Farmworker and Conservation Comments on Chlorpyrifos Revised Human Health Risk Assessment

Earthjustice

League of United Latin American Citizens

Pesticide Action Network North America

Natural Resources Defense Council

California Rural Legal Assistance Foundation

Farmworker Association of Florida

Farmworker Justice

GreenLatinos

Labor Council for
Latin American Advancement

Learning Disabilities Association of America

National Hispanic Medical Association

Pineros y Campesinos Unidos del Noroeste

United Farm Workers

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LIST OF EXHIBITS

NO.	TITLE
1.	Petition to Suspend and Cancel Chlorpyrifos Uses
2.	Declaration of Philip J. Landrigan, M.D., M.SC. in Support of Petition to Suspend and Cancel Chlorpyrifos Uses
3.	Final Report for the FIFRA SAP Virtual Meeting held on Sept. 15-18, 2020, at EPA-HQ-OPP-2020-0263-0054
4.	Transmittal of Meeting Minutes and Final Report of the Federal Insecticide, Fungicide and Rodenticide Act, Scientific Advisory Panel (FIFRA SAP) Virtual Meeting held on September 15-18, 2020 (Dec. 14, 2020) ("2020 NAM Report")
5.	Janie F. Shelton et al. Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study. 122 Environmental Health Perspectives 1107 (2014), https://ehp.niehs.nih.gov/doi/10.1289/ehp.1307044
6.	López-Gálvez N, Wagoner R, Quirós-Alcalá L, et al. Systematic Literature Review of the Take-Home Route of Pesticide Exposure via Biomonitoring and Environmental Monitoring. Int J Environ Res Public Health. 2019;16(12):2177. Published 2019 Jun 19. doi:10.3390/ijerph16122177
7.	First Declaration of Dr. Frank Ackerman, filed in UFW v. EPA, No. CV04-0099-RSM (W.D. Wash. filed 2005
8.	Second Declarations of Dr. Frank Ackerman, filed in *UFW v. EPA*, No. CV04-0099-RSM (W.D. Wash. filed 2007)
9.	Bellanger M, Demeneix B, Grandjean P, Zoeller RT, Trasande L. Neurobehavioral deficits, diseases and associated costs of exposure to endocrine disrupting Chemicals in the European Union. J Clin Endocrinol Metab. 2015
10.	Attina TM, Hauser R, Sathyanarayana S, Hunt PA, Bourguignon J-P, Myers JP, DiGangi J, Zoeller RT, Trasande L. Exposure to endocrine-disrupting chemicals in the USA: a population-based disease burden and cost analysis. Lancet Diabetes Endocrinol. 2016
11.	Gaylord A, Osborne G, Ghassabian A, Malits J, Attina T, Trasande L, Trends in Neurodevelopmental Disability Burden Due to Early Life Chemical Exposure in the USA from 2001 to 2016: A Population-Based Disease Burden and Cost Analysis, 502 Mol. Cell. Endocrinol. 110666 (Feb. 15, 2020)

INTRODUCTION

These comments are submitted on behalf of the League of United Latin American Citizens ("LULAC"), Pesticide Action Network North America ("PANNA"), Natural Resources Defense Council ("NRDC"), California Rural Legal Assistance Foundation, Farmworker Association of Florida, Farmworker Justice, GreenLatinos, Labor Council for Latin American Advancement, Learning Disabilities Association of America, National Hispanic Medical Association, Pineros y Campesinos Unidos del Noroeste, and United Farm Workers (collectively "LULAC"). We urge the Environmental Protection Agency ("EPA") to take swift, long-overdue action to end use of chlorpyrifos.

Chlorpyrifos is unsafe at any level of exposure. It causes acute pesticide poisonings and at far lower levels of exposure it damages children's brains, leaving children with learning disabilities and reduced IQ. When EPA has tried to protect children from learning disabilities and permanent brain-based disorders, it has found all exposures unsafe, whether from food, drinking water, pesticide drift, or worker exposures.

EPA should take the following actions to protect children. First, EPA should act immediately to protect our food supply by revoking all chlorpyrifos food tolerances. Because aggregate exposures to chlorpyrifos from its use on food are unsafe, EPA cannot make the required regulatory finding that there is reasonable certainty of no harm and cannot retain chlorpyrifos tolerances. Once the food tolerances are revoked, use of chlorpyrifos on our food will end. Second, EPA should cancel all uses of chlorpyrifos because all uses cause unreasonable adverse effects to workers and others exposed to the pesticide.

PROCEDURAL POSTURE

These comments are submitted to inform upcoming EPA decisions in two overlapping regulatory processes: (1) registration review of chlorpyrifos; and (2) EPA's recently announced review of its denial of objections and a 2007 petition to cancel food uses of chlorpyrifos.

I. REGISTRATION REVIEW DECISION

In early December 2020, EPA opened a public comment period on its Proposed Interim Registration Review Decision for Chlorpyrifos ("PID") and its Third Revised Human Health Risk Assessment for Chlorpyrifos (Sept. 21, 2020) ("2020 HHRA" or "2020 health risk assessment"). 85 Fed. Reg. 78,849 (Dec. 7, 2020) (notice of availability and request for comment).

Under the Federal Insecticide, Fungicide, and Rodenticide Act ("FIFRA"), EPA must review pesticide registrations every fifteen years, in a process called registration review. 7 U.S.C. § 136a(g)(1)(A)(i), (iii)(II). The purpose of this review is to ensure that pesticide uses meet applicable standards under both FIFRA and the Federal Food, Drug, and Cosmetic Act ("FFDCA"). This round of registration reviews of older pesticides began in 2007 and must be completed by October 1, 2022. EPA had prioritized chlorpyrifos in the registration review

process with final regulatory actions scheduled for 2015. *See* 84 Fed. Reg. 35,555, 35,558 (July 24, 2019).

The 2020 health risk assessment and PID are part of the EPA docket for registration review of chlorpyrifos, which was opened in 2008. EPA-HQ-OPP-2008-0850. This docket contains EPA's 2011 preliminary human health risk assessment, and its 2014 and 2020 revised human health risk assessments. The 2020 HHRA relies on these prior risk assessments, as well as on its 2016 revised human health risk assessment and its 2015 Literature Review on Neurodevelopmental Effects & FQPA Safety Factor Determination for the Organophosphate Pesticides. 2020 HHRA at 6; PID at 7.[1] EPA's risk assessments also rely on several reviews by EPA's Scientific Advisory Panel ("SAP") that examined neurodevelopmental harm caused by chlorpyrifos through low-level exposures. *See infra* at 17 n.19-21. Because EPA's 2020 HHRA and PID rely on these EPA assessments and SAP reviews, the record for EPA's registration review decision must include their underlying records.

In addition, in 2016, many of the commenters here filed a petition to cancel all chlorpyrifos uses because they pose unacceptable risks to workers. The petition also sought suspension of many uses, but that portion of the petition was withdrawn. EPA never opened a docket for this petition, which is attached as Exhibits 1 and 2 (petition and supporting Declaration of Philip J. Landrigan, M.D., M.Sc. (Sept. 2016)). EPA also never sought public comment on it. Nonetheless, it is before the agency and should be considered as EPA makes its registration review decision.

Also, both the 2020 health risk assessment and the PID float the possibility of further eliminating the Food Quality Protection Act ("FQPA") tenfold safety factor based on new testing methodologies that EPA presented to the SAP in September 2020. After EPA released the risk assessment and PID for public comment, the SAP issued its report, concluding that the new methods could not be used to reduce safety factors. Final Report for the FIFRA SAP Virtual Meeting held on Sept. 15-18, 2020, at EPA-HQ-OPP-2020-0263-0054 (Exhibit 3). The record for registration review should include the record of this SAP review.

II. EPA ACTION ON 2007 PETITION TO CANCEL FOOD USES OF CHLORPYRIFOS

After EPA published the request for comment, President Biden was inaugurated on January 20, 2021. On his first day in office, the President issued an Executive Order on Protecting Public Health and the Environment and Restoring Science to Tackle the Climate Crisis (Jan. 20, 2021).[2] Section 1 of the Executive Order provides:

> Policy. Our Nation has an abiding commitment to empower our workers and communities; promote and protect our public health

[1] The 2016 risk assessment is in the docket for EPA's proposed revocation of chlorpyrifos tolerances, discussed below. EPA-HQ-OPP-2015-0653.

[2] https://www.whitehouse.gov/briefing-room/presidential-actions/2021/01/20/executive-order-protecting-public-health-and-environment-and-restoring-science-to-tackle-climate-crisis/

> and the environment; and conserve our national treasures and monuments, places that secure our national memory. Where the Federal Government has failed to meet that commitment in the past, it must advance environmental justice. In carrying out this charge, the Federal Government must be guided by the best science and be protected by processes that ensure the integrity of Federal decision-making. It is, therefore, the policy of my Administration to listen to the science; to improve public health and protect our environment; to ensure access to clean air and water; to limit exposure to dangerous chemicals and pesticides; to hold polluters accountable, including those who disproportionately harm communities of color and low-income communities; to reduce greenhouse gas emissions; to bolster resilience to the impacts of climate change; to restore and expand our national treasures and monuments; and to prioritize both environmental justice and the creation of the well-paying union jobs necessary to deliver on these goals.
>
> To that end, this order directs all executive departments and agencies (agencies) to immediately review and, as appropriate and consistent with applicable law, take action to address the promulgation of Federal regulations and other actions during the last 4 years that conflict with these important national objectives, and to immediately commence work to confront the climate crisis.

The White House released a non-exclusive list of agency actions subject to this review, which includes: "Chlorpyrifos; Final Order Denying Objections to March 2017 Petition Denial Order," 84 Fed. Reg. 35,555 (July 24, 2019) ("Objections Denial Order" or "Wheeler Order"). This order was issued by then-EPA Administrator Andrew Wheeler as part of EPA's review and action on a petition filed in 2007 by NRDC and PANNA. The 2007 petition sought to ban food uses of chlorpyrifos based on evidence of neurodevelopmental harm to children at exposures below those that cause cholinesterase inhibition. NRDC and PANNA, represented by Earthjustice, went to court five times and obtained court deadlines for EPA to act on this petition based on the Ninth Circuit Court of Appeals' findings that EPA's delay in acting on the 2007 petition was "egregious" and "objectively extreme," in light of its pressing human health effects. *In re PANNA v. EPA*, 798 F.3d 809, 811, 813 (9th Cir. 2015); *In re PANNA*, 840 F.3d 1014, 1015 (9th Cir. 2016).

Both in response to the petition and as part of registration review, EPA found that chlorpyrifos causes damage to children's developing brains at exposures far below its regulatory endpoint designed to prevent acute poisonings that are caused by cholinesterase inhibition. Specifically, after two reviews by its SAP, EPA released its 2014 revised human health risk assessment in which it found, based on laboratory and human studies, that chlorpyrifos disrupts children's brain development and can result in loss of IQ, and learning and developmental

disorders, including attention deficit/hyperactivity disorder and autism at low-level exposures. That risk assessment found that children would face unsafe exposures to chlorpyrifos in drinking water. In 2015, EPA proposed revoking all tolerances and ending food uses of chlorpyrifos based on drinking water contamination. 80 Fed. Reg. 69,080 (Nov. 6, 2015). EPA stated that it "is unable to conclude that the risk from aggregate exposure from the use of chlorpyrifos meets the safety standard of the Federal Food, Drug, and Cosmetic Act (FFDCA)." *Id*.; *see also id*. at 69,081 ("EPA cannot, at this time, determine that aggregate exposure to residues of chlorpyrifos, including all anticipated dietary exposures and all non-occupational exposures for which there is reliable information, are safe."). The proposed rule acknowledged that the 2014 risk assessment underestimates the harm to children because learning disabilities occur at exposures below EPA's cholinesterase inhibition endpoint. EPA planned to continue reviewing the evidence of long-lasting damage to children's brains from low-level exposures to try to identify a safe level of exposure for children.

In November 2016, EPA updated its risk assessment using an endpoint that would prevent damage to children's developing brains. EPA found that chlorpyrifos presents unacceptable safety risks through exposures from food, drinking water, spray drift, and worker activities. EPA reiterated that "it can only retain chlorpyrifos tolerances if it is able to conclude that such tolerances are safe" and its updated analysis "continues to indicate that the risk from the potential aggregate exposure does not meet the FFDCA safety standard." 81 Fed. Reg. 81,049, 81,050 (Nov. 17, 2016).

EPA had a March 31, 2017 court-imposed deadline to take final action on the 2007 petition. It had drafted a final revocation rule, which would have ended food uses of chlorpyrifos by October 2017. Instead, on March 29, 2017, then-Administrator Scott Pruitt issued an order denying the 2007 petition. Even though ten years had passed since NRDC and PANNA filed their petition, EPA claimed it needed to continue studying the science before finalizing the proposed revocation or taking an alternative regulatory path. It indicated that it would put off chlorpyrifos tolerance decisions until completion of registration review of older pesticides. 82 Fed. Reg. 16,581 (April 5, 2017) ("Pruitt Order").

The health, farmworker, and civil rights groups submitting these comments, joined by several states led by New York, challenged the Pruitt Order in court and in administrative objections filed with the agency. The full Ninth Circuit ordered EPA to decide administrative objections by July 19, 2019. *LULAC v. Wheeler*, 922 F.3d 443 (9th Cir. 2019) (en banc). By that deadline, then-Administrator Wheeler issued the order denying the objections—the order included on President Biden's list of agency actions under review. The Wheeler Order did not find chlorpyrifos safe. Instead, it reiterated EPA's plan to engage in further study and put off regulatory action until completion of registration review. 84 Fed. Reg. 35,555 (July 24, 2019). EPA indicated that it would release an updated risk assessment and proposed regulatory action for comment in 2020, the documents that are the subject of these comments.

LULAC *et al*. and New York, California, Washington, Maryland, Vermont, Massachusetts, Hawaii, Oregon, and the District of Columbia have challenged the 2019 Wheeler Order. *LULAC v. Wheeler*, No. 19-71979 (9th Cir.); *New York v. Wheeler*, No. 19-71982 (9th

Cir.). The consolidated cases were argued before the Ninth Circuit Court of Appeals on July 28, 2020.

Because these comments will inform EPA's review of the Wheeler Order, the record must include the dockets for the 2007 petition to cancel chlorpyrifos food uses (EPA-HQ-OPP-2007-1005) and the proposed revocation order (EPA-HQ-OPP-2015-0653).

EXECUTIVE SUMMARY

Chlorpyrifos is an organophosphate pesticide used on a wide variety of food and feed crops, including foods eaten by children like apples, peaches, nectarines, pears, grapes, cherries, oranges, and strawberries. It is acutely toxic, causing poisonings by suppressing cholinesterase, an enzyme that regulates nerve impulses. EPA re-registered chlorpyrifos in 2006 using 10% cholinesterase inhibition as its regulatory endpoint based on its view that no poisonings or other harm would occur from exposures causing that effect.

I. NEURODEVELOPMENTAL HARM FROM LOW-LEVEL EXPOSURES

A growing body of scientific evidence demonstrates that chlorpyrifos impairs children's brain development at exposures far below those that produce 10% cholinesterase inhibition. In response to the 2007 petition to ban food uses of chlorpyrifos, EPA submitted its reviews of the scientific literature to its SAP.

EPA and the SAP focused specifically on a long-term study conducted by the Columbia Center on Children's Environmental Health that correlated chlorpyrifos levels in African American and Dominican pregnant women in New York City with adverse neurodevelopmental effects in their children. The study began in 1997, before EPA initiated the phase out of all home uses of chlorpyrifos beginning in 2000, in response to evidence that children who crawled on carpets or hugged pets who had been treated with chlorpyrifos were exposed to unsafe levels of the pesticide.[3] The children born after the ban had dramatically lower chlorpyrifos levels than those born before the ban. A series of published peer reviewed articles found statistically significant delays in motor and mental development, attention disorders, and reduced IQ. EPA and the SAP considered the Columbia and other epidemiology studies finding similar results to be high quality studies conducted in accordance with EPA policies. Based on these and other studies, the SAP found, in 2008, 2012, and 2016, that chlorpyrifos causes learning disabilities and harms children's brains at exposures below those that cause 10% cholinesterase inhibition.

[3] Rauh, V., Arunajadai, S., Horton, M., Perera, F., Hoepner, L., Barr, D. B., & Whyatt, R. (2011). Seven-year neurodevelopmental scores and prenatal exposure to chlorpyrifos, a common agricultural pesticide. Environmental Health Perspectives, 119(8), 1196-1201, https://doi.org/10.1289/ehp.1003160; Perera FP, Illman SM, Kinney PL, et al. The challenge of preventing environmentally related disease in young children: community-based research in New York City. Environmental Health Perspect. 2002;110(2):197-204. doi:10.1289/ehp.02110197

In 2014, EPA issued a revised human health risk assessment that acknowledged the SAP findings and the growing body of studies that correlate chlorpyrifos exposure with neurodevelopmental harm. Based on its review of both laboratory and human studies, EPA found that chlorpyrifos causes learning disabilities and other damage to children's developing brains at exposures lower than those that cause 10% cholinesterase inhibition. EPA has never deviated from this finding. Nor has the SAP. Both the 2020 HHRA and the PID reiterate that chlorpyrifos damages children's brains at such low-level exposures.

II. THE 2020 RISK ASSESSMENT DOES NOT PROTECT CHILDREN FROM LEARNING DISABILITIES AND IMPAIRED BRAIN DEVELOPMENT

The 2020 risk assessment uses the same methodology as its 2014 predecessor. First, it continues to use 10% cholinesterase inhibition as the regulatory endpoint even though children suffer from learning disabilities and other neurodevelopmental harm at lower exposure levels. In doing so, EPA violated its own policy of using the most sensitive endpoint in assessing pesticide risks.

Second, EPA retained the tenfold safety factor for infants, children, youth, and women of child-bearing years because of the neurodevelopmental harm to children and uncertainties as to the specific exposure level at which chlorpyrifos damages children's brains. While the PID considered eliminating the FQPA tenfold safety factor based on new scientific methods, the SAP soundly rejected this possibility in December 2020. The FQPA tenfold safety factor must be retained.

Third, EPA used a model developed by Dow Agrosciences (DAS) based on human studies to try to pinpoint the exposures that correspond to 10% cholinesterase inhibition. Because the model measures effects directly in people, EPA eliminated the tenfold safety factor that is ordinarily in place to account for uncertainties in extrapolating from animal studies to people and shrunk another tenfold safety factor that accounts for variations among human populations. It is indefensible to eliminate this safety factor when 10% cholinesterase inhibition is not the most sensitive endpoint. Even using that endpoint, EPA found in both 2014 and 2020 that agricultural chlorpyrifos uses would result in exposures that exceed EPA's drinking water levels of concern.

EPA recognized that its 2014 risk assessment was not sufficiently protective because damage to children's developing brains occurs from lower exposures. EPA could not find chlorpyrifos safe based on the 2014 risk assessment's findings of unsafe drinking water contamination. It, therefore, proposed in 2015 to revoke all tolerances and end food uses of chlorpyrifos. 80 Fed. Reg. 69,080 (Nov. 6, 2015). The proposed rule acknowledged that the risks to children are far greater than those identified in the 2014 risk assessment because chlorpyrifos exposures damages the developing brain even if there is little or no cholinesterase inhibition.

It is long-standing EPA policy, when assessing developmental effects, that "the most sensitive developmental effect (i.e., the critical effect) from the most appropriate and/or sensitive

mammalian species is used for determining the NOAEL, LOAEL, or the benchmark dose."[4] Similarly, EPA's guidance on the process for developing a reference dose/concentration states that it should be based on a "critical effect," defined as "the first adverse effect, or its known precursor, that occurs to the most sensitive species as the dose rate of an agent increases."[5] In keeping with these policies, EPA during the Obama administration tried to identify a safe level of exposure for children, even after it proposed revoking all chlorpyrifos tolerances. In November 2016, EPA updated its risk assessment using an endpoint intended to prevent harm to children's developing brains. EPA found that chlorpyrifos presents unacceptable safety risks through exposures from food, drinking water, spray drift, and worker activities. Food-only exposures for chlorpyrifos were found to be unsafe for all populations, with young children facing the highest risks of concern. While adults face estimated exposures that are 62 times higher than the safe level, children ages 1-2 face predicted exposures that are more than 140 times higher than safe levels. EPA continued to find that "the majority of estimated drinking water exposures from currently registered uses, including water exposures from non-food uses, continue to exceed safe levels even taking into account more refined drinking water exposures." In EPA's words, the updated risk analysis "indicates that expected residues of chlorpyrifos on most individual food crops exceed the 'reasonable certainty of no harm' safety standard" and that most drinking water exposures "continue to exceed safe levels . . . ." 81 Fed. Reg. 81,049, 81,050 (Nov. 17, 2016).

The 2020 assessment abandons EPA's prior attempt to find a safe exposure level that would prevent harm to children's brains. It reverts to the framework of the 2014 assessment, which used 10% cholinesterase inhibition as the regulatory endpoint. Despite nearly four years of delay, EPA conducted no additional peer review of the scientific evidence. Nor did it undertake any additional effort to identify a safe exposure level.

Instead, it cobbled together reasons to justify not using its 2016 risk assessment, embracing arguments made by Dow that run counter to EPA policies and the requirements of the law. The top line message of the 2020 assessment (repeated no fewer than 9 times) is: "The science addressing neurodevelopmental effects remains unresolved." While the precise exposure level and mechanism by which chlorpyrifos damages children's brains is uncertain, the link between this pesticide and debilitating learning disabilities and impaired brain development is well-established. Under EPA policy, the agency cannot ignore evidence of harm simply because it has not yet determined the mode of action.

EPA also claims that it can disregard the Columbia epidemiology study because the underlying raw data have not been made public, even though it acknowledges that releasing the raw data would violate the privacy rights of the study participants. In accepting Dow's argument that the raw data must be made public, EPA deviated from its policies that allow the agency to

[4] EPA, Guidelines for Developmental Toxicity Risk Assessment. U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC, (1991), https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=23162

[5] EPA, A Review of the Reference Dose and Reference Concentration Processes at G-2 (Dec. 2002), https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf

rely on peer review and its iterative SAP reviews to ensure the validity of the studies. EPA previously had adhered to agency policies in finding the Columbia study sound, carefully designed, and well-executed and in determining based on the weight of the evidence that chlorpyrifos harms children's brains at low-level exposures.

By trying to turn scientific uncertainties into a reason to continue delaying regulatory action to protect children, workers, and our food supply, the 2020 risk assessment (and PID based on it) violate the law. The FQPA requires EPA to make an affirmative safety finding in order to leave a pesticide tolerance in place—that is, EPA must find that there is a "reasonable certainty of no harm" for every age group, specifically children, and based on all aggregate chlorpyrifos exposures for each food use for which it establishes a tolerance. Here, EPA acknowledges (and the SAP has likewise found) that harm to children's brains occurs at exposure levels far below 10% cholinesterase inhibition—as such, basing tolerances on 10% cholinesterase inhibition is underprotective and unsafe.

EPA gave short shrift to other ways it could have tried to protect children from neurodevelopmental harm. In 2018, California's Department of Pesticide Regulation ("CDPR") designated chlorpyrifos a toxic air contaminant. It did so based on a risk assessment that relied on animal studies that documented neurodevelopmental harm at exposure levels below those that cause 10% cholinesterase inhibition. The designation and subsequent cancellation proceedings led to the end of 99% of chlorpyrifos uses in California by the end of 2020. The 2020 risk assessment reviewed the animal studies and found two were adequate for use in risk assessments, yet it never tried to use them.

EPA's 2016 risk assessment established a safe exposure level 650 times lower than EPA's 10% cholinesterase inhibition trigger. The regulatory endpoint in California's risk assessment is 150 times lower. It is inexcusable for EPA to continue to use 10% cholinesterase inhibition in the face of such powerful evidence that doing so is unsafe for children.

The underprotective endpoint infects the entirety of the 2020 risk assessment. Even using this endpoint, EPA finds risks of concern in drinking water and to workers. If it had used an endpoint that would protect children from neurodevelopmental harm, it would have found, as it did in 2016, that chlorpyrifos also poses risks of concern in food and pesticide drift.

III. THE 2020 DRINKING WATER ASSESSMENT UNDERESTIMATES EXPOSURES.

The 2020 assessment also updated EPA's drinking water assessment, using the underprotective 10% cholinesterase inhibition endpoint. EPA focused specifically on 11 crops identified by Dow/Corteva or EPA staff as the most important to growers—alfalfa, apples, asparagus, cherries, citrus, cotton, peaches, soybean, sugar beet, strawberries, and wheat. The revised drinking water assessment purports to find that use of chlorpyrifos on these crops would not exceed the drinking water levels of concern, while the remaining uses would. Of course, the drinking water levels of concern are based on the underprotective endpoint and are invalid for that reason. If EPA used an endpoint that would be safe for children's brain development, it

would have found unsafe drinking water contamination from all uses of chlorpyrifos on food, as it found in the 2015 proposed revocation rule.

EPA's new drinking water modeling is also flawed because it underestimates exposures. It fails to consider groundwater, is based on case studies drawn from only one part of the country and small sample sizes, and produced more low-confidence rankings than high-confidence ones. EPA acknowledged that real-world water monitoring has detected chlorpyrifos at levels above EPA's drinking water levels of concern. EPA indicated it is unable to determine whether the drinking water contamination is from any of the uses Corteva is seeking to retain. It also noted that drinking water levels of concern might be exceeded if chlorpyrifos is used on more than one crop in the watershed. The water monitoring data demonstrate that EPA's new drinking water assessment is seriously underprotective and cannot support a finding of reasonable certainty of no harm from drinking water exposures.

Even with its underprotective exposure assessment and safety level, EPA's drinking water assessment reveals drinking water levels of concern would be exceeded in 4 of the 11 regions of the country. Over 200 community water systems could have unsafe levels of chlorpyrifos. Since EPA's drinking water assessment is underprotective, unsafe drinking water is, in fact, even more ubiquitous.

IV. THE 2020 RISK ASSESSMENT UNDERESTIMATES WORKER EXPOSURES

Even using an underprotective endpoint, the 2020 risk assessment continues to find unacceptable risks to handlers from over 140 activities, including aerial spraying and certain formulations used in greenhouses, even if more personal protective equipment ("PPE") or engineering controls are required. The assessment purports to find that use of engineering controls would adequately mitigate the risks to handlers from an additional 45 activities. The risks from airblast spraying in tractors without enclosed cabs are extraordinarily high, one or sometimes two orders of magnitude above EPA's risk of concern level.

The risks of concern are far more severe and extensive because the worker risk assessment is based on an underprotective endpoint. The risk assessment also overstates the effectiveness of PPE. And the risk assessment suggests that risks of concern could be reduced or eliminated with maximum PPE—double layers of clothing, long-sleeves, long-pants, and respirators—even though EPA acknowledges this may lead to heat and respiratory stress in many parts of the country where chlorpyrifos is used.

EPA also finds that some field workers who re-enter the fields soon after pesticide applications will face risks of concern. For 30 activities performed by workers, such as hand harvesting, thinning, irrigation, and scouting, the bar on re-entering the fields after spraying would need to be extended by several more days than is currently required.

The risk assessment is underprotective for another reason. It fails to account for the risks workers face from aggregate exposures to chlorpyrifos from their jobs, pesticide drift, residues

that remain on their clothes and introduce chlorpyrifos-laden dust into their homes, and drinking water from both crop and other uses of chlorpyrifos.

V. THE PID FAILS TO CONDUCT A CREDIBLE BALANCING OF ALL OF THE RISKS AND BENEFITS IN PURPORTING TO JUSTIFY EXPOSING WORKERS TO RISKS OF CONCERN.

Because EPA cannot find chlorpyrifos safe on food, it must revoke all chlorpyrifos tolerances and cancel the FIFRA registrations for food uses. The controlling food safety standard in FFDCA and FIFRA is health-based. Congress decided that our food should be safe, particularly for children, and that safety cannot be sacrificed for any reason. Nonetheless, EPA's crop benefits assessment estimates the costs of shifting from chlorpyrifos to alternative pesticides. EPA admitted that it cannot find chlorpyrifos safe for food crops and therefore these uses must end no matter the benefits.

For food, FIFRA's risk-benefit balancing standard applies only if EPA could find reasonable certainty of no harm from use of chlorpyrifos on a particular food crop, which it cannot. If EPA could make a safety finding for the 11 crops it featured in its drinking water assessment, it would need to find that there are no unreasonable adverse effects on the environment, "taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." 7 U.S.C. § 136(bb). EPA would need to make the same finding for non-food uses.

The PID fails to undertake a credible balancing of all the costs and benefits of using chlorpyrifos. First, while EPA prepared benefits assessments, those assessments focus only on currently registered chemical pesticide alternatives, thereby ignoring organic farming and less toxic pesticides that may be developed in the future. Second, EPA never considered the other side of the ledger—the costs of acute poisonings, missed work, educating children who have learning disabilities, and lower earning potential from reduced IQ. It treats all worker risks the same even if the risk from one activity is an order of greater magnitude than from another.

When it comes to balancing the risks and the benefits, EPA is a no-show. The PID repeatedly justifies not stopping use of chlorpyrifos or not requiring the most effective mitigation possible short of cancellation by calling the use a "high benefit use." It uses this label indiscriminately every time it indicates that EPA might not require maximum PPE or engineering controls. It assigns this label to crops identified as high benefit in its crop benefits assessment, but even more often, for crops not so identified. And it acts as if reciting this mantra is the end of the inquiry. It never balances the benefits of chlorpyrifos for growing a particular crop or for any other use with the harm the pesticide causes to workers and children.

The PID would allow use of chlorpyrifos to continue without considering the disproportionate impacts on communities of color. The PID has no environmental justice analysis. The 2020 HHRA's one-paragraph environmental justice analysis does not satisfy EPA's obligations under the 1994 Environmental Justice Executive Order or the 1997 Children's Health Executive Order. Even though EPA failed to complete an adequate environmental justice

analysis for chlorpyrifos, the record before EPA clearly demonstrates that chlorpyrifos must be banned. If EPA somehow concludes that some uses of chlorpyrifos can remain, then EPA must engage in a robust environmental justice analysis of those remaining uses. A proper environmental justice assessment must look at the disproportionate impacts of drift, food, drinking water, and worker exposures (including take-home exposures), and the assessment should be used to inform EPA's regulatory decisions and mitigation measures.

By failing to prevent unacceptable risks to workers, EPA is denying farmworkers the same level of protection afforded other workers. The Occupational Safety and Health Administration adheres to the hierarchy of controls in setting workplace standards for industrial workers, which prioritizes eliminating harmful exposures over engineering controls and views PPE as the least effective mitigation measure and the last resort. EPA turns the hierarchy of controls on its head by turning first to PPE and considering engineering controls only if PPE cannot, in its view, eliminate the risks of concern. Eliminating the exposure by ending the application method comes last. And the PID purports to justify rejecting all of these options by calling the use "high benefit."

EPA cannot find reasonable certainty of no harm from food uses of chlorpyrifos. Nor can it find that other uses avoid causing unreasonable adverse health effects. Accordingly, EPA must finalize the tolerance revocation rule and cancel all chlorpyrifos registrations.

CONTROLLING STATUTES

I. THE FFDCA MANDATES ELIMINATION OF HARMFUL PESTICIDES FROM OUR FOOD SUPPLY.

EPA regulates allowable contaminants, including pesticides, in our food supply under the FFDCA. For a pesticide to be permitted on food and imported or sold in interstate commerce, EPA must issue a tolerance that establishes 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) (emphasis added). Under the Food Quality Protection Act ("FQPA"), which passed unanimously in 1996 and amended the FFDCA, "safe" means that EPA can "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).

The 1996 passage of the FQPA responded to a seminal 1993 National Academy of Sciences ("NAS") report criticizing EPA for regulating pesticides based on the effects on a 150-pound adult male.[6] It documented the ways that children are not "little adults" but have unique exposures from the foods they eat, their play, and their metabolism. For example, a 6-month old child drinks seven times more per body weight than an adult, inhales twice as much air, and puts its hands in its mouth more than is common later in life. The report also highlighted the

[6] National Research Council, *Pesticides: Diets of Infants and Children* (1993) ("NAS Report"), https://www.nap.edu/catalog/2126/pesticides-in-the-diets-of-infants-and-children.

windows of vulnerability — *in utero*, infancy, and adolescence — where children are particularly susceptible to the impacts of chemicals on their development. Chemical exposures can damage the developing brain at exposures less than those that affect adults.

The NAS recommended that EPA revamp and strengthen its regulation of pesticides 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 toxicity to infants and children and where data are incomplete on such toxicity or on children's exposures:

> 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.

NAS Report at 9-10.

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 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*.

This tenfold safety factor (called the FQPA safety factor or kids safety factor or 10X) is in addition to two safety factors that 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."[7]

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."[8] 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, increased to 1000X when the FQPA safety factor is added.[9]

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." NAS Report 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 <u>from aggregate exposure</u>" to a pesticide from all sources. 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)(D)(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 contacting pesticide residues in and around the home or other places where people can be exposed. *Id.* § 346a(b)(2)(A)(ii), (C)(i)(I), (D)(vi). The FQPA also requires EPA to assess and protect against unsafe risks posed by cumulative exposures to all pesticides that share a "common mechanism of toxicity," as is the case with pesticides in the organophosphate family. *See id*. § 346a(b)(2)(C)(i)(III)-(D)(v).

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[7] EPA, Office of the Science Advisor, An Examination of EPA Risk Assessment Principles and Practices (Mar. 2004) at 30, https://nepis.epa.gov/Exe/ZyPDF.cgi/100045MJ.PDF?Dockey=100045MJ.PDF.

[8] *Id.*

[9] 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).

II. EPA MUST ENSURE THERE ARE NO UNREASONABLE ADVERSE EFFECTS FROM USE OF PESTICIDES IN THE UNITED STATES.

While the FFDCA regulates whether pesticides residues are allowed on food, a different statute – the Federal Insecticide, Fungicide, and Rodenticide Act ("FIFRA") – regulates whether and, if so, how a pesticide may be used in the United States. EPA must register a pesticide for each allowable use, and to do so, EPA must find the pesticide use will not generally cause "unreasonable adverse effects on the environment." 7 U.S.C. §136a(c)(5); *see also id*. §136d(b) (providing for cancellation of registrations for uses that pose unreasonable adverse effects).

