No. 24-1068

IN THE

# Supreme Court of the United States

MONSANTO COMPANY,

*Petitioner,*

*v.*

JOHN L. DURNELL,

*Respondent.*

**ON WRIT OF CERTIORARI TO THE MISSOURI COURT OF APPEALS, EASTERN DISTRICT**

## BRIEF OF CHILDREN'S HEALTH DEFENSE AS *AMICUS CURIAE* IN SUPPORT OF RESPONDENT


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## STATEMENT OF INTEREST[1]

Over the last thirty years, chronic illness among children has reached epidemic proportions.[2] Children's Health Defense ("CHD") is a nonprofit organization whose mission is to end the childhood chronic disease epidemic by reducing the toxic environmental exposures that underlie it, especially where those exposures are involuntary. CHD seeks to hold responsible parties accountable and to establish safeguards to prevent future harm to children's health.

CHD is interested in the outcome of this case for two primary reasons. First, glyphosate is the most widely used biocide on the planet and infants and young children are the sub-populations most exposed to glyphosate and particularly vulnerable to its harms. Second, children's health is undermined when federal preemption works in tandem with regulatory failure to protect children from toxic agents, as is threatened in this case.

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1. Under Supreme Court Rule 37.6, CHD states that no counsel for any party authored this brief in whole or in part and no entity or person, aside from CHD, its members, and counsel, made any monetary contribution toward the preparation or submission of this brief.

2. *See* Christopher B. Forrest et al., *Trends in US children's mortality, chronic conditions, obesity, functional status, and symptoms, 334* JAMA 6:509–516 (2025); Enrique Rivero, *Pediatric chronic disease has risen nearly 30% in the last 20 years*, UCLA Health (March 10, 2025), https://www.uclahealth.org/news/release/pediatric-chronic-disease-prevalence-has-risen-nearly-30; CDC, Managing Chronic Conditions, https://www.cdc.gov/school-health-conditions/chronic-conditions/index.html.

Since the 1990s, use of glyphosate-based pesticides has skyrocketed. Indeed, it is now so widespread that glyphosate residue is regularly ingested by children through food and drink, as well through play on treated parks and lawns, in swimming pools and ponds where runoff may occur, and through oral contact with unwashed hands and toys and other objects that are contaminated with residue.

Mounting scientific evidence suggests that such exposure harms children. Yet since 1998, based on a handful of short-term, industry-funded, unpublished animal studies conducted mostly before the turn of the century, the EPA has concluded that glyphosate poses no more risk to infants and children than to adults, and refuses to provide children with the default-level of extra protection required under federal law.

Policymakers must use extra caution when evaluating the impact of environmental exposures on children because children are not merely little adults; rather, their biology leaves them more susceptible to health effects from chemical exposures, and those effects may be lifelong. The EPA's outdated science notwithstanding, the States must be able to use the tort system to protect their youngest and most vulnerable citizens from known pesticide risks by requiring warning labels that acknowledge the full extent of those risks.

## INTRODUCTION AND SUMMARY OF ARGUMENT

The EPA has failed to protect children from risks associated with glyphosate, a now-ubiquitous pesticide chemical that presents special and increasingly evident dangers to children. A question presented by this case is, should the federal government preclude states from requiring pesticide manufacturers to warn of those risks?

Biology and other factors render children more susceptible than adults to injuries from toxins, and experts recognize that extra caution must be used when managing children's exposure to potential toxins. The Food Quality Protection Act ("FQPA") writes this recognition into federal law, requiring EPA to apply an additional tenfold (10X) safety factor to protect infants and children when it conducts the risk assessments that underlie pesticide residue tolerance limits on agricultural commodities, unless reliable data justify a different factor. *See* 21 U.S.C. § 346a(b)(2)(C).

The EPA has ignored both the needs of children and federal law when it comes to glyphosate. When assessing the risks of glyphosate to children and setting residue tolerance limits for glyphosate, the EPA has opted not to study developmental neurotoxicity and other potentially serious effects of glyphosate on children. Instead, relying on a handful of unpublished, industry-funded, decades-old animal studies with endpoints limited to a small set of immediately observable, physical effects such as skeletal malformation, the Agency has determined that glyphosate exposure poses no more risks to children than to adults. Thus, since 1998, the Agency has assessed glyphosate

risk and set tolerance limits without using the 10X FQPA safety factor that would provide crucial added protection for children.

The use of glyphosate has increased dramatically over the years,[3] and infants and children under five are the populations most highly exposed. Despite the EPA turning a blind eye to glyphosate's effects on children, a growing body of evidence suggests that glyphosate poses a greater risk to children than to adults, including negative effects that can be both profound and permanent. The EPA has failed to identify these effects for the simple reason that it has refused to look for them.

Where the EPA has failed to adequately account for the effects of glyphosate on children, and in the face of children's increasing and long-term exposure to this chemical and mounting evidence of harm, states must

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3. The U.S.G.S. Pesticide National Synthesis Project provides pesticide use maps graphically depicting the increased use of glyphosate. *See* https://water.usgs.gov/nawqa/pnsp/usage/maps/show_map.php?year=1992&map=GLYPHOSATE&hilo=L. Glyphosate was originally patented in 1964 as a chelating agent, and used to strip mineral deposits in commercial hot-water systems. *Toxic Legacy: How the Weedkiller Glyphosate is Destroying our Health and the Environment - One Scientist's Determined Quest to Reveal the Truth*, by Stephanie Seneff, PhD (Chelsea Green Publishing 2021) ("*Toxic Legacy*"), 9; *see also* U.S. Patent No. 316032, awarded Dec. 8, 1964. Use as an herbicide began in the early 1970s, and in the mid 1990s, with the advent of "Roundup Ready" crops genetically-engineered to resist glyphosate, glyphosate's use began to increase dramatically. In more recent years, use has expanded from weedkilling to harvest-aid, and it is now sprayed as a desiccant on many types of crops immediately before harvest. *See Toxic Legacy,* 9-11.

retain their power to require that pesticide warning labels are adequate to protect children's health. There are sound legal arguments as to why the failure-to-warn claim in this case is not preempted;[4] in addition, this Court should uphold the lower court's decision to ensure that states retain the ability to protect their most vulnerable citizens.

