WATER TRUCK AND TURNED AWAY WHEN HE SAW A 'CLOUD' FROM THE PESTICIDE APPLICATION. HE DID NOT FEEL PESTICIDE DRIFT ON HIM BUT DEVELOPED SYMPTOMS 15 MINUTES LATER. HE WAS TAKEN FOR CARE.</p>	Possible
2009	892	Chlorpyrifos	Oranges	KERN	<p>UPSET STOMACH, DIARRHEA, NAUSEA, COUGHING, DIZZINESS, SORE AND ITCHY THROAT WHICH LASTED TWO DAYS.</p>	<p>31-KER-09. SEE 2009-888. SHE WORKED IN A GUARDSHACK BUT WOULD OCCASIONALLY STEP OUTSIDE FOR BRIEF PERIODS. SHE SAID THAT SHE SAW A MIST OVER THE ORCHARD BUT DID NOT FEEL PESTICIDE DRIFT ON HER. SHE WENT HOME AFTER WORK, SHOWERED THEN SOUGHT CARE.</p>	Possible
2009	893	Chlorpyrifos	Oranges	KERN	<p>SORE AND IRRITATED THROAT, BURNING AND ITCHY EYES AND A HEADACHE THAT PERSISTED FOR 2 DAYS.</p>	<p>31-KER-09. SEE 2009-888. SHE WORKED IN A GUARDHOUSE BUT WOULD OCCASIONALLY WALK OUTSIDE TO CHECK TRUCKS. SHE SAID THAT SHE DID NOT FEEL ANY PESTICIDE DRIFT ON HER BUT THAT, OVER TIME, HER 'SKIN FELT STICKY'. SHE SOUGHT CARE.</p>	Possible
2009	1053	Chlorpyrifos	Grapes	FRESNO	<p>COUGHING, EYE AND THROAT IRRITATION. AFTER TWO DAYS, SHE COULD HARDLY TALK.</p>	<p>FROM A FRONT PORCH, FOUR RELATIVES SAW AN AIRBLAST APPLICATION TO A VINEYARD ACROSS THE STREET. THEY SAID THE APPLICATOR CONTINUED SPRAYING DURING TURNS, WHICH HE DENIED. ONE OF TWO SAMPLES SHOWED A SMALL AMOUNT OF PESTICIDE. SEE 2009-1054 - 1026.</p>	Probable

2009	1054	Chlorpyrifos	Grapes	FRESNO	SORE THROAT, DIFFICULTY BREATHING, SWELLING AROUND THE EYES. SYMPTOMS BEGAN ABOUT THREE TO FOUR HOURS AFTER EXPOSURE. SWELLING RESOLVED IN A FEW DAYS, BREATHING DIFFICULTY IN ABOUT A WEEK, AND SORE THROAT AFTER ABOUT TWO WEEKS.	SEE 2009-1053. INVESTIGATORS FOUND THAT FIELD POSTING SIGNS WERE INCOMPLETE AND SPACED TOO FAR APART, THAT THE APPLICATOR HAD NOT BEEN GIVEN CHEMICAL-RESISTANT BOOTS (REQUIRED BY THE LABEL), AND THE APPLICATION STILL HAD NOT BEEN REPORTED AFTER 7 MONTHS.	Probable
2009	1055	Chlorpyrifos	Grapes	FRESNO	SORE THROAT, LASTED FOUR OR FIVE DAYS.	SEE 2009-1053.	Probable
2009	1056	Chlorpyrifos	Grapes	FRESNO	SLIGHT SORE THROAT.	SEE 2009-1053.	Probable
2010	856	Chlorpyrifos	Citrus	KERN	FELT TIRED, "FELT HE COULD NOT BREATHE WITH RESPIRATOR ON." FELT NAUSEATED, DIZZY AND VOMITED AFTER HE REMOVED HIS RESPIRATOR AND SMELLED CHEMICAL ODOR. AFTER HOURS OF WAITING AT THE ER, HE FELT BETTER, SO HE LEFT BEFORE BEING SEEN.	AS AN APPLICATOR WALKED DOWN ROWS OF CITRUS TREES BROADCASTING PESTICIDES, HE BEGAN TO FEEL ILL. HE WAS TAKEN FOR CARE. THE EMPLOYER DID NOT DOCUMENT THAT THE WORKER RECEIVED MEDICAL EVALUATION OR A RESPIRATORY FIT TEST PRIOR TO USING A RESPIRATOR.	Probable
2011	126	Chlorpyrifos	Lemons	VENTURA	HEADACHE, AND EYE PAIN AND IRRITATION. AT THE TIME OF CONSULT THE NEXT DAY, SYMPTOMS HAVE RESOLVED AND THE DOCTOR GAVE A DIAGNOSIS OF "NO TOXIC EXPOSURE." ON EXAM, A DOCTOR NOTED SYMPTOMS RESOLVED	PLANT WORKERS NOTED IRRITATING ODORS AROUND 8:00 AM, & FIRE DEPT. WAS CALLED. 3 WORKERS WENT HOME WITH HEADACHES, OTHERS WITH BURNING EYES & NAUSEA. AN APPLICATION WAS TAKING PLACE ACROSS THE STREET, & THE AIR WAS NOT MONITORED FOR ODORS. SEE 2011-600.	Probable