Under FIFRA, EPA may register or maintain a registration of a pesticide only if EPA determines that it will not have "unreasonable adverse effects on the environment." 7 U.S.C. § 136a(c)(5). An "unreasonable adverse effect[] on the environment" includes "any unreasonable risk to [people] or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." *Id*. § 136(bb).

The FQPA amended FIFRA's definition of "unreasonable adverse effects" to include "a human dietary risk from residues that result from a use of a pesticide in or on any food inconsistent with the standard under the FFDCA." 7 U.S.C. § 136(bb). If a pesticide fails to meet the FFDCA "reasonable certainty of no harm" standard, it cannot be used on food and registrations for such uses must be cancelled.

EPA must review pesticides every fifteen years, in a process called registration review, to ensure they meet both FFDCA and FIFRA legal standards. 7 U.S.C. § 136a(g)(1)(A)(i), (iii)(II). Registration review is designed to "ensure[] that older pesticides meet contemporary health and safety standards."[10] As passed in 1996, registration review had a hortatory 15-year goal. FQPA, Pub. L. 104-170, § 106(b), 110 Stat. 1489 (Aug. 3, 1996). A 2007 amendment replaced that goal with a hard 15-year deadline for registration review and set the deadline for review of the older pesticides as October 1, 2022. Pesticide Registration Improvement Renewal Act, Pub. L. No. 110-94, § 3, 121 Stat. 1000 (Oct. 9, 2007).

CHLORPYRIFOS HARMS CHILDREN'S BRAIN DEVELOPMENT AT LEVELS OF EXPOSURE THAT DO NOT CAUSE >10% RED BLOOD CELL ACETYLCHOLINESTERASE INHIBITION.

When EPA re-registered chlorpyrifos and the other organophosphates in 2006, it used acetylcholinesterase (AChE) inhibition as the regulatory endpoint. 80 Fed. Reg. 69079, 69086 (Nov. 6, 2015). AChE inhibition is the mechanism by which chlorpyrifos and other

[10] EPA, Evaluation of the U.S. EPA Pesticide Product Reregistration Process: Opportunities for Efficiency and Innovation at 1-1 (Mar. 2007), https://www.epa.gov/sites/production/files/2015-09/documents/eval-epa-pesticide-product-reregistration-process.pdf; 40 C.F.R. § 155.40.

organophosphate pesticides cause acute poisonings. *Id.*[11] When AChE, an enzyme that regulates nerve impulses, is inhibited, nerves are over-stimulated, causing people to experience symptoms such as headaches, nausea, dizziness, difficulty breathing, vomiting, diarrhea, muscle spasms, seizures, skin rashes, and at high levels of exposure, convulsions, respiratory paralysis, and even death. *Id.* EPA ignored the growing body of scientific evidence that chlorpyrifos harms children's brain development at exposures far below those associated with its regulatory endpoint designed to prevent acute poisonings. This scientific evidence, as described below, has become overwhelming in the 14 years since re-registration. Yet EPA has continued to ignore children's brain development and leave kids at serious risk of harm.

I. DOZENS OF EPIDEMIOLOGIC AND TOXICOLOGIC STUDIES DEMONSTRATE THAT PRENATAL CHLORPYRIFOS EXPOSURE HARMS CHILDREN'S BRAIN DEVELOPMENT.

Scientists describe the period of early development of the brain and nervous system as a "critical window" of susceptibility, when the fetus is undergoing rapid cell growth, migration, differentiation, nutrition uptake, and formation of the final organ structure. For this reason, the entire period of neurodevelopment – beginning in the womb and extending throughout childhood – is considered a critical window of increased vulnerability to toxic chemicals. Experts warn that exposure to harmful chemicals at any time during neurodevelopment, even at low levels or for only a short time, may lead to long-lasting physical, cognitive, and behavioral impairments.[12] The increased risk from pesticides is described in detail in the landmark 1993 NAS Report: "[s]tudies in animals suggest that the nature of an injury is determined by the stage of brain development at the time of exposure rather than by the relationship of the insult to the time of the birth event." NAS Report at 60. That is, it is not only the dose that makes the poison, but also the timing during critical windows of development. Additionally, it matters whether or not exposure occurs during prenatal development because the placenta is not an adequate barrier to passage of many toxic chemicals like chlorpyrifos from the mother to the fetus (Whyatt *et al.* (2005); Rauh *et al.* (2011)).

There are dozens of epidemiologic studies in pregnant women and children and toxicologic studies in laboratory animals demonstrating that prenatal exposure to chlorpyrifos leads to long-term and likely permanent adverse impacts on children's brain development and function. These include studies of chlorpyrifos and of other organophosphate pesticides. In a published systematic review, "all but one of the 27 studies evaluated showed some negative

[11] *See also* Hertz-Picciotto I et al. (2018). Organophosphate Exposures During Pregnancy and Child Neurodevelopment: Recommendations for Essential Policy Reforms. PLoS Medicine 15(10): 2, https://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.1002671.

[12] Heindel JJ et al., Developmental Origins of Health and Disease: Integrating Environmental Influences, 156 (10) Endocrinology 3416-21 (Oct. 2015), doi: 10.1210/EN.2015-1394, https://bit.ly/3b3ibNZ; Bennett D et al., Project TENDR: Targeting Environmental NeuroDevelopmental Risks The TENDR Consensus Statement, 124 (7) Environ Health Perspect. A118-22 (Jul. 1, 2016), doi: 10.1289/EHP358, https://bit.ly/2W1UgtR.

effects of [organophosphate] pesticides on neurobehavioral development."[13] In newborns, organophosphate pesticides are associated with abnormalities in primitive reflexes, suggesting harm to the development of the central nervous system (Engel, *et al*. (2007); Young, *et al*. (2005)). In children, they are associated with reduction in motor function (Eskenazi, *et al*. (2007); Rauh, *et al*. (2006); Grandjean, *et al*. (2006); Handal, *et al*. (2008); Harai, *et al*. (2010); Rauh, *et al*. (2015)), decreases in working and visual memory, processing speed, verbal comprehension, perceptual reasoning, and full scale IQ (Bouchard, *et al*. (2011); Engel, *et al*. (2011); Rauh, *et al*. (2011); Handal, *et al*. (2008)), and increases in problems including ADHD, pervasive developmental disorder, and behaviors typical of autism spectrum disorders (Rauh, *et al*. (2006); Marks, *et al*. (2010); Furlong *et al*. (2014)). These studies have found that certain subpopulations have greater susceptibility, including children of farmworkers (Castorina, *et al*. (2010); Engel, *et al*. (2015)) and those who have reduced capacity to detoxify the organophosphates (Engel, *et al*. (2015)).

EPA has focused on studies from prospective birth cohorts based at Columbia University, Mount Sinai School of Medicine, and the University of California-Berkeley. 80 Fed. Reg. at 69,091. For these studies, investigators enrolled pregnant women, measured exposure to one or more organophosphate pesticides during pregnancy or at delivery, and assessed neurodevelopment in their children at multiple time points after birth. *Id*.

The Columbia Center on Children's Environmental Health found that chlorpyrifos levels in African American and Dominican pregnant women in New York City were associated with adverse neurodevelopmental effects in their children. The study began before and continued after the residential chlorpyrifos ban. The mothers of children born after the ban had dramatically lower chlorpyrifos levels than mothers exposed before the ban. Peer-reviewed scientific articles document that, at age three, the highly exposed children had statistically significant delays in motor and mental development, and attention and behavior problems (Rauh *et al.* (2006)). At age seven, they experienced reduced IQ and loss of working memory (Rauh *et al.* (2011)). At age 11, the children had more arm tremors and reduced fine motor control that affected the children's ability to draw shapes (Rauh *et al.* (2015)). Subsequent testing using magnetic resonance imaging ("MRI") revealed physical brain abnormalities in an area of the brains of highly exposed children linked to learning, cognition, and social behaviors (Rauh *et al.* (2012)).

The Columbia study's findings are consistent with those of two other mother-child pair studies conducted by University of California-Berkeley and Mount Sinai School of Medicine, which found that prenatal exposures to organophosphate pesticides were associated with neurodevelopmental harm to children. The UC Berkeley study followed a cohort of children born to farmworkers in Salinas Valley, California, and found reduced IQ, verbal comprehension, perceptual reasoning, and working memory (Bouchard *et al.* (2011)). The Mount Sinai study observed a New York City Hispanic population and found similar effects in the exposed children (Engel *et al.* (2011)).

[13] Muñoz-Quezada MT et al. (2013). Neurodevelopmental effects in children associated with exposure to organophosphate pesticides: A systematic review. *NeuroToxicology* 39:158.

EPA reported, "[A]cross these three children's environmental health studies, authors consistently identified associations with neurodevelopmental outcomes in relation to [organophosphate] exposure." 80 Fed. Reg. at 69,092. EPA has noted many of the outcomes listed above, including abnormal reflexes in newborns, mental and psychomotor developmental delays and attention and behavior problems in early childhood, and impaired cognition in middle childhood. *Id*. at 69,091-93.

The epidemiologic evidence is consistent with data from toxicologic studies that evaluated the neurodevelopmental effects of pre- and/or post-natal exposure to organophosphate pesticides in experimental animals. In 2015, EPA found "a considerable and still-growing body of literature on the effects of chlorpyrifos on the developing brain of laboratory animals." *Id*. at 69,090. The consistent results across epidemiologic and toxicologic studies are notable because the strengths of epidemiologic studies tend to balance the limitations of toxicologic studies, and *vice versa*.[14] For example, unlike toxicologic studies in experimental animals, epidemiologic studies evaluate exposures in the populations of interest — in this case, pregnant women and children. However, unlike epidemiologic studies in human beings, toxicologic studies can assign exposure at random to ensure that any differences between exposed and control animals are due to chlorpyrifos. These complementary lines of evidence show that chlorpyrifos harms children's brain development.

II. EPA AND THE FIFRA SCIENTIFIC ADVISORY PANEL REVIEWS AGREE THAT CHLORPYRIFOS HARMS CHILDREN'S BRAIN DEVELOPMENT.

EPA has conducted weight-of-evidence analyses that integrate multiple lines of evidence from both epidemiology and toxicology.[15] EPA has concluded again and again that prenatal exposure to chlorpyrifos harms children's brain development. Indeed, the agency has stated that, if anything, the studies *underestimate* the harm to children. In 2012, EPA wrote:

> Overall, these are well performed studies which are shielded from several major sources of bias in the interpretation of results due to the strong design, conduct and analyses utilized in these investigations. While factors are present across these studies which may have led to either false positive or negative associations, it is notable that positive associations were observed as EPA believes the possibility of under-estimation of effect size is more likely than factors that would lead to over-estimation of effect size.[16]

[14] EPA, Preamble to the Integrated Science Assessments at 14 (2015), http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=526136.
[15] EPA, Literature Review on Neurodevelopment Effects & FQPA Safety Factor Determination for the Organophosphate Pesticides (Sept. 15, 2015) at 76, https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0062-0055.
[16] EPA, Draft Issue Paper: Scientific Issues Concerning Health Effects of Chlorpyrifos, for Meeting of FIFRA Scientific Advisory Panel (April 2012) ("2012 FIFRA SAP Issue Paper") at 71, https://www.regulations.gov/document?D=EPA-HQ-OPP-2012-0040-0002.

And in 2015, the agency wrote, "EPA believes these are strong studies which support a conclusion that [organophosphates] likely played a role in these outcomes." 80 Fed. Reg. at 69,091. The agency again reviewed the threats to inference that can arise in epidemiologic research, but it "believes that random or systematic errors in the design, conduct, or analysis of these studies were unlikely to fully explain observed positive associations between in utero [organophosphate] exposure and adverse neurodevelopmental effects observed at birth and through childhood (age 7 years)." *Id*.

Sound epidemiologic studies like these can support EPA risk assessments. EPA's *Guidelines for Developmental Toxicity Risk Assessment* state, "Good epidemiologic studies provide the most relevant information for assessing human risk."[17] As noted in the Draft Framework for Incorporating Human Epidemiologic & Incident Data in Health Risk Assessment, epidemiologic studies provide extremely valuable information to inform risk assessments:

> Specifically, these types of human information provide insight into the effects caused by actual chemical exposures in humans and thus can contribute to … hazard/risk characterization. In addition, epidemiologic and human incident data can guide additional analyses or data generations (e.g., dose and endpoint selection for use in in vitro and targeted in vivo experimental studies), identify potentially susceptible populations, identify new health effects or confirm the existing toxicological observations.[18]

In addition, the FIFRA Scientific Advisory Panel (SAP) has conducted three reviews of EPA's conclusions about the neurodevelopmental toxicity of organophosphate pesticides and chlorpyrifos in particular and affirmed the agency's conclusions. In 2008, the SAP concluded that maternal exposure to chlorpyrifos is associated with adverse neurodevelopmental outcomes in children.[19] In 2012, the SAP reviewed epidemiologic and toxicologic studies, and wrote, "In summary, these lines of evidence suggest that chlorpyrifos can affect neurodevelopment[.]"[20] In 2016, the SAP "agree[d] that both epidemiology and toxicology studies suggest there is evidence for adverse health outcomes associated with chlorpyrifos exposures below levels that result in

[17] EPA, Guidelines for Developmental Toxicity Risk Assessment 23 (1991), https://www.epa.gov/sites/production/files/2014-11/documents/dev_tox.pdf.
[18] EPA OPP, Draft Framework for Incorporating Human Epidemiologic & Incident Data in Health Risk Assessment (Jan. 7, 2010) at 7, https://www.regulations.gov/document?D=EPA-HQ-OPP-2009-0851-0004.
[19] SAP Minutes of September 16-18, 2008 Meeting on Agency's Evaluation of Toxicity Profile of Chlorpyrifos ("2008 SAP Report") at 13, https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0274-0064.
[20] EPA, Transmittal of Meeting Minutes of the FIFRA Scientific Advisory Panel Meeting held April 10-12, 2012 on "Chlorpyrifos Health Effects" (2012) ("2012 SAP Report") at 53, https://www.epa.gov/sites/production/files/2015-06/documents/041012minutes.pdf.

10% RBC AChE inhibition."[21] In 2016, the agency "agree[d] with the 2016 [SAP] (and previous [SAPs]) that there is a potential for neurodevelopmental effects associated with chlorpyrifos exposure." 81 Fed. Reg. 81,049, 81,050 (Nov. 17, 2016). In short, dozens of studies and years of review by EPA and the SAP have established that prenatal exposure to chlorpyrifos harms children's brain development.

III. CONTINUED RELIANCE ON >10% RED BLOOD CELL ACETYLCHOLINESTERASE INHIBITION FOR RISK ASSESSMENTS OF CHLORPYRIFOS IS UNDER-PROTECTIVE OF CHILDREN'S HEALTH.

EPA's continued reliance on greater than 10 percent (>10%) red blood cell acetylcholinesterase ("RBC AChE") inhibition as the critical effect for chlorpyrifos risk assessment is scientifically inappropriate because harm to children's brain development occurs at much lower levels of exposure. This is demonstrated by EPA dose-reconstruction and dose-response analyses as well as the peer-reviewed literature. By relying on 10% RBC AChE inhibition, the agency is deriving population adjusted doses or reference doses — effectively, acceptable levels of exposure — that are many times larger than true safe levels. This leads to false conclusions that dangerous levels of exposure are safe.

The essence of a risk assessment is a comparison of an acceptable level of exposure — known as a population adjusted dose or reference dose — with the level of exposure that is expected to occur in a population of interest. If the expected level of exposure falls below the acceptable level, EPA concludes there is reasonable certainty of no harm. However, the agency errs if the acceptable level is too high, which can occur when it is not based on the most sensitive effect, *i.e.*, the effect that occurs at the lowest level of exposure. EPA's *Guidelines for Developmental Toxicity Risk Assessment* state that the most sensitive effect should be used.[22] EPA, however, has continued to utilize 10% RBC AChE inhibition for organophosphate pesticides despite compelling evidence and its own conclusions that harm to children's brains occurs at lower levels of exposure.

A. Peer-reviewed Literature.

The evidence that harm to children's brain development is the most sensitive effect includes epidemiologic and animal studies in which both types of effect were measured and, while neurodevelopmental effects were observed, AChE inhibition was <10% or absent. In CHAMACOS, the cohort established at University of California, Berkeley, for example, EPA notes that investigators "measured AChE activity and showed that no inhibition in AChE activity

[21] EPA, Transmittal of Meeting Minutes of the April 19-21, 2016 FIFRA SAP Meeting Held to Consider and Review Scientific Issues Associated with "Chlorpyrifos: Analysis of Biomonitoring Data" (2016) ("2016 SAP Report") at 52-53, https://www.epa.gov/sites/production/files/2016-07/documents/chlorpyrifos_sap_april_2016_final_minutes.pdf.

[22] EPA, Guidelines for Developmental Toxicity Risk Assessment at 42 (1991), https://www.epa.gov/sites/production/files/2014-11/documents/dev_tox.pdf.

[was] observed." 2016 HHRA at 13. In a literature review, the primary investigators of the three major birth cohorts concurred with EPA: "Generally, levels of exposure in these studies are too low to induce measurable depression of cholinesterase in adults."[23] Furthermore, the review notes, "[E]ffects on cognition, motor activity, and social behaviors were repeatedly demonstrated in rodents dosed in early life with concentrations of [organophosphates] eliciting little to no inhibition of AChE in the brain."[24]

B. EPA's 2014 Human Health Risk Assessment.

EPA itself has concluded that it is "unlikely" that pregnant women exposed to chlorpyrifos in epidemiologic studies experienced RBC AChE inhibition. 2014 HHRA at 41. In 2014, the agency conducted a dose-reconstruction analysis "to help characterize the extent to which participants in the [Columbia University] cohort may or may not have experienced RBC AChE inhibition." *Id.* at 40. The analysis concluded RBC AChE inhibition was just 0.0012% for women applying chlorpyrifos and just 0.45% for women exposed after the pesticide was applied. *Id*. at 41. EPA wrote, "The Agency's dose reconstruction analysis supports a qualitative conclusion that it is unlikely that >10% RBC AChE inhibition would have occurred in the [Columbia University cohort] participants." *Id*. EPA retained the FQPA safety factor of 10X to account for uncertainty over neurodevelopmental toxicity. *Id*. at 49. However, as explained below, simply retaining this safety factor is woefully inadequate to protect children.

The SAP reviewed this issue repeatedly and agreed with EPA's conclusions. In 2012, the SAP noted "multiple lines of evidence suggesting that adverse neurodevelopmental effects may be attributed to chlorpyrifos doses lower than those that elicit a 10% inhibition of AChE."[25] In 2016, it stated: "The Panel agrees that both epidemiology and toxicology studies suggest there is evidence for adverse health outcomes associated with chlorpyrifos exposures below levels that result in 10% RBC AChE inhibition (i.e., toxicity at lower doses)."[26]

1. EPA's 2016 Human Health Risk Assessment

In 2016, EPA's risk assessment for chlorpyrifos found that acceptable levels of exposure to organophosphate pesticides based on harm to children's brain development are dramatically lower than acceptable levels based on >10% RBC AChE inhibition, which was used in EPA's 2014 risk assessment for chlorpyrifos. For the 2014 risk assessment, the agency derived population adjusted doses — the acceptable levels of exposure — for steady-state exposure to chlorpyrifos residues on food of 0.78 to 2.6 mcg/kg/day (Table 1). 2014 HHRA at 76. In 2016, when EPA assessed risks from chlorpyrifos based on neurodevelopmental toxicity, the population adjusted doses were 0.0012 to 0.002 mcg/kg/day — *three to four orders of magnitude*

[23] Hertz-Picciotto et al. (2018) at 2.
[24] Hertz-Picciotto et al. (2018) at 2.
[25] 2012 SAP Report at 50.
[26] 2016 SAP Report at 18.

lower than when the acceptable level was based on >10% RBC AChE inhibition (Table 1). 2016 HHRA at 23.[27]

Table 1: Steady-state Population Adjusted Doses (mcg/kg/day) for Food Exposure to Chlorpyrifos

	AChE Inhibition (2014/2020)	Neurodevelopment (2016)
Infants	2.6	0.002
Children	2.5	0.0017
Youths	2.2	0.0012
Adults	0.78	0.0012

The 2016 population adjusted doses for neurodevelopmental toxicity presented in the table may still be too high by an order of magnitude. EPA based these metrics on a level of exposure believed to have harmed children's brain development. The agency therefore used a lowest observed adverse effect level (LOAEL) as a point of departure. 2016 HHRA at 21-22. The use of a LOAEL instead of a no observed adverse effect level (NOAEL) requires an additional uncertainty factor of 10X,[28] but EPA did not include one. *Id*. This means that the acceptable level based on neurodevelopmental toxicity may be *four to five orders of magnitude* lower than the acceptable level based on AChE inhibition.

The stark contrast in population adjusted doses, or acceptable levels, for 10% RBC AChE inhibition and neurodevelopmental toxicity in EPA's risk assessments indicate that continuing to base risk assessments for chlorpyrifos on the former endpoint is under-protective — even when the FQPA safety factor of 10X is retained. If the point of departure and thus the population adjusted dose for the neurodevelopmental toxicity of chlorpyrifos could be >1,000X lower than what EPA has derived for AChE inhibition, relying only on the FQPA safety factor of 10X to protect children from neurodevelopmental harm is plainly inadequate.

EPA has attempted to undermine the results of its 2016 risk assessment by saying the population adjusted doses derived for that risk assessment and presented in the table above were based on results from an epidemiologic study conducted at Columbia University, and that the agency cannot use these results because it does not have access to raw study data. 2020 HHRA

[27] In its 2020 chlorpyrifos human health risk assessment and in arguments made to the Ninth Circuit Court of Appeals, EPA has erroneously stated that the 2016 risk assessment used cord blood from the Columbia study and was the subject of criticism by the 2016 SAP. This is incorrect. A spring 2016 EPA white paper proposed using cord blood from the Columbia study and a 2% decline in working memory to establish a regulatory endpoint. A majority of the 2016 SAP disfavored using a single data point from a single study to establish the regulatory endpoint and instead urged EPA to reconstruct the exposures based on pest control methods used in the pregnant women's homes. EPA heeded this advice in the risk assessment it produced in the fall of 2016.

[28] EPA, Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment at 9 (2002), https://www.epa.gov/sites/production/files/2015-07/documents/determ.pdf; EPA, A Review of the Reference Dose and Reference Concentration Processes at 4-44 (2002), https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf.

at 5, 89-90. However, EPA did not use any results or data from that study in a quantitative manner. Rather, based on feedback from the SAP, the agency derived the points of departure underlying the population adjusted doses three steps. 2016 HHRA at 13-14. First, EPA selected an exposure scenario that was typical of how pregnant women in the Columbia study were exposed, specifically indoor use of chlorpyrifos in cracks and crevices. *Id*. at 14. Second, EPA used a physiologic based pharmacokinetic (PBPK) model developed by the chlorpyrifos registrant to predict the concentration of chlorpyrifos in umbilical cord blood (a measure of fetal exposure) that would result from this use of chlorpyrifos. *Id*. Finally, EPA used the same PBPK model to estimate the levels of exposure by other pathways (*e.g.*, food crops, golf courses) that would result in the same concentration of chlorpyrifos in cord blood. *Id*. These levels of exposure were the points of departure utilized in the 2016 Human Health Risk Assessment. *Id*. Access to raw study data from Columbia is irrelevant to EPA's conclusions.

2. California's 2018 Toxic Air Contaminant Evaluation

The California Department of Pesticide Regulation ("CDPR") also concluded that prenatal exposure to chlorpyrifos can elicit neurodevelopmental toxicity at levels of exposure that do not result in >10% RBC AChE inhibition. In 2018, when evaluating whether chlorpyrifos is a toxic air contaminant under California law, CDPR noted, "Recent in vivo animal studies provide evidence of neurotoxicity to developing organisms at chlorpyrifos doses below those causing cholinesterase inhibition."[29] The agency based its evaluation on developmental neurotoxicity rather than AChE inhibition: "These studies, along with epidemiological studies, are the impetus for CDPR considering developmental neurotoxicity as the critical endpoint for chlorpyrifos."[30]

CDPR considered five toxicologic studies reporting neurodevelopmental effects at low doses that did not elicit meaningful AChE inhibition.[31] It derived reference doses from them, found chlorpyrifos unsafe, and initiated cancellation proceedings, which phased out of 99% of chlorpyrifos use by the end of 2020.[32] Table 2 compares CDPR's references doses for neurodevelopmental toxicity from acute oral exposure to EPA's population adjusted doses for AChE inhibition from acute dietary exposure. CDPR's acceptable levels are 47-150X lower than EPA's, which further suggests that EPA's approach is under-protective of children's health.[33]

[29] California Department of Pesticide Regulation ("CDPR"), Final Toxic Air Contaminant Evaluation of Chlorpyrifos at 9-10 (2018), https://www.cdpr.ca.gov/docs/whs/pdf/chlorpyrifos_final_tac.pdf.
[30] *Id*.
[31] EPA, Chlorpyrifos: Review of 5 Open Literature Studies Investigating Potential Developmental Neurotoxicity Following Early Lifestage Exposure (2020).
[32] CDPR, Final Toxic Air Contaminant Evaluation of Chlorpyrifos at 9-10; *see also* CDPR, Agreement Reached to End Sale of Chlorpyrifos by February 2020 (Oct. 9, 2019), https://www.cdpr.ca.gov/docs/pressrls/2019/100919.htm.
[33] CDPR, Final Toxic Air Contaminant Evaluation of Chlorpyrifos at 82; 2014 HHRA at 75; 2020 HHRA at 34-35.

Table 2: Acute Reference Doses and Population Adjusted Doses (mcg/kg/day) for Chlorpyrifos

	CDPR (2018)	EPA (2014/2020)
Infants	0.1	15
Children	0.1	14
Youths	0.1	13
Adults	0.1	4.7

In 2019, when EPA denied objections to its decision not to revoke all tolerances and cancel all registrations for chlorpyrifos, the agency said its decision to delay would allow it to consider these five studies. 84 Fed. Reg. at 35,566. Yet, inexplicably, EPA does not even try to use these toxicologic studies to identify a population adjusted dose that would protect children's brain development. EPA has discussed the studies utilized by CDPR in a memorandum accompanying the 2020 Human Health Risk Assessment.[34] The agency states that while three of five studies did not meet its quality criteria, two that did reported effects on motor activity in mice and anxious behavior in rats.[35] However, the two studies the agency believes meet it quality criteria play no role whatsoever in EPA's derivation of population adjusted doses and it offers no reason for failing to incorporate them.

The peer-reviewed literature, as well as dose-reconstruction and dose-response analyses for chlorpyrifos, demonstrate what EPA has admitted: "[T]he use of 10% RBC AChE inhibition for deriving [points of departure, the bases for population adjusted doses] may not provide a sufficiently protective human health risk assessment." 2016 HHRA at 13. If the agency bases its risk assessments for chlorpyrifos on >10% RBC AChE inhibition, it could conclude that an exposure is safe even when it exceeds a true safe level by a thousand-fold or more. EPA should base its chlorpyrifos risk assessment on neurodevelopmental toxicity.

THE 2020 HUMAN HEALTH RISK ASSESSMENT AND PID DO NOT USE THE BEST SCIENCE AND FAIL TO PROTECT CHILDREN.

I. THE 2020 HUMAN HEALTH RISK ASSESSMENT AND PID PROPOSALS ARE BASED ON A REGULATORY ENDPOINT THAT IS GROSSLY UNDERPROTECTIVE OF CHILDREN

The 2020 risk assessment reiterates EPA's findings that chlorpyrifos harms children's brains at exposures below 10% cholinesterase inhibition. It cites the Columbia and other cohort studies, which it calls high-quality, well-executed studies, as support for this finding, along with laboratory studies and the consistent findings of the SAP from 2008, 2012, and 2016.

The 2020 assessment reduces protection for children by abandoning EPA's prior attempt to find a safe exposure level that would prevent damage to children's brains. It reverts to the

[34] EPA, Chlorpyrifos: Review of 5 Open Literature Studies Investigating Potential Developmental Neurotoxicity Following Early Lifestage Exposure (2020).
[35] *Id*. at 4, 11.

framework of the 2014 assessment, which used 10% cholinesterase inhibition as measured in red-blood cells as the regulatory endpoint. The 2020 assessment is not predicated on a level of exposure that is safe because it does not use the most sensitive endpoint for neurodevelopmental harm to children and it shrinks safety factors, rather than expand them to be protective. Using this endpoint, EPA indicated that 1% of children born each year would experience 10% or greater cholinesterase inhibition. 2020 HHRA at 28. One percent is approximately 38,000 babies. Given that chlorpyrifos causes neurodevelopmental harm at far lower exposures, EPA is putting a huge number of children at risk.

As to the endpoint, EPA deemed this regulatory endpoint unsafe in its 2014 risk assessment and 2015 proposed tolerance revocation. EPA could not find a reasonable certainty of no harm to children using 10% cholinesterase inhibition because of the unbroken findings by EPA and the SAP that chlorpyrifos damages children's brains at exposures far below 10% cholinesterase inhibition. EPA never disavows these findings and even acknowledges in the 2020 assessment that scientific studies document neurodevelopmental harm from chlorpyrifos at exposures below those that cause 10% cholinesterase inhibition. 2020 HHRA at 85-86, 88; *see also* 84 Fed. Reg. at 35,563-64 (order denying objections). By continuing to use 10% cholinesterase inhibition, EPA is violating its own policies to use the most sensitive endpoint in risk assessments. Because brain-based disorders, such as learning disabilities, IQ deficits, autism, and ADHD, occur at lower exposures, 10% cholinesterase inhibition is not a no adverse effect level, but instead is a low adverse effect level, yet EPA did not add an additional tenfold safety factor for use of a LOAEL as its policies require.

Efforts to find an exposure level that would not damage children's brains have led to a regulatory endpoint far lower than 10% cholinesterase inhibition. For example, EPA's 2016 risk assessment identified an exposure that will not damage children's brains that is 650X lower than what is used in the 2020 risk assessment. Using this endpoint, EPA found all exposures to chlorpyrifos unsafe. The 2020 assessment contends that scientific uncertainty prevents it from using this risk assessment, which is addressed below, but it then reverts to an acute poisoning endpoint that admittedly does not protect children's brains.

Similarly, the 2020 risk assessment acknowledges that the California risk assessment identified a safe exposure level for neurodevelopmental harm to children based on animal studies. The safe exposure level for infants from the California risk assessment is 150X lower than what the 2020 risk assessment is using. The California risk assessment led to cancellation proceedings and to the end of 99% of the uses of chlorpyrifos in California by the end of 2020. EPA reviewed those studies and found two of them adequate to be used either qualitatively or quantitatively in risk assessment. Inexplicably, the 2020 risk assessment doesn't even try to use these studies to identify an exposure level that would protect children's brains.

Alternatively, EPA could have used additional safety/uncertainty factors to try to account for the scientific uncertainties and fact that damage to children's brains occurs from exposures below those that cause 10% cholinesterase inhibition. It made no attempt to do so. And if it had retained the full suite of default safety factors totaling 1000X and added another 10X safety

factor to reflect the fact that 10% cholinesterase inhibition is a low adverse effect level, it would have a total uncertainty factor of 10,000X. EPA guidance provides that the agency should not attempt to estimate a reference dose if the total uncertainty factor would be 10,000X or more, because such large uncertainty factor means the data are too uncertain to provide a valid estimation of risk.[36]

Instead, it did the opposite. The 2020 assessment reduces the default safety factors based on a model developed by Dow that uses human data and tries to pinpoint the precise exposures that cause 10% cholinesterase inhibition in various human populations. On this basis, EPA eliminated the interspecies safety factor altogether and shrunk the safety factor that accounts for human variability. 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 because the Dow model lacked sufficient data to be used for pregnant women. EPA cannot shrink safety factors based on the Dow model because that model does not use the most sensitive endpoint. 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. In addition, as described more fully in our comments on the 2014 risk assessment, 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, and it improperly relies on human dosing studies that EPA's advisors have criticized on both scientific and ethical grounds and that EPA admits have not been fully reviewed in accordance with its recently strengthened regulations. 2015 Farmworker Comments at 28-42.[37]

The 2014 risk assessment similarly reduced safety factors, but EPA rightfully found in its 2015 proposed tolerance revocation that it could not make a reasonable certainty of no harm finding based on the 2014 assessment. Likewise, EPA cannot make a reasonable certainty of no harm finding based on the 2020 risk assessment because chlorpyrifos impairs children's brain development at exposures far lower than what the 2020 risk assessment would allow.

Further illustrating its callous disregard for children, EPA even floats the possibility of further eliminating the FQPA tenfold safety factor based on new testing methodologies for developmental neurotoxicity. After EPA released the 2020 assessment, however, the SAP issued a scathing review of EPA's proposal to shrink safety factors based on these new methods, referred to as "New Approach Methodologies," or "NAMs." Relying on NAMs to allow

[36] EPA, A Review of the Reference Dose and Reference Concentration Processes at 4-41 (2002), https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf ("The Technical Panel recommends...avoiding the derivation of a reference value that involves application of the full 10-fold UF in four or more areas of extrapolation [*i.e.*, 10,000].").

[37] Farmworker and Conservation Comments on Chlorpyrifos Revised Human Health Risk Assessment (Apr. 30, 2015) ("2015 Farmworker Comments"), https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0850-0848.

increased uses and risks from chlorpyrifos would be underprotective of children, in part, because the testing failed to adequately capture the full range of physiological conditions that contribute to neurodevelopmental harms. These new methods thus cannot be used to replace or downgrade the overwhelming scientific evidence of low-exposure neurodevelopmental harm from chlorpyrifos.