4. In addition to respondent's arguments as to why the failure-to-warn claim is not preempted, federal preemption of any state remedy for damages from nonconsensual exposure without inclusion of an adequate federal remedy for those harms would present significant substantive and procedural due process problems. *See Pruneyard Shopping Ctr. v. Robins*, 447 U.S. 74, 93-94 (1980) (Marshall, concurring) ("Quite serious constitutional questions might be raised if a legislature attempted to abolish certain categories of common-law rights in some general way. Indeed, our cases demonstrate that there are limits on governmental authority to abolish 'core' common-law rights, including rights against trespass, at least without a compelling showing of necessity or a provision for a reasonable alternative remedy."); *United Construction Workers v. Laburnum Construction Corp.*, 347 U.S. 656, 663-64 (1954) ("Here Congress has neither provided nor suggested any substitute for the traditional state court procedure for collecting damages for injuries caused by tortious conduct. For us to cut off the injured respondent from this right of recovery will deprive it of its property without recourse or compensation."); *Duke Power Co. v. Carolina Envt'l. Study Group*, 438 U.S. 59, 87-92 (1978) (sustaining federal statute preempting state law remedies and providing *alternative* federal remedy with reasonable liability caps).

## STATEMENT

### I. Children Are Uniquely Vulnerable to Harm from Chemical Exposures, so Extra Caution is Required in Protecting Children from the Risks Posed by Pesticides.

No one would dispute that extra caution is required in protecting children from the harmful effects of chemical exposures, because children are *not* simply little adults; rather, profound biological differences exist between children and adults that leave children even more vulnerable than adults to injuries from chemical exposures. Even the EPA acknowledges "the scientific understanding that children may be at greater risk to environmental pollutants than adults due to differences in behavior and biology, and that the effects of early life exposures may also arise in adulthood or in later generations." *U.S. Environmental Protection Agency Policy on Children's Health*, (March 2026).

In its 1993 publication *Pesticides in the Diets of Infants and Children* ("*NRC Pesticides*"), the National Research Council ("NRC") thoroughly explored special characteristics of children that render them uniquely vulnerable to injuries from toxic exposures, and the corresponding need for extra caution in assessing and managing the risks created by such exposures. More recently, in its 2006 *Principles for Evaluating Health Risks in Children Associated with Exposure to Chemicals* ("*WHO Principles*"), the World Health Organization ("WHO") discussed both the basis for children's vulnerability and recommendations for assessing and managing risks created by chemical exposures.

As explained by the WHO:

> Children have different susceptibilities during different life stages owing to their dynamic growth and developmental processes as well as physiological, metabolic, and behavioural differences. From conception through adolescence, rapid growth and developmental processes occur that can be disrupted by exposures to environmental chemicals. These include anatomical, physiological, metabolic, functional, toxicokinetic, and toxicodynamic processes. Exposure pathways and exposure patterns may also be different in different stages of childhood. Exposure can occur in utero through transplacental transfer of environmental agents from mother to fetus or in nursing infants via breast milk. Children consume more food and beverages per kilogram of body weight than do adults, and their dietary patterns are different and often less variable during different developmental stages. They have a higher inhalation rate and a higher body surface area to body weight ratio, which may lead to increased exposures. Children's normal behaviours, such as crawling on the ground and putting their hands in their mouths, can result in exposures not faced by adults. Children's metabolic pathways may differ from those of adults. Children have more years of future life and thus more time to develop chronic diseases that take decades to appear and that may be triggered by early environmental exposures.

> They are often unaware of environmental risks and generally have no voice in decision-making.

*WHO Principles,* 2.

Along with more current studies, *WHO Principles* and *NRC Pesticides* confirm that infants and children are uniquely susceptible to harm from toxic chemical exposures in at least four ways. First, unlike adults, they may be exposed to toxins during critical windows of development during which development of organs and systems may be disrupted, leading to a cascade of harms. *See, e.g.*, *NRC Pesticides*, 3, 24 ("toxicity to a functional system . . . prior to its full development may permanently affect that system, resulting in altered function . . . in the mature animal"); *WHO Principles,* 39-128 (discussing "Developmental State-Specific Susceptibilities and Outcomes"). Second, infants and young children have immature defense systems, rendering them less able to eliminate some toxins and metabolites that can affect development of important systems. *See, e.g.*, *WHO Principles*, 26, 33; *NRC Pesticides*, 78, 87, 90, 95-96. Third, children may receive higher relative doses of toxins per exposure than adults due to lesser body weight, greater relative surface area, and other factors. *See, e.g., NRC Pesticides*, 4; *WHO Principles*, 31-33 and 36-38. Finally, the ill-effects of toxic exposures on children may unfold over time and be difficult to ascertain through short-term observation or testing. *See, e.g.*, *WHO Principles,* 78 (discussing evidence that "neurotoxic insults during development that result in no observable phenotype at birth or during childhood could manifest later in life as earlier onset of neurodegenerative diseases, such as Parkinson disease"); *NRC Pesticides*, 7 (noting possibility of chronic effects

that are expressed only after long latency periods, such as cancer, neurodevelopmental impairment, and immune dysfunction); National Academies of Sciences ("NAS"), *Environmental Neurotoxicology* 4 (1992) (observing that neurotoxic effects of environmental exposures are difficult to ascertain because "[t]he changes are often subtle and subclinical, and months or years can elapse between exposure to a neurotoxicant and the appearance of dysfunction or disease").

The following more recent studies address children's unique vulnerability to toxic insult and differences between children and adults that may explain this vulnerability:

- Ertuğrul Kıykım, *Neurodevelopmental Impact of Pesticides: A Silent Threat*, 60 Turk. Arch. Pediatrics 114 (2025) (discussing evidence that chronic low dose exposure to organophosphate pesticides interferes with various biochemical pathways, causing impaired neurodevelopment and cognitive deficits);
- Ersen Karakiliç et al., *Endocrine Disruptors and Children's Health*, 55 Turk. J. Med. Sci. 1671 (2025) (reviewing risks posed by endocrine disruptors and factors making early-life exposures more harmful to children, including immature blood–brain barrier and detoxification enzymes, higher intake of air, food, and water per kg, thinner skin, and higher surface-to-volume ratio);[5]

5. Although EPA holds that glyphosate is not an endocrine disruptor (*see* EPA, *Glyphosate: Response to Comments on the Human Health Draft Risk Assessment*, Decision No. 542736 (Apr.