2011	428	Chlorpyrifos	Oranges	KERN	COUGHING, IRRITATED THROAT, MUCOSAL BURNING, AND DIFFICULTY BREATHING. 2 WEEKS LATER: DIFFICULTY BREATHING, IRRITATION, AND CHEST PAINS. HE SAID THAT THE PULMONARY SPECIALIST WHO SAW HIM 2 WEEKS FROM THE EXPOSURE DIAGNOSED HIM WITH	A SECURITY GUARD DEVELOPED SYMPTOMS WHILE PATROLLING THE RANCH PREMISES DURING A PESTICIDE APPLICATION HALF A MILE AWAY. HE SOUGHT CARE BUT HIS SYMPTOMS PERSISTED TWO WEEKS AFTER EXPOSURE. THE FARM SUPERVISOR HAD INFORMED HIM OF THE APPLICATION.	Possible
2011	600	Chlorpyrifos	Lemons	VENTURA	A FACILITY MANAGER TOLD AN INVESTIGATOR THAT WORKERS EXPERIENCED HEADACHES, BURNING EYES AND NAUSEA. THIS WORKER'S MEDICAL RECORD'S COMPLAINTS SECTION WAS NOT MADE	REF. 2011-126. APPLICATORS, & SUPERVISORS SAID THERE WAS NO WIND, & TEMPERATURE WAS AT 81 DEGREES ON A BLOCK THEY HAVE BEEN WORKING ON FOR 3 DAYS. THE COMPANY WAS CITED FOR FAILURE TO CHECK WEATHER CONDITIONS, & PREVENTION OF CONTAMINATION TO PROPERTY.	Probable
2012	784	Chlorpyrifos	Wheat	KERN	STOMACH ACHE, CHEMICAL TASTE IN MOUTH, BURNING SENSATION IN EYES AND THROAT, AND FELT "ITCHY". NO COUGH. EXAM AND LABS WERE WITHIN NORMAL LIMITS. DECONTAMINATED AT CLINIC.	16-KER-12. AS A BUS PICKED UP CHILDREN ON ITS MORNING ROUTE, A CROPDUSTER FLEW ACROSS THE HIGHWAY AND SPRAYED THE BUS WITH CHLORPYRIFOS. SEE 2012-785 TO 819.	Possible
2012	785	Chlorpyrifos	Wheat	KERN	NOTICED A "BAD" SMELL, FELT "ITCHY", STOMACHACHE, BURNING EYES, HER EARS "HURT", HEADACHE. DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. SAID SHE WAS STILL ITCHY AFTER SHOWERING.	16-KER-12. REF 2012-784. AFTER THE PLANE FLEW OVER, THE DRIVER CALLED THE DISTRICT WHEN STUDENTS COMPLAINED OF ODOR. HE RETURNED TO THE SCHOOL AS DIRECTED, AND PICKED UP 10 MORE STUDENTS ALONG THE WAY.	Possible

2012	786	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. HE WAS SITTING ON THE DRIVER'S SIDE NEAR AN OPEN WINDOW. HE SAID HE SMELLED "YUCKY AIR", HAD A HEADACHE, FELT SLEEPY, AND BECAME ITCHY. LATER THAT MORNING HE SAID HE FELT BETTER BUT HIS HEAD STILL HURT.	16-KER-12. REF 2012-784. OF THE 29 STUDENTS ABOARD WHEN THE DRIFT OCCURRED, 14 REPORTED SYMPTOMS AND 18 SMELLED AN ODOR. THE BUS DRIVER ALSO NOTED AN ODOR. NONE OF THE 10 CHILDREN PICKED UP AFTER THE EXPOSURE REPORTED ODOR OR SYMPTOMS.	Possible
2012	787	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. NOTED A "WEIRD" SMELL, ITCHY SKIN AND BURNING EYES.	16-KER-12. REF 2012-784. EMERGENCY RESPONDERS ARRIVED ON THE SCENE AND DECONTAMINATED 27 PEOPLE INCLUDING THE DRIVER AT THE SCHOOL'S SWIMMING POOL SHOWERS. CLOTHING WAS BAGGED SEPARATELY AND GIVEN TO COUNTY STAFF FOR LAB ANALYSIS.	Possible
2012	788	Chlorpyrifos	Wheat	KERN	PICKED UP BY MOM AND SHOWERED AT HOME. REPORTED A STINGING SENSATION AND A "WEIRD" SMELL. SAID HE HAD ITCHY STOMACH SKIN, EYES, AND LEGS. HE REMAINED ITCHY ON INTERVIEW LATER THAT MORNING.	16-KER-12. REF 2012-784. FIVE OF THE CHILDREN DECONTAMINATED BY FIRE DEPARTMENT STAFF WERE STUDENTS PICKED UP AFTER THE DRIFT OCCURRED, AND ONE WAS A CHILD IN THE SCHOOL OFFICE (2012-818) WHO REPORTED SYMPTOMS BUT WAS NEVER ON THE BUS.	Possible
2012	789	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. SHE REPORTED NOT FEELING ANY SPRAY, BUT FELT ITCHY AND LIGHTHEADED WHEN SHE GOT OFF THE BUS. SHE SAID HER SYMPTOMS REMAINED ON INTERVIEW LATER THAT	16-KER-12. REF 2012-784. EIGHT STUDENTS WERE PICKED UP BY THEIR PARENTS AND WERE DECONTAMINATED AT HOME. ONE CHILD (2012-784) WAS TAKEN BY HER PARENTS TO HER DOCTOR AND SHOWERED THERE. SHE WAS THE ONLY PERSON KNOWN TO SEEK MEDICAL ATTENTION.	Possible