II. SCIENTIFIC UNCERTAINTY DOES NOT ALLOW EPA TO RETAIN TOLERANCES.

Congress established a statutory standard that precludes denying protection, particularly to children, solely because there is some scientific uncertainty as to the full extent of the harm. Under the FQPA standard, uncertainty compels revocation of tolerances since "safe" means that EPA "has determined that there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue," 21 U.S.C. § 346a(b)(2)(A)(ii), and EPA can "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) (emphasis added). Congress also specifically directed EPA to assume that children face a ten times greater risk than adults unless it has reliable data showing a different margin will be safe for infants and children. 21 U.S.C. § 346a(b)(2)(C); *Nw. Coal. for Alts. to Pesticides*, 544 F.3d 1043, 1046 (9th Cir. 2008). If uncertainty prevents EPA from making an affirmative safety finding, EPA must revoke the tolerances.

The top line message of the 2020 assessment (repeated no fewer than nine times) and in the PID is: "The science addressing neurodevelopmental effects remains unresolved." Under FQPA standard, the scientific uncertainty prevents EPA from finding chlorpyrifos safe.

In claiming uncertainty, EPA ignores how much progress has been made in assessing the large body of scientific evidence and substantiating the linkage between chlorpyrifos and damage to children's developing brains at exposures far lower than EPA's regulatory endpoint. The administrative record demonstrates how, over the past 12 years, EPA has obtained a series of peer reviews of studies demonstrating a relationship between chlorpyrifos exposure and damage to children's brains at extremely low exposure levels. EPA and the SAP have determined that the Columbia and other epidemiology studies are scientifically sound and robust and that they correlate harm to children's brains to chlorpyrifos exposures at levels far below what EPA currently allows. EPA and independent reviews have reduced uncertainties to the point where EPA has concluded that the human population studies more likely under-estimate, rather than over-estimate, the association between chlorpyrifos and children's brain development. *See, e.g.*, 2012 FIFRA SAP Issue Paper at 71.

Since at least 2008, EPA and multiple SAPs have found that the Columbia and other epidemiology studies are sound and of high quality. While EPA notes that the mechanism by which chlorpyrifos damages children's brains is uncertain, the link between this pesticide and neurodevelopmental harm is well-established. Under EPA policy, the agency cannot ignore evidence of harm simply because it has not yet determined the mode of action. *See* 2014 HHRA at 48-49; *see also Am. Trucking Assocs. v. EPA*, 175 F.3d 1027, 1055-56 (D.C. Cir. 1999) (EPA

is not required to identify the biological mechanism by which a pollutant affects public health), *opinion modified on reh'g*, 195 F.3d 4 (D.C. Cir 1999), *and aff'd in part, rev'd in part on other grounds sub nom., Whitman v. Am. Trucking Assocs.*, 531 U.S. 457 (2001). EPA also points to uncertainties about the precise exposure level that damages children's brains. Even if there is uncertainty in identifying a safe exposure level, EPA and its SAP have long found that neurodevelopmental harm is correlated with exposures far below the current endpoint of 10% cholinesterase inhibition. Moreover, under the FQPA, any uncertainty should weigh in favor of affording children more protection, not less.

III. EPA CANNOT DOWNPLAY THE RELATIONSHIP BETWEEN CHLORPYRIFOS AND DAMAGE TO CHILDREN'S BRAINS BY CLAIMING THE RAW DATA FROM THE LEADING EPIDEMIOLOGY STUDY NEED TO BE PUBLIC.

EPA has made much of the supposed "uncertainty" of the science because EPA has not obtained the raw data from the Columbia University epidemiology study that repeatedly linked chlorpyrifos exposure and damage to children's brains. This purported need for the raw data ignores EPA policies, the harm to personal privacy that public release of the data would cause, the outstanding offer to make the data available to EPA in a secure location, and a misreading of the 2016 SAP review and risk assessment. Indeed, EPA called the Columbia and related epidemiology studies "strong" and "well-performed" studies and of "high quality." 2014 HHRA at 33; 2012 FIFRA SAP Issue Paper at 71, 99-100. Moreover, EPA acknowledges that it "does not have a specific reason to believe that CCCEH have inappropriately handled the data or statistical analysis." 2020 HHRA at 89. EPA cannot dismiss the findings of the Columbia study simply because the raw data is not publicly available.

First, EPA policies allow it to use epidemiology studies and articles in the open literature in EPA health assessments where the agency cannot access the raw data due, for example, to compelling interests in safeguarding personal privacy; it can employ other checks, such as peer review, to ensure the robustness of the analytical results.[38] EPA adhered to its policies and utilized a series of reviews to ensure the validity and robustness of the Columbia and other studies. Not only were the articles from the Columbia and other epidemiology studies peer reviewed before publication in academic journals, but EPA also convened the SAP. The SAP called the Columbia study in particular "sound," the "best available," "carefully designed," and "well-executed." 2008 SAP Report at 12; 2012 SAP Report at 22. Beginning in 2008, the SAP concluded that learning disabilities and other damage to children's brains occurred at chlorpyrifos exposures far below the levels that EPA's tolerances allow. The successive EPA and SAP reviews reduced uncertainties to the point that EPA found that any errors would be

[38] EPA, Guidance for Considering and Using Open Literature Toxicity Studies to Support Human Health Risk Assessment at 9-10 (2012), https://www.epa.gov/sites/production/files/2015-07/documents/lit-studies.pdf; EPA, Plan to Increase Access to Results of EPA-Funded Scientific Research at 11 (2016), https://www.epa.gov/sites/production/files/2016-12/documents/epascientificresearchtransperancyplan.pdf; EPA, Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the EPA at 21 (2002), https://www.epa.gov/sites/production/files/2019 08/documents/epa-info-qualityguidelines_1.pdf.

more likely to underestimate, rather than overestimate, the association between chlorpyrifos exposures and harm to children's brains. 2012 FIFRA SAP Issue Paper at 71, 75, 99-100.

Second, EPA has acknowledged that releasing the raw data from the Columbia study would compromise the privacy of the study participants. From the 2020 HHRA (at 91-92):

> Following the June 2018 conference call with CCCEH, EPA contacted the CDC in July 2018 to discuss HIPAA and data de-identification issues as it relates to the CCCEH. The CDC representative noted that even after taking out personally identifiable information (PII) from the dataset, the data that remain can still pose identification issues because of the possibility of linking it with information currently in the public domain. The CDC representative further noted there are some datasets that cannot be deidentified given the nature of the data and specified that geographic location is one of the variables that makes something highly identifiable. In the case of CCCEH, the study participants live within a small geographical range with New York City. The CDC representative noted that for those cases, there is the possibility of allowing the data to be viewed in a secure data center.

Third, Columbia has offered EPA the opportunity to review the raw data in a secure setting, but EPA has not availed itself of that opportunity.[39] Indeed, it has made no attempts to obtain or view the raw data since July 2018. 2020 HHRA at 92. Instead, EPA has tried to minimize robust peer-reviewed studies that correlate low-level chlorpyrifos exposure with harm to children's brains. This not only violates the law and EPA's own policies, it also led to a risk assessment and proposed registration review decision that vastly understate the risks posed by chlorpyrifos, particularly to children.

Nothing in the law or EPA's policies required EPA to obtain raw data.[40] The raw data are utterly unnecessary to validate the peer-reviewed published studies in light of the peer

[39] Letter from Linda P. Fried, Dean, Mailman School of Public Health, Columbia University, to Jack E. Housenger, Director, Office of Pesticide Programs, EPA (May 18, 2016), https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0850-0928.

[40] EPA's recently vacated Censoring Science Rule, 86 Fed. Reg. 469 (Jan. 6, 2021), cannot provide a basis for discounting the Columbia study. *See Envtl. Def. Fund v. EPA*, No. 4:21-CV-03-BMM, ECF No. 38 Order Granting Vacatur and Remand (Feb. 1, 2021); *see also Envtl. Def. Fund v. EPA*, No. 4:21-CV-03-BMM, 2021 WL 270246 (D. Mont. Jan. 27, 2021). Even if the Censoring Science Rule were lawful and in effect, it would not apply here as the 2020 HHRA and PID preceded the Censoring Science Rule's effective date. *See* 40 C.F.R. § 30.3(a) ("The provisions of this part apply to significant regulatory actions for which a proposed rule was published in the Federal Register after January 6, 2021 and influential scientific information submitted for peer review after January 6, 2021.").

reviews preceding their publication, the validation EPA has already conducted in accordance with its policies, and the fact that EPA has done nothing to view the data in a secure location over the many years Columbia has indicated it could do so. EPA appears to be pushing for the raw data so industry product defense firms can pick the studies apart, try to cast doubt on the findings, and continue to delay the long-overdue ban on chlorpyrifos. *See* 2020 HHRA at 89-90.

Finally, in its rationale for discounting the Columbia study, EPA suggests there might be possible selection bias and weaknesses in the statistical analysis in Rauh *et al.* (2011), which found a reduction in working memory. 2020 HHRA at 89-90. EPA used this study in its spring 2016 white paper, which correlated cord blood measurements from the Columbia study with a 2% loss of working memory at age seven.[41] Some members of the 2016 SAP questioned this approach because it used a single measurement from a single study to derive the regulatory endpoint.

Importantly, the fall 2016 risk assessment did not use that approach or rely specifically on Rauh *et al.* (2011) to derive the regulatory endpoint. Instead, it followed the SAP's recommendation and reconstructed exposures based on the pest control methods used in the public housing where the Columbia study participants lived. The 2016 HHRA explained that its approach to dose-response assessment "does not directly rely on quantitative measures of chlorpyrifos in cord blood obtained from the CCCEH, which was the source of uncertainty identified by the 2016 SAP" and, moreover, "does not directly rely on quantitative measures of chlorpyrifos in cord blood obtained from the CCCEH, and thus, **the lack of access to the raw data from the CCCEH is less of an uncertainty**." 2016 HHRA at 14 (emphasis added). In other words, Rauh *et al*. (2011) is not central to the 2016 HHRA because, in that assessment, EPA did not use any epidemiologic data in a quantitative manner. Rather, the agency took the following approach:

1. Select an exposure scenario likely experienced by pregnant women in the Columbia study

2. Predict the concentration of chlorpyrifos in cord blood that resulted from that exposure scenario using a model

3. Estimate the levels of exposure by other pathways (e.g., dietary, golf) that would result in the same concentration of chlorpyrifos in cord blood

The levels of exposure estimated in step 3 were the points of departure to which uncertainty factors were applied in the 2016 HHRA. EPA did not rely on data from Rauh *et al*. (2011) or any other epidemiologic study for the 2016 HHRA.

[41] EPA OPP, Chlorpyrifos Issue Paper: Evaluation of Biomonitoring Data from Epidemiology Studies (Mar. 11, 2016) at 40-41, https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0062-0005.

IV. IT WOULD BE INDEFENSIBLE FOR EPA TO ELIMINATE OR REDUCE THE FQPA 10X BASED ON NEW METHODOLOGIES FOUND WANTING BY THE SAP.

In its 2020 risk assessment and PID, EPA indicated that it was considering eliminating the FQPA 10X based on new methodologies in development. Specifically, EPA is in the process of developing a suite of *in vitro* assays to assess the developmental neurotoxicity ("DNT") potential of individual organophosphate pesticides ("OPs"), including chlorpyrifos. EPA plans to use information from this suite of tests, referred to as new approach methodologies ("NAMs"), "in the future as part of the weight of evidence evaluation of neurodevelopmental toxicity potential for OPs." 2020 HHRA at 51. This will include using NAMs to evaluate inter- and intra-species variability, a purpose that no government body has ever employed to date.

Scientists and subject matter experts, including those from the FIFRA SAP, have voiced serious substantive problems with EPA's proposed use of NAMs in the context of human health risk assessment. The SAP report makes it indefensible to rely on NAMs in EPA's risk evaluation for chlorpyrifos.

EPA selected OPs, including chlorpyrifos, as its first test case in using NAMs because all OPs inhibit activity of the acetylcholinesterase enzyme ("AChEi").[42] This is a baseless approach given that scientists agree — and EPA's own findings concur — that prenatal OP exposure increases the risk of neurodevelopmental harm at levels well below those that induce AChEi.[43] EPA charged the SAP with assessing the application of a selected suite of NAMs for predicting the DNT potential of OPs, and further, to justify stripping away protective uncertainty factors. EPA's charge to the SAP went so far as to propose stripping the congressionally mandated default FQPA tenfold safety factor that EPA retained for most of the OPs due to demonstrated risk of neurodevelopmental harm from prenatal exposures.

The SAP met in September 2020 and issued its final report in December 2020, less than 2 months ago. It was a scathing critique of EPA's proposed use of the NAMs. *See* 2020 NAM Report.[44] Among its many pointed criticisms, the SAP warned that the *in vitro* cell culture assays, "may not reflect *in vivo* conditions" and "lack some features that are known to be critical in the development of the nervous system" like hormonal and neurotransmitter signaling, sex differences, the presence of glial cells, inter-organ and intra-tissue communications, and peripheral influences from the maternal environment. *Id*. at 12-13. The SAP further stated that

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[42] EPA: "in vitro acetylcholinesterase (AChE) inhibition data has been generated for OP compounds. The OPP is considering the potential use of these data to develop interspecies and/or intraspecies data-derived extrapolation factors (DDEFs) in accordance with EPA's 2014 Guidance for Applying Quantitative Data to Develop DDEFs for Interspecies and Intraspecies Extrapolation." (EPA Issue Paper, p. 5)

[43] Scientific Letter on Chlorpyrifos and Neurodevelopmental Harm (Feb. 3, 2021), in EPA-HQ-OPP-2008-0850; EPA, Literature Review on Neurodevelopment Effects & FQPA Safety Factor Determination for the Organophosphate Pesticides (Sept. 15, 2015).

[44] *See* Transmittal of Meeting Minutes and Final Report of the Federal Insecticide, Fungicide and Rodenticide Act, Scientific Advisory Panel (FIFRA SAP) Virtual Meeting held on September 15-18, 2020 (Dec. 14, 2020) ("2020 NAM Report"), https://www.regulations.gov/document?D=EPA-HQ-OPP-2020-0263-0054 (Ex. 4).

these limitations precluded the proposed NAMs assays from representing "…many processes and mechanisms that could cause developmental neurotoxic events." *Id.* at 12. The SAP concluded that the proposed NAMs *in vitro* assays would neither derive "…a meaningful point of departure useful in predicting a disease state in humans…" nor "…contribute to any understanding of mechanisms." *Id*. at 13. As such, the Panel suggested that these assays would serve best as preliminary toxicity screening tools, but "…wondered about their utility in their proposed use to ultimately define a safe level of exposure." *Id*. Overall, the SAP blasted EPA's proposed use of the NAMS as inappropriate and indefensible, given the lengthy list of data gaps, uncertainties, and limitations.

The SAP additionally raised several concerns in response to EPA's proposed use of NAMs to extrapolate effects from *in vitro* to *in vivo*, and in doing so, eliminate the use of uncertainty factors. Namely, the Panel criticized the lack of robustness of analyses conducted by Exponent, a third-party consulting group hired by a consortium of three agrochemical companies (AMVAC, FMC, and Gowan) to model "data derived extrapolation factors" ("DDEFs").[45] Exponent's analyses relied on the presumption that the active site on the AChE molecule has the same structure across species, and therefore has the same function and activity. A memo from EPA toxicologist Dr. Stephanie Padilla refutes this assumption.[46] EPA noted a range in variability of 3% to 97% in AChEi among human samples tested.[47] This far-ranging variability renders the results practically meaningless and is likely due to the extremely small sample size of the study. This point was noted by the SAP that further criticized Exponent's intraspecies models for relying on "…too few sample data points" and an "…absence of samples representing certain ethnic and racial groups…." 2020 NAM Report at 14 n.4. The SAP concluded that these factors resulted in analyses that were "underpowered," thus lacking statistical support, and were unreliable and meaningless. *Id*. at 15.

Even apart from these methodological deficiencies, Exponent's analysis was doomed by design because it relies on the false presumption that AChEi is the most sensitive endpoint, when scientific studies and the weight of the evidence prove that it is not. EPA describes the flawed origin of this work as follows: "In 2016, three OP pesticide registrants (AMVAC, FMC, and Gowan) and their consultant (Exponent) and Dr. Janice Chambers from Mississippi State

[45] Exponent submitted the following two papers to EPA: a whitepaper (MRID 50773504) that provides Exponent's summary of existing knowledge regarding AChE in rats and humans, including amino acid sequence alignments and 3D structures; and, a separate report was submitted on using these data to calculate pharmacodynamic DDEFs (MRID 50773504). The Exponent papers are discussed by EPA in: EPA 2020. Use of New Approach Methodologies to Derive Extrapolation Factors and Evaluate Developmental Neurotoxicity for Human Health Risk Assessment, https://www.regulations.gov/document?D=EPA-HQ-OPP-2020-0263-0006.

[46] EPA Memorandum from Stephanie Padilla. Comments on the Exponent Whitepaper Regarding Pharmacodynamic Parameters of Human and Rat Acetylcholinesterase Inhibition by Direct-Acting Organophosphorus (OP) Insecticides or Active Metabolites. Stephanie Padilla, EPA-ORD., July 9, 2020, https://www.regulations.gov/document?D=EPA-HQ-OPP-2020-0263-0005.

[47] September 2020 SAP Charge Questions, Charge Question #8, https://www.regulations.gov/document?D=EPA-HQ-OPP-2020-0263-0018.

University to develop an experimental plan to determine if differences exist in AChE inhibition between rats and humans and estimate intra-human variability and these differences."[48] By 2016, it was well-established in the scientific literature, and even in EPA's own analyses, that AChEi was an insensitive and unprotective endpoint, not appropriate for use in EPA's chlorpyrifos risk evaluation.

In light of the SAP's withering criticisms of the current suite of NAMs tests to assess DNT, they cannot be used to justify stripping away uncertainty factors or weakening protections.

THE 2020 RISK ASSESSMENT UNDERESTIMATES EXPOSURES.

I. EPA'S DRINKING WATER ASSESSMENT FINDS PERVASIVE RISKS OF CONCERN AND, IF IT FULLY ASSESSED THE RISKS, ALL RISKS WOULD BE OF CONCERN.

For nearly 10 years, EPA has repeatedly identified elevated health risks due to chlorpyrifos contamination of drinking water. It expressed these concerns in the 2011 preliminary HHRA, the 2014 HHRA, and the 2016 revisions to the 2014 HHRA. It conducted modeling that estimated drinking water exposures above its levels of concern, and it compared the model results with detections in monitoring, which confirmed that its models were conservative. EPA is now abandoning its health protective approach to drinking water, along with its use of established standard models and methods. Instead, EPA has conducted a new drinking water assessment, based on three new models developed in the past two years that have not previously been used in risk assessment, have a number of significant limitations, strip away health-protective assumptions from previous models, and underpredict exposures when compared with real-world data. EPA goes through the exercise of validating the model output against real-world monitoring data, but then downplays the evidence that shows its modeling is underprotective.

A. 2011 and 2014 Preliminary and Revised HHRA

The drinking water risk estimates in EPA's 2011 preliminary HHRA indicated 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 2011 preliminary assessment noted that EPA had come to believe that its previous modeling results (using SCI-GROW) "used in 2000 likely underestimate the potential exposure," so in 2011 EPA instead used a range of surface water EDWCs derived from the PRZM-EXAMS model. 2011 HHRA at 12.

[48] EPA 2020. Use of New Approach Methodologies to Derive Extrapolation Factors and Evaluate Developmental Neurotoxicity for Human Health Risk Assessment at 68, https://www.regulations.gov/document?D=EPA-HQ-OPP-2020-0263-0006.

The 2014 HHRA used different modeling, but continued to find that a substantial amount of chlorpyrifos uses will result in exceedances of EPA's drinking water levels of concern.[49] These assessments, like the 2020 HHRA, are based on an underprotective drinking water level of concern ("DWLOC"), because it uses 10% cholinesterase inhibition as the regulatory endpoint and therefore fails to protect infants and children from neurodevelopmental impacts. Even using the flawed DWLOC, EPA found that existing label regulations are inadequate to prevent unsafe chlorpyrifos contamination of drinking water, particularly to infants, and that numerous chlorpyrifos application scenarios cause contamination of drinking water at levels exceeding the DWLOC.

EPA proposed revoking all chlorpyrifos tolerances because drinking water contamination prevented it from finding chlorpyrifos safe. Using its standard drinking water assessment methods, EPA found that many, if not most, label uses of chlorpyrifos result in drinking water contamination levels that exceed EPA's levels of concern for infants and children. 80 Fed. Reg. at 69,082, 69,083.[50] EPA found:

> [W]hen growers use maximum application rates, or even rates much lower than maximum, chlorpyrifos oxon concentrations in drinking water could pose an exposure concern for a wide range of chlorpyrifos uses.

Id. at 69,106. As a result, EPA "cannot make a safety finding based on drinking water exposures." *Id*.

In addition to showing EPA's regulatory endpoint is underprotective, comments filed on the 2014 HHRA made the following points:

1. 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.

[49] EPA, Office of Chemical Safety and Pollution Prevention, Chlorpyrifos: Updated Drinking Water Assessment for Registration Review (Dec. 23, 2014), https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0850-0198.

[50] EPA used the steady state concentrations, which tend to be much lower than the acute levels, *e.g.*, 3.9 ppb vs. 24 ppb for infants. 80 Fed. Reg. at 69,101. EPA notes that "it is possible that for some limited numbers of use scenarios, the EDWC could result in an exceedance of the acute DWLOC, but not the steady state DWLOC." *Id*. at 69,101. If EPA relies on any refined watershed assessments, it would need to guard against any such acute exceedances.

2. EPA noted that its drinking water assessment likely understated the exceedances of the DWLOC. For example, it did not evaluate higher application rates in the South Atlantic Gulf and noted that "more exceedances are expected for higher application rates." Indeed, the other evaluations above of the high application rate showed that almost all resulted in exceedances. It also conducted the national and regional modeling 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."

3. EPA appropriately determined that the identification of chlorpyrifos drinking water exceedances is an appropriate and defensible finding 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 concluded that "[t]his analysis demonstrates that the model estimated concentration reasonably compare to measured concentrations." The concordance with monitoring data bolsters confidence that EPA's predictions of exceedances are realistic and accurate.

2015 Farmworker Comments at 73-76.

B. 2016 HHRA

EPA finalized a refined drinking water assessment for chlorpyrifos in April 2016, which served to "combine, update and complete the work presented in the 2011 and 2014 drinking water assessments..." 2016 Chlorpyrifos Refined Drinking Water Risk Assessment for Registration Review ("2016 DWA") at 6.[51] The 2016 drinking water assessment results were consistent with the previous assessments and suggested "potential exposure to chlorpyrifos or chlorpyrifos-oxon in finished drinking [sic] based on currently labeled uses." *Id*. Unsurprisingly, higher concentrations of chlorpyrifos and the more potent chlorpyrifos-oxon are likely to be found in areas with higher chlorpyrifos use and areas that are more vulnerable to runoff. *Id*. at 7. Thus, agricultural communities, including farmworkers and their families, are more likely to have their drinking water contaminated by chlorpyrifos. EPA's revised

[51] The 2016 Chlorpyrifos Refined Drinking Water Risk Assessment for Registration Review can be found at EPA-HQ-OPP-2015-0653-0437.

assessment did not result in any changes to its finding that "the majority of estimated drinking water exposures from currently registered uses, including water exposures from non-food uses, continue to exceed safe levels even taking into account more refined drinking water exposures." 81 Fed. Reg. 81,049, 81,050 (Nov. 17, 2016).

EPA had already found that food exposures alone exceeded risks of concern, but it calculated "no food" drinking water concentrations that would alone exceed its level of concern. 2016 RHHRA at 24. It assessed potential chlorpyrifos drinking water exposures based on national modeling, regional modeling and monitoring data. All three analyses showed that drinking water concentrations across the country exceed the drinking water level of concern.

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

Table 3. Comparison of EPA's national-level estimated chlorpyrifos drinking water concentrations[52] to the "no food" drinking water level of concern.

		1-in-10-year concentration (ug/L)			
	Absolute Peak (ug/L)	**Peak**	**21-day average**	**Annual average**	**30-year annual average (ug/L)**
High end scenario (Michigan tart cherries)	172	129	83.8	39.2	29.7
Exceedance of "no food" drinking water level of concern	**12,286**	**9,214**	**5,986**	**2,800**	**2,121**
Low end scenario (Georgia bulb onions)	8.5	6.2	3.1	1.2	0.8
Exceedance of "no food" drinking water level of concern	**607**	**443**	**221**	**86**	**57**

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

[52] From Table 1 of the 2016 DWA at 7.

found that chlorpyrifos drinking water contamination is likely and that such contamination is unsafe.

C. 2020 Drinking Water Assessment and RHHRA

The 2020 Refined Drinking Water Assessment ("2020 DWA") is a startling departure from all EPA's previous pesticide drinking water assessments – for chlorpyrifos, for other OP insecticides, and for most other pesticides – which applied peer reviewed and tested models and monitoring data. EPA has abandoned its long-established drinking water PRZM/EXAMs model, and instead has employed three new, untested, and inadequately vetted models. *See* 2020 DWA at 9, 82. All three models were introduced in 2019 and 2020 and have not been used in any other pesticide evaluations.[53] This is especially troubling given that the models – all of which are limited to drinking water exposure from surface water sources – introduce significant uncertainties and limitations that are likely to underestimate exposures and result in a modeled output that fails to protect all Americans. In short, EPA has relied on untested new methods to strip away surface water protections, putting our Nation's pregnant women and children at risk from unsafe drinking water.

Here we briefly summarize the concerns with each of the three new models:

The Percent Cropped Area (PCA) and Percent of Crop Treated (PCT) model continues to incorporate the PCA as have previous models, but it added a value for PCT which was not included in previous models.[54] This means that EPA is abandoning its longstanding protective assumption built into the PRZM/EXAMs that 100 percent of the field is treated, instead using smaller values.[55] This makes the model less conservative, and the output less protective. It also adds considerable uncertainty, given that the percent of the field that is treated is highly changeable, depending on pest-pressures that can be very localized and different throughout the year and from year to year, and even between neighboring fields. EPA itself noted the complexities when it attempted to develop PCT values in 2002: "Pesticide use is a dynamic process that is subject to unpredictable factors such as weather, pest population, and the pesticide market itself. ... Modeling the complex relationships between these factors and the applicators' decision-making process, in order to forecast PCT, would require overwhelming amounts of

[53] *See* EPA, About Water Exposure Models Used in Pesticide Assessments, https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/about-water-exposure-models-used-pesticide#names.
[54] *Id*.
[55] *See* EPA, Development of Community Water System Drinking Water Intake Percent Cropped Area Adjustment Factors for use in Drinking Water Exposure Assessments: 2014 Update (Sept. 9, 2014) at 10 n.4 ("PRZM/EXAMs model inputs include values for certain physical-chemical properties of the pesticide, application practices, crop agronomic information, precipitation, and soil properties. In the standard scenarios used to estimate pesticide exposure, it is assumed that 100% of the field is treated."), https://www.epa.gov/sites/production/files/2015-07/documents/development_and_use_of_community_water_system.pdf

information."[56] Peer reviewers reiterated this concern in their review of the 2020 model: "PCTs based on historical usage data are not predictive of the potential for increased usage in the future."[57] In other words, the percentage of a field that was pesticide-treated last year cannot be used to predict the percentage that will be treated next year, or the year after. In their report, peer reviewers provided examples of glyphosate and dicamba, both of which have had dramatically increased use each year. The peer review report provides no real recommendations for solving this problem because the problem is largely unsolvable, since it is inherently speculative to make this type of forecast of future behaviors that hinge on so many unknowable factors. The peer reviewers also raise substantive specific issues with the model: "the process by which min, max, and average PCT values are calculated and the benefit of having this range of values did not come across clearly. It is also unclear that the upper and lower distribution methods will provide useful information as in most [Community Water System] watersheds, these are likely to apply the pesticide to all or none of the eligible crop areas, respectively."[58] The most generous conclusion we can draw is that EPA's use of this new model introduces significant uncertainties. Further, the uncertainty is likely to be in the direction of underpredicting exposure by using a lower-than-100 percent value for PCT.

The Method for the Development of New Scenarios for Use in the Pesticide in Water Calculator, builds new scenarios (a combination of crop, soil type, and weather data) used in the Pesticide in Water Calculator (PWC). Scenarios are defined at the 90th percentile exposure value for each crop or group of crops for each of the 18 hydrologic unit regions (as outlined by the U.S. Geological Survey) in the contiguous United States. This introduces a number of new variables, including soil type and weather data, that can be transient, localized, and therefore uncertain. In addition to these uncertainties, use of the 90th percentile exposure value will underestimate exposure for highest 10 percent crops.

The two methodologies discussed above – the PCA/PCT model and the Pesticide Water Calculator – were reviewed by peer reviewers once only. There were no public comments, and the peer review report emphasized that a detailed description of the Quality Assurance/Quality Control (QA/QC) procedures was lacking and should be provided by EPA.[59] There is no record that a detailed QA/QC report has been provided to peer reviewers or the public, and in any case, the models have not been subjected to further review, despite these concerns.

Most concerning is EPA's reliance on yet another newly-developed model, *Approaches for the Quantitative Use of Surface Water Monitoring Data in Drinking Water Assessments*. This model is employed by EPA to use surface water monitoring data to estimate pesticide

[56] EPA, Development of a Methodology for Projecting Domestic Percent Crop Treated (2002), https://archive.epa.gov/scipoly/sap/meetings/web/pdf/developmentofamethodologyforprojectingdomestic.pdf
[57] Summary Report External Peer Review of Two Methods for Improving Pesticide Drinking Water Assessments (Mar. 3, 2020) at 34, EPA-HQ-OPP-2020-0279-0004.
[58] *Id*. at 39.
[59] *Id*. at 11.

concentrations in its drinking water assessments. The model uses the USGS SEAsonalWAVEQ with EXtended capabilities model (SEAWAVE-QEX). To address temporal challenges with available monitoring data, EPA developed methods to derive and integrate pesticide-specific sampling bias factors ("SBF") for four reference pesticides: atrazine, carbaryl, chlorpyrifos, and fipronil.[60] The SBF values are critical to the model's predictive accuracy, as they are used to calculate an upper bound prediction interval (for example, the 95th percentile) for a given measured concentration value where sampling was not frequent enough to catch the concentration peaks (SAP, p. 14). Although this method was peer reviewed by the Scientific Advisory Panel in November 2019, the SAP raised a number of significant concerns that do not appear to have been addressed by EPA.[61] These are summarized as follows:

- EPA's evaluation of SEAWAVE-QEX geographically limited to Midwest - The white paper that EPA presented to the SAP used case studies that were entirely drawn from the Midwest, despite the fact that most of the crop uses that the registrant has identified as "Critical," or that EPA has identified as "High Benefit," are grown outside of that region. The SAP noted that, "further evaluation was needed for application of SEAWAVE-QEX and SBFs to small watersheds with 'flashy' streams or to static, non-flowing systems" (SAP, p. 14). In its 2020 DWA, the EPA has considered sites in all regions (all 2-digit HUCs) for this assessment (2020 DWA at 41), but whether and how those were included is unclear. Nor is it apparent that the peer review concerns have been addressed. How have small watersheds with flashy streams been included? What about static, non-flowing systems? The 2020 DWA mentions this limitation, but not whether it has been addressed. 2020 DWA at 63.

- EPA's evaluation of SEAWAVE-QEX limited to data from tile drained fields - Agriculture tile drains are in roughly half of the agriculture fields of the Corn Belt and Great Lake states, whereas the rest of the nation uses them sparingly if at all.[62] The SAP noted in its report that, "this suggests uncertainty as to whether the maximum SBF values for… Midwestern sites would be protective for streams in other environmental settings." (SAP, p. 14). The 2020 DWA mentions this limitation, but similarly not whether it has been addressed to the Panel's satisfaction. 2020 DWA at 63.

[60] Transmittal of Meeting Minutes and Final Report for the Federal Insecticide Fungicide and Rodenticide Act, Scientific Advisory Panel (FIFRA SAP) Meeting Held on November 19-21, 2019. Peer Review of the Approaches for Quantitative Use of Surface Water Monitoring Data in Pesticide Drinking Water Assessments. Report, Feb 18, 2020. Document ID EPA-HQ-OPP-2019-0417-0019, https://www.epa.gov/sap/meeting-information-november-19-22-2019-scientific-advisory-panel.

[61] Related documents including EPA's response to the SAP comments can be accessed on the docket at EPA-HQ-OPP-2019-0417.

[62] See Acres Tiled as a Share of Cropland Acres, U.S. 2017. https://farmdocdaily.illinois.edu/2019/08/use-of-tile-2017-us-census-of-agriculture.html#:~:text=Tile%20in%202017&text=Acres%20tiled%20were%2014%25%20of,share%20was%2010%25%20for%20Wisconsin.

- The SEAWAVE-QEX model is based on an inadequate sample size - The SAP recommended that EPA increase the number of subsamples to at least 30 to better distinguish between within-sample and between-sample variability in the estimated maximum concentration." (SAP, p. 59). However, EPA reports that there are no sites with that many samples. 2020 DWA at 59. Instead, EPA included sites with as few as 12 samples per year. 2020 DWA at 41. EPA discusses some of the implications of such limited sampling, including noting that, "as sample collection increases, the detection frequency also increases" and that infrequent sampling will "reduce the likelihood of measuring peak concentrations." 2020 DWA at 59. EPA also notes that "most of the water" sampling comes from "grab samples," which is a single sample collected over a short time (15 minutes or less), in contrast to the more robust "compost sample" method which involves taking multiple samples over a longer period of time, usually 24 hours. 2020 DWA at 59. Both of these limitations – low frequency and short duration of sampling – will reduce the statistical power of the data to detect the pesticide and will almost surely fail to detect peak concentrations. In short, they will lead to underestimates of contamination.

- Lack of monitoring data, low confidence in model output - EPA notes that there are no SEAWAVE-QEX sites in HUC-10 and 11, and in most other HUCs there is only one SEAWAVE-QEX site. 2020 DWA at 60. Out of 13 monitoring sites that EPA has determined have acceptable SEAWAVE-QEX models, only 2 are rated as high confidence, whereas 4 are low confidence (5 are "medium" and 2 are unrated). 2020 DWA at 62, Table 18. Twice as many sites are ranked "low confidence" as "high confidence." The limitations, information gaps, and failures that lead to such a low or spotty confidence in the model output are also carried through to EPA's proposed registration review decisions, which should also be considered unreliable, unsupported, and indefensible.