- Giada Di Pietro et al., *Endocrine Disruptor Chemicals and Children's Health*, 24 Int'l J. Molecular Sci. 2671 (2023) (discussing differences between adults and children that potentially render health effects from early-life exposure to endocrine-disrupting chemicals more severe and long-lasting than exposure in adulthood);
- Grant McFarland et al., *Acute Exposure and Chronic Retention of Aluminum in Three Vaccine Schedules and Effects of Genetic and Environmental Variation*, 58 J. Trace Elem. Med. Biol. 126444 (2020) (analyzing expected acute exposures and longer-term whole-body accumulation/clearance across three vaccination schedules, indicating, *inter alia*, that children experience greater health risks than adults due to immature detoxification, renal, gut, and neurological systems);
- Joseph M. Braun, *Early-Life Exposure to Endocrine Disrupting Chemicals and Role in Childhood Obesity and Neurodevelopment*, 13 Nature Rev. Endocrinology 161 (2017) (reviewing epidemiological studies of endocrine disrupting chemicals, indicating fetuses and children may have greater vulnerability

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23, 2018), 5-6), some evidence suggests otherwise. *See* Juan P. Munoz et al., *Glyphosate and the Key Characteristics of an Endocrine Disruptor: A Review*, 270 Chemosphere 128619 (2020) (review of research indicating glyphosate exhibits 8 of 10 key characteristics of an endocrine disruptor).

due to hormone-dependent developmental processes occurring only in early life, lower levels of detoxification enzymes, and higher intake of food and water per kilogram, increasing risk of obesity, metabolic disorders, and neurodevelopmental conditions);

- Anthony Samsel & Stephanie Seneff, *Glyphosate, Pathways to Modern Diseases II: Celiac Sprue and Gluten Intolerance*, 6 INTERDISCIP. TOXICOL. 159 (2013) (indicating glyphosate exposure poses greater health risk to children than adults in part due to immature detoxification, renal, gut, and neurological systems, resulting in various biochemical disruptions most consequential during fetal/early childhood development when these systems are forming);
- Maria J. Carroquino et al., *Environmental Toxicology: Children at Risk*, in ENVTL. TOXICOL. 239 (Edward A. Laws ed., Springer 2013) (orig. pub. online 2012) (discussing findings from several major scientific reports that risks to children differ from adults, due to developmental timing of organ formation, underdeveloped metabolism and excretion capabilities, and higher exposure per body weight);
- Carol J. Burns et al., *Pesticide Exposure and Neurodevelopmental Outcomes: Review of the Epidemiologic and Animal Studies*, 16 J. TOXICOL. & ENVTL. HEALTH,

Part B 127 (2013) (synthesizing human and animal studies on pesticide exposure during development; finding developing animals and children can be more sensitive to pesticide neurotoxicity than adults, causing permanent neurological changes);

- Jianghong Liu & Erin Schelar, *Pesticide Exposure and Child Neurodevelopment: Summary and Implications*, 60 WORKPLACE HEALTH & SAF. 235 (2012) (review indicating even low levels of pesticide exposure in pre-natal and early childhood is linked to cognitive deficits, behavioral problems and developmental delays, due in part to vulnerable windows of development and higher exposure relative to body weight);

- James R. Roberts & Catherine J. Karr, *Pesticide Exposure in Children*, 130 PEDIATRICS e1765 (2012) (Council on Envtl. Health) (2013) (comprehensive American Association of Pediatrics technical report reviewing scientific evidence on pesticide exposure in children, finding children are uniquely vulnerable to uptake and adverse effects of pesticides because of developmental, dietary, and physiologic factors);

- María Teresa Muñoz-Quezada et al., *Neurodevelopmental Effects in Children Associated with Exposure to Organophosphate Pesticides: A Systematic Review*, 39 NEUROTOXICOLOGY 158 (2013)

(synthesis of reported evidence over previous decade on neurodevelopmental effects of organophosphate pesticide exposure in children, indicating critical developmental windows make children more susceptible than adults to neurotoxic effects of pesticides);

- Norman R. Saunders et al., *Barrier Mechanisms in the Developing Brain*, 3 FRONT. PHARMACOL. 46 (2012) (scientific analysis and review of literature suggesting developing brains may be more vulnerable to drugs, toxins, and pathological conditions, contributing to cerebral damage and later neurological disorders);

- Peter D. Sly & Felicity Flack, *Susceptibility of Children to Environmental Pollutants*, 1140 ANN. N.Y. ACAD. SCI. 163 (2008) (concluding children more likely to suffer adverse health outcomes from same exposure level, due to exposures during critical developmental windows where toxins can cause permanent structural or functional damage; discussing immature metabolic and detoxification pathways; and higher internal doses for the same environmental concentration);

- Robert Scheuplein et al., *Differential Sensitivity of Children and Adults to Chemical Toxicity: I. Biological Basis*, 35 REGUL. TOXICOL. PHARMACOL. 429 (2002) (recognizing dietary exposure is

compounded by children's immature livers and excretory systems, which may be unable to effectively remove pesticide metabolites);

- Philip J. Landrigan, *Environmental Hazards for Children in USA*, 11 Int'l J. Occupational Med. & Envtl. Health 189 (1998) (analysis of biological sensitivities indicating children are generally more susceptible to environmental toxicants than adults, due in part to rapid organ growth and development).

As the WHO notes, due to the potential for dramatically different effects of chemical exposures on children, "the full spectrum of effects from childhood exposures cannot be predicted from adult data," and adult-based safety data relating to such exposures often cannot be readily applied to the pediatric population. *WHO Principles,* 2. Thus, risk assessment paradigms must "evaluate exposures relevant to children from preconception to adolescence, taking into account the specific susceptibilities at each developmental stage" and "[r]isk assessment approaches for exposures in children must be linked to life stages." *Id*. Along similar lines, the NRC notes it is important to "enhance understanding of developmental toxicity, especially in humans, during critical periods of postnatal development, including infancy and puberty." *NRC Pesticides*, 9.

The NRC adds "[o]f particular importance are tests for neurotoxicity and toxicity to the developing immune and reproductive systems. Extrapolation of toxicity data from adult and adolescent laboratory animals to young humans may be inaccurate." *Id.* The NRC also cautions

that because of differences in maturation rates across species, "cross-species comparisons of potential toxicity for pesticides in the very young animal, although helpful, cannot be used in the same manner that cross-species comparisons are used with adult animals because of differences in developmental patterns." *Id.*, 25.

The *WHO Principles* recommend various child-protective risk-assessment approaches, including the following:

- Conduct prospective cohort studies of pregnant women, infants, and children with longitudinal capture of exposures at critical windows and sensitive health end-points along the continuum of human development.

- Continue to develop and enhance population-based surveillance systems for real-time capture of sentinel health end-points.