2012	790	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. REPORTED AN ODOR AND STOMACHACHE. SHE SAID SHE FELT BETTER ON INTERVIEW LATER THAT MORNING.	16-KER-12. REF 2012-784. OF NINE SAMPLES COLLECTED FOR LABORATORY ANALYSIS, ALL WERE POSITIVE FOR PESTICIDE RESIDUE. THE CONTROL SAMPLE DID DETECT A SLIGHT AMOUNT OF RESIDUE, BUT A SUBSEQUENT LABORATORY CONTROL WAS NEGATIVE FOR CHLORPYRIFOS.	Possible
2012	791	Chlorpyrifos	Wheat	KERN	DECONTAMINATED AT HOME. HE REPORTED A "FUNNY" SMELL, LIKE "GROUND". HE FELT ITCHY, BUT SAID HE FELT BETTER ON INTERVIEW LATER THAT MORNING.	16-KER-12. REF 2012-784. SAMPLES WERE TAKEN FROM INSIDE AND OUTSIDE THE BUS AND WELL AS THREE STUDENT T-SHIRTS. ONE T-SHIRT SAMPLE (2012-786) CONTAINED 90 TIMES THE MINIMUM DETECTABLE LIMIT OF RESIDUE.	Possible
2012	792	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. REPORTED A STRONG SMELL AND HEADACHE.	16-KER-12. REF 2012-784. THE AVERAGE AGE OF THE CHILDREN WAS 9.8 YEARS. THEIR INTERVIEW ACCOUNTS OF THE EXPOSURE VARY WIDELY, BUT MANY REPORTED HEARING OR SEEING A PLANE. THE MOST COMMON SYMPTOM WAS ITCHY SKIN, FOLLOWED BY STOMACHACHE AND	Possible
2012	793	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. HE REPORTED SMELLING AN ODOR AND FEELING ITCHY "IN ABOUT 5 MINUTES". HE REPORTED FEELING BETTER AFTER	16-KER-12. REF 2012-784. THE CROPDUSTER PILOT WAS ISSUED A VIOLATION FOR FAILURE TO USE THE PRODUCT PURSUANT TO THE LABEL INSTRUCTIONS BY NOT ENSURING PERSONS WOULD NOT BE CONTACTED DIRECTLY OR THROUGH DRIFT.	Possible
2012	794	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. SHE REPORTED AN ODOR THAT "SMELLED FUNNY LIKE SWEET BUT STUNK". SHE SAID SHE FELT ITCHY AND REMAINED ITCHY AFTER SHOWERING.	16-KER-12. REF 2012-784.	Possible
2012	795	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. REPORTED A STRONG SMELL AND ITCHY ARMS.	16-KER-12. REF 2012-784.	Possible