EPA acknowledges that its modeled data underestimates contamination and that critical and high benefit uses may exceed DWLOC. EPA acknowledges that attempts to validate the model estimated concentrations of chlorpyrifos by comparing them to real-world monitoring data demonstrates that the model is underestimating contamination. "Model estimated concentrations indicate that for the subset of assessed uses concentrations of chlorpyrifos and chlorpyrifos-oxon are not expected to be above the DWLOCs with or without the retention of the FQPA safety factor. However, monitoring data suggest that in some areas of the country concentrations may exceed the DWLOC with and without the FQPA safety factor when all uses currently registered are considered since available monitoring data represent usage of chlorpyrifos." 2020 DWA at 76. It should be considered unacceptable for EPA to apply a model that underestimates exposure, and therefore risk.

At sites with more than 13 samples per year – those with more robust data and therefore higher confidence in the model and monitoring results – EPA reports that there are five sites, all in HUC-17, with a potential for DWLOC exceedances. 2020 DWA at 76. It is especially

concerning that EPA cannot determine whether the contamination is from the 11 Critical or High Benefit uses that the registrant is seeking and EPA is proposing to retain. 2020 DWA at 76. That is, the uses that will remain may be contributing to surface water contamination exceeding the DWLOC.

Monitoring data show that several sites across the U.S. could exceed the DWLOCs, but EPA speculates (without evidence) that contributions from other currently registered uses (not considered in this assessment), *i.e.*, non-food uses, may be contributing to the exceedances. 2020 DWA at 9. "[A] thorough analysis of monitoring data was completed and indicates that there are several monitoring sites across the United States that could have concentrations higher than the DWLOCs (with and without the retention of the FQPA safety factor). However, the contribution of other currently registered uses of chlorpyrifos (*i.e.*, uses not considered in this assessment) could not be ruled out, nor could a definitive conclusion be made that the measured concentration data correlated to one of the specific uses evaluated in this assessment." 2020 DWA at 81. However, this is irrelevant, since the FQPA "risk cup" of aggregate exposures that will be safe needs to include all uses, including non-food uses. EPA's phased-down withdrawal of sulfuryl fluoride, a pesticide that breaks down into fluoride and is commonly used in food storage and processing facilities, is illuminating. "Although sulfuryl fluoride residues in food contribute only a very small portion of total exposure to fluoride, when combined with other fluoride exposure pathways, including drinking water and toothpaste, EPA has concluded that the tolerance (legal residue limits on food) no longer meets the safety standard under the Federal Food, Drug, and Cosmetic Act (FFDCA) and the tolerances for sulfuryl fluoride should be withdrawn."[63]

It is not only chlorpyrifos that is polluting our drinking water, but also chlorpyrifos-oxon, which is roughly a thousand times more toxic than the parent compound; it is produced in drinking water from chlorpyrifos as a result of chlorination during routine drinking water treatment.[64] EPA identifies exceedances for the 21-day average chlorpyrifos-oxon concentrations in the source surface water of 4 of 11 regions: Great Lakes; Upper Mississippi; Souris-Red-Rainy; Pacific Northwest. *See* 2020 DWA at 13, Table 1. Further EPA's analysis reveals that 232 community water system watersheds may have chlorpyrifos-oxon concentrations above the 21-day DWLOC for upper bound application rates. *See* 2020 DWA at 50, Table 14. This was determined by counting the number of community water systems with PCAs above the critical PCA for each respective region. No information is provided as to how many people are served by these systems, but EPA reports that they comprise over 70% of the systems in two of the regions (Great Lakes HUC 04 and 09) and half of the systems in one region (HUC 07), but less than one percent of the systems in one region (HUC 17). So, presumably in three of the four HUC regions the majority of households may be pulling their drinking water from systems that are unsafe for at least a portion of the year.

[63] EPA Proposes to Withdraw Sulfuryl Fluoride Tolerances, https://archive.epa.gov/oppsrrd1/registration_review/web/html/evaluations.html

[64] Wu J, Laird DA. Abiotic transformation of chlorpyrifos to chlorpyrifos oxon in chlorinated water. Environ Toxicol Chem. 2003 Feb;22(2):261-4. PMID: 12558155.

Not only does EPA's drinking water assessment use an underprotective endpoint, but it estimates exposure based on three new models that have serious flaws identified in their peer reviews that have not been addressed. EPA acknowledges that monitoring data show exceedances of its DWLOC.

II. EPA'S PROPOSAL WILL NOT PROTECT PEOPLE FROM SPRAY DRIFT

People living in agricultural communities are at particular risk from chlorpyrifos spray drift, especially children who are exposed to drift near their schools and day cares, in their homes, and at playgrounds. As with drinking water contamination, farmworkers and their families are disproportionately exposed to toxic chlorpyrifos drift – they are, quite literally, getting hit from all sides. Spray buffers are currently in place for chlorpyrifos, but those buffers are far too small to protect people from drift. *See* PID 19, 61; 2020 HHRA at 9, 48. As noted throughout these comments, the PID will not protect children from neurodevelopmental harm because EPA is using an underprotective regulatory endpoint. Departing from its 2016 risk assessment, which sought to protect from damage to children's brains, EPA reverts to a 10% cholinesterase endpoint in the 2020 HHRA and PID. *See* 2020 HHRA at 48. When EPA accounted for neurodevelopmental harm to children in its 2016 risk assessment, EPA found unsafe levels of chlorpyrifos from the field's edge to distances of more than 300 feet from where the pesticide is sprayed. 2016 HHRA at 31. The risks presented by spray drift weigh in favor of a ban on chlorpyrifos because, when neurodevelopmental harm is properly accounted for, all uses lead to risks of concern and necessitate buffers in excess of 300 feet.[65]

A. Current buffers do not protect children and pregnant women from unsafe exposures.

The buffers and drift mitigation measures proposed in the PID will not protect people from unsafe chlorpyrifos exposures. *See* PID at 19, 60-61. EPA conducted its drift assessment without taking into account the demonstrated neurodevelopmental damage to children from chlorpyrifos. It previously set buffers to guard against cholinesterase inhibition, even though it has found that damage to children's brains occurs at lower doses. 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, EPA would have determined that larger buffers are needed to protect children. Indeed, when EPA accounted for neurodevelopmental harm in its 2016 risk assessment it found that buffers in excess of 300 feet were necessary. Having reverted to using an endpoint that it recognizes as underprotective, EPA now purports to find no risks of concern with the current spray buffers, and asserts that even smaller buffers might be sufficient. PID at 19.

[65] Indeed, it is unclear how large buffers would actually need to be to adequately protect children because EPA's spray drift modeling does not go beyond 300 feet.

In 2012, pursuant to its policy of mitigating risks that emerge during registration review even before that review has been completed, EPA pressed registrants to amend their labels to require buffers around schools, homes, and other populated areas to protect bystanders from pesticide drift outside of the registration review process. *See* 2014 HHRA at 82; Spray Drift Mitigation Decision for Chlorpyrifos (July 2012) ("Where risks are identified early in the registration review process and opportunities for early mitigation exist, the Agency will pursue those opportunities as they arise, rather than waiting for completion of a chemical's registration review in order to mitigate the risks.").[66] By December 2012, chlorpyrifos 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. 2020 HHRA at 48. 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."[67] 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
>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[1]	10	10
>2 – 3	medium	100[1]	10	10
>3 – 4	medium or coarse	NA[2]	25	10
>4	medium or coarse	NA	50	10

[1] Aerial application of greater than 2 lb ai/A is only permitted for Asian Citrus Psylla control, up to 2.3 lb ai/A.
[2] "NA" means "not allowed."

Banning chlorpyrifos is the only way to adequately protect against the pesticide's harms, but as a mitigation measure, no-spray buffers are a proven and effective safeguard to lessen harmful pesticide exposures. The current chlorpyrifos buffers, however, are based on the underprotective endpoint of 10% cholinesterase inhibition and are vastly inadequate. As another mitigation measure, the PID proposes label changes for application such as wind speed and release height, but these mitigation measures are also based on 10% cholinesterase inhibition. *See* PID at 60. As stated above, the no-spray buffers established for chlorpyrifos are far too

[66] 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) (EPA-HQ-OPP-2008-0850-0105).
[67] *Id.* at 3.

small as even buffers of a half-mile may not be adequately protective. Moreover, EPA did not validate that the current 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 should be developed." [68] When proposing mitigation measures, agencies must evaluate their effectiveness.[69]

The PID proposes to leave in place current buffers of 10-100 feet even though, in 2016, EPA concluded that spray uses "require buffer distances of > 300 feet to [result in exposure] below the level of concern." 2016 HHRA at 6. Notably, EPA did not assess buffer distances greater than 300 feet and therefore could not identify safe buffer distances. In 2018, the California Environmental Protection Agency (CalEPA) concluded that risks from aerial spraying extended to 1,320 feet (about ¼ mile) in most children and adults and to 2,608 feet (about ½ mile) in infants.[70] CalEPA did not consider distances beyond 2,608 feet and therefore, like EPA, could not identify a safe buffer distance. Furthermore, as CalEPA acknowledged, "[I]t is possible to detect concentrations of chlorpyrifos in ambient air at levels at or above the analytical limit of detection at distances farther downwind from an application than ½ mile (2640 feet)."[71] The safe distance may be well in excess of ½ mile. Indeed, one study found associations between autism spectrum disorder in children and chlorpyrifos applications within 1.5 kilometers (about 1 mile) of maternal residence in the second trimester of pregnancy.[72]

Other documented incidents also demonstrate that harmful spray drift occurs outside of the current buffer. In 2015, 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. [73] The workers were dispersed with their distance from the edge of the pear orchard ranging from 30 to more than 350 feet. The Washington Department of Health's incident investigations also attest to this fact. In March 2015, a pesticide drifted from airblast spraying onto school grounds 90 feet away and sickened three people, including a pregnant elementary school teacher. And in April 2014, drift from airblast spraying 260 feet away made two people sick at their home residence.[74] While

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[68] *Id*. at 43.
[69] *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)).
[70] CDPR, Final Toxic Air Contaminant Evaluation of Chlorpyrifos at 81.
[71] *Id*. at 80.
[72] Janie F. Shelton et al. Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study. 122 *Environmental Health Perspectives* 1107 (2014), https://ehp.niehs.nih.gov/doi/10.1289/ehp.1307044 (Ex5).
[73] 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). *See also* Comment Letter on 2014 HHRA from Washington Department of Health attaching summaries of incidents investigated by the Department of Health and found to be definitely, probably, or possibly due to chlorpyrifos exposure (May 8, 2015) ("Washington Department of Health Incident Investigation Summaries"), https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0850-0842.
[74] Washington Department of Health Incident Investigation Summaries, *supra* note 73.

these examples are not chlorpyrifos-specific, they demonstrate the prevalence of pesticide poisoning incidents due to drift.[75]

Specific to chlorpyrifos, in March 2014, a farmworker in Washington State became sick when chlorpyrifos drifted from airblast spraying a quarter-mile away.[76] Likewise, California's poisoning incident reports also contain numerous incidents from chlorpyrifos alone at distances from 80-feet up to a half-mile from the field where the application occurred.[77]

For its part, EPA reviewed chlorpyrifos poisoning incidents in the PID, but seems not to have reviewed any data from the past decade. *See* PID at 29-31. Moreover, EPA dismissed all incidents where the person was exposed to chlorpyrifos along with other pesticides. At the very least, EPA should have considered exposures to chlorpyrifos along with other organophosphates as they all suppress cholinesterase and are associated with damage to children's brains.

In the face of evidence and findings like those in the CDC report and state incident reports, 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. Moreover, EPA must account for—and protect from—neurodevelopmental harm that will result from spray drift exposures.

B. Air monitoring shows chlorpyrifos levels in agricultural communities that pose a risk to children and pregnant women.

In the 2016 HHRA, EPA appropriately evaluated inhalation exposures from chlorpyrifos drift and thereby filled an important exposure gap that was ignored in the 2014 HHRA. *See* 2016 HHRA at 7, 30-35. Unfortunately, EPA ignores its own 2016 evaluation of air monitoring data in the 2020 HHRA and PID. The multiple air monitoring studies conducted in agricultural communities, summarized in the 2016 HHRA, show that chlorpyrifos is regularly detected in the ambient air where children and pregnant women are exposed (e.g., in communities and at schools). In addition, research studies have shown that chlorpyrifos is found in the air at considerable distance from where it was applied and persists for multiple days – for example, one study found strong correlations with detections of chlorpyrifos in the air with applications

[75] EPA has recognized that additional prescriptions are needed in addition to the current label prohibition on applying a pesticide in a way that will contact workers or other people directly or through drift. The label prohibition, alone, has not prevented toxic drift and poisoning incidents. EPA, Pesticides; Agricultural Worker Protection Standard Revisions, 80 Fed. Reg. 67,496, 67,521-22 (Nov. 2, 2015) ("additional measures are needed" because the label "do not contact" language has proven "insufficient" to prevent exposure of workers to drift). EPA found it necessary to afford additional protection in the form of application exclusion zones ("AEZs") that prevent workers from being in treated areas during applications. *Id*. at 67,521-22. The requirement to suspend applications in AEZs when workers or other people not handling the application are present, however, has been weakened by recent amendments to the Worker Protection Standard. 85 Fed. Reg. 68,780 (Oct. 30, 2020).

[76] Washington Department of Health Incident Investigation Summaries, *supra* note 73.

[77] From http://www.cdpr.ca.gov/docs/whs/pisp.htm (query for chlorpyrifos 2001-2013), attached as Appendix 1 to 2015 Farmworker Comments.

made within 1.5 miles and up to 4 days prior to the sampling event.[78] This is consistent with previous analysis finding that chlorpyrifos detections and air concentrations are correlated with amount of use within a 5 mile (8 km) area around the monitoring site.[79] EPA's evaluation of these studies to consider inhalation exposures is critical to understanding exposures in agricultural communities and should be relied upon in registration review.

Even in the absence of comprehensive modeling of volatilization and transport from treated fields under different atmospheric conditions, the ambient monitoring data illustrates that real-world exposures in agricultural communities do not meet the safety standard due to inhalation exposure alone. When aggregate dietary and spray drift exposures are also considered, the risk faced in these communities is staggering. For example, the Shafter Air Monitoring Site is located at a school in close proximity to almond orchards where chlorpyrifos is used. The most recent published data available (2015) from the California Department of Pesticide Regulation showed that chlorpyrifos was detected in nearly two-thirds (61%) of the samples taken at this site.[80] In 2014, the closest field application site was 0.3 miles from the monitoring site, and a total of 13,837 pounds of chlorpyrifos were used within 5 miles of the monitoring site.[81] EPA's evaluation of the data from this air monitoring site found both acute and steady-state risks of concern with MOEs below 10. For children attending this school and living nearby, the inhalation exposures are compounded with the potential for spray drift and dietary exposure.

When using an endpoint meant to protect against neurodevelopmental harm, EPA found that the peak values recorded in all 11 air monitoring data sets result in acute inhalation exposure that do not meet the safety standard for children, and the vast majority do not meet the safety standard for pregnant women. It is clear from this analysis that the levels of chlorpyrifos routinely measured in the air in agricultural communities pose a significant threat to public health. Indeed, California recently classified chlorpyrifos as a toxic air contaminant and initiated cancellation proceedings of the pesticide. California and Dow/Corteva agreed to settlement that resulted in a ban of 99% of chlorpyrifos uses in the state effective December 31, 2020.

C. The current chlorpyrifos buffers are arbitrary.

Remarkably, the buffers that EPA now says are sufficiently protective of human health are smaller than current buffers put in place by EPA or other regulators. EPA's 2001 IRED for

[78] Harnly, M., McLaughlin, R., Bradman, A., Anderson, M., and Gunier, L. (2005). Correlating Agricultural Use of Organophosphates with Outdoor Air Concentrations: A Particular Concern for Children. Environmental Health Perspective, 113(9): 1184-1189.
[79] Wofford, P., Segawa, R., Schreider, J., Federighi, V., Neal, R., and Brattesani, M. (2014). Community Air Monitoring for Pesticides. Part 3: Using Health-Based Screening Levels to Evaluate Results Collected for a Year. Environ. Monit. Assess., 186(3):1355-1370.
[80] CDPR. 2016. 2015 Draft Air Monitoring Network Report. http://www.cdpr.ca.gov/docs/emon/airinit/amn_2015_report_draft.pdf.
[81] CDPR. 2016. Correlating Agricultural Use with Ambient Air Concentrations Of Chlorpyrifos and Chlorpyrifos-Oxon During The Period of 2011-2014. http://www.cdpr.ca.gov/docs/emon/airinit/2560_chlorpyrifos_final.pdf.

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[82]

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

Chlorpyrifos has also been the subject of an Endangered Species Act 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. In December 2017, NOAA Fisheries proposed mitigation options to avoid this prohibited result, which include buffers. The mitigation included 150-meter buffers for ground applications and 300-meter buffers for aerial applications.[83] 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.

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

[82] Chlorpyrifos IRED at 208.
[83] NOAA Fisheries, Biological Opinion on the Environmental Protection Agency's Registration of Pesticides containing Chlorpyrifos, Diazinon, and Malathion, https://repository.library.noaa.gov/view/noaa/16997.

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, in its 2015 revision of the Worker Protection Standard (WPS), EPA included Application Exclusion Zones (AEZs) of 25-100 feet around application equipment during applications. 40 C.F.R. §§ 170.405 and 170.505. Applicators are required to cease spraying if other workers or people enter the AEZ, but with the recent weakening of the standard, the AEZ is limited to the boundaries of the agricultural establishment. *Id.*; 40 C.F.R. § 170.505. Still, the AEZs remain larger than the current chlorpyrifos buffers. Again, when using the appropriate endpoint of neurodevelopmental harm, EPA found that buffers greater than 300 feet are necessary to protect people from harmful impacts from chlorpyrifos, and those findings are based only on dermal risks (and incidental oral risks for children) from indirect exposures to drift. Bystanders and workers in other fields need more protection than the AEZs provide.

D. EPA's Proposal Will Not Protect People from Volatization

Commenters have repeatedly raised concerns about exposures from chlorpyrifos volatilization, but EPA has yet to sufficiently address the issue. Moreover, in the 2020 HHRA, EPA noted that "[c]hlorpyrifos has been detected in air samples, and so volatilization may play more of a role in dissipation than laboratory studies indicate." 2020 HHRA at 17. If EPA allows any chlorpyrifos uses to remain after registration review, the agency must conduct an adequate assessment of volatilization risks.

In the 2011 HHRA, EPA found that risks of concern are exceeded for bystanders. 2011 HHRA at 55. 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 of the acute application site concentrations and most of the short- and intermediate-term application site concentration assessments.

In 2013, drawing on methods used to assess bystander inhalation risks from fumigant pesticides and recommendations from a December 2009 Scientific Advisory Panel meeting, EPA conducted an assessment of volatilization risks from chlorpyrifos. EPA found that chlorpyrifos applied to fields can volatilize and harm people nearly a mile away (and likely farther): "Given the current available information and the state of the science concerning the volatilization of pesticides, this preliminary risk assessment indicates risks of concern are exceeded for

bystanders."[84] EPA identified buffer zones that would be required to reduce off-site concentrations to safe levels. For example, for oranges, the average application rate is so high (greater than 2 pounds of active ingredient/acre) that the maximum buffers would need to be between 1,476 and 4,724 feet and whole field buffers would need to range from 623 to 2,838 feet, so large that continued use of chlorpyrifos would be infeasible.[85]

In the 2014 HHRA, EPA reversed course and ignored all volatilization exposures based on two Dow studies, which purport to show that people will not experience adverse effects from volatilization exposures. It is also important to note that EPA evaluated the studies against the wrong endpoint of 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.[86]

Public comments objected to EPA's use of the Dow studies without subjecting them to peer review. 2015 Farmworker Comments at 32-33. Comments explained that the Dow studies ignored the effects of temperature, soil moisture, and individual variation and submitted biomonitoring and incident reports showing poisoning incidents at distances as far away as one-half mile from the application site. *Id*. at 50-58. Comments also pointed out the lack of controls in the Dow study that demonstrated that the experiment was capable of successfully producing or detecting cholinesterase inhibition. Without such controls, the study results cannot be interpreted or used to claim that chlorpyrifos volatilization does not produce cholinesterase inhibition. *Id*. at 51. 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 dismissing volatilization exposures from chlorpyrifos.

[84] Chlorpyrifos: Preliminary Evaluation of the Potential Risks from Volatilization (Jan. 31, 2013) at 55 (assessment based on a study that measured the effects of aerosolized chlorpyrifos – the form chlorpyrifos takes when applied as a spray – and not the vapor form it takes after volatilization), https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0850-0114.
[85] *Id*. at 32-46.
[86] *See* Slides in docket at https://www.regulations.gov/document?D=EPA-HQ-OPP-2008-0850-0189.

III. EPA FAILS TO ACCOUNT FOR EXPOSURE TO DUST FROM TAKE-HOME RESIDUES.

EPA fails to account for exposures to chlorpyrifos in dust brought home on a worker's clothing and gear. Numerous studies document that levels of chlorpyrifos in indoor dust are correlated to nearby agricultural use, including a recent systematic review by university environmental health researchers of relevant studies from around the world.[87] The authors used a systematic review method to cull through roughly 1,700 English-language published articles focusing on agriculture workers or farmworker families. Of these, the authors selected 39 articles that were of high quality and included measurements of pesticides and/or metabolites in biological (blood or urine) or environmental samples. Most of the studies were conducted on U.S. populations (25 studies, 64%), and most included organophosphate data (32 studies, 82%). After carefully scrutinizing all the data, the authors concluded that the majority of the reviewed articles (34 studies, 87%) "provided strong evidence that supports the take-home pathway."[88] Further, the studies showed that rural populations and farmworkers have higher concentrations of pesticides in blood and urine, and in house dust, compared with their non-rural or non-farmworker counterparts.[89] The authors noted that only a small portion of the studies (4 studies, 10%) did not identify take-home exposures as a relevant pathway. In short, a detailed systematic review of the publicly available scientific literature overwhelmingly demonstrates that off-target movement of chlorpyrifos and deposition of indoor dust is a significant pathway of exposure for harmful pesticides, including chlorpyrifos and other OP insecticides.

In the systematic review, three of the selected studies evaluated urine metabolites based on age, finding that OP residues and metabolites (creatinine-corrected total DAP; TCPy which is specific for chlorpyrifos; DMAP; AChE activity) were all inversely correlated with age (higher levels in younger children).[90]

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.[91] In a California study, chlorpyrifos detection was

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[87] López-Gálvez N, Wagoner R, Quirós-Alcalá L, et al. Systematic Literature Review of the Take-Home Route of Pesticide Exposure via Biomonitoring and Environmental Monitoring. *Int J Environ Res Public Health.* 2019;16(12):2177. Published 2019 Jun 19. doi:10.3390/ijerph16122177 (Ex. 6); *see also* Martha Harnly et al., *Pesticides in Dust from Homes in an Agricultural Area*, 43 Envtl. Sci. and Tech. 8767 (2009); Robert B. Gunier *et al*., Determinants of Agricultural Pesticide Concentrations in Carpet Dust, 119 Envtl. Health Persp. 970 (2011).

[88] López-Gálvez N, Wagoner R, Quirós-Alcalá L, *et al.* Systematic Literature Review of the Take-Home Route of Pesticide Exposure via Biomonitoring and Environmental Monitoring. *Int J Environ Res Public Health.* 2019;16(12):2177. Published 2019 Jun 19. doi:10.3390/ijerph16122177.

[89] *Id*.

[90] *Id*.

[91] Gloria D. Coronado et al., *Organophosphate Pesticide Exposure and Residential Proximity to Nearby Fields: Evidence for the Drift Pathway*, 53 J. Occup. and Envtl. Med. 884 (2011).

ubiquitous in indoor dust and found in close to 100% of the homes tested.[92] Because they crawl, play on the floor, and constantly put their hands in their mouths, young children have greater exposures to indoor dust.[93] 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.[94] 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.[95]

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.

IV. EPA MUST TAKE ACTION TO REDUCE WORKER RISKS.

A. EPA Has Documented Worker Risks from Chlorpyrifos For Years.

1. Acute Poisonings

Chlorpyrifos is one of the pesticides most often identified as the culprit when workers and bystanders suffer acute pesticide poisonings. This trend is particularly significant given the widespread under-reporting of pesticide poisonings due to such factors as inadequate reporting systems, fear of retaliation, and reluctance to seek medical treatment. In its proposed Worker Protection Standard revisions, EPA rightly acknowledges that "[u]nderreporting of pesticide

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[92] 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), *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 Envtl. Epidemiology 331 (2006).

[93] Jacqueline Moya and Linda Phillips, *A Review of Soil and Dust Ingestion Studies for Children*, 6 J. Exposure Sci. and Envtl. Epidemiology 545 (2014).

[94] Beti Thompson et al., *Variability in the Take-Home Pathway: Farmworkers and Non-Farmworkers and Their Childre*n, 24 J. Exposure Sci. and Envtl. Epidemiology 522 (2014).

[95] 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.)

incidents is a challenge," and assumes that only 25% of acute pesticide incidents are reported.[96] 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.[97] Other factors contributing to under-reporting 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.[98]

The 2020 risk assessment provides some data on pesticide poisonings from chlorpyrifos, but it is based on the 2011 preliminary human health risk assessment and therefore has no information after 2010, even though public comments have submitted more current information. The risk assessment also discounts poisoning incidents unless the only exposure was to chlorpyrifos. *See* PID at 29-31.

The 2016 petition to cancel and suspend chlorpyrifos uses due to unacceptable worker risks attached excerpts from California's pesticide exposure incident database that identify 289 definite, probably, or possible chlorpyrifos exposure incidents from 2001 through 2013.[99] Some reported poisoning incidents provide cursory descriptions of the illnesses:

1. In 2007, 26 vineyard workers in Tulare County, California, who were poisoned by drift from a nearby almond orchard, experienced nausea, vomiting, dizziness, difficulty breathing, blurred vision, rashes, throat irritation, and numbness in their fingers and tongues.[100]

[96] Worker Protection Standard Revisions, 79 Fed. Reg. 15,444, 15,453, 15,459 (Mar. 19, 2014). Focus groups conducted by the Washington Department of Health revealed that 75% of the workers reported that they or someone close to them had become ill from pesticides at work and often they did not seek medical care because they could not afford losing wages, feared losing their jobs, didn't know worker's compensation would pay for the visit, or mistrusted the health care providers as being aligned with the employers. Washington State Department of Health, *Learning from Listening: Results of Yakima Farmworker Focus Groups About Pesticides and Health Care* (2004).

[97] U.S. Gen. Accounting Office, Pesticides on Farms: Limited Capability Exists to Monitor Occupational Illnesses and Injuries 9 (1993), http://archive.gao.gov/t2pbat4/150612.pdf; *see also* Geoffrey M. Calvert et al., Acute Pesticide Poisoning Among Agricultural Workers in the United States, 1998-2005, 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).

[98] *Id.*

[99] From http://www.cdpr.ca.gov/docs/whs/pisp.htm (query for chlorpyrifos 2001-2013), attached as Appendix 1 to 2015 Farmworker Comments.

[100] Tulare County Report of Case 2007-689.

2. In 2012, a worker spraying chlorpyrifos developed neurological, gastrointestinal, and respiratory symptoms, even though he was wearing complete personal protective equipment.

3. In 2014, a worker air blasting chlorpyrifos had chest pains and exacerbated asthma when a branch ensnared his respirator and it fell off.

4. In 2014, a field worker tying apple tree branches across a field from airblast spraying to bare apple trees went to the emergency room for neurological and gastrointestinal symptoms.

5. In 2014, a worker pruning in a vineyard went to the hospital for gastrointestinal and respiratory symptoms due to drift from airblast spraying in adjacent fields.[101]

6. On May 5, 2017, chlorpyrifos traveled one-half mile from a farm, sickening dozens of people who were harvesting cabbage at a farm that does not use chlopyrifos. The Kern County Department of Agriculture and Measurement Standards found that chlorpyrifos drifted one-half mile from a farm. An applicator implicated in this drift incident was assessed penalties of more than $30,000.[102] 2002 Interim Re-Registration Eligibility Decision and Underlying Risk Assessment.

In 2001, after ending all home uses due to harm to children, EPA re-registered chlorpyrifos, allowing its use to continue in agriculture, despite finding numerous risks of concern to farmworkers that went unmitigated. When EPA assessed risks to workers who handle chlorpyrifos as part of the re-registration process, it identified risks of concern from a variety of activities, including mixing and loading various pesticide formulations and applications using certain types of equipment like airblast sprayers and backpack sprayers. To reduce risks, the risk assessment indicated that the labels would need to be amended to require additional personal protective clothing, enclosed cockpits for aerial applications, contained packaging for some formulations, and reductions in some application rates. Some risks of concern would remain even with such added mitigation. The risk assessment found risks of concern to field workers that it believed could be eliminated with reduced application rates or longer re-entry intervals. EPA lacked sufficient information to assess fully risks from seed treatment and to workers who enter greenhouses after pesticide spraying. Interim Reregistration Eligibility Decision for Chlorpyrifos (Feb. 2002).

UFW and others challenged EPA's re-registration of chlorpyrifos in part because of the unmitigated worker risks. A near-final settlement of that case fell apart when intervening precedent deprived the court of jurisdiction. *UFW v. EPA*, No. 07-3950-JF (N.D. Cal. Filed

[101] Washington Department of Health Incident Investigation Summaries, *supra* note 73.
[102] https://www.panna.org/sites/default/files/Copus-Road%20Incident-May-Press%20Release.pdf.

Aug. 1, 2007). The focus then shifted to the registration review process and advocacy to spur EPA action on the 2007 petition to ban chlorpyrifos.

2. Risk Assessments and Proposed Revocation – 2014-2016

In its 2014 risk assessment (which used an underprotective endpoint based on acute poisoning), EPA identified over 125 of 285 scenarios where workers face risks of concern from various handling tasks. It stated that 27 could be mitigated with additional engineering controls and on a few occasions with additional protective gear. EPA found that unacceptable risks would remain for 126 exposure scenarios, 32 seed treatment scenarios, and numerous greenhouse worker activities, even with maximum protective clothing and gear and engineering controls. EPA found that risks of concern could not be mitigated to acceptable levels for handlers who mix and load various formulations for aerial spraying, chemigation (irrigation), air blast, ground boom, and tractor-drawn spreaders, nor for workers applying some chlorpyrifos formulations by handwands, backpack sprayers, handguns, hand dispersal, and belly grinders, or for flaggers.

EPA found risks of concern to field workers who enter the fields to perform various tasks. To protect field workers, EPA establishes prohibitions on entering the fields during a re-entry interval ("REI") after the pesticide application. The 2014 risk assessment found that many REIs would need to be longer than the currently required re-entry periods.

EPA initiated discussions with chlorpyrifos registrants in an attempt to convince them to agree to measures to reduce risks to workers.[103] By June 2015, the negotiations with industry had broken down. EPA then told the Ninth Circuit that regulatory action would be necessary. EPA Status Report, *In re Pesticide Action Network North America v. EPA,* No. 14-72794 (9th Cir. June 30, 2015).

In October 2015, EPA proposed revoking all chlorpyrifos food tolerances, which would, if finalized, have ended all food uses within six months, thereby eliminating the associated risks to workers. In 2016, farmworkers, health, labor, and civil rights advocates petitioned EPA to cancel all uses of chlorpyrifos with the primary goal of protecting workers.[104] The petition sought to protect workers not only from risks associated with producing food crops, but also from nonfood uses of chlorpyrifos. Of course, EPA stalled all action to curtail the untenable risks from chlorpyrifos throughout the Trump administration. Six growing seasons have passed since EPA found pervasive unacceptable risks to workers.

[103] Letter to A. Colangelo from J. Housenger, Director OPP, at 4 (Mar. 26, 2015), https://www.regulations.gov/document?D=EPA-HQ-OPP-2007-1005-0099.

[104] The 2016 Petitioners are: United Farm Workers, League of United Latin American Citizens, Labor Council for Latin American Advancement, National Hispanic Medical Association, Farmworker Association of Florida, Pineros y Campesinos Unidos del Noroeste, Farm Labor Organizing Committee, California Rural Legal Assistance Foundation, Migrant Clinicians Network, and Learning Disabilities Association of America. The petition also sought suspension of the uses that pose unacceptable risks to workers, but that part of the petition was subsequently withdrawn.

B. 2020 HHRA Reiterates Findings of Unacceptable Risks to Workers

EPA's 2020 human health risk assessment covers no new ground with respect to worker risks. It uses the same occupational handler scenarios, exposure assumptions, and inputs as the 2014 assessment and makes comparable risks findings with a few modest tweaks. Like the 2014 risk assessment, the 2020 one uses an underprotective endpoint and fails to protect children from neurodevelopmental harm. EPA's failure to use an endpoint to protect against neurodevelopmental harm to children exposes pregnant women to levels of chlorpyrifos that could cause serious learning disabilities and reduced IQ in their children.

While the 2020 HHRA conducts the risk analysis both retaining and dispensing with the FQPA 10X safety factor, the recently issued SAP report eviscerates any argument that EPA can eliminate the FQPA 10X safety factor for chlorpyrifos or any other organophosphate pesticides. These comments therefore address only the risk assessments with retention of the FQPA 10X.

1. Handlers

Using the underprotective 10% cholinesterase inhibition endpoint, EPA found pervasive unacceptable risks to workers. EPA identified 119 non-seed treatment scenarios with risks of concerns for handlers and an additional 45 scenarios that present risks of concern that EPA believed could be mitigated with engineering controls. 2020 HHRA at 52. EPA also identified 22 seed treatment scenarios with risks of concern with current PPE requirements and when the 10X is retained. *Id*.; Appendix 10-2.