- Strengthen exposure monitoring efforts in children during different developmental stages, including assessment of aggregate and cumulative exposures.

- Develop validated, sensitive, and cost-effective biomarkers of exposure, susceptibility, and effects, particularly during early developmental stages.

- Improve characterization of differences in toxicokinetic and toxicodynamic properties of xenobiotics at different developmental stages. Develop databases of stage-

specific physiological and pharmacokinetic parameters in both human and animal studies.

- Study mechanisms of action during different developmental stages by which exposures may cause adverse outcomes.

- Develop endpoints that can be used to assess organ system functions in both humans and animal species and to identify analogous periods of development across species.

- Improve characterization of windows of susceptibility of different organ systems in relation to structural and functional endpoints.

- Develop and validate biological models and animal testing guidelines that can address health outcomes at different developmental stages.

*WHO Principles*, 4-6.

The NRC offers this precautionary principle as an overall guide: "***In the absence of data to the contrary, there should be a presumption of greater toxicity to infants and children***." *NRC Pesticides*, 9 (emphasis added).

## II. Because of Children's Vulnerability to Harm from Chemical Exposures, the EPA is Required to Use a Presumptive 10X Safety Factor to Protect Children from the Risks Posed by Pesticides.

The EPA is required to set tolerance limits for pesticide chemical residue on food, including raw agricultural commodities and processed foods. *See* 21 U.S.C. § 346a(a). Residue tolerance limits are "*the single most important tool by which the U.S. Government regulates pesticide residues in food.*" *NRC Pesticides*, 18 (emphasis in original).

The EPA is required to set tolerance limits such that "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)(i) and (ii). Additionally, the limits must ensure "a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residue." *See* 21 U.S.C. § 346a(b)(2)(C)(ii).

In 1996, through the Food Quality Protection Act, Congress wrote into federal law the recognition of children's unique vulnerabilities and the need to specially protect them from harmful pesticide exposures. *See* FQPA, Pub. L. No. 104-170, 110 Stat. 1489 (Aug. 3, 1996). The FQPA directs the EPA to use "an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure . . . 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).

In other words, the EPA is required to assume that the risk to children from the use of a particular pesticide is ten times greater than for adults when assessing risks and setting residue tolerance limits for pesticide chemicals like glyphosate. The EPA may "use a different margin of safety for the pesticide chemical residue *only if, on the basis of reliable data,* such margin will be safe for infants and children." *Id.* (emphasis added).

## III. Since 1998, the EPA has Declined to Use the 10X Safety Factor to Protect Children from Harmful Levels of Glyphosate Exposure Based on a Handful of Outdated Animal Studies.

The EPA has long recognized that infants and children under five years old are the population most highly exposed to glyphosate, through multiple routes of exposure.[6] *See* U.S. Envtl. Prot. Agency, *Glyphosate Human Health Risk Assessment for Proposed Use on Indian Mulberry and Amended Use on Pea, Dry*, Decision No. 360557 (2006) ("*2006 Risk Assessment*"), 3, 21. However, according to EPA documents, in 1998, the EPA concluded that infants

6. They are exposed not only through what they eat and drink, but also through outdoor play in treated areas and where runoff may accumulate, such as lawns, turf, playgrounds, and pools, and through incidental exposures, including hand-to-mouth and object-to-mouth exposures. *See* EPA, *Children Are at Greater Risks from Pesticide Exposure, https://archive.epa.gov/pesticides/regulating/laws/fqpa/web/html/kidpesticide.html.*

and children are no more vulnerable to glyphosate than adults, and should be afforded no extra protection. Based on this conclusion, the EPA reduced the FQPA children's safety factor for glyphosate from the presumptive 10X to 1X, thereby eliminating the statute's additional tenfold child-protective safety margin. *See id.*, 17-18.

The 1998 decision to eliminate the children's safety factor was based primarily on a handful of rat and rabbit studies where pregnant animals were exposed to varied levels of glyphosate, and the lowest observed adverse events levels ("LOAEL") for fetuses and offspring were compared with the LOAELs for the mothers. From these studies, the Agency concluded, "***There is no evidence of quantitative or qualitative increased susceptibility of the young demonstrated in the prenatal developmental studies in rats and rabbits and pre/post natal reproduction study in rats.***" *Id.* (emphasis added). According to the EPA, the additional bases for eliminating the safety factor were: "2. The toxicology database is complete; 3. A developmental neurotoxicity study is not required; and 4. The dietary (food and drinking water) exposure assessments will not underestimate the potential exposures for infants and children." *Id.*, 17-18, 11-13, 16 (discussing developmental and reproductive toxicity studies available in the toxicity database).

Since 1998, as use of glyphosate has increased, the EPA has maintained its decision to eliminate the 10X FQPA safety factor each time it has added residue tolerance limits for new types of foods and agricultural commodities or increased existing limits, apparently based on the same set of rat and rabbit studies. *See, e.g.*, Glyphosate Pesticide Tolerances Rule, 65 Fed. Reg.

57957, 57961-63 (Sept. 27, 2000) (maintaining FQPA factor at 1X while expanding and restructuring tolerances); Glyphosate Pesticide Tolerances Rule, 69 Fed. Reg. 65081, 65085 (Nov. 10, 2004) (maintaining FQPA factor at 1X while setting new and increased tolerances); Glyphosate Pesticide Tolerances Rule, 71 Fed. Reg. 76180, 76183 (Dec. 20, 2006) (maintaining FQPA factor at 1X while setting new tolerances); Glyphosate Pesticide Tolerances Rule, 73 Fed. Reg. 73586, 73591 (Dec. 3, 2008) (maintaining FQPA factor at 1X while setting new and increased tolerances); Glyphosate Pesticide Tolerances Rule, 78 Fed. Reg. 25396, 25398 (May 1, 2013) (maintaining FQPA factor at 1X while setting new and increased tolerances).

In its 2017 draft glyphosate risk assessment, EPA reiterated the conclusion that "there is no evidence of quantitative or qualitative fetal susceptibility in rats or rabbits following *in utero* exposure in the developmental studies" and maintained its elimination of the FQPA safety factor.[7] U.S. Envtl. Prot. Agency, *Glyphosate: Draft human health risk assessment in support of registration review*, Decision No. 487242, (2017) ("*2017 Risk Assessment*"), 4.