2012	796	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL AND SHOWERED AT HOME. HE WAS INTERVIEWED TWICE. IN ONE HE REPORTED AN ODOR AND ITCHING; IN THE NEXT INTERVIEW HE SAID NOTHING GOT ON	16-KER-12. REF 2012-784.	Possible
2012	797	Chlorpyrifos	Wheat	KERN	DECONTAMINATED BY EMERGENCY RESPONDERS AT SCHOOL. SHE SAID SOME MIST GOT IN THE WINDOW, BUT NONE GOT ON HER. SHE REPORTED FEELING A LITTLE ITCHY, AND SAID SHE WAS STILL ITCHY ON INTERVIEW LATER THAT	16-KER-12. REF 2012-784.	Possible
1999	97	Chlorpyrifos, Copper Hydroxide, Petroleum Oil	Almonds	FRESNO	HEADACHE, VOMITING, DIARRHEA, NAUSEA, BURNING AND WATERING EYES, THROAT IRRITATION.	WHILE REMOVING DEAD ARMS FROM GRAPEVINES, WORKERS NOTICED AN APPLICATION IN THE ADJACENT ALMOND ORCHARD AND DETECTED A STRONG ODOR. SEVERAL WORKERS DEVELOPED SYMPTOMS, BUT ONLY 4 SOUGHT MEDICAL ATTENTION THE NEXT MORNING. SEE 1999-98 TO 100.	Probable
1999	98	Chlorpyrifos, Copper Hydroxide, Petroleum Oil	Almonds	FRESNO	HEADACHE, DIARRHEA, NAUSEA, SLIGHTLY IRRITATED EYES, RUNNY NOSE.	SEE 1999-97.	Probable
1999	99	Chlorpyrifos, Copper Hydroxide, Petroleum Oil	Almonds	FRESNO	HEADACHE, VOMITING, DIARRHEA, NAUSEA, WATERING EYES.	SEE 1999-97.	Probable
1999	100	Chlorpyrifos, Copper Hydroxide, Petroleum Oil	Almonds	FRESNO	NAUSEA, VOMITING, DIARRHEA, HEADACHE, BURNING THROAT.	SEE 1999-97.	Probable
1998	1440	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	BURNING SENSATION OF THE EYES, NOSE AND THROAT, ITCHING FACIAL SKIN, DIARRHEA.	32-MER-98. WHILE REMOVING TREES ADJACENT TO A NECTARINE ORCHARD, 12 WORKERS DEVELOPED SYMPTOMS AFTER BEING DRIFTED ON BY AN APPLICATION TO THE ORCHARD. THEY WERE ALL DECONTAMINATED AND EXAMINED AT A HOSPITAL EMERGENCY ROOM. SEE 1998-1441 TO 1451.	Probable

1998	1441	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	IRRITATED AND WATERING EYES, RUNNY NOSE, CHEST PAIN, BREATHING DIFFICULTY, HEADACHE, LOSS OF APPETITE, VOMITING, DIARRHEA, ITCHING SKIN.	32-MER-98. SEE 1998-1440.	Probable
1998	1442	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	ITCHING AND BURNING SKIN ON FACE AND NECK, LIGHTHEADEDNESS, WEAKNESS, HEADACHE, LOSS OF APPETITE, SCRATCHY AND SWOLLEN THROAT.	32-MER-98. SEE 1998-1440.	Probable
1998	1443	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	EYE IRRITATION, HEADACHE, LIGHTHEADEDNESS, NAUSEA, SHORTNESS OF BREATH.	32-MER-98. SEE 1998-1440.	Probable
1998	1444	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	ITCHING ON THE FACE AND HANDS.	32-MER-98. SEE 1998-1440. THIS WORKER FELT MIST ON HIS HANDS AND FACE.	Probable
1998	1445	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	HEADACHE, SORE THROAT, BURNING AND ITCHING SKIN ON THE FACE AND HANDS, LOOSE	32-MER-98. SEE 1998-1440.	Probable
1998	1446	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	BURNING EYES AND NOSE, ITCHING OF THE FACIAL SKIN, IRRITATED THROAT, HEADACHE, DECREASED APPETITE.	32-MER-98. SEE 1998-1440.	Probable
1998	1447	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	BURNING EYES, SORE THROAT, RUNNY NOSE, ITCHING AND RASH ON THE NECK AND WRIST.	32-MER-98. SEE 1998-1440.	Probable
1998	1448	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	NAUSEA, MILD SORE THROAT, BURNING EYES, ITCHY RASH WITH SKIN DISCOLORATION ON THE FACE, NECK AND CHEST.	32-MER-98. SEE 1998-1440.	Probable
1998	1449	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	BURNING EYES, SORE THROAT, ITCHING.	32-MER-98. SEE 1998-1440.	Probable
1998	1450	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	ITCHING, SKIN ON THE FACE AND NECK, NAUSEA, HEADACHE, DIZZINESS, COUGHING, DECREASED APPETITE.	32-MER-98. SEE 1998-1440.	Probable