The magnitude of the risks documented by EPA is breathtaking for many uses. EPA's occupational risk assessments focus on acute poisonings. To determine risks of concern, EPA purports to identify a no-adverse-effect exposure level. For organophosphates, EPA identified 10% red-blood cell cholinesterase inhibition as an effect that would be short of what would poison the worker. Of course, this is not a no observable adverse effect level for neurodevelopmental harm to children, which occurs at lower exposure levels. To prevent exposures that cause 10% cholinesterase inhibition, EPA uses safety factors that it multiplies by the no-adverse-effect level. For chlorpyrifos, EPA set the margin of exposure ("MOE") at 100 based on its retention of the FQPA 10X and its elimination of the inter-species 10X due to its use of the Dow model, which uses human data. A margin of exposure that is less than 100 poses a risk of concern. The smaller the MOE, the closer the expected exposure is to the level that causes 10% cholinesterase inhibition, an unacceptable outcome from a public health perspective.[105]

[105] EPA uses the terms Level of Concern ("LOC") and MOE in assessing worker risks. EPA determines the LOC by multiplying safety factors. For example, if EPA applies a 10X safety factor for interspecies variability (extrapolating from a rodent study to human risk), and another 10X for intraspecies variability (differences between individual people across a diverse population), multiplying the two produces a total LOC of 100. When a 10X to protect children is added, the LOC becomes 1000X. The MOE is calculated as the point of departure divided by the actual or projected environmental exposure of interest. If the MOE is less than the LOC, EPA finds a risk of concern.

In its 2020 risk assessment, as in its 2014 one, EPA often found risks an order of magnitude more severe than its MOE. For example, EPA identified MOEs that were less than 10 for certain types of applications to almonds, walnuts, pecans, apples, corn, cotton, alfalfa, cherries, strawberries, citrus, asparagus, sugar beets, soybean, and wheat. Appendix 10-1. The risks from airblast applications are alarming, with some MOEs of less than 5 and some even less than 1. *Id*. Recalling that the MOE is designed to prevent exposures close to the regulatory endpoint, an MOE of 10 poses a tenfold greater risk than an MOE of 100, and an MOE of 1 exposes the worker to the actual dose that EPA's regulatory approach is designed to avoid. The 2020 HHRA continued to find that certain types of application methods are particularly dangerous, including aerial spraying, chemigation, air blast spraying, and ground boom applications.

2. Field Workers

EPA found risks of concern to field workers who enter the fields to perform various tasks. To protect field workers, EPA establishes prohibitions on entering the fields during a re-entry interval ("REI") after the pesticide application. Most chlorpyrifos labels have a 1-day REI, although some have an REI of up to 5 days.

The 2020 HHRA found that, with the FQPA 10X, most post-applications scenarios are not of concern after 24 hours. 2020 HHRA at 53. However, some activities such as irrigation, hand harvesting, scouting, and thinning result in risks of concern for many additional days. *Id*. at 53. The PID indicates that 30 activities lead to risks of concern without longer REIs. PID at 59. For the 11 crops uses that the PID suggests could be retained, longer REIs would be needed as follows to avoid unacceptable post-application risks to field workers:

Crop	REI Needed To Avoid Risks of Concern
Strawberries	4 days
Corn	1-3 days
Apples	2-5 days
Cherries	2-5 days
Peaches	2-6 days
Sour cherries	2-6 days
Citrus	2 days
Alfalfa	2 days
Cotton	2-4 days

2020 HHRA at 54-58 & Appendix 11; PID Appendix D1 & D2. Various activities in forestry and on ornamental trees outdoors would need REIs of 2-5 days. PID at 98, 100-01, 104-06 Appendix D2.

For ornamental production in greenhouses, EPA found that an REI increase of up to 5 days may be needed to alleviate risks of concern to workers from chlorpyrifos exposures. Appendix 11. EPA also assessed exposures to the chlorpyrifos oxon in greenhouses because of

the formation of the oxon in indoor environments, slower deactivation indoors, and the greater toxicity compared to the parent chlorpyrifos compound. 2020 HHRA at 59; *see also* 2014 Updated Occupational Assessment at 11; 2015 Proposed Revocation, 80 Fed. Reg. at 69,082. While the 2011 HHRA sought additional data to measure chlorpyrifos oxon residues on leaf surfaces, no such data have been provided. EPA estimated oxon concentrations instead of using actual data. EPA estimates that the REI may need to be up to 6 days to alleviate chlorpyrifos oxon risks from non-microencapsulated formulations and possibly more than 35 days for microencapsulated formulations. 2020 HHRA at 60-61; 2014 Updated Occupational Assessment at 11, 40-41. These risk calculations are based on dermal exposures only, even though EPA recognizes that inhalation exposures must be considered for greenhouse exposures. 2020 HHRA at 61-62.

C. EPA's Worker Risk Assessment Understates Worker Exposures.

In addition to dramatically understating the risks because it uses the underprotective 10% cholinesterase inhibition regulatory endpoint, EPA's 2020 worker risk assessment fails to account for all of the ways workers are exposed to chlorpyrifos.

1. EPA's Assumptions Under-Estimate Worker Exposures.

EPA under-estimates risks to workers because it makes assumptions that are at odds with the real-life circumstances of the workers. For example, EPA assumes a body weight of 152 pounds, yet many female workers weigh less than that. It also assumes an 8-hour work day and 5-day work week. In 2008, the USDA reported that 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 worked more than 40 hours per week.[106] 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.[107] One of the reported incidents in California involved a mixer/loader/applicator who became sick after handling chlorpyrifos for 9 days, and another involved an applicator who became sick after several weeks of 14-hour days. California Case 2007-571 and 1996-1293. Not only do farmworkers work longer days and weeks than EPA assumed, but their exposures do not stop at the end of work day because most workers lack access to showering and laundering facilities that would end the exposure and prevent the workers from taking home chlorpyrifos-laden dust on their clothing.[108]

[106] William Kandel, USDA, *A Profile of Hired Farmworkers, A 2008 Update* at 16 (July 2008), http://www.ers.usda.gov/publications/err-economic-research-report/err60.aspx.
[107] 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.
[108] Quirina M. Vallejos *et al.*, Migrant Farmworkers' Housing Conditions Across an Agricultural Season in North Carolina, 54 Am. J. Indus. Med. 533 (2011) (most labor camps in North Carolina lacked

2. EPA Overstates the Efficacy of Protective Clothing and Gear.

EPA over-estimates the efficacy of protective gear in the face of evidence that significant exposures remain from mixing, loading, and applying pesticides. For example, although the Worker Protection Standard allows 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.[109] "Black light and fluorescent tracers dramatically demonstrate the extent to which pesticide exposure may occur, even with the use of PPE."[110] 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."[111]

Protective clothing and gear can increase heat and respiratory stress. When promulgating the revised Worker Protection Standard, EPA found that "heat stress can be a problem for workers…when employees must wear PPE." 80 Fed. Reg. at 67,527. 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.[112] Thus, if pesticide handlers wore full PPE while mixing and loading pesticides, there would be a real risk that heat stress symptoms would reduce their alertness, creating a potential hazard, or otherwise cause physical harm.[113]

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; the most common reason was that the employer

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adequate bathing and laundry facilities); 80 Fed. Reg. at 67,533 (Worker Protection Standard revisions do not require that employers provide showers for handlers).

[109] Washington State Department of Health Pesticide Incident Reporting & Tracking Panel, 2000-2001 Annual Report (2002), http://www.doh.wa.gov/Portals/1/Documents/Pubs/334-293.pdf; *see also* Washington State Department of Health., Pesticide Incident Reporting & Tracking Panel, 2009, Annual Report 61-64 (2009).

[110] Frederick M. Fishel, Exposing Pesticide Exposure Using Fluorescent Tracer Dyes (2014), available at http://edis.ifas.ufl.edu/pdffiles/PI/PI19900.pdf.

[111] 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).

[112] 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).

[113] *Id.*

did not provide it.[114] 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.[115]

3. The Worker Risk Assessment Fails to Account for Direct Pesticide Drift.

EPA's worker risk assessment and PID ignore exposures from direct pesticide drift. Every year, workers are poisoned by pesticides when they move offsite onto neighboring fields and people inhale the droplets, particles, or vapors that have drifted offsite. The California Pesticide Illness Database identified 147 pesticide incidents in 2013 from drift, and 1297 from 2001-2013.[116] In Washington, 150 workers were impacted by drift incidents in 2007-2011, and the number of reported drift incidents increased substantially from 2012 to 2014.[117]

EPA ignores field workers' exposure to drift because the pesticide labels prohibit spraying pesticides directly on people by including a "do not contact" instruction. EPA, Pesticides; Agricultural Worker Protection Standard Revisions, 80 Fed. Reg. 67,496, 67,521 (Nov. 2, 2015). EPA acknowledges that the label "do not contact" direction and other label requirements are not "by themselves sufficient to protect workers and bystanders from being directly contacted by pesticides that are applied," *id.*, yet it does not account for these exposures and drift incidents in its risk assessments. Instead, EPA looks only at drift that deposits in a field and exposures to the pesticide residues from later touching the treated crop. This approach captures only a small fraction of the harm from pesticide drift and ignores the reality that the "do not contact" label statement is inadequate to prevent drift-induced poisonings and neurodevelopmental effects.

Indeed, in strengthening the work protection standard in 2015, EPA found that the "do not contact" and other label requirements are not "by themselves sufficient to protect workers and bystanders from being directly contacted by pesticides that are applied." *Id*. EPA deemed it necessary to afford additional protection in the form of application exclusion zones ("AEZs") that prohibit workers from being in treated areas during pesticide applications. *Id*. at 67,521-22 ("additional measures are needed" because the label "do not contact" language has proven "insufficient" to prevent worker exposures to drift).

EPA adopted a provision requiring that applications be suspended when workers or other people not handling the application are present in an AEZ. The AEZ is 100 feet for aerial spraying, air blast application, use as a fumigant, smoke, mist, or fog, and 25 feet for some other application methods. 40 C.F.R. §§ 170.405, 170.505. However, on October 30, 2020, EPA finalized amendments that weaken the AEZ requirements by limiting them to the boundaries of the farm where the spraying is taking place. 85 Fed. Reg. 68,760 (Oct. 30, 2020). Even though it has been weakened, EPA's adoption of AEZs demonstrates that it cannot rely on a "do not

[114] Washington PIRT 2010 Report, at 61.
[115] Washington PIRT 2010 Report, at 62.
[116] http://apps.cdpr.ca.gov/calpiq/calpiq_detail.cfm (bystander drift incidents in 2013 involving agricultural spraying of pesticides).
[117] *Pesticide Data Report Washington State* at 28.

contact" label mandate to prevent harm to workers when common practices are insufficient to prevent such harm.

FIFRA requires EPA to ensure that a pesticide use will not cause unreasonable adverse effects "when used in accordance with widespread and commonly recognized practice." 7 U.S.C. § 136a(c)(5). When it required no-spray buffers around schools, homes, and other places people gather, EPA recognized that widespread and common practices were insufficient to prevent exposures to toxic drift at these locations. EPA made a similar determination when it required AEZs. EPA needs to undertake an analogous assessment of the propensity of pesticides to drift to adjacent fields and contact people directly, not only through subsequent dermal contact with deposited residues. It has refused to do so, calling such drift incidences misuse or enforcement matters. 84 Fed. Reg. at 35,567 (order denying chlorpyrifos objections). Pesticide handlers must wear PPE because of the serious risks of concerns they face. Field workers, who are not required to wear PPE, may be working in areas where pesticide drift may travel. EPA must examine how far toxic drift will travel and require measures that will prevent harmful exposures to workers in harm's way.

4. EPA Fails to Account for Aggregate and Cumulative Exposures.

EPA also understates the risks because it has not accounted for all aggregate exposures. To set or maintain a tolerance, EPA must determine that the pesticide is "safe," meaning "there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information." 21 U.S.C. § 346a(b)(2)(A)(ii). This provision requires that EPA consider all aggregate exposures to food use pesticides in setting tolerances. *See also id.* § 346a(b)2)(C)(ii)(I) & (D)(vi). EPA must also assess cumulative exposures to all organophosphates because they have a common mechanism of toxicity. 21 U.S.C. § 346a(b)(2)(C)(i)(III), (D)(v).

These standards are incorporated into FIFRA for food uses of pesticides. 7 U.S.C. § 136(bb). In addition, a 2009 EPA policy determined that these risk assessment approaches had become the norm and "'sound science' now calls upon [EPA] to consider such risk assessment factors for any pesticide risk assessment." EPA, Revised Risk Assessment Methods for Workers, Children of Workers in Agricultural Fields, and Pesticides with No Food Uses at 2 (Dec. 7, 2009), https://www.regulations.gov/document?D=EPA-HQ-OPP-2009-0889-0002. More specifically, EPA must consider aggregate and cumulative exposures for nonfood pesticide uses as well as food uses, and it must consider aggregate and cumulative exposures to workers and their families.

a. Aggregate exposures

In addition to their exposures to chlorpyrifos at work, workers are exposed to chlorpyrifos drift when they leave the fields and go home or gather with their families or communities in places in close proximity to the fields. Farmworkers are likely to be within the zone of danger for pesticide drift because of where they work and often where they live to be

close to their work. EPA must aggregate workers' exposures through their jobs with the exposures they face from pesticide drift. And they, like other consumers, are exposed to chlorpyrifos on their food and in their drinking water.

Drinking water contamination is a greater risk for drinking water sources near the fields. EPA must assess the combined risks to workers from all exposures. By way of example, the PID proposes to allow uses of chlorpyrifos to continue on tart cherries, citrus, and peaches. EPA's risk assessment documents unacceptable risks from use of chlorpyrifos on each of these crops for pesticide handlers from most application methods and for field workers entering the fields to perform pruning, scouting, and transplanting as well as thinning and harvesting for one of the crops. 2020 HHRA Appendix 10-1; Appendix 11. EPA must consider the combined risks workers face from their work and the air they breathe, the food they eat, and the water they drink.

b. Cumulative exposures

EPA must also assess cumulative exposures to chlorpyrifos and other organophosphates because organophosphates have a common mechanism in that they all suppress cholinesterase and cause acute pesticide poisonings. For that reason, EPA conducted a cumulative organophosphate risk assessment in 2006 as part of its re-registration process. Moreover, in September 2015, EPA determined that the scientific evidence documents neurodevelopmental effects on children from organophosphate pesticides as a class and that the FQPA safety factor must be retained for all organophosphates.[118]

In its registration review process, EPA is conducting risk assessments for each organophosphate pesticide individually, and it is consistently finding acute poisoning risks of concern to workers. As with chlorpyrifos, EPA has not, to date, eliminated the uses that pose such risks. As a result, workers are being exposed to the same types of risks from multiple organophosphate pesticides they handle or encounter in their work. Pesticide handlers, who face the highest risks, typically travel from one field to the next applying a variety of pesticides. They often apply one organophosphate one day and another the next. Their cumulative exposures within the 21-day period used by EPA to assess steady state risks will often be far greater than their exposure to chlorpyrifos alone.

EPA will need to assess cumulative risks from exposures to organophosphates as a class to discharge its registration review obligations, and this cumulative risk assessment will need to address neurodevelopmental harm from low-level exposures. EPA should, however, not delay taking action to protect people from chlorpyrifos during the time it will take to complete a cumulative risk assessment. Instead, it should follow the practice it employed during re-registration where it finalized interim re-registration eligibility decisions for individual organophosphate and ended uses, revoked tolerances, and required mitigation when each interim

[118] EPA OPP, Literature Review on Neurodevelopmental Effects & FQPA Safety Factor Determination for the Organophosphate Pesticides (Sept. 15, 2015), https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0119-0023.

decision was made. When EPA completes a cumulative risk assessment at a later date, it should then take additional actions to revoke tolerances and cancel or modify registrations as required by that cumulative assessment.

D. EPA Is Relying on Flawed Benefits Assessments.

EPA has prepared benefits assessments – one for crop usage and the other for non-crop usage – to use in conducting the risk-benefit balancing required to make unreasonable adverse effects determinations under FIFRA. EPA's crop benefits assessment concludes that, for most crops, there are adequate alternatives to control the pests targeted by chlorpyrifos. Crop Benefits Assessment ("Crop BA") at 5. Its assessment focuses on the additional costs of alternative registered pesticides for some uses and reduced yields and quality losses where the alternative pesticides are less effective. It presents the cost of alternative chemical pest control per acre and a range of total costs for the crop.

The non-crop benefits assessment focuses on non-crop settings like nurseries/greenhouses, turf, mosquito control, and golf courses. This assessment draws on less data on the costs of alternatives than the crop benefits assessment. For usage information, EPA relied on surveys from 2011 for turf, nurseries, and greenhouses and 2015 for mosquito control. Surveys conducted in 2016 for other registered uses reported no usage. Overall, EPA found that chlorpyrifos is no longer recommended or heavily used for important pests for most non-crop uses. It noted exceptions where chlorpyrifos constitutes a substantial percentage of the market by weight for turf (58%) or a far lower percentage in the case of nurseries and greenhouses (8.3%). EPA also focused on public health issues like ticks at golf courses or adult mosquitoes where a small percentage of chlorpyrifos is used and there are other alternatives, but EPA sees benefits in having numerous options to address pest resistant that develops to alternative chemical pesticides.

The benefits assessments are seriously flawed for three reasons: (1) the crop assessment and the livestock portion of the non-crop assessment include food uses of chlorpyrifos, but Congress adopted a health-based standard that prohibits use of a pesticide on food if EPA cannot find it safe regardless of the pesticide's economic benefits to growers; (2) EPA included uses that have been or are being banned by states; and (3) the assessments look only at other registered pesticides as alternatives and make unsupported assumptions.

1. The Crop Benefits Assessment and PID Present the Costs of Pesticide Alternatives to Chlorpyrifos for Unsafe Food Uses When Such Costs are Legally Irrelevant.

When Congress unanimously passed the Food Quality Protection Act in 1996, it adopted a uniform health-based standard for food uses of pesticides. It amended the FFDCA to require that EPA affirmatively find a pesticide safe in order to set or maintain a tolerance, and it defined "safe" to mean "there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other

exposures for which there is reliable information." 21 U.S.C. § 346a(b)(2)(A)(ii). Congress embedded this same standard into FIFRA by defining "unreasonable adverse effects" to include residues of the pesticide on food are inconsistent with the FQPA's safety standard. 7 U.S.C. §136(bb). This is strictly a health-based standard. If EPA cannot find a pesticide use safe, it cannot allow the pesticide to be used on food.

This standard is in sharp contrast to FIFRA's standard for non-food uses of pesticides, which directs EPA to take into account "the economic, social, and environmental costs and benefits" of the pesticide use. *Id*. No such balancing comes into play for food uses.

The crop benefits assessment recites the "reasonable certainty of no harm" standard and acknowledges that it establishes a more stringent standard for pesticides used on food. Crop BA at 3. It nonetheless quantifies the costs of using other pesticides and presents the costs as if they have a role to play in all decisions before the agency.

To be relevant to and inform the decisions before EPA, the benefits assessment must be tied to EPA's health risk assessment for food uses. In that assessment, EPA struggles to find risks that are below levels of concern by shrinking or eliminating safety factors, but admits it cannot do so for most food uses. That finding answers the only question before EPA under the FFDCA and FIFRA for these food uses of chlorpyrifos; chlorpyrifos is unsafe and cannot be used. The crop benefits assessment has no bearing as to such uses. *See, e.g.*, Crop BA at 20, 22, 27, 29 (broccoli, cabbage, cauliflower, sweet cherries, cranberries, grapes).

For a discrete set of food uses, EPA purports to find that risks are below its risk of concern level. These findings are invalid because harm to children's brains occurs below EPA's regulatory endpoint. If, however, some exposures could be safe, EPA would need to determine the total amount of exposure that would be safe, which EPA previously did by calling this amount of exposure a risk cup. A crop benefits assessment could then inform which uses and types of aggregate exposures should continue and which should be discontinued, provided the total exposures would fit within the risk cup.

2. The Crop Benefits Assessment and PID Consider the Costs of Shifting Away from Uses Banned or Soon-To-Be Banned by States.

When EPA refused to finalize the revocation of chlorpyrifos tolerances in 2017, states stepped in to fill the void. EPA sets tolerances and states are powerless to keep foods with chlorpyrifos residues from entering their states. However, states retain the power to regulate pesticide use, which includes the power to ban, phase-out, or otherwise impose limits on a pesticide's use to protect people or the environment in the state. Many states have exercised this authority with respect to chlorpyrifos.

Hawaii was the first state to act, passing a law that phases out chlorpyrifos in that state.[119] In California, the state ended almost all (99%) uses of chlorpyrifos by the end of 2020. *Supra* n.32. Oregon recently finalized a chlorpyrifos phase-out and the development of such a phase-out in New York is well underway.[120] Several other states are considering taking similar actions to phase out chlorpyrifos.[121]

Despite these bans and phase-outs, the crop benefits assessment includes the costs of shifting to alternative pesticides in these states. For example, it includes in its cost estimates what it dubs the "high" costs of shifting to alternatives to chlorpyrifos use on California oranges and lemons. Crop BA at 2, 5, 32, 35-37, 39, 43, 55; *see also* Crop BA at 15-16 (alfalfa and almonds); Crop BA at 19 (asparagus); Crop BA at 25-26 (cotton); Crop BA at 34 (mint). It does so even though it acknowledges that, in light of the California phase out, these costs reflect the past and not any future costs of phasing out chlorpyrifos. Crop BA at 2. The crop benefits assessment similarly focuses on the use of chlorpyrifos on strawberries to address a pest present only in Oregon, *see* Crop BA at 12, 13, 46-48, even though Oregon is phasing out chlorpyrifos use on strawberries along with almost all other crops by December 31, 2023. Permanent Chlorpyrifos Rule, OAR 603-057-0545 (adopted December 15, 2020); *see also* Crop BA at 17 (apples in New York); Crop BA at 34 (mint in Oregon).

3. The Crop Benefits Assessments Suffer from Other Methodological Flaws.

EPA based its assumptions about chlorpyrifos use on surveys in which growers identified the pesticides they use on which crops and the pests they are targeting. Crop BA at 11. In other words, past grower preferences and practices as reflected in the surveys became the baseline.

For alternatives, EPA looked only at chemical pesticides that are currently registered for use on the crops. In this way, EPA discounted non-pesticide alternatives and the prospect that new alternatives would be developed. Crop BA at 11, 13. 18. A credible economic assessment of benefits must consider the full range of alternatives, which would not only include alternative registered chemical pesticides, but also biological controls, integrated pest management, and organic approaches.

Where there is a less costly alternative, EPA did not consider it because it assumed a grower would choose to use a less expensive chemical if it were as effective. Crop BA at 11. The pitfalls of EPA's alternatives assumptions have borne out over time. For two crops

[119] Haw. Sen. Bill No. 3095, 29th Leg. (2018), https://www.capitol.hawaii.gov/session2018/bills/SB3095_CD1_.htm (prohibitions imposed in 2019 with temporary permits possible through 2022).
[120] Or. Admin. R. 603-057-0545 (2020), Limitations on Pesticide Products Containing Chlorpyrifos; Chlorpyrifos Rulemaking, https://www.dec.ny.gov/docs/materials_minerals_pdf/chlorpyrifos.pdf (notice of New York proposed rulemaking that would effectively ban chlorpyrifos use in the state).
[121] The comments submitted by New York and other states describe (at 19-25) the legislative and regulatory actions being taken or proposed to ban or restrict chlorpyrifos in additional states, as well as in other countries.

(Brassica and sugar beets), EPA predicted far greater costs of alternatives in 2016 than turned out to be the case. Crop BA at 5. In fact, for Brassica crops (broccoli, cabbage, and cauliflower), growers told EPA there were no feasible alternatives, but they have since largely stopped using chlorpyrifos, indicating that alternatives indeed are available. Crop BA at 7-8, 20.

4. EPA Considered Benefits on Crops Beyond Those Warranted by its Benefits Assessment.

Based on its crop benefits assessment, EPA identified a set of what it calls "high benefit uses," consisting of apples and soybeans nationwide, asparagus in Michigan, tart cherries in Michigan, peaches in in Georgia and South Carolina, and strawberries in Oregon. PID at 39, 42; Crop BA at 2. Its rationale for deeming a use "high benefit" is the lack of pesticide alternatives, substantially more expensive alternatives, or less efficacious alternatives. Crop BA at 2; PID at 39.

In its drinking water assessment and PID, EPA tries to justify retaining additional uses based on benefits that are not supported by the benefits assessment. Specifically, EPA considered an additional set of crops that Corteva identified as "critical uses." The criteria used by Corteva to make that designation have not been disclosed. Corteva identified the following as critical uses: alfalfa, citrus, cotton, soybean, sugar beets, and wheat.

Corteva's "critical use" crops include alfalfa and wheat, where, according to the crop benefits assessment, use of alternative pesticides would result in only a relatively small additional cost (*e.g.*, $0-$1 per acre) and chlorpyrifos is currently used as one of many pesticides used to produce those crops. Crop BA at 6-7; *see also id.* (soybeans estimated $1-$4 per acre; cotton at $0-$14 per acre).

The PID also calls out additional crops that have high usage of chlorpyrifos measured in pounds per year, even if only a small percentage of the crop is treated with chlorpyrifos. PID at 11. For example, a large quantity of chlorpyrifos is used on corn, but only 6% of corn nationwide is treated with chlorpyrifos. *See id*. (corn, almonds, grapes, peanuts, pecans, walnuts). EPA's crop benefit assessment did not identify these uses as "high benefit uses," yet EPA nonetheless included them in its drinking water assessment because Corteva called them "critical."

Moreover, as discussed below, the PID justifies continuing to expose workers to risks of concern by claiming the use is a "high benefit use," even where that characterization is not borne out by the crop benefit assessment. This justification is contrary to EPA's crop benefits assessment and lacks support in the record.

E. The PID Proposes to Retain Chlorpyrifos Uses and Continue Exposing Workers to Unacceptable Risks from Chlorpyrifos.

At long last, EPA is proposing to take steps to reduce worker risks, but the proposed steps are far too little and tragically come after years of delay and inexcusable harm. The PID contains two sets of food use cancellations drawn from the health risk assessment. The first would cancel all uses except the 11 food uses for which the drinking water assessment purported to find no risks of concern with the FQPA 10X. The second would retain additional uses based on the elimination of the FQPA 10X. For this latter set of uses, EPA was keeping the door open to retaining tolerances and registrations based on the new scientific methods it floated last year, but the SAP recommendations closed that door. Retaining these uses lacks any rational basis.

The PID proposes additional PPE and engineering control requirements for all crops, even the food uses that it acknowledges must be eliminated. Since such uses cannot continue, these comments will focus only on the 11 food crops that the health risk assessment purports to find pose no risks of concern, as well as nurseries and greenhouses that pose high risks for workers and that the PID suggests may continue.[122] As explained above, EPA's health risk assessment is underprotective as to these uses because EPA used 10% chlolinesterase inhibition as the regulatory endpoint and improperly reduced safety factors based on the use of Dow's model, when chlorpyrifos harms children's brains at exposures far lower than 10% cholinesterase inhibition.

1. EPA Is Proposing to Retain Uses Based on Economic Benefits to Growers Without Balancing those Benefits Against the Harm to Human Health and the Environment.

The PID approaches handler risks in two ways: (1) crop-by-crop; and (2) by particularly harmful application methods. These comments will first address the crop-by-crop proposals and then the application methods.

In its crop-by-crop risk assessment and proposals, EPA identifies risks of concern from use of chlorpyrifos on many crops and the mitigation it believes is needed to reduce or eliminate such risks. For others posing comparable risks of concern, EPA calls the use a "high benefit use," implying that this characterization is the end of the matter.

EPA's discussion of citrus is illustrative. It identifies risks of concern for airblast applications without closed cabs as ranging from 0.55 to 4.2 MOE (far more than an order of magnitude greater than EPA's level of concern). Requiring the use of closed cabs still results in

[122] While EPA acknowledges that it can retain food uses only if there are no risks of concern, PID at 40-41, it inexplicably has a section recommending changes to all existing tolerances to reflect rounding practices and even to add new tolerances for several uses. Of course, EPA cannot retain or add tolerances for the many uses it does not even purport to find safe.

risks of concern, but EPA justifies continued use of chlorpyrifos on citrus with closed cabs because it calls it a "high benefit use." PID at 43.

EPA adopts the very same rationale for pecans with the same result – it proposes to allow continued airblast applications of chlorpyrifos on pecans in closed cabs because it dubs pecans a "high benefit use." PID at 43. The defect in this rationale is that the crop benefits assessment did not find pecans a "high benefit use." Indeed, Corteva did not even include pecans among the uses it called "critical."

The PID is indiscriminate in justifying continuing uses by calling them "high benefit uses." This conclusory statement is attached to all uses, regardless of whether the crop benefits assessment supports this characterization or even whether Corteva characterized the use as critical. *See, e.g.*, Crop BA at 44 (justification for allowing ground boom applications on radishes with closed systems even though risks of concern remain). Sometimes EPA makes this cryptic, conclusory statement for an individual crop. And for the remainder, it provides tables identifying the MOEs for crops with additional PPE or engineering controls with a footnote indicating that every use where the MOEs would continue to exceed the level of concern is a "high benefit use." Crop BA at 43-50.

Not only does EPA's characterization of most crops as high benefit lack rational support in the record, but basing its decision solely on this characterization also runs afoul of the law. Even where EPA is relying on findings in its benefits assessments, EPA is proposing to allow risks of concern to workers to continue based solely on its assessment of the benefits of chlorpyrifos to growers or for other types of pest control. Its characterization of uses that will continue to pose risks of concern as "high benefit uses" constitutes EPA's entire rationale for exposing workers to risks of concern, cutting off any further inquiry.

Under FIFRA, however, EPA cannot rely solely on the benefits of a pesticide for a particular use. FIFRA requires EPA to prevent unreasonable adverse effects on health and the environment, "taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." 7 U.S.C. § 136(bb). Once EPA finds risks of concern, EPA must cancel the pesticide use unless it finds that the benefits of continued use outweigh the risks. *Envtl. Defense Fund, Inc. v. EPA*, 548 F.2d 998, 1012 (D.C. Cir. 1976). To make this finding, EPA must engage in a balancing of the risks and the benefits. EPA has not done so. It recites the mantra that the use is "high benefit," regardless of the magnitude of the risks workers face, the number of workers at risk, or the serious and irreversible harm chlorpyrifos can cause to children.

EPA similarly gives undue weight to relatively small costs of engineering controls that can reduce harm to workers. EPA believes risks of concern from many mixing and loading activities would be reduced or eliminated with the use of closed mixing and delivery systems. It estimates that the cost of such a system is around $300, but EPA believes growers would be unwilling to incur this expense if chlorpyrifos is the only chemical uses in the field that would require a closed system. PID at 54. EPA treats a $300 expense as the end of the story without

considering it in the context of the horrific and extreme harm chlorpyrifos causes to workers and their families.

2. In Addressing Risks and Benefits, EPA Sidelines the Harm to People and the Environment.

EPA's benefits assessments focus myopically on the cost of alternative chemical pesticides to growers and other pesticide users. The crop benefits assessment comes up with the costs per acre and an estimate of aggregated annual costs. But it stops there.

Even though chlorpyrifos causes acute poisonings and damage to children's brains and EPA has found risks of concern to workers and bystanders from chlorpyrifos exposures, it never quantifies the harms to human health. It lacks any basis to make registration review decisions based on a quantitative balancing of both the risks and the benefits. Nor did it purport to undertake any qualitative balancing.

For all nonfood uses and worker risks, EPA must consider all of the risks and benefits from the pesticide. When UFW, Farmworker Justice, NRDC, Earthjustice, and others challenged EPA's reregistration of other organophosphates – azinphos-methyl and phosmet – Dr. Frank Ackerman, then the Director of the Research and Policy program at the Global Development and Environment Institute at Tufts University, submitted two expert economics declarations critiquing EPA's benefits assessment. First and Second Declarations of Dr. Frank Ackerman, filed in *UFW v. EPA*, No. CV04-0099-RSM (W.D. Wash. filed 2005 and 2007) (Ex. 7 & 8.) Dr. Ackerman included his analysis in his book *Poisoned for Pennies: The Economics of Toxics and Precaution* at 113-28 (2008).

Dr. Ackerman found that "EPA's minimal effort to conduct a cost benefit analysis was completely inadequate and that the agency's analysis fails to utilize relevant information and does not provide a sound basis for making a risk-benefit determination." Ackerman Decl. ¶ 7. More specifically:

> EPA fails to account adequately for the costs to farmworkers and their families associated with continued use of AZM and phosmet. The agency mentions impacts on farmworkers, but makes no attempt to determine whether the costs to farmworkers outweigh the benefits to growers. Instead, the EPA analysis simply cites the benefits to growers as justification for the continued use of the pesticides. It is not clear whether EPA believes that the benefits to growers obviously exceed the value of the costs to farmworkers, or whether the agency has chosen to consider only one side of the equation. Either alternative is of course unacceptable; explicit evaluation of the effects on farmworkers is necessary. By failing to discuss the comparison between benefits to growers and costs to

> workers, EPA's current treatment of the issue implies that the value of workers' health is negligible or irrelevant.

Id. ¶ 9.[123]

In order to conduct risk-benefit balancing for chlorpyrifos, EPA must account for the full impacts to workers and their families and the impacts to wildlife and the environment. For chlorpyrifos, EPA must address the impacts of acute poisonings, neurodevelopmental harm to children, and harm to salmon and other wildlife.

a. Acute poisonings

Chlorpyrifos causes acute poisonings of workers and bystanders every year. The precise number of poisonings is unknown as there is no comprehensive pesticide incident reporting network and the reports that are made represent only a fraction of the poisonings that occur.

Nonetheless, EPA must address acute poisonings in its risk-benefit balancing. When workers become sick from acute poisonings, they may need medical care, miss work, and bring residues of chlorpyrifos home on their clothing where they can make family members sick. Since some medical costs are paid for by Medicaid or subsidized care at federally subsidized migrant clinics, the health costs imposed by pesticides are also, in part, costs that are borne by the nation as a whole. Ackerman Decl. ¶ 23. Some people experience long-term neurological and neuropsychological impairment in the poisoned individuals.[124] Such impairments can lead to reduced productivity or additional medical costs, which must be accounted for.

b. Neurodevelopmental Harm to Children

Since chlorpyrifos also causes damage to children's brains, autism, attention deficit/hyperactivity disorder, reduced IQ, and other learning disabilities, EPA must account for this harm in its risk-benefit balancing. The social costs of learning disabilities have been

[123] Further assessments of azinphos-methyl led EPA to phase out its use in the United States by September 2013.