7. The EPA discussed risk in its *Glyphosate Interim Registration Review Registration Decision* of 2020, but in 2022, the human health portion of the Decision was vacated because the Agency did not adequately consider whether glyphosate causes cancer. *See NRDC v. United States EPA*, 38 F.4th 34 (9th Cir. 2022). The Agency subsequently withdrew the 2020 Decision, leaving the 2017 Draft Risk Assessment as the most current non-withrawn glyphosate risk assessment. *See* U.S. EPA Memorandum, Withdrawal of the *Glyphosate Interim Registration Review Decision*, Sept. 21, 2022.

The Agency's 2017 conclusion relied on "two prenatal developmental toxicity studies in rats and rabbits and three reproductive toxicity studies (two studies performed for two generations, 1 study performed for three generations)," which the Agency characterized, collectively, as "Evidence of Sensitivity/Susceptibility in the Developing or Young Animal." *2017 Risk Assessment*, 12-14.

Four of the studies underlying the 2017 conclusion are the same ones the Agency has relied on since eliminating the 10X safety factor in 1998, and of those, three predate the EPA's 1983 adoption of Good Laboratory Practice standards ("GLP"). The most recent study is dated 2007. All of the studies were industry-funded.

As shown in Table 1, the studies underlying the 2017 conclusions regarding glyphosate effects on infants and children considered only immediate, physically observable adverse effects on offspring of maternal exposure to glyphosate.

**Table 1: Studies Referenced in EPA's 2017 Draft Risk Assessment as Evidence that Developing and Young Children are No More Sensitive/Susceptible to Glyphosate's Adverse Effects than Adults Are**[8]

| **Study Date Sponsor EPA MRID #** | **Study Type** | **Study Endpoints: Offspring Effects Considered at LOAEL** |
|---|---|---|
| 1980 (pre-GLP) Monsanto #46362 | Rat: Developmental Toxicity | Fetuses examined after gestational day 20 for visceral soft-tissue and skeletal alterations |
| 1980 (pre-GLP) Monsanto # 46363 | Rabbit: Developmental Toxicity | Fetuses examined after gestational day 28 for visceral and skeletal malformations |
| 1982 (pre-GLP) Monsanto #105995 | Rat: Three Generation Reproduction | Offspring examined for sex distribution, body weights, survival, and gross postmortem findings. |

8. *See* 2017 Risk Assessment, 30-31; *see also* U.S. Envtl. Prot. Agency, Office of Chemical Safety and Pollution Prevention, *EDSP Weight of Evidence Conclusions on the Tier 1 Screening Assays for the List 1 Chemicals* (June 29, 2015), 27-29; 47-48; 50-52.

| | | |
|---|---|---|
| 1990<br>Monsanto<br>#41621501 | Rat:<br>Two Generation<br>Reproduction | Offspring examined for mortality, clinical signs (e.g., soft stool), body and organ weight, food-consumption; reproduction-fertility indices (pre-coital interval, gestation length, litter size, dead pups/litter, pup survival), and selected tissues. |
| 2007<br>Safepharm Labs, Ltd.<br>#48865101 | Rat:<br>Two Generation<br>Reproduction | Offspring organs weighed and tissues examined for gross and microscopic pathology; also considered mortality, clinical signs, body weight, food consumption, reproduction-fertility indices, developmental landmarks and reflexes, ano-genital distance. |

There are many sound bases for critiquing the Agency's decision to eliminate the FQPA safety factor and the animal studies that underlie it.[9] Here, suffice it to say that

9. Indeed, there are many sound bases for challenging the Agency's conclusions about glyphosate safety for human beings and other life forms. For comprehensive discussion of the human and environmental impacts of glyphosate and the ways glyphosate

crucial studies are missing from the EPA's risk assessment for children.

To begin, absent are studies of developmental neurotoxicity, which would assess cognitive, behavioral, and neurobiological effects over time, for different exposure windows. *See generally*, NAS, *Environmental Neurotoxicology*, 18, 54 (1992) ("a chemical cannot be regarded as free of neurotoxicity merely because data on its toxicity are lacking"' and "[t]o maximize detection of toxicity, some toxicity studies encompassing the full life span of experimental animals should be encouraged"). These studies are important; as the EPA recognizes, "The developing nervous system is especially vulnerable to certain chemicals, and exposures may result in altered neural development with consequences that may be quite unlike the chemical's effects in an adult nervous system." *See* NAFTA Technical Working Group on Pesticides *Developmental Neurotoxicity Study (DNT) Guidance Document*, 1 (Dec. 2016); *see also* Phillipe Grandjean & Philip Landrigan, *Neurobehavioural effects of developmental toxicity,* 13 LANCET 330, 334 (2014) (brain damage caused by developmental neurotoxicity is "too often untreatable and frequently permanent").

Also absent are studies of endpoints where children may have unique vulnerabilities, including oxidative stress and other biochemical effects; endocrine, immune system,

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fails to meet FIFRA's safety standard, see *Petition to cancel all registrations of glyphosate herbicide*, filed with the EPA on Dec. 13, 2023 by six farmworker, environmental, and agriculture public interest organizations. For another comprehensive discussion of glyphosate's harmful effects on all life forms and proposed biochemical mechanisms of harm, see *Toxic Legacy*, supra, n. 3.

and microbiome-mediated effects; and adult-onset disease following early-life exposure. Also missing: studies of the long-term or cumulative effects of glyphosate on infants and children and of real-world exposures to glyphosate formulations that include surfactants and adjuvants.

## IV. Studies of the Past 20 Years Strongly Suggest Glyphosate Has Neurodevelopmental and Other Harmful Effects on Children.

The EPA's failure to meaningfully study the effects of glyphosate on the health of infants and children hardly means that health effects do not occur.[10] Indeed, over the last two decades, a growing body of literature points to myriad ill effects of glyphosate on infants and children, from both prenatal and post-natal exposure. The EPA has not acknowledged these effects for the simple reason that it has refused even to consider them. Relevant literature includes the following:

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10. The EPA's dereliction of duty has resulted in an underestimate of the risks posed by glyphosate. However, Monsanto plays a role here too by its "deceptive efforts to defend the safety of its top-selling Roundup herbicide." *See* Carey Gillam, *How Monsanto manipulates journalists and academics*, The Guardian, June 2, 2019; *see also* Warren Cornwall, '*Journal retracts weed killer study backed by Monsanto, citing 'serious ethical concerns,'* Science Advisor, Dec. 5, 2025; *Monsanto Papers*, https://www.wisnerbaum.com/toxic-tort-law/monsanto-roundup-lawsuit/monsanto-papers/ (internal documents obtained in discovery "reveal a systematic, decades-long campaign" by Monsanto to "manipulate scientific research, corrupt regulatory processes, and deceive the public about the safety of its flagship herbicide, Roundup"). Monsanto's malfeasance is beyond the scope of this brief, but should not be ignored.