1998	1451	Chlorpyrifos, Copper Sulfate, Mineral Oil	Nectarines	MERCED	BURNING AND ITCHING EYES, NOSE, THROAT AND SKIN ON THE ARMS AND FACE, STOMACH ACHE, HEADACHE, NAUSEA.	32-MER-98. SEE 1998-1440.	Probable
2005	311	Chlorpyrifos, Copper Sulfate, Mineral Oil	Peaches	SUTTER	VOMITING, HEADACHE, STINGING FACIAL SKIN, CHEST TIGHTNESS, SHORTNESS OF BREATH, COUGHING, SCRATCHY THROAT, CHILLS, DISTURBED SLEEP, GENERAL FATIGUE, LOOSE STOOLS, LOSS OF APPETITE. THE DOCTOR NOTED POSSIBLE REACTIVE AIRWAYS DISEASE SECONDARY TO EXPOSURE.	WHILE MAKING AN APPLICATION, AN AG PCO DRIFTED PESTICIDES ONTO A WOMAN DRIVING HOME. SHE FELT SPRAY MIST AGAIN WHEN SHE EXITED HER TRUCK AND DEVELOPED SYMPTOMS. A CLOTHING SAMPLE AND SWAB SAMPLES TAKEN FROM HER TRUCK AND FENCE CONFIRMED DRIFT.	Probable
1997	166	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Apples	MADERA	EYE IRRITATION, HEADACHE.	SPRAY FROM AN AIR BLAST SPRAYER DRIFTED ACROSS A ROAD AND INTO A DRIVER'S OPEN WINDOW. A SWEATSHIRT AND VEHICLE SAMPLES CONFIRMED PESTICIDE CONTAMINATION. THE DRIVER SAID THE RIG DID NOT SHUT OFF WHILE TURNING AT THE END OF THE ROW.	Definite
2005	510	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, NAUSEA, DIZZINESS, SORE THROAT.	29-TUL-05. 12 BOX-MAKING PLANT EMPLOYEES DEVELOPED SYMPTOMS AFTER SMELLING AN ODOR. THERE WAS AN ONGOING CHLORPYRIFOS APPLICATION 1/4 MILE AWAY. THEY WERE TOLD TO SEEK MEDICAL ATTENTION, BUT EVERYONE REFUSED. SEE 2005-511 TO 521.	Probable
2005	511	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, CHEST PAIN, SORE THROAT.	29-TUL-05. SEE 2005-510. AFTER INVESTIGATION, CAC ISSUED A NOTICE OF VIOLATION FOR NOT HAVING WIND-SPEED MEASURING EQUIPMENT DURING THEM APPLICATION. HOWEVER, AFTER THE WEATHER STATION REPORTED NO SUBSTANTIAL WIND PRESENT, CAC DECIDED	Probable
2005	512	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, NAUSEA, NASAL IRRITATION.	29-TUL-05. SEE 2005-510.	Probable
2005	513	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, BODY ACHES, DIARRHEA, FEVER.	29-TUL-05. SEE 2005-510. THIS WORKER'S SYMPTOMS MAY HAVE INITIALLY BEEN DUE TO SMELLING THE ODOR, BUT SUBSEQUENT SYMPTOMS MAY HAVE BEEN DUE TO ANOTHER CAUSE.	Probable