[124] Savage, Eldon P., *et al.*, "Chronic Neurological Sequelae of Acute Organophosphate Pesticide Poisoning." Archives of Environmental Health, Vol. 43, No.5 (1988); Steeland, Kyle, *et al*., "Chronic Neurological Sequelae of Organophosphate Pesticide Poisoning." *American Journal of Public Health*, Vol. 84, No. 5 (May 1994); Reidy, Thomas J., *et al*., "Pesticide Exposure and Neuropsychological Impairment in Migrant Farm Workers." *Archives of Clinical Neuropsychology*, Vol. 7, pp.85-95 (1992); Rosenstock, Linda, *et al*., "Chronic Central Nervous System Effects of Acute Organophosphate Pesticide Intoxication." *The Lancet*, Vol. 338 (July 27, 1991); Lundberg, Miranda, *et al*., "Onset of Grip and Pinch Strength Impairment After Acute Poisonings with Organophosphate Insecticides." 8 *Intl. J. Occupational Envtl. Health* 1 (2002); Wassailing C., *et al*., "Long-term Neurobehavioral Effects of Mild Poisoning with Organophosphate and N-Methyl-Carbamate Pesticides among Banana Workers." *International Journal of Occupational and Environmental Health*, 8(1); 27-34; Jamal, G.A., "Neurological Symptoms of Organophosphorous Compounds." *Adverse Drug React Toxicol Rev*, Vol. 16, pp. 133-70 (1997).

described by prestigious physicians in a scientific paper published in Lancet Neurology: "Neurodevelopmental disabilities, including autism, attention-deficit hyperactivity disorder, dyslexia, and other cognitive impairments, affect millions of children worldwide, and some diagnoses seem to be increasing in frequency. Industrial chemicals that injure the developing brain are among the known causes for this rise in prevalence… All these disabilities can have severe consequences - they diminish quality of life, reduce academic achievement, and disturb behavior, with profound consequences for the welfare and productivity of entire societies."[125] The contribution of pesticides, and particularly those that impair acetylcholine, are specifically called out by the authors: "Some pesticides inhibit cholinesterase function in the developing brain, thereby affecting the crucial regulatory role of acetylcholine before synapse formation."[126]

These disabilities have economic consequences. For example, it costs more to educate a child with a learning or developmental disability. Parents lose workdays when they need to care for children who have special needs.

In addition, lifetime productivity losses (in terms of future income foregone) can be estimated. Dr. Philip Landrigan, a leading pediatrician and epidemiologist who is a member of the faculty of the Icahn School of Medicine at Mount Sinai, submitted a declaration in support of the 2016 petition to cancel and suspend chlorpyrifos uses, attached as Exhibit 2. Dr. Landrigan described ways to quantify the costs of brain impairments:

> Preventing exposures to chemicals can yield great economic savings. While it is difficult to precisely quantify the harm from neurodevelopmental disorders and the cost savings that result from their prevention, several studies suggest that both are quite large. To estimate the contribution of environmental pollutants to the prevalence and costs of disease in American children, investigators at Mount Sinai School of Medicine examined four categories of illness: lead poisoning, asthma, cancer, and neurobehavioral disorders. Based on prevalence, the environmentally attributable fraction of each disease, and national economic data, they calculated that the total annual costs of these diseases attributable to environmental exposures is $54.9 billion (range $48.8 billion to $64.8 billion): $43.4 billion for lead poisoning, $2.0 billion for asthma, $0.3 billion for childhood cancer, and $9.2 billion for neurobehavioral disorders. Because of the difficulties inherent in assessing the full economic consequences of neurobehavioral impairments, it is likely that these estimates are low.

[125] Grandjean P, Landrigan PJ. Neurobehavioural effects of developmental toxicity. Lancet Neurol. 2014 Mar. 13(3):330-8. doi: 10.1016/S1474-4422(13)70278-3. Epub 2014 Feb 17. PMID: 24556010; PMCID: PMC4418502. https://www.thelancet.com/action/showPdf?pii=S1474-4422%2813%2970278-3.
[126] *Id.*

> After the phase-out of lead in gasoline from 1976 and 1990, the mean blood lead level of American children decreased by more than 90% (to below 2 micrograms per deciliter today), and the incidence of childhood lead poisoning also fell by more than 90%. A further consequence of the reduction in exposure to lead was that the mean IQ of American children has increased. Children born in the United States today are estimated to have IQ scores that, on average, are 2.2–4.7 points higher than those of children born in the early 1970s. And because each 1-point gain in population mean IQ is associated with an estimated 2% increase in productivity over a lifetime, the gain in population IQ is estimated to have produced a national economic benefit of $110–$319 billion in each annual cohort of babies born in the United States since the 1980s.

Id. ¶¶ 34, 35. Bellanger *et al.* (2015) calculated the annual costs to European Union populations at €146 ($171) billion from IQ losses due to chlorpyrifos and other organophosphate exposures during pregnancy.[127] A similar calculation from the U.S. by Attina *et al.* (2016) suggested annual costs of $45 billion.[128] Building on these data, in 2020, a team led by physician and policy expert, Dr. Leo Trasande valued the cost to the U.S. economy from neurodevelopmental disabilities due to OPs alone from 2001 to 2008 to be roughly 26.6 million lost IQ points, with an associated economic loss of around $30-50 billion annually.[129]

c. Environmental Harm

EPA also fails to account for environmental harm from chlorpyrifos. It has released a draft ecological risk assessment ("ERA"), which finds that chlorpyrifos is toxic to mammals, birds, and fish, with citrus and tart cherries posing some of the highest risks. ERA at 4. It also notes that chlorpyrifos is associated with "notable incidents" such as significant fish kills, large numbers of bird deaths, and bee kills. *Id.*

[127] Bellanger M, Demeneix B, Grandjean P, Zoeller RT, Trasande L. Neurobehavioral deficits, diseases and associated costs of exposure to endocrine disrupting Chemicals in the European Union. J Clin Endocrinol Metab. 2015;100(4):1256–66 (Ex. 9).

[128] Attina TM, Hauser R, Sathyanarayana S, Hunt PA, Bourguignon J-P, Myers JP, DiGangi J, Zoeller RT, Trasande L. Exposure to endocrine-disrupting chemicals in the USA: a population-based disease burden and cost analysis. Lancet Diabetes Endocrinol. 2016;4(12):996–1003 (Ex. 10).

[129] Gaylord A, Osborne G, Ghassabian A, Malits J, Attina T, Trasande L, *Trends in Neurodevelopmental Disability Burden Due to Early Life Chemical Exposure in the USA from 2001 to 2016: A Population-Based Disease Burden and Cost Analysis*, 502 Mol. Cell. Endocrinol. 110666 (Feb. 15, 2020), doi: 10.1016/j.mce.2019.110666 (using EPA standard assumptions that each IQ point loss incurs an economic cost of $22,268), https://bit.ly/3fik1gZ. (Ex. 11).

The environmental harms from chlorpyrifos are pervasive. Surface waters in many states are in violation of water quality standards due chlorpyrifos contamination.[130] Chlorpyrifos is also toxic to bees, through direct application and drift. The Washington State Department of Agriculture found chlorpyrifos to be among the insecticides involved in the majority of bee kill incidents between 1992 and 2005.[131] In 2016, EPA released its biological evaluation of chlorpyrifos, which found that chlorpyrifos may affect nearly all species on the Endangered Species Act (ESA) list.[132] The biological evaluation initiated consultation with the expert fish and wildlife agencies to determine the full extent of the harm and needed mitigation.

The consultation with NOAA Fisheries on the effects of chlorpyrifos on Pacific salmon and other marine species led to issuance of a biological opinion in January 2018.[133] In that biological opinion, NOAA Fisheries found that chlorpyrifos is likely to jeopardize the survival and recovery of listed Pacific salmon, other listed fish, and endangered Southern Resident Killer Whales, which depend on listed salmon as their prey. The biological opinion recommends protective measures to avoid jeopardizing salmon survival, including stopping use in salmon habitat and large buffer zones around salmon streams. EPA has implemented none of the recommended mitigation. Instead, in response to pressure from Dow and other registrants, it asked NOAA Fisheries to revise the biological opinion, which NOAA Fisheries has agreed to do by June 2022. *See Makhteshim Agan of N. Am., Inc. v. NMFS*, No. PWG-18-961, 2019 WL 5964526 (D. Md. Oct. 18, 2019).

EPA's incessant delays in bringing its registrations of chlorpyrifos into compliance with the ESA has economic consequences. In the Northwest (and California), chlorpyrifos is used on orchards, other crops, and for forestry in watersheds that are essential to salmon. The Washington Governor's Salmon Recovery Office's recently issued report, 2020 State of Salmon in Watersheds, documents the critical importance of salmon and orcas to that state's economy, growth, and prosperity.[134] For example, commercial and recreational fishing in Washington is estimated to support 16,000 jobs and $540 million in personal income, and an estimated $1.5 billion is spent annually on equipment and trip-related costs by people fishing and harvesting shellfish recreationally in Washington, supporting many rural families and businesses. And

[130] *See, e.g.,* California 303(d) List of Water Quality Limited Segments in the Greater Monterey County Integrated Regional Water Management Region, at http://www.greatermontereyirwmp.org/wp-content/uploads/2012/08/AppendixG_303dList.pdf; Washington Water Quality Assessment 305(b) Report and 303(d) List (approved by EPA on July 22, 2016), at https://fortress.wa.gov/ecy/approvedwqa/ApprovedSearch.aspx.
[131] http://agr.wa.gov/PestFert/Pesticides/docs/PollinatorSLNSect18.pdf.
[132] Biological Evaluation Chapters and Effects Determinations for Chlorpyrifos Endangered Species Act Assessment (April 2016) (likely to adversely effect findings for 1725 of 1782 listed species), at https://www.epa.gov/endangered-species/biological-evaluation-chapters-chlorpyrifos-esa-assessment.
[133] NOAA Fisheries, Biological Opinion on the Environmental Protection Agency's Registration of Pesticides containing Chlorpyrifos, Diazinon, and Malathion (Dec. 29, 2017), *available at* https://repository.library.noaa.gov/view/noaa/16997.
[134] http://teststateofsalmon.wa.gov/wp-content/uploads/2020/12/StateofSalmonExecSummary2020.pdf.

salmon are an important source of food, particularly for Northwest Indian Tribes who have Treaty rights to salmon.

EPA must account for these and other environmental harms in balancing the risks and benefits. And where the survival of endangered species is at stake, Congress already struck the balance in favor of protecting endangered species. *Tennessee Valley Authority v. Hill*, 437 U.S. 153, 188 n.34 (1978). Other than saying it will someday complete ESA consultations for chlorpyrifos, EPA disregards the documented harm chlorpyrifos causes to salmon and other threatened and endangered species in purporting to find that benefits to growers outweigh the risks.

3. EPA Must End Particularly Harmful Application Methods and Require the Most Effective Mitigation.

The PID appropriately comes to the conclusion that many current application methods, like aerial spraying, chemigation, airblast, and ground boom, pose unacceptable risks to workers. EPA is considering ending some application methods and requiring engineering controls or additional PPE for others. Even with the mitigation it is proposing, it admits that many risks of concern will remain. Since EPA is using an underprotective endpoint, the unacceptable risks are far greater than EPA's risk assessment indicates. EPA cannot find food uses of chlorpyrifos safe and therefore must revoke all food tolerances and cancel all food use registrations. EPA must then address risks of concern to workers from nonfood uses under FIFRA's risk-benefit balancing standard.

This section first urges EPA, in deciding on appropriate mitigation, to follow the hierarchy of controls and afford farmworkers the same level of protection as other workers. Next, it urges EPA to follow through with its proposals to end particularly harmful application methods and require the most effective mitigation for others.

a. By Failing to Prevent Unacceptable Chlorpyrifos Risks to Workers, EPA is Denying Farmworkers the Same Level of Protection Afforded Other Workers.

For most types of workers in the United States, the Occupational Safety and Health Administration ("OSHA") establishes and enforces workplace standards under the Occupational Safety and Health Act of 1970 ("OSH Act"). Mandatory OSHA standards dealing with toxic materials are required to:

> [S]et the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life.

29 U.S.C. § 655(b)(5). OSHA has promulgated many standards to protect workers from toxic chemicals on the job that would, if applicable, reduce farmworkers exposures and harm from pesticides.[135]

The OSHA standards, however, do not cover farmworkers. It is EPA, rather than OSHA, that addresses farmworkers' exposures to pesticides, and it does so under FIFRA, rather than the OSH Act. The D.C. Circuit ruled in 1975 that EPA's entry into the field preempted OSHA regulation. *Organized Migrants in Community Action, Inc. v. Brennan*, 520 F.2d 1161, 1163 (D.C. Cir. 1975). Preemption of OSHA is premised on the assumption that the substitute agency has specialized expertise in the particularized field that it will bring to bear in affording workers "comparable" occupational health and safety protection. *Baltimore & O.R.R. v. Occupational Safety and Health Review Comm'n*, 548 F.2d 1052, 1054 (D.C. Cir. 1976); *accord Ensign-Bickford Co. v. Occupational Safety and Health Review Comm'n*, 717 F.2d 1419, 1421 (D.C. Cir. 1983); *see also Reich v. Muth*, 34 F.3d 240, 243 (4th Cir. 1994) (the purpose of OSH Act's negative preemption provision is to avoid duplicative regulation by ceding responsibility for occupational standards in particularized fields to the regulatory bodies specifically tasked with their oversight and control, while leaving to OSHA the remaining general field of regulation outside specialized areas demanding specialized expertise").

In its worker risk assessments on chlorpyrifos and other individual pesticides, EPA prioritizes protective clothing and equipment to mitigate risks of concern. Only if that mitigation fails to eliminate the risks of concern will EPA consider engineering controls, and only if those controls fall short, will it consider stopping the pesticide use.

This priority scheme runs counter to the best and most prudent occupational health practices. These industrial hygiene practices, called the "hierarchy of controls," are designed to prevent harmful exposures as the first line of defense. The American National Standards Institute/American Industrial Hygiene Association Z10 2005 standard[136] provides that employers shall implement and maintain a process for feasible risk reduction based on the following preferred order of controls:

1. Elimination
2. Substitution of less hazardous materials
3. Engineering controls
4. Administrative controls; and
5. Personal protective equipment.

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[135] *See, e.g.*, 29 C.F.R. § 1910.1000 (limiting hours employees can work with specific toxic air contaminants, including pesticides); 29 C.F.R. § 1910.1003 (calling for detailed safeguards against exposures, including closed systems, in workplaces where a range of carcinogens are used); 29 C.F.R. § 1910.134 (requiring respiratory protection when engineering controls and substitution of less toxic chemicals are not feasible).

[136] Fred A. Manuele, ANSI/AIHA Z10-2005: The New Benchmark for Safety Management Systems, Pub. Safety, Feb. 2006, at 25, http://www.asse.org/publications/standards/z10/docs/25-33Feb2006.pdf.

The hierarchy of controls prioritizes hazard elimination and substitution over less protective controls, like engineering controls, while EPA does just the opposite. In fact, EPA prioritizes the least effective measure – PPE. An assessment of the efficacy of industrial hygiene controls to limit worker pesticide exposures found: "personal protective equipment (PPE) is always considered a last resort and should only be used as a method of exposure control when all other controls have been implemented and have not sufficiently reduced the hazard."[137]

The ANSI hierarchy of controls prioritize engineering controls over PPE, as does OSHA. OSHA regulations adopt a hierarchy of controls to prevent employee inhalation, ingestion, skin absorption or contact with harmful amounts of toxic substances:

> [A]dministrative or engineering controls must first be implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section.

29 C.F.R. § 1926.55(b); *see also id*. § 1910.134(a)(1) (prioritizing engineering controls over respirators to reduce toxic air exposures); 43 Fed. Reg. 52,952 (Nov. 14, 1978) (preamble to lead standard finding repeatedly that respirators are ineffective because they do not eliminate the exposure, they provide inadequate protection, and they create additional hazards by interfering with vision and mobility). The PID's option of requiring closed systems for mixing and loading pesticides and requiring air blast applications to be done in closed cabs are preferable as engineering controls to PPE. Similarly, restricting re-entry into a recently sprayed field to the extent necessary to eliminate risks of concern is preferred as an administrative control.

b. EPA Should Adopt the Most Effective Mitigation, Which Often Will Be Ending the Application Method.

Most uses of chlorpyrifos will not pass muster under the FFDCA or FIFRA quite apart from worker risks and must end. For any remaining uses, EPA should adhere to the hierarchy of controls and prohibit the application methods it has found extremely dangerous. It should require enclosed cabs with functioning air conditioning and ventilation systems for airblast spraying and closed mixing and loading systems it has found necessary to eliminate risks of concern for ground boom applications. And it should lengthen REIs whenever field workers would otherwise face risks of concern. EPA should avoid relying on PPE unless it can ensure it would be effective and would avoid causing heat or respiratory stress, which is highly unlikely.

i. Aerial Spraying and Chemigation Should Be Banned.

Some application methods, such as aerial spraying, cannot be done safely and should be prohibited. Aerial spraying (and chemigation) pose risks of concerns even with PPE and

[137] Justine L. Weinberg et al., *Application of Industrial Hygiene Hierarchy of Controls to Prioritize and Promote Safer Methods of Pest Controls: A Case Study*, 124 Pub. Health Rep. 53-62 (2009).

engineering controls. PID at 21-22. Flaggers face risks of concern even with additional PPE and have largely been replaced by GPS. PID at 56. EPA is considering ending all aerial applications and almost all chemigation applications. PID at 55. Given the risks, EPA should end these types of application methods.

ii. Air Blast Applications Should Require Enclosed Cabs With Working Air Conditioning and Ventilation Systems.

All airblast applications pose risks of concern. PID at 22. For some crops, the risks are incredibly severe with MOEs less than 10 or even less than 1, as in the case of citrus and pecans. PID at 22. It should come as no surprise that EPA's risk assessments found such prevalent and serious risks from air blast spraying. EPA found unacceptable risks from air blast spraying in 2001 and did not impose mitigation to eliminate the risks. In the interim 20 years, air blast spraying has been the culprit in numerous pesticide poisonings. In Washington State, air blast spraying is the largest source of drift exposure, comprising more than half the drift illness enforcement actions in recent years.[138]

EPA believes most risks of concern can be mitigated with the use of enclosed cabs. Accordingly, the PID proposes requiring enclosed cabs for all airblast applications. PID at 55. EPA should adopt this requirement.

However, in order to be effective, the closed cabs would need to have air conditioning and ventilation systems. Without air conditioning and ventilation, workers might open the windows in extreme summer heat, which would nullify the efficacy of closed cabs. Unless EPA requires that the closed cabs will have ventilation and air conditioning systems and that those systems will be in working order, enclosed cabs will be ineffective.

The PID rightly considers prohibiting airblast applications unless done in an enclosed cab. PID at 55. As EPA recognizes, a grower who does not own a tractor with an enclosed cab could hire a commercial applicator who does. PID at 55. And if an orchard did not lend itself to enclosed cabs, the grower could shift to alternative pesticides or pest control methods. PID at 55. Given the nature of the risks and harm, EPA should require all airblast applications to be done in enclosed cabs.

Some risks of concern, such as for citrus and pecans, will remain even with closed cabs. EPA admittedly cannot find use of chlorpyrifos on pecans safe, so it must end this use. EPA purports to find drinking water contamination from citrus below levels of concern if application rates are lowered. However, given that risks of concern to handlers would remain even with closed cabs and handlers who mix and load chlorpyrifos for airblast applications also face risks of concern, EPA should end this use.

[138] Washington Department of Agriculture Pesticide Enforcement Actions 2014-2015, http://agr.wa.gov/pestfert/enforcementactions.aspx.

iii. EPA Should Require Closed Systems For Mixing and Loading To Eliminate Risks of Concern.

EPA finds risks of concern to workers who mix and load many chlorpyrifos formulations for airblast and ground boom applications. PID at 22-23. It is considering requiring additional PPE or closed systems for mixing and loading. PID at 43-44.

EPA should adhere to the hierarchy of controls and require closed systems rather than additional PPE. As discussed above, PPE is far less effective than engineering controls. Moreover, EPA has noted that workers could suffer from heat and respiratory stress with any additional PPE, 2014 HHRA at 100, which would particularly be the case in places like the Central Valley or Wenatchee where heat stress is already a problem for workers in the summer. During 2020, EPA and many states relaxed PPE requirements or enforcement during the pandemic and PPE became less available, further underscoring the precariousness of relying on PPE.

Closed systems are far more effective than PPE. In the 1970s, after California required closed mixing and loading systems to be used for Category 1 liquid pesticides (although not for chlorpyrifos), mixers and loaders reported incidents at 1/5 of the previous levels.[139] Other studies have likewise documented dramatically reduced harm to workers when closed systems replaced hand pouring.[140] Since California has the largest share of U.S. receipts for agricultural crops, the mandatory use of closed systems there demonstrates the economic and technological feasibility of closed systems.

In its risk estimates, EPA indicates that closed mixing systems could reduce risks of concern from mixing and loading activities for various chlorpyrifos formulations to what EPA deems acceptable risk levels. EPA should immediately require the use of closed systems for these mixing and loading activities.

For the mixing and loading activities that still produce risks of concern even with closed mixing systems, EPA should prohibit those activities. For airblast applications to citrus (and pecans), workers face risks of concern from both mixing and loading activities and airblast applications. EPA must consider all of these risks in the aggregate and end use of chlorpyrifos on citrus.

For ground boom applications, risks of concern remain for alfalfa, corn, cotton, tree nuts, turf farms, and soybean for mixing and loading some formulations. PID at 23-24, 44, 56. EPA is considering prohibiting these uses and should do so.

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[139] Rutz, *Closed System Acceptance and Use in California*, *in* Pesticide Formulations and Application Systems, at 28-34 (G.B. Beestman & R.D. Vander Hooven eds. 1987).
[140] James B. Knaak et al., *Safety and Effectiveness in Preventing Exposure to Pesticides*, 24 Archives Envtl. Contamination & Toxicology, at 231, 244-245 (1980).

EPA is also considering prohibiting tractor-drawn spreaders, handheld application methods, microencapsulated formulations on ornamentals in nurseries and greenhouses where REIs would need to be more than a month, which is impractical, and seed treatments where workers face risks of concern from multiple activities. PID at 57-58. It should follow through and end these uses.

Finally, EPA has identified approximately 30 activities where longer REIs are needed to prevent risks of concern. EPA is considering requiring the longer REIs. EPA should do so.

V. THE 2020 HHRA AND PID FAIL TO ANALYZE AND PROTECT AGAINST ENVIRONMENTAL JUSTICE IMPACTS.

The discussion of environmental justice in the 2020 HHRA is limited to a single paragraph that leaves unaddressed and unmitigated numerous environmental justice concerns. 2020 HHRA at 18. Instead of engaging in a robust analysis of environmental justice impacts, the 2020 HHRA 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. The 2020 HHRA provides no analysis of those burdens. While EPA failed to complete an adequate environmental justice analysis for chlorpyrifos, the record before EPA clearly demonstrates that chlorpyrifos must be banned. As EPA continues to review other pesticides for registration review, particularly other organophosphates, it must analyze and consider disproportionate impacts—of which there are many, particularly to farmworkers and their families. Likewise, if EPA somehow concludes that some uses of chlorpyrifos can remain, then EPA must engage in a robust environmental justice analysis of those remaining uses.

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 pets—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. EPA ignored drift under the FQPA in its 2006 re-registration decisions, later acknowledging its FQPA obligation to protect children from drift in response to the 2007 Petition and the 2009 Kids' Petition. 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 HHRA 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 failed to fully consider and protect against the broad impacts chlorpyrifos has directly and pervasively on low-income and minority children who live near the fields. Indeed, EPA maintains a double standard by protecting children from urban and residential uses through use cancellation, while settling for inadequate buffers and other mitigation measures that allow continued exposures for 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 must assess the environmental justice impacts of drift, food, drinking water, and worker exposures (including take-home exposures) and that assessment should inform EPA's regulatory decisions and mitigation measures.

Because EPA does no more than pay lip service to environmental justice impacts in the 2020 HHRA, it is in violation of these two executive orders and has acted arbitrarily. While the 2020 HHRA suggests that EPA may have looked at food consumption data and spray drift exposure as part of an environmental justice analysis, the assessment contains no details or conclusions from such an analysis. 2020 HHRA at 18. EPA noted that, at some point, it may also develop tools to consider "other types of possible bystander exposures and farm workers as well as lifestyle and traditional dietary patterns among specific subgroups," but EPA provides no details or timeline for that effort. *Id*. If EPA is contending that it has engaged in the required environmental justice analysis, then it must—at a minimum—explain which data it used and why, how those data were analyzed, and what conclusions the agency reached. EPA's boilerplate assertions that it considered environmental justice concerns (or may at some point in

the future) do not satisfy EPA's obligation to address the disproportionate impacts that chlorpyrifos uses have on low-income and minority communities.

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.[141] At the same time, water monitoring showed chlorpyrifos in 17.7% of samples and exceeding the concentration limit in 9.9% of samples. *Id.* Moreover, California 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.[142] 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.[143] Latino/a 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.[144]

Despite this overwhelming evidence of disproportionate impacts, EPA states without further explanation that "[s]pray drift can also potentially result in post-application exposure and it was considered in this analysis." 2020 HHRA at 18. This conclusory statement does not suggest that EPA conducted the necessary full and robust assessment of environmental justice

[141] *See* Letter from Tracey Brieger, Californians for Pesticide Reform et al., to Arsenio Mataka, Assistant Secretary for Environmental Justice and Tribal Affairs, Cal. Envtl. Prot. Agency at 1 (Aug. 26, 2014) ("Coalition Letter"). *See also* Daniel J. Hicks, *Census Demographics and Chlorpyrifos Use in California's Central Valley, 2011-15: A Distributional Environmental Justice Analysis*, Int'l J. Envtl. Research & Pub. Health (Apr. 10, 2020), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7177971/ (finding that "Hispanic communities in California's Central Valley are associated with higher local chlorpyrifos use, and so higher potential chlorpyrifos exposure").

[142] California Environmental Health Tracking Program, *Agricultural Pesticide Use Near Public Schools in California* ("Schools Report"), http://cehtp.org/projects/ehss01/pesticides_and_schools/Pesticides_Schools_Report_April2014.

[143] *See* Coalition Letter at 5.

[144] *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.

impacts. Nor does the 2020 HHRA have any discussion of which populations are most impacted by poisoning incidents. 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."[145] Children are also at 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.[146]

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.[147] 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.[148]

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[145] NAS, Pesticides in the Diets of Infants and Children 3-7 (1993).
[146] *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).
[147] The 2007 Petition likewise called on EPA to address chlorpyrifos drift and volatilization. 2007 Petition at 17-21, https://www.regulations.gov/document?D=EPA-HQ-OPP-2007-1005-0005.
[148] *Pesticides in the Air—Kids at Risk: Petition to EPA to Protect Children from Pesticide Drift* (Oct. 13, 2009) (the "Kids' Petition"), at EPA-HQ-OPP-2009-0825. After the Kids' Coalition brought a mandamus action to compel a response, *Pesticide Action Network of N. Am. v. U.S. Envtl. Prot. Agency*,

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 that 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. CDPR, 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."[149] Monitoring and modeling studies confirm pesticide drift poses significant health risks to children who live near fields.

In light of the evidence of exposure and poisoning incidents, the discussions of chlorpyrifos drift and volatilization do not fulfill that obligation as EPA has failed to identify the disproportionate impacts chlorpyrifos has on minority and low-income populations.

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

Likewise, the analysis did 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 $20,000-$24,999.[150] In the top five agricultural counties in Texas (the state with the most acres of agriculture), between 21.2 to 35.2 percent of children live in poverty.[151] 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%).[152]

Case No. 13-72616 (9th Cir. filed Mar. 29, 2014), 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. *Pesticide Action Network of N. Am. v. U.S. Envtl. Prot. Agency*, No. 14-71514, 654 Fed. Appx. 887, 2016 WL 3619950 (9th Cir. July 5, 2016) (upholding EPA's denial).

[149] 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* (2005).

[150] Findings from the National Agricultural Workers Survey (NAWS) 2015-2016: A Demographic and Employment Profile of United States Farmworkers, Research Report No. 13 (Jan. 2018), https://www.dol.gov/sites/dolgov/files/ETA/naws/pdfs/NAWS_Research_Report_13.pdf.

[151] U.S. Department of Agriculture, 2018 County-Level Poverty Rates for Texas, https://data.ers.usda.gov/reports.aspx?ID=17826; *see also* USDA 2017 Census of Agriculture State Profile: Texas, https://www.nass.usda.gov/Publications/AgCensus/2017/Online_Resources/County_Profiles/Texas/cp99048.pdf (listing top counties with land in farms as Pecos, Hudspeth, Brewster, Webb, and Presidio).

[152] Alice Larson, Migrant and Seasonal Farmworker Enumeration Profiles Study: California (Sept. 2000).

The vast majority of U.S. farmworkers are of Latin American origin—approximately 83 percent of U.S. farmworkers are of Latin American ancestry.[153] 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.[154]

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.[155] 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—as many as 88 percent of acute poisoning incidents are not reported to public health authorities.[156] Moreover, a 2014 study showed that farmworkers had higher residue concentrations of chlorpyrifos dust.[157] 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.[158]

When working on revisions to the Worker Protection Standard, EPA portrayed the plight of these workers:

> 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. 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. Approximately 44% could not speak English at all, and 53% could not read any English.
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> . . .

[153] NAWS (2015-16).
[154] Environmental Working Group, *Every Breath You Take: Airborne Pesticides in the San Joaquin Valley* (Jan. 2001).
[155] 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).
[156] *See* Joanne Prado, *et al*., *Acute Pesticide-Related Illness Among Farmworkers: Barriers to Reporting to Public Health Authorities*, 22(4) J. Agromedicine 395 (2017), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5846675/pdf/nihms939406.pdf.
[157] Beti Thompson, *et al*., *Variability in the Take-Home Pathway: Farmworkers and Non-Farmworkers and Their Children*, 24 J. Exposure Sci. and Envtl. Epidemiology 522 (2014).
[158] 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.

> 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. Over 20% of respondents lived in housing provided by their employer and 58% rented housing from someone other than their employer. 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. 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.[159]

Yet EPA's cryptic treatment of environmental justice in the 2020 HHRA 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 2020 HHRA. While EPA acknowledges that several studies have found that chlorpyrifos exposures cause long-term brain-based disorders to children exposed *in utero*, EPA failed to assess the race, ethnicity, or income of the most impacted populations, even though the cohort studies documenting such harm involved participants who are predominantly poor and people of color. Similarly, in assessing drinking water impacts, EPA found exceedances of its drinking water levels of concern in watersheds across the country—it is highly likely that the impacted areas are 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. EPA's 2020 Drinking Water Assessment and 2020 HHRA make new assumptions and shrink safety factors—EPA's environmental justice assessment should determine who will suffer from this reduced protection if the agency allows any chlorpyrifos uses to remain. And EPA identifies risks of concern to workers, but it fails to reveal that most of the workers are low-income and Latino/a 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 is required to afford analogous protection to workers under FIFRA, it has failed to do so with respect to chlorpyrifos by failing to adhere to the hierarchy of controls used under OSHA for industrial settings and by proposing to allow numerous risks of concern to continue under the 2020 PID. EPA 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

[159] 79 Fed. Reg. at 15,444, 15,452 (internal citations omitted).

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.

CONCLUSION

More than five years ago, the Ninth Circuit called EPA's delay in protecting people from chlorpyrifos "egregious" and imposed deadlines "necessary to end this cycle of incomplete responses, missed deadlines, and unreasonable delay." *In re PANNA v. EPA*, 798 F.3d 809, 811, 813 (9th Cir. 2015). Unfortunately, the Trump administration evaded the law and defied the science to avoid taking action to protect workers from acute poisonings and children from learning disabilities and IQ deficits. With the change in administrations, EPA is now revisiting the Wheeler order, which refused to grant the 2007 petition to ban food uses of chlorpyrifos. These comments are submitted to inform that review and EPA's interim registration review decision for chlorpyrifos.

The science is unequivocal. Chlorpyrifos causes harm to the developing brain, leaving children with learning disabilities, reduced IQ, and other neurodevelopmental impairments. It is long past time to protect our children from chlorpyrifos. Because EPA cannot find reasonable certainty of no harm from chlorpyrifos, it must finalize its proposed rule revoking all chlorpyrifos tolerances and make that rule effective within 180 days, as proposed. EPA should thereafter cancel all uses of chlorpyrifos because the food uses are unsafe and other uses cause unreasonable adverse effects to workers and others exposed to the pesticide.

Sincerely,

Patti A. Goldman
Marisa Ordonia
Earthjustice
810 Third Avenue, Suite 610
Seattle, Washington 98104
(206) 343-7340
pgoldman@earthjustice.org
mordonia@earthjustice.org

DECLARATION OF PHILIP J. LANDRIGAN, M.D., M.SC. IN SUPPORT OF PETITION TO SUSPEND AND CANCEL CHLORPYRIFOS USES

I, Philip J. Landrigan, M.D., M.Sc., hereby declare and state as follows:

1. I submit this declaration in support of the petition to cancel and suspend chlorpyrifos uses that is being submitted by Earthjustice on behalf of United Farm Workers and other farmworker advocates.

PROFESSIONAL BACKGROUND AND EXPERTISE

2. I am a pediatrician and epidemiologist, and I am board certified in occupational medicine, general preventive medicine and pediatrics. I have been a member of the faculty of the Icahn School of Medicine at Mount Sinai since 1985 and am currently a professor of preventive medicine and a professor of pediatrics. I am also the Dean for Global Health, a position I have held since 2010.