- Bryan Gonzalez et al., *Scoping Review of the Relationship Between Glyphosate-Based Herbicide Exposures and Autism Spectrum Disorder*, 204 Food Chem. Toxicol. 115621 (2025) (scientific review demonstrates strong relationship between deleterious effects of exposure to glyphosate herbicides and autism spectrum disorder in children, particularly during critical neurodevelopmental windows);

- Débora Dummer Meira et al. , *Laying the Groundwork: Exploring Pesticide Exposure and Genetic Factors in South-Eastern Brazilian Farmers*, 8 Curr. Res. Toxicol. 100215 (2025) (finding link between glyphosate use by agricultural workers in Brazil and high blood pressure, heart disease, miscarriages, and ADHD);

- Haley M. Jenkins et al., *Gestational Glyphosate Exposure and Early Childhood Neurodevelopment in a Puerto Rico Birth Cohort*, 246 Envtl. Res. 118114 (2024) (cohort study measuring glyphosate exposure during pregnancy finds association between prenatal exposure and developmental delays in early childhood);

- Katiuska Marins et al., *Maternal Exposure to Glyphosate Increased the Risk of Adverse Neurodevelopmental Outcomes in Rodent Offspring: A Systematic Review*, 363 Envtl. Pollution 125086 (2024)

(comprehensive examination of behavioral, biochemical, morphological, and genetic alterations resulting from perinatal glyphosate exposure, finding exposure during development can cause oxidative stress, neuroinflammation, mitochondrial dysfunction, and behavioral changes, increasing susceptibility to neurological disorders later in life);

- Samantha K. Bartholomew et al., *Glyphosate Exposure Exacerbates Neuroinflammation and Alzheimer's Disease-Like Pathology Despite a 6-Month Recovery Period in Mice*, 21 J. NEUROINFLAMM. 316 (2024) (finding that despite an extended recovery period, glyphosate exposure elicits long-lasting pathological consequences; mice dosed with glyphosate for 14 days showed glyphosate and major metabolite in brain tissue even after six months, with corresponding increases in pro-inflammatory factor that is elevated in neurodegenerative disorders such as Alzheimer's);

- Stephanie Seneff et al., *Is Autism a PIN1 Deficiency Syndrome? A Proposed Etiological Role for Glyphosate*, 168 J. NEUROCHEM. 2124 (2024) (early-life glyphosate exposure produces permanent programming effects not seen with equivalent adult exposure: prenatal/perinatal exposure reduces melatonin in rat pups, disrupts PIN1 signaling,

impairs autophagy, produces autism-like morphological features and behaviors; causes glutamate neuroexcitotoxicity, and oxidative stress);

- Daiane Cattani et al., *Long-Term Effects of Perinatal Exposure to a Glyphosate-Based Herbicide on Melatonin Levels and Oxidative Brain Damage in Adult Male Rats*, 12 Antioxidants (Basel) 1825 (2023) (pregnant rats exposed to glyphosate, where offspring showed reduction in melatonin, increased oxidative stress and cellular brain damage markers associated with increased risk of neurodegenerative disorders);

- Carmen Costas-Ferreira et al., *Toxic Effects of Glyphosate on the Nervous System: A Systematic Review*, 23 Int'l J. Mol. Sci. 4605 (2022) (analyzing mechanisms of glyphosate neurotoxicity, finding glyphosate may influence neurotransmitter signaling, oxidative stress pathways, neuroinflammatory processes);

- Bożena Bukowska et al., *Glyphosate Disturbs Various Epigenetic Processes In Vitro and In Vivo - A Mini Review*, 851 Sci. Total Environ. 158259 (2022) (finding epigenetic changes caused by glyphosate can persist over time, be passed on to offspring in the next generation, and even in third generation can result in some disorders such as prostate disease or obesity);

- Jacqueline A. Barnett et al., *Is the Use of Glyphosate in Modern Agriculture Resulting in Increased Neuropsychiatric Conditions Through Modulation of the Gut-Brain-Microbiome Axis?*, 9 FRONT. NUTR. 827384 (2022) (perspective review finding glyphosate-resistant microbes in the gut have potential to increase production of pro-inflammatory cytokines and reactive oxygen species, and can affect immune and neurodevelopment across generations through maternal transfer of gut microbiome);

- Muhammad Irfan Masood et al., *Environment Permissible Concentrations of Glyphosate in Drinking Water Can Influence the Fate of Neural Stem Cells from the Subventricular Zone of the Postnatal Mouse*, 270 ENVTL. POLLUT. 116179 (2021) (*in vitro* study discussing glyphosate's mechanisms of neurotoxicity, suggesting glyphosate interferes with maturation of new neurons in the brain);

- Yaoyu Pu et al., *Maternal Glyphosate Exposure Causes Autism-Like Behaviors in Offspring Through Increased Expression of Soluble Epoxide Hydrolase*, 117 PROC. NATL. ACAD. SCI. U.S.A. 11753 (2020) (mouse study in which glyphosate administered in maternal drinking water during pre- and post-natal period associated with autism-like behavioral abnormalities in juvenile

offspring and other biological changes including disruption of gut microbiome);

- Mingjun Cao et al., *Melatonin Rescues the Reproductive Toxicity of Low-Dose Glyphosate-Based Herbicide During Mouse Oocyte Maturation Via the GPER Signaling Pathway*, 70 J. Pineal Res. e12718 (2021) (study regarding toxic effects of low-dose glyphosate-based herbicide exposure on mature mouse oocytes during meiotic maturation revealed that low-dose glyphosate has adverse effect on oocyte maturation and early embryo cleavage, highlighting protective role of melatonin);

- María Florencia Rossetti et al., *Epigenetic Changes Associated With Exposure to Glyphosate-Based Herbicides in Mammals*, 12 Front. Endocrinol. (Lausanne) 671991 (2021) (scientific review summarizing epigenetic changes produced by glyphosate and like chemicals, finds such changes could be heritable leading to disease long after the exposure has ended);

- Corina Lesseur et al., *Maternal Urinary Levels of Glyphosate During Pregnancy and Anogenital Distance in Newborns in a US Multicenter Pregnancy Cohort*, 280 Envtl. Pollut. 117002 (2021) (finding statistically significant correlation between levels of glyphosate in urine of mothers during late pregnancy and anogenital

distance in female offspring; long anogenital distance associated with a greatly increased risk to polycystic ovary syndrome, the most common cause of female infertility);