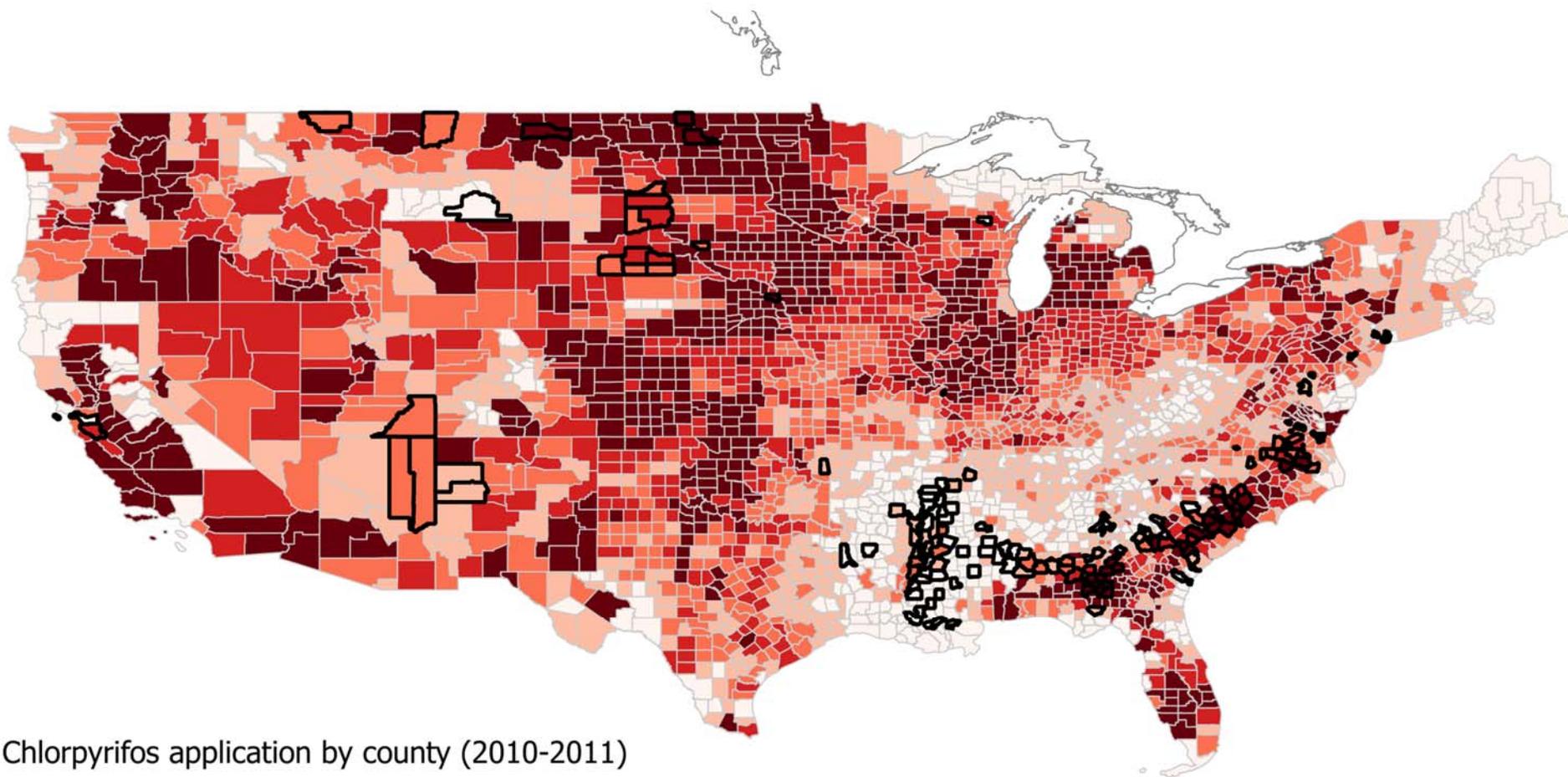
2005	514	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, STUFFY NOSE.	29-TUL-05. SEE 2005-510.	Probable
2005	515	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, STUFFY NOSE.	29-TUL-05. SEE 2005-510.	Probable
2005	516	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, NAUSEA, DIZZINESS.	29-TUL-05. SEE 2005-510.	Probable
2005	517	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	NAUSEA, DIZZINESS.	29-TUL-05. SEE 2005-510.	Probable
2005	518	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, NAUSEA.	29-TUL-05. SEE 2005-510.	Probable
2005	519	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, NAUSEA, SHORTNESS OF BREATH, VOMITING.	29-TUL-05. SEE 2005-510.	Probable
2005	520	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	VOMITING.	29-TUL-05. SEE 2005-510.	Probable
2005	521	Chlorpyrifos, Copper Sulfate, Petroleum Oil	Oranges	TULARE	HEADACHE, VOMITING (3 TIMES).	29-TUL-05. SEE 2005-510.	Probable
1992	144	Chlorpyrifos, Cuprous Oxide, Petroleum Oil	Plums	TULARE	NAUSEA, HEADACHE, VOMITING, UPSET STOMACH.	FOUR WORKERS IN A FLOWER PACKING SHED BECAME ILL AFTER SMELLING A STRONG PESTICIDE ODOR FROM AN APPLICATION BEING MADE IN AN ADJACENT FIELD. THEY SAID THE APPLICATOR DID NOT SHUT OFF THE SPRAY NOZZLES WHILE MAKING TURNS. SEE 92-145 TO 147.	Possible
1992	145	Chlorpyrifos, Cuprous Oxide, Petroleum Oil	Plums	TULARE	NAUSEA, HEADACHE, VOMITING.	SEE 92-144.	Possible
1992	146	Chlorpyrifos, Cuprous Oxide, Petroleum Oil	Plums	TULARE	NAUSEA, LIGHTHEADEDNESS, HEADACHE.	SEE 92-144.	Possible
1992	147	Chlorpyrifos, Cuprous Oxide, Petroleum Oil	Plums	TULARE	HEADACHES, NAUSEA, LIGHTHEADEDNESS.	SEE 92-144.	Possible
1999	211	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	HEADACHE, BURNING EYES, IRRITATED SKIN ON THE ARMS AND FACE.	5 YARDS. 8-MAD-99. A BUS DRIVER CARRIED 26 STUDENTS HOMEWARD. AS THEY PASSED A VINEYARD, THEY FIRST SMELLED A BAD ODOR, THEN SAW TRACTORS SPRAYING. THE DRIVER & 5 STUDENTS FELT SPRAY MIST HIT THEM. SEE 1999-212 TO 237.	Probable
1999	212	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	BURNING EYES.	5 YARDS. 8-MAD-99. SEE 1999-211. BUS & CLOTHING SAMPLES WERE POSITIVE FOR CHLORPYIFOS. THIS STUDENT SAT AT THE BACK OF THE BUS ON THE PASSENGER SIDE. SHE NOTICED A TRACTOR SPRAYING AT THE END OF A ROW, BESIDE THE ROAD. SHE ONLY NOTICED THE ODOR.	Probable