3. I obtained my medical degree from Harvard Medical School in 1967. I completed an internship at Cleveland Metropolitan General Hospital and a residency in pediatrics at Boston Children's Hospital. In 1977, I received a Diploma of Industrial Health from the University of London and a Master of Science degree in Occupational Medicine from the London School of Hygiene and Tropical Medicine. My CV is attached as Exhibit 1.

4. I served for 15 years as an Epidemic Intelligence Service Officer and medical epidemiologist at the Centers for Disease Control and Prevention (CDC) and the National Institute for Occupational Safety and Health (NIOSH). I directed the national program in occupational epidemiology for NIOSH from 1979-1985. I have been awarded numerous honors throughout my career, including the Meritorious Service Medal of the U.S. Public Health Service in 1985.

5. From 2000 to 2002, I served on the Armed Forces Epidemiological Board, and from 1996 to 2005, in the Medical Corps of the U.S. Naval Reserve. I retired from the United States Navy in 2005 at the rank of Captain (O-6) and continue to serve as Surgeon General of the New York Naval Militia, the naval component of the New York National Guard.

6. I was elected a member of the Institute of Medicine of the National Academy of Sciences in 1987. I have chaired committees at the National Academy of Sciences (NAS) on Environmental Neurotoxicology and on Pesticides in the Diets of Infants and Children. From 1997 to 1998, I served as Senior Advisor on Children's Health to the Administrator of the U.S. Environmental Protection Agency (EPA) and was instrumental in helping to establish a new Office of Children's Health Protection at EPA.

7. I am editor in chief of the *Annals of Global Health*, deputy editor of the *American Journal of Industrial Medicine*, and an associate editor of *Environmental Health Perspectives.*

8. I have studied the impacts of toxic chemicals, including pesticides, on children's health for over thirty years. I have published more than 500 scientific papers and five books, on subjects including epidemiology, occupational health, environmental neurotoxicity, and children's health. I have extensive knowledge and expertise in environmental and occupational medicine, epidemiology, environmental neurotoxicity, and the effects of pesticides and other chemicals on children through my education, training, professional experience, involvement in applicable peer-reviewed research, and my ongoing review of the pertinent medical and scientific literature.

CHILDREN'S VULNERABILITY TO PESTICIDES

9. A key policy breakthrough occurred over the past three decades with the discovery that children are far more sensitive than adults to toxic chemicals in the environment.

This finding led to the recognition that chemical exposures early in life are significant yet preventable causes of disease in children and adults.

10. In the 1970s, my research showed that 60% of children living within one mile of ASARCO's El Paso smelting plant had elevated blood lead levels and that even small amounts of lead exposure lowered a child's IQ. My research showed that lead can cause brain damage to children at levels too low to clinically detect signs and symptoms. This phenomenon is now called "subclinical toxicity." These studies contributed importantly to the U.S. federal government's decision to phase out lead components from gasoline and regulate the lead content of paint in the 1970s.

11. I led a five-year study as chair of the NAS Committee that published *Pesticides in the Diets of Infants and Children* in 1993.[1] This pivotal study showed that infants and children, including infants in the womb, are much more sensitive to pesticides and other toxic chemicals than adults and documented four differences between children and adults that contribute to children's heightened susceptibility to chemicals in the environment. The following description of this work is taken from an article that I co-authored with Dr. Lynn R. Goldman, "Children's Vulnerability to Toxic Chemicals: A Challenge and Opportunity to Strengthen Health and Environmental Policy," *Health Affairs* 30, no.5 (2011): 842-850 (Exhibit 2):

> First, children have greater exposures to toxic chemicals for their body weight than adults. A six-month-old infant drinks seven times more water per pound than an adult, and children take in three to four times more calories per pound than adults. The air intake per pound of an infant is twice that of an adult. These differences result in children being disproportionately exposed to toxic chemicals in air, food, and water. Children's hand-to-mouth behavior and play on the ground further magnify their exposures.

[1] National Research Council. Pesticides in the Diets of Infants and Children. Washington, DC: National Academy Press, 1993.

> Second, children's metabolic pathways are immature, and a child's ability to metabolize toxic chemicals is different from an adult's. In some instances, infants are at lower risk than adults because they cannot convert chemicals to their toxic forms. More commonly, however, children are more vulnerable because they lack the enzymes needed to break down and remove toxic chemicals from the body.
>
> Third, children's early developmental processes are easily disrupted. Rapid, complex, and highly choreographed development takes place in prenatal life and in the first years after birth, continuing more slowly throughout childhood into puberty. In the brain, for example, billions of cells must form, move to their assigned positions, and establish trillions of precise interconnections. Likewise, development of the reproductive organs is guided by a complex and precisely timed sequence of chemical messages and is shaped by maternal and fetal hormones.
>
> Recent research in pediatrics and developmental toxicology has elaborated the concept of "windows of vulnerability." These are critical periods in early development when exposures to even minute doses of toxic chemicals—levels that would have no adverse effect on an adult—can disrupt organ formation and cause lifelong functional impairments. . . .These windows of vulnerability have no equivalent in adult life.
>
> Fourth, children have more time than adults to develop chronic diseases. Many diseases triggered by toxic chemicals, such as cancer and neurodegenerative diseases, are now understood to evolve through multistage, multiyear processes that may be initiated by exposures in infancy.

12. Since the 1993 publication of the NAS report, peer-reviewed research continues to document the developing human brain's unique vulnerability to toxic chemical exposures, and to confirm that major windows of developmental vulnerability occur *in utero,* during infancy, and in early childhood. During these sensitive life stages, exposure to pesticides and other chemicals can cause permanent brain injury at levels of exposure far below those which would have an effect in adults.

13. A fetus in the womb is at risk of exposure to pesticides and other toxic chemicals because of both exposure and vulnerabilities. In terms of exposure, the placenta does not block the passage of many toxic chemicals from the maternal to the fetal circulation. In fact, more than 200 chemicals have been detected in infants' umbilical cord blood, meaning they have passed from the mother's circulation to the baby's circulation prior to birth. In terms of susceptibility,

several prenatal developmental processes have been shown to enhance the vulnerability of the fetus in the womb to toxic chemicals.

14. Prior to the publication of the NAS report, virtually all environmental policy in the United States had focused on assessment of risk to the average adult man weighing 150 pounds. Little attention was paid to the unique risks faced by infants, children, or other vulnerable groups within the population.

15. The core findings and recommendations of the NAS report were incorporated into the 1996 Food Quality Protection Act (FQPA), which revamped federal pesticide laws. The FQPA changed risk assessment by requiring the use of child-protective safety factors to account for children's exposures and unique susceptibilities and to account also for gaps in data, and by requiring consideration of aggregate exposures to a pesticide via multiple routes, including diet, drinking water, and interaction with pesticide residues through play and other activities. It also required evaluation of cumulative effects of multiple pesticides that have the same mechanism of toxicity.

16. Implementation of the new standards led to bans on residential applications of two very widely used organophosphate insecticides: chlorpyrifos and diazinon. These bans were triggered by recognition of these compounds' neurodevelopmental toxicity to children and documentation of their long residence time in indoor environments. FQPA implementation also led to a cumulative risk assessment for all organophosphates because they have a common mechanism of toxicity, as discussed below.

NEURODEVELOPMENTAL HARM TO CHILDREN'S BRAINS FROM CHLORPYRIFOS AND OTHER ORGANOPHOSPHATES

17. Chlorpyrifos, like other organophosphate pesticides (OPs), causes acute poisonings by inhibiting the enzyme acetylcholinesterase (AChE), which regulates nerve

impulses. When cholinesterase is inhibited, it leads rapidly to overt symptoms of cholinergic hyperstimulation. The symptoms include nausea, headaches, skin rashes, eye irritation, vomiting, dizziness, seizures, coma, and death, depending on the dose and the toxicity of the product. When EPA conducted risk assessments on the organophosphates in the 1990s through 2006, it set human exposure limits based on detection of AChE inhibition. Specifically, it uses 10% red-blood cell AChE inhibition as its regulatory endpoint, called its point of departure.

18. A growing body of scientific evidence has documented neurodevelopmental harm to the developing brain from organophosphates, including chlorpyrifos. This evidence comes both from animal and epidemiology studies. EPA has compiled and reviewed the published studies in its Revised Human Health Risk Assessment for Chlorpyrifos Registration Review (Dec. 29, 2014) (RHHRA), and in its Literature Review on Neurodevelopmental Effects & FQPA Safety Factor Determination for the Organophosphate Pesticides (Sept. 15, 2015).

19. Numerous scientific studies have documented neurodevelopmental harm from prenatal and early postnatal exposures to chlorpyrifos. Animal studies have found disruption in neuronal development, neurotransmitter systems and synaptic function, as well as behavioral and cognitive impairments following low-dose perinatal chlorpyrifos exposure. Neurobehavioral effects include impairment on maze performance, locomotion, and balance in neonates exposed *in utero* or during postnatal life.

20. Direct evidence that chlorpyrifos can cause neurodevelopmental harm to children's brains comes from three epidemiology studies conducted respectively at Columbia University, University of California-Berkeley, and Mount Sinai School of Medicine. These universities conducted this research through their Centers for Children's Environmental Health and Disease Prevention Research.

21. These Centers are part of an NIH-funded, competitively awarded national network of such Centers established to increase scientific understanding of the impacts of toxic exposures on children. The Berkeley study studied children of farmworkers in the Salinas Valley of California, the Mount Sinai study observed a New York City Hispanic population whose exposures were primarily residential, and the Columbia study examined African-American and Dominican children in New York City, whose exposures were similarly residential.

22. These three Centers have been conducting long-term birth-cohort studies in which pregnant women are enrolled during their pregnancies. Their environmental exposures during pregnancy are recorded through objective measures like blood and urine samples, dust and air samples, and cord blood. Chlorpyrifos exposure during pregnancy was measured through analysis of chlorpyrifos' metabolic breakdown products in maternal urine samples. Even though these three studies were conducted in distinct geographic regions of the country, on different populations, with different routes of exposure, and using different biomarkers, they produced strongly convergent results. All studies found cognitive impairments that persist into school years from OP exposures. The Columbia study was specific to chlorpyrifos. It found that prenatal exposure to chlorpyrifos resulted in the birth of babies with reduced head circumference. Reduction in head circumference at birth is a measure of delayed or reduced brain growth during pregnancy and is an effect seen also in infants exposed in the womb to Zika virus. In the Columbia study, the degree of reduction in head circumference was proportional to the degree of maternal exposure to chlorpyrifos during pregnancy. The impact of chlorpyrifos on head circumference was no longer observed after the ban on residential application of chlorpyrifos was imposed.

23. Follow-up studies of the babies in these three studies have found that prenatal exposures have persistent deleterious effects on cognitive function through 7 years of age. The brain impairments observed in these infants and children include reduction in motor function, decreases in working and visual memory, processing speed, verbal comprehension, perceptual reasoning, and diminished IQ. The studies also documented neurobehavioral problems, including increased risk of attention deficit hyperactivity disorder, pervasive developmental disorder, and behaviors typical of the autism spectrum. Certain subpopulations demonstrate greater susceptibility, including children of farmworkers and those who have reduced capacity to detoxify OPs. Some studies found elevated risks of respiratory symptoms consistent with asthma. And recently, a study using magnetic resonance imaging found that even low to moderate levels of prenatal exposure to chlorpyrifos may lead to long-term, potentially irreversible changes in the structure of the developing brain, causing thinning of the cerebral cortex.

24. These studies found damage to children's brains from exposures to chlorpyrifos that produced no or less than 1% red-blood cell cholinesterase inhibition. In other words, the harm to the developing brain and nervous systems occurred at exposures substantially below EPA's regulatory limit, which is based on exposures that are high enough to inhibit cholinesterase in adults. EPA acknowledged in its 2014 revised human health risk assessment on chlorpyrifos that the neurodevelopmental harm to children's brains occurred at lower doses than its regulatory endpoint.

EPA'S RISK ASSESSMENTS DO NOT PROTECT AGAINST BRAIN DAMAGE TO CHILDREN

25. Even though EPA has acknowledged that neurodevelopmental harm to children occurs at exposures that produce no or only minimal cholinesterase inhibition, EPA has

continued to set its exposure limits based on cholinesterase inhibition. It continues to use 10% red-blood cell cholinesterase inhibition as the endpoint in its risk assessments, even though the mothers in the Columbia study who gave birth to infants with brain injury exhibited less than 1% cholinesterase inhibition or no inhibition at all.

26. Safety factors are used in risk assessment and standard-setting to account for uncertainties. In setting a standard or tolerance for a pesticide, EPA will begin the risk assessment by identifying an exposure level that produces no adverse effect as its endpoint. This is called the no observable adverse effect level. If some adverse effects are observed at that exposure level, EPA will add a three-fold safety factor. EPA then typically uses a tenfold safety factor to account for uncertainties in extrapolating from animal studies to people, and a second tenfold safety factor to account for differences among human populations due to such factors as genetic predisposition and other stressors. Finally, the FQPA requires EPA to use a third tenfold "child-protective" safety factor when there is either evidence that children are especially vulnerable to a chemical or when there are gaps in data concerning children's exposures or vulnerabilities. For OPs, EPA has retained a 10X child-protective FQPA safety factor because of the published evidence that these chemicals cause neurodevelopmental harm to infants and children.

27. For chlorpyrifos, however, EPA departed from this usual practice and instead relied on the Dow Agrosciences Company's pharmacokinetic-pharmacodynamic (PBPK) model of OP toxicity, which tries to pinpoint the exposures that will produce 10% cholinesterase inhibition. The Dow model is drawn largely from human studies that included deliberate dosing of people. Many of these studies were conducted in countries outside of the United States. Use of human studies in risk assessment poses significant ethical and scientific issues, and the Dow

human studies have been criticized for not meeting the informed consent standards that would be required in the US and also for scientific deficiencies. Because the Dow model uses human data, it obviates the need to extrapolate data from animals to humans. In relying on the Dow data, EPA therefore dispensed with the 10X inter-species safety factor for all populations except for women of child-bearing years. For women of child-bearing years, EPA retained the 10X intra-species safety factor because Dow did not have human data for this population.

EPA'S RISK ASSESSMENTS DO NOT PROTECT WORKERS OCUPATIONALLY EXPOSED TO CHLORPYRIFOS AND DO NOT PROTECT THE CHILDREN IN THE WOMB OF PREGNANT WOMEN WORKERS

28. In their assessments of risk from occupational exposures to chlorpyrifos, EPA identified risks of concern for over half of the handler exposure scenarios. EPA states that additional engineering controls or protective gear could eliminate the risks of concern for 27 of these activities, but notes that 126 would remain of concern regardless of the level of personal protective equipment or environmental controls. EPA also found that protection of agricultural field workers against chlorpyrifos toxicity would need longer re-entry intervals to reduce risks.

29. For many of the handler exposure scenarios, EPA found Margins of Exposure (MOEs) of less than 10 and for some scenarios the MOEs were close to or even less than 1. In other words, EPA estimates that worker exposures from these activities likely would result in 10% cholinesterase inhibition. In these scenarios, the current EPA standard manifestly fails to protect worker health or to comply with the fundamental intent of the Occupational Safety & Health Act of 1970 (OSHA) which states that every worker has the "right to a safe and healthful workplace."

30. EPA has acknowledged that its regulatory end point is underprotective. It has proposed using umbilical cord blood chlorpyrifos levels from the Columbia study to develop a more protective end point based on loss of working memory. It convened a Scientific Advisory

Panel (SAP) to review this proposal. The SAP did not support developing a point of departure based on a single study, but it did agree that EPA's approach of using 10% cholinesterase inhibition as the regulatory endpoint was underprotective.

31. The California Department of Pesticide Regulation (DPR) prepared its own risk assessment of chlorpyrifos which was modeled on EPA's approach and like EPA incorporated 10% cholinesterase inhibition, Dow's PBPK model, and the reduced safety factors. California's Office of Environmental Health Hazard Assessment (OEHHA), which routinely reviews pesticide standards proposed by DPR to ensure that they protect worker health, conducted a scientific peer review of DPR's human health risk assessments on chlorpyrifos and released its review in June 2016. OEHHA found that the 10% cholinesterase inhibition end point and the reduced safety factors proposed by the DPR failed to adequately protect human health and therefore failed to comply with occupational safety and health legislation. OEHHA recommended using a total uncertainty factor of 1000X or 3000X to protect the health of workers occupationally exposed to chlorpyrifos.

32. Any occupational exposure standard for chlorpyrifos needs to take cognizance of the fact that the workforce may include pregnant women workers (who may not yet realize that they are pregnant) and that pregnant women workers who are occupationally exposed to chlorpyrifos will unwittingly pass any chlorpyrifos that they absorb into the bodies of their unborn children where the chlorpyrifos will cause irreversible brain damage. To prevent this sequence of events, EPA should at a minimum use safety factors that total 1000X. Moreover, an additional 3X uncertainty factor is warranted over and above the 1000X safety factor because 10% cholinesterase inhibition cannot be considered a "no observable adverse effect level" in

light of the finding that neurodevelopmental harm to the fetus can result at exposure levels below this outdated limit value.

PREVENTING BRAIN DAMAGE TO CHILDREN FROM TOXIC CHEMICAL EXPOSURES YIELDS SIGNIFICANT COST SAVINGS

33. Neurobehavioral development disorders affect 10-15% of births in the United States, and the prevalence of attention deficit hyperactivity disorder, autism and other neurodevelopmental disorders is increasing in the US and worldwide. Subclinical decrements in brain function are even more common. All of these disabilities can have serious consequences for individuals, such as diminished quality of life, reduced academic achievement, behavioral disruptions, and they also have consequences for society in the form of the diminished economic productivity of affected children and the increased risk that these children will grow up to become unemployed, underemployed and institutionalized or incarcerated adults. Environmental exposures play a role in many, if not most, of these developmental disorders as genetic factors account for only approximately 30-40% of them.

34. Preventing exposures to chemicals can yield great economic savings. While it is difficult to precisely quantify the harm from neurodevelopmental disorders and the cost savings that result from their prevention, several studies suggest that both are quite large. To estimate the contribution of environmental pollutants to the prevalence and costs of disease in American children, investigators at Mount Sinai School of Medicine examined four categories of illness: lead poisoning, asthma, cancer, and neurobehavioral disorders. Based on prevalence, the environmentally attributable fraction of each disease, and national economic data, they calculated that the total annual costs of these diseases attributable to environmental exposures is $54.9 billion (range $48.8 billion to $64.8 billion): $43.4 billion for lead poisoning, $2.0 billion for asthma, $0.3 billion for childhood cancer, and $9.2 billion for neurobehavioral disorders.

Because of the difficulties inherent in assessing the full economic consequences of neurobehavioral impairments, it is likely that these estimates are low.

35. After the phase-out of lead in gasoline from 1976 and 1990, the mean blood lead level of American children decreased by more than 90% (to below 2 micrograms per deciliter today), and the incidence of childhood lead poisoning also fell by more than 90%. A further consequence of the reduction in exposure to lead was that the mean IQ of American children has increased. Children born in the United States today are estimated to have IQ scores that, on average, are 2.2–4.7 points higher than those of children born in the early 1970s. And because each 1-point gain in population mean IQ is associated with an estimated 2% increase in productivity over a lifetime, the gain in population IQ is estimated to have produced a national economic benefit of $110–$319 billion in each annual cohort of babies born in the United States since the 1980s.

36. Dr. David Bellinger, a professor of neurology at Harvard Medical School, published a paper in 2012, which estimated that Americans have collectively forfeited 41 million IQ points as a result of exposure to lead, mercury, and OPs. He calculated a total loss of 16.9 million IQ points due to exposure to OPs.[2]

EPA'S APPROACH TO WORKER RISK MITIGATION IS UNDERPROTECTIVE AND AT ODDS WITH STANDARD OCCUPATIONAL HEALTH PRACTICE

37. When EPA identifies a risk of concern, it explores as a first priority whether use of personal protective equipment will eliminate the risk. If personal protective equipment is found not to be protective, EPA then asks whether engineering controls or administrative controls such as restricted re-entry intervals will eliminate the risk. Only if the risk of concern

[2] D.C. Bellinger, "A Strategy for Comparing Contributions of Environmental Chemicals and Other Risk Factors to Neurodevelopment of Children," *Environmental Health Perspectives*, Vol. 120, No. 4, pp.501-507 (April 2012).

remains after implementation of all such mitigation does EPA explore eliminating the exposure or shifting to less harmful alternative chemicals or application methods.

38. EPA's approach is backwards and wrong. It violates standard, long-established occupational health practice. It fails to protect worker health.

39. In the field of occupational safety and health, regulators adhere to a hierarchy of controls that prioritizes prevention of exposure – not use of personal protection. Regulators start by asking whether the exposure can be eliminated altogether or whether other less toxic chemicals can be substituted. If those approaches are found not to be feasible, the regulator will look to engineering controls such as machine-guarding or administrative controls such as longer re-entry times to sprayed fields. The regulator will turn to personal protective equipment only as a last resort, because personal protective equipment has been shown repeatedly over the decades to be far less effective at worker protection than product substitution, engineering controls and administrative controls. A final reason for not relying on personal protective equipment is that such equipment degrades workers' ability to function and increases risk of heat stress and heat stroke. Thus double-layers of clothing, gloves, and respirators likely impede mobility and contribute to heat and respiratory stress of pesticide handlers working in hot temperatures during summer growing seasons.

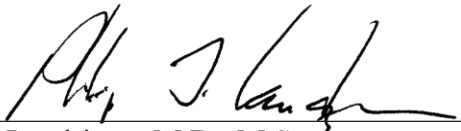
40. OSHA has adhered to this prioritization for decades. The lead standard is illustrative. EPA refused to rely on personal protective equipment, and on respirators in particular, because they fail to eliminate exposure, provides inadequate protection, and creates additional hazards by interfering with vision and mobility. The 1978 lead standard is replete with findings that respirators afford inadequate protection. OSHA required respirators **in**

addition to engineering controls to afford workers additional protection during the time it would take to fully implement the controls. 43 Fed. Reg. 52,952 (Nov. 14, 1978).

41. For decades, EPA has adopted a wrong-headed strategy for mitigating worker exposures to chlorpyrifos and other toxic pesticides that relies first and foremost on personal protective equipment. By relying on this inadequate strategy and by relying on personal protective equipment that has been shown to confer highly inadequate protection, EPA has allowed workers to be exposed to harmful levels of chlorpyrifos. By relying on this ineffective strategy, EPA has allowed pregnant women workers to be occupationally exposed to levels of chlorpyrifos that can result in fetal brain damage to infants in the womb. Sound occupational health principles require engineering or administrative controls, where effective, or elimination of the exposure, where engineering or administrative controls are not effective.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on this 7th day of September 2016, in New York, New York.



Philip J. Landrigan, M.D., M.Sc.

Exhibit 1

CURRICULUM VITAE

Philip J. Landrigan, M.D., M.Sc., D.I.H., F.A.A.P., F.A.C.P.M.

ACADEMIC APPOINTMENTS

Current: **Icahn School of Medicine at Mount Sinai,** Dean for Global Health, 2010-Present
Icahn School of Medicine at Mount Sinai, Professor, Department of Preventive Medicine, 1990-Present
Icahn School of Medicine at Mount Sinai, Professor of Pediatrics, 1985-Present

Previous: **Icahn School of Medicine at Mount Sinai,** Ethel H. Wise Professor and Chairman, Department of Preventive Medicine, 1990-2015.
Icahn School of Medicine at Mount Sinai, Director, Division of Environmental and Occupational Medicine, Department of Community and Preventive Medicine, 1985-1990.
U.S. Environmental Protection Agency, Senior Advisor to the Administrator on Children's Health and the Environment, 1997-1998. (Sabbatical position)
National Institute for Occupational Safety and Health, Director, Division of Surveillance, Hazard Evaluations and Field Studies, 1979-1985.
Centers for Disease Control and Prevention

- Chief, Environmental Hazards Activity, Bureau of Epidemiology, 1974-1979.
- Director, Research and Development, Bureau of Smallpox Eradication, 1973-1974.
- Epidemic Intelligence Service (EIS) Officer, 1970-1973.

Adjunct Positions:

Harvard School of Public Health, Adjunct Professor of Environmental Health, 2010-present; Visiting Lecturer on Occupational Health, 1981-2010
Harvard Medical School, Clinical Instructor in Pediatrics, 1969-1970; Visiting Lecturer on Preventive Medicine and Clinical Epidemiology, 1982-Present
University of Washington School of Public Health and Community Medicine, Auxilary Clinical Professor of Environmental Health, 1983-2013
University of Cincinnati, Department of Environmental Health, College of Medicine, Assistant Clinical Professor of Environmental Health, 1981-1986
London School of Hygiene and Tropical Medicine, Visiting Fellow, TUC Institute of Occupational Health, 1976-1977

EDUCATION

High School: Boston Latin School, 1959
College: Boston College, A.B. (magna cum laude), 1963
Medical School: Harvard Medical School, M.D., 1967

POSTDOCTORAL TRAINING

Internship: Cleveland Metropolitan General Hospital, 1967-1968
Residency: Children's Hospital Medical Center, Boston, (Pediatrics), 1968-1970
Post Graduate: London School of Hygiene & Tropical Medicine, 1976-77
Diploma of Industrial Health (England), 1977
Master of Science in Occupational Medicine, University of London (with distinction), 1977

CERTIFICATION

American Board of Pediatrics - 1973
American Board of Preventive Medicine:
General Preventive Medicine - l979
Occupational Medicine - 1983

MEDICAL LICENSURE

Massachusetts #31277, 1967 - present
New York #162034, 1985 - present

INSTITUTE OF MEDICINE

Institute of Medicine, National Academy of Sciences, Elected to membership, 1987

HONORS/AWARDS

Asbestos Disease Awareness Association – Dr. Irving Selikoff Lifetime Achievement Award, 2016
Grassroots Environmental Education, Award for Outstanding Leadership in Children's Environmental Health, 2015
Boston College – Distinguished Alumni Research Award, 2014
Boston Latin School - Distinguished Graduate Award, 2014
University of Medicine & Dentistry of New Jersey - Senator Frank R. Lautenberg Annual Award in Public Health, 2011
Hearst Foundation, The Daily Green – The Heart of Green Award, 2010
The New York Academy of Medicine – The Stephen Smith Medal for Lifetime Achievement in Public Health, 2009
U.S. Environmental Protection Agency, Region II – Environmental Quality Award on behalf of Mount Sinai Medical Center, 2009
Westchester County (NY). Sustainability Award for Service on Westchester County Global Warming Task Force, 2009
Student Physicians for Social Responsibility. Lifetime Achievement Award, 2009
Women's City Club of New York. Civic Spirit Award, 2009
Boston College. Alumni Award for Professional Excellence, 2008
Collegium Ramazzini. Irving J. Selikoff Award, 2008
Healthy Schools Network, Inc. Healthy Schools Hero Award, 2008
Westchester Children's Association. Edith Macy Award for Distinguished Service, 2008
Children's Health Environmental Coalition. Lifetime Achievement Award, 2006
U.S. Environmental Protection Agency. Child Health Champion Award, 2006
Huntington Breast Cancer Action Coalition. Humanities Award for Children's Health Protection, 2005
Icahn School of Medicine at Mount Sinai. J. Lester Gabrilove Award, 2005
American College of Occupational and Environmental Medicine. Health Achievement in Occupational Medicine Award, 2005
National Nutritional Foods Association. Rachel Carson Environmental Award, 2005
Federated Conservationists of Westchester County. Super Hero Award for Children's Health, 2005
Physicians for Social Responsibility, Los Angeles Chapter. Socially Responsible Medicine Award, 2004
***Organic Style Magazine*.** Environmental Power List, 2004
Finnish Institute for Occupational Health. Jorma Rantanen Award, 2003
American Public Health Association, David P. Rall Award for Advocacy in Public Health, 2003
Castle Connolly Ltd.-America's Top Doctor. Preventive Medicine. New York Metropolitan Area and United States 2001, 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014 and 2015

HONORS/AWARDS (cont)

Public Health Association of New York City, Haven Emerson Award, 2002
National Institute for Occupational Safety & Health, James Keogh Award, 2002
Icahn School of Medicine at Mount Sinai, Jacobi Medallion, 2002
Environmental Advocates (New York), Award for Environmental Advocacy on Behalf of Children, 2000
American Conference of Governmental Industrial Hygienists, William Steiger Memorial Award, 2000
Russian Academy of Medical Science, Elected as Foreign Member, 2000
Earth Day New York, Award for Excellence in Environmental Medicine, 1999
Mothers & Others for a Livable Planet, Award for Advocacy on Behalf of the Health of Children, 1999
American College of Preventive Medicine, Katherine Boucot Sturgis Award, 1999
International Society for Occupational and Environmental Health, Vernon Houk Award, 1998
New Jersey Environmental Federation Certificate of Recognition. Environmental Achievement Award, 1998
Physicians for Social Responsibility, Broad Street Pump Award in Environmental Health, 1996
International Association of Fire Fighters, Occupational Health and Safety Award, 1995
American Public Health Association, Herbert L. Needleman Medal and Award for Scientific Contributions and Advocacy on Behalf of Children, 1995
United Brotherhood of Carpenters, William Sidell Presidential Award, 1995
New England College of Occupational and Environmental Medicine, Harriet Hardy Award, 1993
New York Committee for Occupational Safety and Health, Annual Honoree, 1985
United States Navy

- Navy & Marine Corps Commendation Medal (3 awards), 2002, 2003 and 2005
- National Defense Service Medal, 2003
- Secretary of Defense Medal for Outstanding Public Service, 2002

U.S. Public Health Service

- Meritorious Service Medal, 1985
- Group Citation as Member of Beryllium Review Panel, 1978
- Career Development Award, 1976

U.S. Department of Health, Education and Welfare, Volunteer Award, 1973

HONORARY DEGREES:

Mount Sinai School of Medicine, Doctor of Science (honoris causa), 2007

OTHER PROFESSIONAL APPOINTMENTS:

American College of Preventive Medicine Fellow, 2003-present
Physicians for Social Responsibility, Board of Directors 1996-1999; Board of Sponsors, 1994-95
New York Academy of Medicine, Elected Fellow, 1991
American College of Occupational and Environmental Medicine, Fellow, 1986
Herman Biggs Society, Member, 1986-1992
International Commission on Occupational Health, Member, 1985-present
Collegium Ramazzini, Fellow, 1983-present
President, 1997-present
American College of Epidemiology, Fellow, 1983-present
Board of Directors, 1990 - 1993
American Epidemiological Society, Elected Member, 1982-present
American Public Health Association, Member, 1982-present
Occupational Health Section, Chair, 1989-90

OTHER PROFESSIONAL APPOINTMENTS: (cont)

Society for Epidemiologic Research, Member, 1978-present
Royal Society of Medicine, Elected Fellow, 1977
American Academy of Pediatrics, Fellow, 1975-present
New York Occupational Medicine Association, Member 1985-present
Board of Directors, 1988-1990
New York Academy of Sciences, Fellow 2002-present

COMMITTEES:

The White House

Presidential Advisory Committee on Gulf War Veterans' Illnesses, 1995-1996

American Academy of Pediatrics

Committee on Environmental Hazards, 1976-1987. Chairman, 1983-1987

National Research Council

Institute of Medicine, Chairman, Interest Group (14) Environmental and Occupational Health and Toxicology, 2009-2011
National Academy of Sciences, Board on Sustainable Development, 1995-1998
National Academy of Sciences, Committee on the Scientific Issues Surrounding the Regulation of Pesticides in the Diets of Infants and Children, Chairman, 1988-1992
National Academy of Sciences, Committee on Neurotoxicology in Risk Assessment, 1987-1989
National Academy of Sciences, Committee on the Epidemiology of Air Pollutants, Vice-Chairman, 1984-1985
National Academy of Sciences, Assembly of Life Sciences, 1981-1982; Commission on Life Sciences, 1982-1984
National Academy of Sciences, Panel on the Proposed Air Force Study of Herbicide Agent Orange, 1979-1980
Institute of Medicine, Committee for a Planning Study for an Ongoing Study of Costs of Environment-Related Health Effects, 1979-1980
National Academy of Sciences, Assembly of Life Sciences. Board on Toxicology and Environmental Health Hazards, 1978-1987; Vice-Chairman, 1981-1984

National Institutes of Health/U.S. Public Health Service

National Institutes of Health, National Institute of Environmental Health Sciences, External Clinical Advisory Council, 2009-present
National Institute of Child Health and Human Development, Federal Advisory Committee to the National Children's Study, 2003-2005
National Institute of Child Health and Human Development, National Children's Study, Executive Steering Committee, 2007-2009
Food and Drug Administration, Ranch Hand Advisory Committee, 2000-2001
National Institute for Occupational Safety and Health, Board of Scientific Counselors, 1995-1997
National Institutes of Health, Study Section on Epidemiology and Disease Control, 1986-1990
National Institute of Environmental Health Sciences, Third Task Force for Research Planning in the Environmental Health Sciences; Chairman, Subtask Force on Research Strategies for Prevention of and Intervention in Environmentally Produced Disease, 1983-1984

Department of Defense

Armed Forces Epidemiological Board, 2000-2002

COMMITTEES: (cont)

State and Local Government

New York State, Governor's Advisory Committee on Safety and Healthy New York Foods, 2015-2016
State of New York, Advisory Council on Children's Environmental Health, Co-Chair 2009-present
State of New York, Advisory Council on Lead Poisoning Prevention, 2009-present
Westchester County, New York, Westchester County Global Warming Task Force, 2006-2008
City of New York, Weapons of Mass Destruction (WMD) Advisory Group, 2002-2008
State of New York, Health Research Science Board, 1997-present
State of New York, Public Health Priorities Committee, 1996
State of New York, New York State Advisory Council on Lead Poisoning Prevention, Chairman, 1993-2004
City of New York, Mayor's Lead Paint Poisoning Advisory Committee, 1991-1993
State of New York, Asbestos Advisory Board, Chair, 1987-present
State of New Jersey, Meadowlands Cancer Advisory Board, Chair, 1987-1989
State of New York, Governor's Blue Ribbon Committee on the Love Canal, 1978-1979

Academic

Cornell University, Dean's Advisory Council in Veterinary Medicine, 1996-1997
Mickey Leland National Urban Air Toxics Research Center, National Advisory Committee, 1994-1995
New York Academy of Medicine, Working Group on Housing and Health, 1987-1989; Chairman, 1989
New York Lung Association, Research and Scientific Advisory Committee, 1986-1989. Board of Directors, 1987-1990
Association of University Programs in Occupational Health and Safety, 1985-Present; President, 1986-1988
Milbank Memorial Foundation, Technical Board, 1986-1988
Harvard School of Public Health, Occupational Health Program, Residency Review Committee, 1981-1983; Chairman, 1981

International Organizations

World Health Organization. Contributor to the WHO Publication: "Guidelines on Studies in Environmental Epidemiology" (Environmental Health Criteria, No. 27), 1984.
International Agency for Research on Cancer, service as member of Working Groups on Cancer Assessment, volume 29 (benezene), 1981; volume 42 (silica), 1986; voume 87 (lead), 2005; volume 98, (firefighting) 2007; volume 100 (asbestos). 2011.