- Juan P. Munoz et al., *Glyphosate and the Key Characteristics of an Endocrine Disruptor: A Review*, 270 CHEMOSPHERE 128619 (2020) (scientific review indicating glyphosate exhibits 8 of 10 key characteristics of an endocrine disruptor, including disruption of thyroid hormone regulation, suppression of testosterone synthesis, inhibition of developmental enzyme for converting testosterone to estrogen, and enhanced estrogenic signaling in breast cancer cells);

- Ondine S. von Ehrenstein et al., *Prenatal and Infant Exposure to Ambient Pesticides and Autism Spectrum Disorder in Children: Population Based Case-Control Study*, 364 BMJ 1962 (2019) (finding women living within 1.2 miles of pesticide spray areas 30 percent more likely to have children with severe autism, with highest odds ratio for children exposed to glyphosate in first year of life);

- Deepika Kubsad et al., *Assessment of Glyphosate Induced Epigenetic Transgenerational Inheritance of Pathologies and Sperm Epimutations: Generational Toxicology,* 9 SCI. REP. 6372 (2019) (study exposing pregnant rats to low

doses of glyphosate between eighth and fourteenth days of gestation, finds dramatic increase in in several health problems, most linked to reproduction, in second and third generation);

- Jakeline Liara Teleken et al., *Glyphosate-Based Herbicide Exposure during Pregnancy and Lactation Malprograms the Male Reproductive Morphfunction in F1 Offspring*, 16 J. of Dev. Orig. Health & Dis. 1 (2019) (male offspring of mice exposed to glyphosate during gestation and lactation suffered serious health effects, including delayed testicular descent, reduced sperm counts, structural alterations within testes);

- Xin Ren et al., *Effects of Chronic Glyphosate Exposure to Pregnant Mice on Hepatic Lipid Metabolism in Offspring*, 254 Envtl. Pollut. 112906 (2019) (offspring of pregnant mice exposed to glyphosate showed fatty liver and perturbations of various blood parameters related to fats, cholesterol and standard liver enzymes, demonstrating that chronic prenatal exposure to glyphosate can result in lipid metabolism disruption in offspring);

- Thu Ha Pham et al., *Perinatal Exposure to Glyphosate and a Glyphosate-Based Herbicide Affect Spermatogenesis in Mice,* 169 Toxicol. Sci. 260 (2019) (male rats exposed to glyphosate products in

utero had low testosterone, deformed testes, decreased sperm count, and fertility problems);

- Fabiana Manservisi et al, *The Ramazzini Institute 13-Week Pilot Study Glyphosate-Based Herbicides Administered at Human-Equivalent Dose to Sprague Dawley Rats: Effects on Development and Endocrine System*, 18 Envtl. Health 15 (2019) (offspring of pregnant rat dams exposed to glyphosate-based herbicides throughout gestation and weaning, at doses considered safe for humans, induced endocrine effects and altered reproductive developmental parameters, including androgen-like effects and increased testosterone in females);

- Yassine Ait Bali et al., *Learning and Memory Impairments Associated to Acetylcholinesterase Inhibition and Oxidative Stress Following Glyphosate Based-Herbicide Exposure in Mice*, 415 Toxicol. 18 (2019) (mouse study finding subchronic and chronic exposure to glyphosate-based herbicides led to numerous cognitive abnormalities and various potentially explanatory biochemical changes);

- Quixing Mao et al., *The Ramazzini Institute 13-Week Pilot Study on Glyphosate and Roundup Administered at Human-Equivalent Dose to Sprague Dawley Rats:*

*Effects on the Microbiome*, 17 ENVTL. HEALTH 50 (2018) (finding exposure to glyphosate-based herbicides in utero and for six days after birth at doses considered safe for humans significantly altered diversity of gut microbiota in rat pups, while mothers were unaffected; describing possibility of significant downstream health effects related to microbiome disruption);

- María-Aránzazu Martínez et al., *Neurotransmitter Changes in Rat Brain Regions Following Glyphosate Exposure*, 161 ENVTL. RES. 212 (2018) (finding oral glyphosate exposure in rats significantly altered central nervous system (CNS) monoaminergic neurotransmitters in a brain regional- and dose-related manner, which could potentially affect mood, memory, and motor control);

- Cristina Eugenia Gallegos et al., *Perinatal Glyphosate-Based Herbicide Exposure in Rats Alters Brain Antioxidant Status, Glutamate and Acetylcholine Metabolism and Affects Recognition Memory*, 34 NEUROTOX. RES. 363 (2018) (concluding exposure to glyphosate-based herbicide during early stages of rat development affects brain oxidative stress markers and activity of enzymes involved in biochemical systems, which contribute to neurotransmission disruption, and cognitive deficits in offspring);

- Maria M. Milesi et al., *Perinatal Exposure to a Glyphosate-Based Herbicide Impairs Female Reproductive Outcomes and Induces Second-Generation Adverse Effects in Wistar Rats*, 92 ARCH.TOXICOL. 2629 (2018) (concluding perinatal exposure to low doses of glyphosate-based herbicide in food impaired female reproductive performance and induced fetal growth retardation and structural congenital anomalies in offspring);

- Daiane Cattani, et al., *Developmental Exposure to Glyphosate-Based Herbicide and Depressive-Like Behavior in Adult Offspring: Implication of Glutamate Excitotoxicity and Oxidative Stress*, 387 TOXICOL. 67 (2017) (investigation regarding effects of subchronic exposure to GBH on some neurochemical and behavioral parameters in immature and adult rat offspring exposed to glyphosate in utero and through drinking water, finds various biochemical disruptions and depressive behaviors);

- Gilles-Eric Séralini et al., *Republished*[11] *study: Long-term Toxicity of a Roundup*

11. The study was retracted after its original publication in a different journal in 2012. *See Retraction notice to "Long term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize*," 63 FOOD & CHEM. TOX. 244 (Jan. 2014). Emails later revealed that prior to the retraction, Monsanto "engaged with a network of scientists and other commentators to spread the message

*Herbicide and a Roundup-tolerant Genetically Modified Maize*, 26 Envtl. Sci. Eur. 14 (2014) (First full-lifespan (2 year) rat study finding long-term exposure to glyphosate-tolerant GM maize and low-dose Roundup in drinking water associated with increased cancer risk, liver and kidney toxicity, and endocrine/metabolic disturbances);