1999	214	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	SORE AND BURNING RIGHT EYE, NAUSEA, DIFFICULTY BREATHING.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS ASTHMATIC STUDENT LAID DOWN IN HER SEAT ON THE PASSENGER SIDE OF THE BUS AND SAW NOTHING. SHE FELT SPRAY MIST ON THE RIGHT SIDE OF HER FACE. ANALYSIS OF HER SHIRT DETECTED THE PRESENCE OF CHLORPYRIFOS AND SULFUR.	Probable
1999	219	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	HEADACHE, STOMACH ACHE, BURNING OF THE EYES, LIPS AND THE SKIN ON THE LEFT HAND.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS STUDENT SAT IN THE FIRST SEAT BEHIND THE DRIVER. SHE FELT SPRAY MIST ON HER FACE AND HANDS. SHE SAW 4 TRACTORS, BUT ONLY 2 WERE SPRAYING. ANALYSIS OF HER CLOTHING DETECTED THE PRESENCE OF CHLORPYRIFOS AND SULFUR.	Probable
1999	221	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	BURNING EYES, STOMACH ACHE.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS STUDENT SAT IN THE THIRD SEAT ON THE PASSENGER SIDE. SHE NOTICED A TRACTOR SPRAYING NEAR THE ROAD, GOING IN THE SAME DIRECTION AS THE BUS. HER WINDOW WAS CLOSED, BUT THE TWO WINDOWS AHEAD OF HER WERE OPEN.	Probable
1999	225	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	EYE IRRITATION OF BRIEF DURATION, STOMACH ACHE.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS STUDENT SAT IN THE TENTH SEAT ON THE PASSENGER SIDE OF THE BUS WITH THE WINDOW CLOSED. THE WINDOW IN FRONT OF HIM WAS OPEN.	Probable
1999	231	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	BURNING EYES, IRRITATED SKIN ON THE FACE AND ARM.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS STUDENT SAT IN THE TENTH SEAT ON THE PASSENGER SIDE OF THE BUS WITH THE WINDOWS IN FRONT OF HIM OPEN. HE SMELLED THE ODOR & FELT SPRAY MIST HIT HIS FACE. ONLY RECORD OF SYMPTOMS ARE IN THE INVESTIGATOR'S INTERVIEW.	Probable
1999	232	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	BURNING, WATERY AND IRRITATED EYES, HEADACHE.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS STUDENT SAT IN THE FIRST SEAT OF THE PASSENGER SIDE OF THE BUS WITH HIS WINDOW OPEN. HE DID NOT NOTICE THE TRACTOR, BUT SMELLED THE ODOR AND FELT SPRAY MIST ON HIS FACE AND CHEST.	Probable
1999	236	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	BURNING LEFT EYE, IRRITATED SKIN ON THE FACE AND LEFT ARM, HEADACHE, CHEST PAIN RESEMBLING WHAT HE REGULARLY FEELS WHEN RUNNING.	5 YARDS. 8-MAD-99. 1999-211. THIS STUDENT SAT IN THE SECOND SEAT BEHIND THE BUS DRIVER. WHILE TURNED AROUND IN HIS SEAT, HE SAW THE TRACTOR, SMELLED THE ODOR AND FELT THE SPRAY MIST WHICH CAME THROUGH THE OPEN WINDOWS ON THE PASSENGER SIDE OF THE BUS.	Probable

1999	237	Chlorpyrifos, Lime-Sulfur	Grapes	MADERA	BURNING EYES.	5 YARDS. 8-MAD-99. SEE 1999-211. THIS STUDENT SAT IN THE SECOND SEAT BEHIND THE BUS DRIVER WITH THE WINDOW CLOSED. HER EYES BEGAN TO BURN ABOUT 5 MINUTES AFTER SMELLING THE PESTICIDE ODOR AND CONTINUED TO BOTHER HER THROUGH THE TIME OF THE INTERVIEW.	Probable
1999	565	Chlorpyrifos, Mineral Oil	Apples	SANTA CRUZ	BURNING NOSE AND THROAT, HEADACHE, NAUSEA, SLIGHT STOMACH PAIN.	AS A GROWER APPLIED PESTICIDES TO HIS APPLE ORCHARD, A BUS DROVE UP TO A NEARBY BUS STOP. THE DRIVER NOTICED SPRAY MIST HIT THE WINDSHIELD. UPON OPENING THE BUS DOOR FOR PASSENGERS, HE DETECTED A STRONG CHEMICAL ODOR. HE BECAME ILL A SHORT TIME LATER.	Probable
1999	919	Chlorpyrifos, Mineral Oil	Apples	SANTA CRUZ	HEADACHE, COUGH, FEVER, INSOMNIA, FATIGUE, MEMORY LOSS.	AN APPLICATOR DRIFTED PESTICIDES ONTO A VEHICLE ON A ROAD ADJACENT TO THE ORCHARD. ALTHOUGH INITIALLY ASYMPTOMATIC, THE DRIVER DEVELOPED SYMPTOMS WITHIN 2 DAYS. LABORATORY ANALYSIS SHOWED CHLORPYRIFOS IN THE SWAB SAMPLES TAKEN FROM THE VEHICLE.	Possible
2006	249	Chlorpyrifos, Mineral Oil	Oranges	TULARE	DIZZINESS, HEADACHE.	100 FEET. A FARM WORKER MONITORED A ROAD FOR TRAFFIC WHILE A CO-WORKER SPRAYED ORANGES. IN SPITE OF WEARING A RESPIRATOR, HE SMELLED THE MATERIAL APPLIED. HE REPORTED CONTINUING DIZZINESS TWO DAYS LATER. INVESTIGATORS FOUND TECHNICAL SHORTCOMINGS.	Possible
1996	681	Chlorpyrifos, Petroleum Oil	Oranges	TULARE	HEADACHE, NAUSEA, DIZZINESS, MUSCLE SPASMS, DROOLING, RUNNY NOSE, BLURRY VISION, TINGLING SKIN, SOB.	AN APPLICATOR DEVELOPED SYMPTOMS SHORTLY AFTER SPRAYING PESTICIDES IN AN ORANGE GROVE. HE CLAIMED TO FEEL SOME SPRAY MIST ON HIS ARMS AND LEGS DURING THE APPLICATION. HE SOUGHT MEDICAL ATTENTION THE NEXT DAY.	Probable
2000	958	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	NAUSEA, ABDOMINAL CRAMS, DIZZINESS, HEADACHE, DIARRHEA.	SEE 2000-952. CHILD WAS EXPOSED TO DRIFT OUTSIDE FOR 10 MINUTES BEFORE SCHOOL, INDOORS THE REST OF THE DAY, DEVELOPING SYMPTOMS LATE. AN UNEXPOSED SIBLING HAD SIMILAR SYMPTOMS. HER MOTHER TOOK HER FOR EVALUATION AFTER SYMPTOMS PERSISTED	Possible
2000	1108	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	HEADACHE, ITCHY AND TEARY EYES, HEAVY NASAL CONGESTION.	SEE 2000-952. THE CHILD'S MOTHER REPORTS SHE WAS FINE WHEN SHE LEFT FOR SCHOOL. SHE DEVELOPED SYMPTOMS WHILE AT SCHOOL AS WELL AS ADDITIONAL SYMPTOMS WHILE AT HOME. HER MOTHER CONSULTED HER PEDIATRICIAN BY	Possible