Environmental Organizations

Healthy Child, Healthy World, Board of Directors, 1996-present
Children's Environmental Health Network, Board of Directors, 1995-present
Environmental Health Foundation, Board of Directors, 1993-1996
INFORM, Board of Directors, 1991-2003

Labor Unions

International Brotherhood of Teamsters, National Health and Safety Advisory Committee, 1994-2002
George Meany Center for Labor Studies, Board of Trustees, 1994-1997
United Brotherhood of Carpenters, National Health and Safety Fund, Medical Advisory Committee, 1990 -2000; Chairman, 1994-2000

COMMITTEES: (cont)

Labor Unions

United Automobile Workers (UAW) - Chrysler Corporation, Joint Scientific Advisory Committee, Member, 1990-2006

International Association of Fire Fighters, John Redmond Foundation, Medical Advisory Committee, 1989-present

Other Organizations

Health Insurance Plan (HIP) of Greater New York, Board of Directors, 1992-1994

American Legion, Science Panel, Chairman, 1988-2000

Editorial Boards

Editor-in-Chief: *Annals of Global Health*, 2013-present

Deputy Editor: *American Journal of Industrial Medicine,* 2007-present

Associate Editor: *Environmental Health Perspectives,* 2002-present

Editorial Board: *Journal of Public Health Management and Practice*, 1995-1996

Editor-in-Chief: *American Journal of Industrial Medicine*, 1992-2006; Consulting Editor, 1979-1992

Editorial Board: *New Solutions*: A Journal of Environmental and Occupational Health Policy, 1990-present

Editorial Board: *The PSR Quarterly*, 1990-1994

Editorial Board: *American Journal of Public Health*, 1987-1993

Editor-in-Chief: *Environmental Research*, 1987-1994

Senior Editor: *Environmental Research*, 1985-1987

Editorial Board: *Annual Review of Public Health*, 1984-1990

Consulting Editor: *Archives of Environmental Health*, 1982-present

National Service

United States Naval Reserve, Medical Corps, 1996-2005
LCDR (0-4) 1996-98; CDR (0-5) 1998 – 2004; CAPT (0-6) 2004-2005. Retired January 1, 2005.

United States Public Health Service, Commissioned Corps, 1970-1995. LCDR (0-4) to CAPT (0-6).

New York Naval Militia 2000-present; CAPT (0-6); Surgeon General.

GRANT SUPPORT

ACTIVE:

Blacksmith Institute (Landrigan, PI) 01/01/12 – 12/23/17
$45,000

Assessing the Disease Burden of Hazardous Waste Sites

The purpose of this contract is to support the development of a series of scientific papers that will assess the health burden associated with human exposure to hazardous waste sites in the developing world.

2011-N-13318 (Lucchini, PI) 07/01/11 – 12/31/16
CDC $28,422,550

World Trade Center Data and Coordination Center

This project is the coordinating center for a multicenter program providing monitoring and treatment to volunteers who assisted in the recovery and cleanup after the 9/11 attack.

Role: Co-Investigator

Completed Research Support:

1T32HD049311 (Landrigan, PI) 05/01/07 - 07/01/13
NICHD $323,002
Research Training Program in Environmental Pediatrics
The goal of this interdisciplinary research training program is to train the next generation of physician-researchers and academic leaders in environmental pediatrics.

C-010124 NYS DoH Landrigan (PI) 4/1/09 – 3/31/12; Lucchini (PI) 4/1/12 – present
World Trade Center Responders Data and Coordinating Center. This program has collected, analyzed and published medical monitoring and treatment data collected clinically on 30,000 9/11 responders evaluated at five Clinical Centers in the New York metropolitan area.

NIH-HHSN27520080031C (Landrigan, PI) 09/28/08 - 09/27/13 (Monroe)
NIH
National Children's Study Vanguard Centers
This project will recruit 1250 live births into a NICHD study of social, behavioral and environmental factors and their impact on childhood health, growth and development. The Queens Vanguard Center is one of the first six sites selected to pilot the NCS, which will follow more than 100,000 children across the United States from birth until age 21.

U10-OH08232 CDC Landrigan (PI) 6/1/04 – 3/31/12; Lucchini (PI) 4/1/12 - present
New York/New Jersey Education Research Center in Occupational Safety & Health. The goal of this multi-institutional program is to train professionals from multiple disciplines - medicine, nursing, industrial hygiene and industrial safety - to be future leaders in occupational health and safety.

Children's Environmental Health Center - Inner City Toxicants, Child Growth and Development
Co-Principal Investigator
EPA RD831711-01 11/1/03 – 10/31/10
NIEHS P01 ES009584 11/1/98 – 10/31/10

ORIGINAL, PEER-REVIEWED PUBLICATIONS

1. Lovejoy FH Jr, Marcuse EK, Landrigan PJ: Two examples of purpura factitia. *Clinica Pediatr* 10:183-184, 1971.
2. Landrigan PJ, Conrad JL: Current status of measles in the United States. *J Infect Dis* 124:620-622, 1971.
3. Landrigan PJ: Epidemic measles in a divided city. *JAMA* 221:567-570, 1972.
4. Hattwick MA, Hochberg FH, Landrigan PJ, Gregg MG: Skunk-associated human rabies. *JAMA* 222:44-50, 1972.
5. Grand MG, Wyll SA, Gehlbach SH, Landrigan PJ, Judelsohn RG, Zendel SA, Witte JJ: Clinical reactions following rubella vaccination: A prospective analysis of joint, muscular, and neuritic symptoms. *JAMA* 220:1569-1572, 1972.
6. Landrigan PJ, Griesbach PH: Measles - In previously vaccinated children in Illinois. *Illinois Med J* 141:367-372, 1972.
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INVITED LECTURES/PRESENTATIONS

Visiting Professorships and Lectureships:

University of Utah, Wallace Stegner Lecturer, 2012
Harvard School of Public Health, The James L. Whittenberger Lecturer, 2009
University of Kentucky, Inaugural John P. Wyatt Lecturer in Environmental Health and Disease, 2008
University of Minnesota, School of Public Health, Richard G. Bond Memorial Lecture, 2007
James P. Keogh, MD Memorial, Lecturer in Occupational Medicine, University of Maryland School of Medicine, 2006
Royal College of Physicians (London), Faculty of Occupational Medicine, Richard Schilling Memorial Lecturer, 2000
University of Rochester, 44th Annual Paul W. Beaven Lecturer, 2000
Centers for Disease Control and Prevention, Langmuir Memorial Lecturer, 1999
Mayo Clinic, Department of Pediatrics, Amberg-Helmholtz Lecturer in Pediatrics, 1998
Duke University Medical School, Visiting Professor, NIEHS Clinical Training Program in Environmental Medicine, 1995
National University of Singapore, Visiting External Examiner in Occupational Medicine, 1994
Medical College of Pennsylvania, Catherine Boucot Sturgis Visiting Professor in Community and Preventive Medicine, March 1992
University of Cape Town Medical School, Visiting Professor, Department of Community Health, March 1992
University of Tokyo, Visiting Professor of the University, July 1990
University of Tokyo, Visiting Professor of the Faculty of Medicine, September 1989

Exhibit 2

By Philip J. Landrigan and Lynn R. Goldman

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Children's Vulnerability To Toxic Chemicals: A Challenge And Opportunity To Strengthen Health And Environmental Policy

Philip J. Landrigan (phil.landrigan@mssm.edu) is the dean for global health and a professor of both preventive medicine and pediatrics at the Mount Sinai School of Medicine, in New York City.

Lynn R. Goldman is the dean of the School of Public Health and a professor of environmental and occupational health at the George Washington University, in Washington, D.C.

ABSTRACT A key policy breakthrough occurred nearly twenty years ago with the discovery that children are far more sensitive than adults to toxic chemicals in the environment. This finding led to the recognition that chemical exposures early in life are significant and preventable causes of disease in children and adults. We review this knowledge and recommend a new policy to regulate industrial and consumer chemicals that will protect the health of children and all Americans, prevent disease, and reduce health care costs. The linchpins of a new US chemical policy will be: first, a legally mandated requirement to test the toxicity of chemicals already in commerce, prioritizing chemicals in the widest use, and incorporating new assessment technologies; second, a tiered approach to premarket evaluation of new chemicals; and third, epidemiologic monitoring and focused health studies of exposed populations.

Recognition of the unique vulnerability of children, infants, and fetuses to toxic chemicals in the environment was a watershed development for health and environmental policy.[1] This discovery catalyzed two further insights: that early life exposures to toxic chemicals are important causes of disease and dysfunction in children and also in adults,[2–4] and that diseases caused by chemicals can successfully be prevented, thus saving lives, enhancing the quality of life, reducing health care and education costs, and increasing national productivity. A notable example is the nation's experience with removing lead from gasoline. This one change reduced lead poisoning by more than 90 percent[5] and produced an estimated annual economic benefit of $110 billion to $319 billion.[6]

These insights have affected risk assessment, regulation, and law.[7] In this article we explore the implications for health and environmental policy.

Children Are Vulnerable To Toxic Chemicals

The realization that children are uniquely sensitive to toxic chemicals was catalyzed by the publication in 1993 of a National Academies report, *Pesticides in the Diets of Infants and Children*.[1] Studies cited in the report found that children are quantitatively and qualitatively different from adults in their sensitivity to pesticides and other chemicals.

Prior to the report's publication, virtually all environmental policy in the United States had focused on assessment of risk to the "average adult." Risk assessment had paid scant heed to exposures that diverged from the norm. Little attention was paid to the unique risks of infants, children, or other vulnerable groups within the population.

The report produced a paradigm shift in that approach to health and environmental policy. It led to new legislative and regulatory initiatives to better protect infants and children against environmental health threats and has been especially

influential in changing the regulation of pesticide and pharmaceutical chemicals.[7]

The report identified four differences between children and adults that contribute to children's heightened susceptibility to chemicals in the environment.

First, children have greater exposures to toxic chemicals for their body weight than adults.[1] A six-month-old infant drinks seven times more water per pound than an adult.[8] Children take in three to four times more calories per pound than adults. The air intake per pound of an infant is twice that of an adult. These differences result in children being disproportionately exposed to toxic chemicals in air, food, and water. Children's hand-to-mouth behavior and play on the ground further magnify their exposures.

Second, children's metabolic pathways are immature,[1] and a child's ability to metabolize toxic chemicals is different from an adult's. In some instances, infants are at lower risk than adults because they cannot convert chemicals to their toxic forms. More commonly, however, children are more vulnerable because they lack the enzymes needed to break down and remove toxic chemicals from the body.[9]

Third, children's early developmental processes are easily disrupted.[1] Rapid, complex, and highly choreographed development takes place in prenatal life and in the first years after birth, continuing more slowly throughout childhood into puberty. In the brain, for example, billions of cells must form, move to their assigned positions, and establish trillions of precise interconnections.[10] Likewise, development of the reproductive organs is guided by a complex and precisely timed sequence of chemical messages and is shaped by maternal and fetal hormones.[11]

Recent research in pediatrics and developmental toxicology has elaborated the concept of "windows of vulnerability."[12] These are critical periods in early development when exposures to even minute doses of toxic chemicals—levels that would have no adverse effect on an adult—can disrupt organ formation and cause lifelong functional impairments.

If, for example, cells in an infant's brain are injured by lead or a pesticide, the consequences can include developmental disabilities in childhood[11,13] and possibly increased risk of neurological degeneration, such as Parkinson's disease, in adult life.[4] If inappropriate hormonal signals are sent to the developing reproductive organs by a synthetic chemical endocrine disruptor—such as certain chemicals commonly found in household products, plastics, and cosmetics (phthalates), and on clothing (flame retardants)—lifelong reproductive impairment may ensue.[11] These windows of vulnerability have no equivalent in adult life.

Fourth, children have more time than adults to develop chronic diseases. Many diseases triggered by toxic chemicals, such as cancer and neurodegenerative diseases, are now understood to evolve through multistage, multiyear processes that may be initiated by exposures in infancy.[1,4] This insight has catalyzed new research to identify how early environmental influences may affect health in childhood and across the human lifespan. Notable research includes the US National Children's Study,[14] the Japan Environment and Children's Study,[15] and the International Childhood Cancer Cohort Consortium.[16]

Rates Of Chronic Diseases In US Children Are Rising

Today in the United States, the principal causes of sickness, disability, and death in children are chronic illnesses. Rates of many of these diseases are high and rising.[2,3] Toxic chemicals in the environment are making important contributions to these disease trends.

Asthma is one of the most common chronic diseases in American children. The prevalence of childhood asthma has more than doubled over the past twenty years, and in 2008, 9 percent of all US children had asthma.[17,18] Asthma is the leading cause of pediatric hospitalization and school absenteeism and a major driver of pediatric health costs.

Birth defects are now the leading cause of infant death and are associated with substantial health and education costs. Certain birth defects, such as those of the male reproductive organs[19] and of the abdominal wall,[20] appear to have increased in frequency.

Neurodevelopmental disorders, including dyslexia, mental retardation, attention deficit hyperactivity disorder, and autism, affect 5–10 percent of the babies born in the United States each year.[21] Autism spectrum disorder is currently diagnosed in one of every 110 American children.[22] The prevalence of attention deficit hyperactivity disorder has also risen, and today 14 percent of US children have been diagnosed with this condition; two-thirds of them also have learning disabilities.[23]

The incidence of leukemia and brain cancer in children younger than age eighteen increased steadily from the 1970s through the 1990s, despite declining mortality.[24] Testicular cancer in males ages 15–30 has increased in incidence by more than 50 percent.[24]

Obesity in children has tripled in prevalence over the past twenty years, from 5 percent to

17 percent.[25] One of its consequences, type 2 diabetes, is occurring earlier in life and at epidemic rates.

Children And The Chemical Environment

The environment in which American children live has changed greatly in the past fifty years, especially in terms of the chemicals to which they are routinely exposed. During this time, more than 80,000 new synthetic chemicals have been invented and are used today in millions of consumer products, ranging from foods and food packaging to clothing, building materials, cleaning products, cosmetics, toys, and baby bottles.[26] Some of these chemicals may pose risks for children's health. The Environmental Protection Agency has identified 3,000 "high–production-volume" chemicals—chemicals produced in quantities of more than a million pounds per year—that are in widest use and therefore have the greatest potential for human exposure. Children are especially at risk for exposure to these chemicals.

In national surveys conducted by the Centers for Disease Control and Prevention, measurable quantities of 200 high–production-volume chemicals have been detected in the blood and urine of virtually all Americans,[27] including pregnant women.[28] The significance of this finding for human health is not fully understood. But it is worrisome, because most of these chemicals have not undergone even minimal assessment for potential toxicity, and only about 20 percent of them have been screened for their potential to disrupt early human development or to cause disease in infants and children.[26] Even less is known about the potential effects of exposure to several of these chemicals simultaneously, or how they may interact with one another in the human body, possibly causing synergistic adverse effects on health.

The absence of information about the possible risks associated with routine exposure to untested synthetic chemicals is fraught with risk for disease and dysfunction. Unless studies are conducted to specifically seek ill effects associated with chemical exposures, dysfunctions can go unrecognized for many years.

The "silent epidemic" of childhood lead poisoning[6,13] is a dramatic case in point. Millions of American children were exposed to excessive levels of lead from the 1940s to the 1970s, when lead was an additive to gasoline. Many suffered unrecognized brain injury before sufficient evidence could be marshaled to mandate removing lead from gasoline, household paint, and consumer products.[5,6]

Failure to evaluate chemicals for potential toxicity reflects the failure of the Toxic Substances Control Act of 1976.[29] At the time of its passage, the act was intended to be pioneering legislation that would require testing chemicals already in commerce for potential toxicity, and would also require premarket evaluation of all new chemicals. The act never fulfilled these intentions. A particularly egregious lapse was a decision by Congress to "grandfather in" 62,000 chemicals already on the market without any toxicity testing requirement.[29,30] These chemicals were presumed to be safe and allowed to remain in commerce, unless and until the Environmental Protection Agency made a finding that they posed an "unreasonable risk."[30]

The "unreasonable risk" standard identified in the Toxic Substances Control Act has created a substantial barrier to the regulation of industrial and consumer chemicals. This standard has been so burdensome that the Environmental Protection Agency has not been able to remove chemicals from the market except when there is overwhelming evidence of potential harm. The result is that only five chemicals have been controlled under the act in the thirty-five years since its passage. These chemicals were polychlorinated biphenyls (PCBs), chlorofluorocarbons, dioxin, asbestos, and hexavalent chromium. Only two of these five were totally banned: PCBs, which were eliminated by an act of Congress and not because the Environmental Protection Agency exercised its authority, and asbestos, a chemical for which there is overwhelming evidence of serious hazard to human health.

Further barriers to enforcement of the Toxic Substances Control Act have resulted from the federal courts' interpretation of the "unreasonable risk" standard. Thus, in a 1991 opinion on the asbestos ban in *Corrosion Proof Fittings v. EPA*, the Fifth Circuit found that the Environmental Protection Agency had failed to show that it was taking the "least burdensome" approach required under the act in formulating its final rule banning asbestos. The court thus overturned the agency's rule. This interpretation has made it virtually impossible since 1991 for the Environmental Protection Agency to regulate dangerous chemicals under the act.[30]

Toxic Chemicals And Disease In Children

Evidence is strong and continuing to accumulate that toxic chemicals are important causes of disease and dysfunction in children. This recognition first arose in studies of lead and mercury.[31–36] In recent years, as research strategies in environmental pediatrics have become more refined, the

pace of scientific discovery has quickened and a series of new associations has been discovered. Examples include the following.

Prenatal exposure to PCBs is associated with reduction in children's intelligence.[37] PCBs are an environmentally persistent class of chemicals that accumulate to high levels in certain species of fish. Human exposure is principally the consequence of maternal consumption of contaminated fish before and during pregnancy. Although PCBs are no longer manufactured in the United States, they were used extensively for many years in manufacturing electrical equipment such as transformers, and they continue to be important contaminants today because they are highly persistent in the environment and because they become concentrated in the tissues of organisms in the food chain.

Prenatal exposure to the commonly used insecticide chlorpyrifos is associated with reduced head circumference at birth [38] and with developmental delays.[39] Small head circumference at birth is an indicator of delayed brain growth during pregnancy. Chlorpyrifos is also linked to pervasive developmental disorder, a form of autism.[39,40]

Baby boys exposed in the womb to phthalates—a chemical compound found in plastics, cosmetics, and many common household products—appear to be at increased risk of behavioral abnormalities that resemble attention deficit hyperactivity disorder.[41] Prenatal exposure to bisphenol A, a synthetic chemical used to manufacture polycarbonate plastics, is linked to behavioral abnormalities in girls.[42] Prenatal exposure to brominated flame retardants is linked to cognitive impairments,[43] and prenatal exposures to arsenic and manganese is associated with neurodevelopmental impairment.[44,45]

Rates of asthma are increased in children exposed to secondhand cigarette smoke and to fine particulate air pollution.[17,18] Risk of respiratory death is increased in infants exposed to fine particulate air pollution.[46]

Prenatal exposure to phthalates has also been linked to shortening of the ano-genital distance in baby boys, a finding indicative of feminization.[47] Prenatal exposure to perfluorinated chemicals (perfluorooctanic acid and perfluorooctane sulfonate) used to make nonstick pans and stain repellents has been linked to decreased birthweight and reduced head circumference in newborn infants.[48]

Diseases Associated With Chemicals Are Costly

Preventing exposures to chemicals can yield great savings. To estimate the contribution of environmental pollutants to the prevalence and costs of disease in American children, investigators at Mount Sinai School of Medicine examined four categories of illness: lead poisoning, asthma, cancer, and neurobehavioral disorders.[49] Based on prevalence, the environmentally attributable fraction of each disease, and national economic data, they calculated that the total annual costs of these diseases attributable to environmental exposures is $54.9 billion (range $48.8 billion to $64.8 billion): $43.4 billion for lead poisoning, $2.0 billion for asthma, $0.3 billion for childhood cancer, and $9.2 billion for neurobehavioral disorders. Because of the difficulties inherent in assessing the full economic consequences of neurobehavioral impairments, it is likely that these estimates are low.

Disease and dysfunction caused by toxic chemicals can be prevented. Prevention is most effectively achieved by assessing chemicals for toxicity through laboratory and human studies and using the data gained in those assessments to guide evidence-based prevention of exposure. Great cost savings can result.

Again, we use the example of phasing out the use of lead in gasoline. This phase-out began in the United States in 1976, was 50 percent accomplished by 1980, and virtually complete by 2000.[5] Prior to 1976, 100,000 tons of tetraethyl lead was added to the US gasoline supply each year to improve engine performance and fuel efficiency. Widespread environmental contamination resulted.

The average US blood lead level peaked in the mid-1970s at 17 micrograms per deciliter,[5] a level significantly above the current Centers for Disease Control and Prevention guideline of 10 micrograms per deciliter and now known to be associated with significant toxic injury to the developing brain. These elevated blood lead levels, found in epidemiologic studies, were associated with reduced intelligence, shortened attention span, and disruptive behavior in children.[33,34] Each increase of 3 micrograms per deciliter in mean blood lead level was shown to be associated with a decline of 0.5–1.0 points in intelligence quotient (IQ).[6] These effects occurred in the absence of any clinical symptoms or obvious illness and were thus termed "silent" lead poisoning.[34]

The discovery that lead could erode children's intelligence even at relatively low levels was not the original justification for the Environmental Protection Agency's decision to remove lead from gasoline. In fact, the decision to remove lead was made in the first instance to protect catalytic converters from damage by lead. However, the discovery did play an important role in reinforcing the decision and in sustaining it over

time. A result of the phase-out was that between 1976 and 1990 the mean blood lead level of American children decreased by more than 90 percent (to below 2 micrograms per deciliter today).[5] The incidence of childhood lead poisoning also fell by more than 90 percent.[5]

A further consequence of the reduction in exposure to lead was that the mean IQ of American children has increased.[6] Children born in the United States today are estimated to have IQ scores that, on average, are 2.2–4.7 points higher than those of children born in the early 1970s.[6] And because each 1-point gain in population mean IQ is associated with an estimated 2 percent increase in productivity over a lifetime,[50] the gain in population IQ is estimated to have produced a national economic benefit of $110–$319 billion in each annual cohort of babies born in the United States since the 1980s.[6]

Consequences For Environmental Policy

The recognition of children's unique vulnerability to toxic chemicals has had far-reaching consequences.

LEGISLATIVE CONSEQUENCES Recognition of children's susceptibility to toxic chemicals strongly influenced the Food Quality Protection Act of 1996, the major federal law governing the use of pesticides. This act became the first federal environmental statute to contain explicit provisions for protecting children's health.

This recognition led also to passage of the Best Pharmaceuticals for Children Act of 2002. This act requires that drugs labeled for use in children undergo studies to specifically examine children's susceptibilities.

CONSEQUENCES FOR RISK ASSESSMENT AND REGULATION A key provision of the Food Quality Protection Act is a requirement that federal pesticide standards ("tolerances") be health-based and that they explicitly consider the effects of pesticides on children's health.[30,51] This requirement represents a diametric change from the previous regulatory regime, in which the health risks of pesticides were balanced against the costs of regulation to agricultural producers in setting standards. This provision of the act forced reexamination of all extant pesticide tolerances to ensure that they met the standard of public health protection. As a result, many uses of pesticides were reduced or dropped altogether.

For example, agricultural use of organophosphate insecticides, a class of pesticide chemicals toxic to brain development, was reduced.[52] The review led also to bans on residential applications of two widely used insecticides—chlorpyrifos and diazinon—that had been used for household pest control.[52]

The Food Quality Protection Act mandates realistic consideration of exposures to multiple pesticides via multiple routes to assess potentially synergistic effects.[53] The law also mandates consideration of exposures to pesticide chemicals that are endocrine disruptors. These are chemicals that exert their toxicity through interactions with the endocrine system, disrupting function of the thyroid or pituitary glands, the ovaries, or the testes, or changing levels of hormones by changing their metabolism.[11]

The new approaches to risk assessment mandated by the Food Quality Protection Act have not yet extended beyond pesticides to include industrial or consumer chemicals.

CONSEQUENCES FOR BIOMEDICAL RESEARCH Recognition of children's vulnerability led to establishment of the Office of Children's Health Protection within the Environmental Protection Agency.[7] It catalyzed a 1997 executive order requiring federal agencies to consider children's special susceptibilities in all policy and rule making.[54] And it led to the creation of a White House Task Force on Children's Health and Safety.

Those programs have, in turn, stimulated substantial investments in children's health research.[7] The resulting initiatives include the following: a national network of Centers for Children's Environmental Health and Disease Prevention Research, supported by the National Institute of Environmental Health Sciences and the Environmental Protection Agency;[55] a network of Pediatric Environmental Health Specialty Units supported by the Centers for Disease Control and Prevention and the Agency for Toxic Substance and Disease Registry;[56] fellowship training programs in environmental pediatrics;[57] and the National Children's Study, a prospective epidemiologic study that will follow a nationally representative sample of 100,000 children from early pregnancy to age twenty-one.[14]

Consequences For Health Policy

The finding that children are uniquely vulnerable to synthetic chemicals indicates the need for fundamental revision of US chemical policy. By default, current policy presumes chemicals to be safe and permits them to enter and remain on the market with minimal evaluation of potential toxicity unless and until they are proved to be overwhelmingly hazardous by the Environmental Protection Agency, using the Toxic Substances Control Act's standard of "unreasonable risk." This policy is neither protective of human health nor consistent with current scientific

understanding of children's vulnerability.

The credible possibility exists that among the hundreds of untested chemicals currently in wide commercial use, there are synthetic chemicals whose toxicity to early childhood development has not yet been discovered.[13] The late David Rall, former director of the National Institute of Environmental Health Sciences, once stated, "If thalidomide [a drug widely used in the 1950s and 1960s to treat morning sickness in early pregnancy] had caused a ten-point loss of IQ instead of obvious birth defects of the limbs, it would probably still be on the market."[58]

To protect human health, and especially the health of infants and children, the paradigm for regulating industrial and consumer chemicals needs to become health-based. The Toxic Substances Control Act's "unreasonable risk" standard needs to be replaced with a new standard that explicitly considers effects of industrial and consumer chemicals on children's health. Such a move would align the strategy used to regulate industrial chemicals with the strategy used under the Food Quality Protection Act to regulate pesticides. It would mark a dramatic change in the current regulatory regime.[30]

A New US Chemical Policy

The linchpin of a new, health-based chemical policy would be a legally mandated requirement that chemicals already on the market be systematically examined for potential toxicity. Such testing will not be an easy task, but it is necessary. It will be far more challenging than updating the tolerances for pesticides proved to be.

To evaluate tens of thousands of chemicals currently in commercial use would require new legislation that directed the Environmental Protection Agency to first address those classes of chemicals that are in the widest use and the most likely to confer risk. Data on the use of chemicals in consumer products, especially products used by young children and pregnant women; data on discharges of chemicals into the air and water; and data on chemicals already widely detectable in the bodies of Americans[27] would help to target the chemicals that most urgently need to be evaluated.[30]

Enhanced evaluation of chemical toxicity would require new, more efficient approaches to toxicity testing. Such approaches are already in development at the National Institute of Environmental Health Sciences and the Environmental Protection Agency.[59] These approaches should incorporate new technologies identified through research in developmental toxicology and consider such complexities as which endpoints to assess, which doses to administer, and which mixtures to evaluate.[59]

A second critical component of a health-based chemical policy would be a legally mandated, strictly enforced requirement that all new chemicals be assessed for potential toxicity before they enter the market. Such assessment could be undertaken in tiered fashion, making use of new rapid assessment methods in computational and in vitro toxicology, taking into account the proposed use patterns of new chemicals, and giving the Environmental Protection Agency latitude to require less extensive evaluation of chemicals and chemical uses judged to be less hazardous to health.[30,59]

As has happened with pesticides, the new approach to the evaluation of industrial chemicals that we propose here would be more likely to result in continued approval for certain uses and withdrawal of approval for others, rather than outright bans of chemicals. For example, the United States, Canada, and the European Union have all recently taken action to ban polycarbonate plastics containing bisphenol A from baby bottles. However, in all of these regions, polycarbonates are still permitted in the manufacture of compact discs, eyeglasses, and other consumer products in which the potential for human exposure is judged to be lower than in uses where the bisphenol A can migrate into foods.

One model approach to health-based chemical policy can be found in the European Union's Registration, Evaluation, Authorisation and Restriction of Chemical Substances legislation, enacted in 2007.[60] This legislation, commonly referred to by its acronym, REACH, places the responsibility on industry to generate substantial amounts of data on potential risks of commercial chemicals and to register this information in a central database that is housed in the European Chemical Agency in Helsinki.[61] The European Chemical Agency not only manages this central database but also coordinates the in-depth evaluation of suspicious chemicals. It is also developing a public database to house and make accessible hazard information relevant to consumers and health and environmental professionals. The first cycle of REACH registrations closed in January 2010 and in February 2011 the European Chemical Agency released its first list of six dangerous substances that are to be phased out by the European Union, through a process that involves scientific analysis and consultation with member states. The European Union is using this information to craft regulations that protect the health of European children, and it has led to bans and restrictions of certain potentially toxic products.[61]

Much of the information collected by the Euro-

pean Union under REACH is claimed as confidential business information and is therefore not available to the US government or to any other entities outside of European Union regulatory authorities. A new, health-based US chemical policy could mandate that industry provide similar data to US regulators. Because these data are already being produced for use in Europe, the marginal costs of providing them to the Environmental Protection Agency should not be great.

A third pillar of a health-based chemical policy would be continued research to examine the impact of chemicals on children's health.[14,55,56] Such research, which includes epidemiologic monitoring of exposed populations as well as specific studies of the effects of particular chemicals, is an essential complement to toxicity testing. It provides direct evidence of the effects of chemicals on human health. It also provides an evidentiary basis for assessing the impact on children's health of policy interventions.

The argument will probably be made that any additional controls on chemicals would cost jobs and harm the economy. However, there is little evidence that environmental protection has to date been harmful to the US economy or to business.[62] To the contrary, there is compelling evidence that the high costs of diseases caused by toxic chemicals are a major, but potentially avoidable, drag on the US economy.[49,63]

Conclusion

Recognition of children's unique vulnerability to toxic chemicals, a vulnerability that receives scant consideration in current US chemical policy, challenges existing policy and creates an opportunity for change.

Creating a new chemical policy explicitly protective of health could prevent disease and dysfunction in childhood and across the lifespan, reduce health and education costs, increase national productivity, and promote better health and well-being for all Americans. ■

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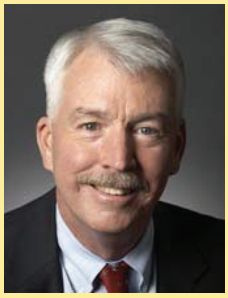
ABOUT THE AUTHORS: PHILIP J. LANDRIGAN & LYNN R. GOLDMAN



Philip J. Landrigan is the dean for global health and a professor of both preventive medicine and pediatrics at the Mount Sinai School of Medicine.

In *Health Affairs* this month, Philip Landrigan and Lynn Goldman review findings that children are far more sensitive to environmental toxins than are adults—and on that basis, the authors argue for an overhaul of our system for regulating chemicals. They call for a requirement that all chemicals to be introduced into the market, as well as those already on the market, be tested for toxicity. What's more, they argue that chemicals' actual or potential impact on all exposed populations, including children, should be taken into account in the testing and review process.

Landrigan, who is also the subject of a "People and Places" article in this issue of *Health Affairs*, is an epidemiologist and pediatrician, and the Ethel Wise Professor of preventive medicine at the Mount Sinai School of Medicine. He is also the school's dean for global health and the director of the Children's Environmental Health Center. He has long been associated with research demonstrating that children are more susceptible than adults to environmental exposures, such as to lead and pesticides.

Landrigan is a recipient of the Meritorious Service Medal of the Public Health Service and a member of the Institute of Medicine. He received his medical degree from Harvard Medical School and a master of science degree in occupational medicine from the University of London.

Lynn R. Goldman is the dean of the School of Public Health and a professor of environmental and occupational health at the George Washington University.

Goldman is the dean of the George Washington University School of Public Health. Previously, she was a professor of environmental health sciences at the Johns Hopkins University's Bloomberg School of Public Health. Before that, she served in the Clinton administration, as assistant administrator for toxic substances in the Environmental Protection Agency. During her time there, the agency overhauled the nation's pesticide laws, expanded right-to-know requirements for release of toxins, reached consensus on an approach to testing chemicals with the potential to disrupt the human endocrine system, developed standards to implement lead screening legislation, and promoted children's health and global chemical safety.

Goldman also worked in environmental health for the California Department of Public Health Services, where she managed a statewide environmental epidemiology program that focused on childhood lead poisoning, birth defects, and occupational health. She is a member of the Institute of Medicine and of the National Academy of Sciences Board on Environmental Sciences and Toxicology.

Goldman earned a master's degree in health and medical science from the University of California, Berkeley; a master of public health degree in epidemiology from the Johns Hopkins University School of Hygiene and Public Health; and a medical degree from the University of California, San Francisco.