2000	1118	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	HEADACHE, RED AND ITCHY EYES.	SEE 2000-952. THIS TEACHER REPORTS BEING EXPOSED AT SCHOOL AS WELL AS POSSIBLY BEING EXPOSED AS ON HER WAY TO THE SCHOOL.	Possible
2000	1121	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	NAUSEA, STOMACH CRAMPS.	SEE 2000-952. ACCORDING TO HIS MOTHER'S COMPLAINT, THIS BOY'S PESTICIDE EXPOSURE OCCURRED AS HE WALKED TO SCHOOL IN THE MORNING.	Possible
2000	1122	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	MUSCLE TWITCHING, STOMACH CRAMPS, HEADACHE, TEARING EYES, CONFUSION, BACKACHE, ASTHMA, EMOTIONAL UPSET.	SEE 2000-952. HIS MOTHER REPORTS HIS EXPOSURE OCCURRED WHILE HE WAS IN FRONT OF THE SCHOOL AS WELL AS IN THE CLASSROOM. SHE ALSO REPORTS TAKING HIM TO SEE A DOCTOR.	Possible
2000	1123	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	HEADACHE, STOMACH ACHE, ASTHMA.	SEE 2000-952. HER MOTHER REPORTS HER EXPOSURE OCCURRED WHILE SHE WAS IN FRONT OF THE SCHOOL AS WELL AS IN THE CLASSROOM. SHE ALSO REPORTS TAKING HER TO SEE A DOCTOR.	Possible
2000	1130	Chlorpyrifos, Petroleum Oil	Lemons	VENTURA	STOMACH ACHE, NAUSEA.	SEE 2000-952. THIS CHILD WAS DROPPED OFF BY HIS PARENT IN FRONT OF THE SCHOOL BEFORE THE END OF THE APPLICATION. THAT EVENING, HE MENTIONED THE AIR SMELLING FUNNY.	Possible
2006	233	Chlorpyrifos, Petroleum Oil	Oranges	TULARE	DIZZINESS, NAUSEA, VOMITING. PREEXISTING STOMACH PAIN UNDER TREATMENT BY PERSONAL PHYSICIAN.	AN APPLICATOR APPLIED CHLORPYRIFOS AND OIL TO AN ORANGE GROVE. HE QUIT SPRAYING THE ORCHARD CLOSE TO AN ADJACENT ORCHARD WHEN A CREW ARRIVED. HE STARTED SPRAYING ONE A SHORT DISTANCE AWAY. TWO ORANGE PICKERS SMELLED THE ODOR AND BECAME	Probable
2006	234	Chlorpyrifos, Petroleum Oil	Oranges	TULARE	NAUSEA, VOMITING, DRY MOUTH, HEADACHE, DIZZINESS.	SEE 2006-233. UPON BEING NOTIFIED BY THE CREW FOREMAN, THE APPLICATOR MOVED FROM THE NEAREST BLOCK OF ORANGES TO THE NEXT ONE OVER. THIS WORKER ARRIVED AS THE FOREMAN TALKED WITH THE APPLICATOR.	Probable

## **APPENDIX 2**



Chlorpyrifos application by county (2010-2011)

kg chlorpyrifos

- 0.0000 - 17.6000
- 17.6000 - 185.6000
- 185.6000 - 658.2000
- 658.2000 - 1545.6000
- 1545.6000 - 116751.7000

>50 non-white population (2010 census)

above 50% non-white