- Daiane Cattani et al., *Mechanisms Underlying the Neurotoxicity Induced by Glyphosate-Based Herbicide in Immature Rat Hippocampus: Involvement of Glutamate Excitotoxicity*, 320 Toxicol. 34 (2014) (investigating whether Roundup leads to neurotoxicity in hippocampus of immature rats following acute and chronic (pregnancy and lactation) exposure, finding Roundup might lead to excessive extracellular glutamate levels and consequently to glutamate excitotoxicity and oxidative stress in rat hippocampus);

- Anthony Samsel & Stephanie Seneff, *Glyphosate's Suppression of Cytochrome P450 Enzymes and Amino Acid Biosynthesis by the Gut Microbiome: Pathways to Modern Diseases* 15 Entropy 1416 (2013)

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that the Seralini paper was bad science and should be retracted." Retraction Watch, *Unearthed emails: Monsanto connected to campaign to retract GMO paper*, https://retractionwatch.com/2017/08/10/unearthed-docs-monsanto-connected-campaign-retract-gmo-paper/.

(linking glyphosate to gut dysbiosis and a cascade of diseases through disruption of critical enzyme, which manifests over time as inflammation, damaging cellular systems throughout body);

- Michael N. Antoniou et al., *Teratogenic Effects of Glyphosate-Based Herbicides: Divergence of Regulatory Decisions from Scientific Evidence*, 4 J. of ENVTL. & ANAL. TOXICOL. 006 (2012) (synthesizing evidence from existing studies showing glyphosate-based herbicides can interfere with crucial developmental pathways, causing birth defects; arguing that European regulatory agencies favor non-public, industry-sponsored data over independent evidence that shows developmental toxicity);

- Eliane Dallegrave et al., *The Teratogenic Potential of the Herbicide Glyphosate–Roundup in Wistar Rats*, 142 TOXICOL. LETT. 45 (2003) (pups exposed to varying doses of glyphosate in utero from days 6-15 of gestation showed congenital deformities at a rate inversely proportional to dosing level; nearly all pups had retarded skeletal development and edema related to organ failure).

If these studies suggest anything with respect to the EPA's glyphosate decisions, it is that the EPA is failing children, and should have increased the 10X safety factor, rather than eliminating it.

## ARGUMENT

### This Court Should Allow States to Protect Children from Exposure to Chemicals, Including Glyphosate, Whose Harmful Effects on Children the EPA has Failed to Recognize.

In *Bates v. Dow Agrosciences L.L.C.*, this Court recognized "the importance of providing an incentive to manufacturers to use the utmost care in the business of distributing inherently dangerous items." 544 U.S. 431, 449-50 (2005). Moreover, the Court noted that in the context of FIFRA, "tort suits can serve as a catalyst" for ensuring and improving product safety:

> By encouraging plaintiffs to bring suit for injuries not previously recognized as traceable to pesticides such as [the pesticide there at issue], a state tort action of the kind under review may aid in the exposure of new dangers associated with pesticides. Successful actions of this sort may lead manufacturers to petition EPA to allow more detailed labelling of their products; alternatively, EPA itself may decide that revised labels are required in light of the new information that has been brought to its attention through common law suits. In addition, the specter of damage actions may provide manufacturers with added dynamic incentives to continue to keep abreast of all possible injuries stemming from use of their product so as to forestall such actions through product improvement.

*Id.* at 431 (brackets in Court's opinion; internal quotation marks and citation omitted).

The need for safety incentives is nowhere greater than in the realm of child welfare, and the rationale from *Bates v. Dow Agrosciences* is fully applicable here. Especially because new information about the ill effects of glyphosate on children continues to emerge on a regular basis, state-law failure-to-warn actions are essential catalysts for safety. States must be allowed to protect children from the risks of glyphosate by requiring warnings fully disclosing those risks.

## CONCLUSION

Over the last thirty years, two trends in the federal government have worked synergistically to undermine protection of children's health: (1) federal preemption of state laws that would otherwise incentivize safety and protect children against potentially harmful products to which they are routinely exposed (often without informed consent); and (2) federal regulators' failure to ensure that these products are safe for children, a failure due in part to the regulators' use of outdated studies that fail to account for children's unique vulnerability or their current level of real-world exposure.

For example, the Telecommunications Act of 1996 is understood to bar states from regulating in connection with health effects of radiofrequency (RF) radiation as long as wireless facilities and devices comply with the Federal Communications Commission's RF radiation emission limits. *See, e.g.*, *Cohen v. Apple Inc.*, 46 F.4th 1012, 1016 (9th Cir. 2022). In 1996, the FCC adopted RF

exposure guidelines that were not designed to protect against what are now well-recognized non-thermal health effects of RF radiation. Since 1996, the deployment of wireless facilities and technology and the population's exposure to RF radiation have skyrocketed, and in 2021, the U.S. Court of Appeals for the District of Columbia ordered the FCC to explain why it ignored compelling evidence of biological harms from exposure to RF radiation when it declined an opportunity to update the 1996 guidelines–in particular, with respect to children. *See Envtl. Health Trust v. FCC*, 9 F. 4th 893 (D.C. Cir. 2021).[12] But despite children's ever-increasing exposure to RF radiation and mounting evidence of harm, the FCC has continued to leave the guidelines in place. Thus, federal preemption coupled with regulatory failure leaves children vulnerable to harms from mounting real-world exposure to wireless radiation.

Vaccines present a structurally similar problem. The 1986 National Childhood Vaccine Injury Act preempts state laws that would otherwise allow lawsuits on behalf of children injured by routine childhood vaccines, and provides essentially blanket liability protection to vaccine manufacturers and those who administer the vaccines. This scheme leaves a regulatory vacuum: vaccine manufacturers have little incentive to monitor and improve the safety of their products. *See Bruesewitz v. Wyeth LLC*, 562 U.S. 223, 250 (2010) (Sotomayor, J., dissenting). Since 1986, the number of vaccines recommended and mandated for routine childhood use has skyrocketed. However, the federal agencies charged with ensuring vaccine safety have failed to study (among other things)

12. CHD was one of several petitioners in *EHT v. FCC*.

the full safety profiles of individual vaccines, exposure to multiple vaccines at once, and cumulative exposure to vaccines over time. Thus, just as with wireless radiation, federal preemption coupled with regulatory failure leaves children vulnerable to harms from mounting real-world exposure to vaccines.

While a similar situation is threatened in the context of pesticides, this case presents an opportunity to interrupt these troubling trends.For the sound legal reasons articulated in the Respondent's brief, along with the policy considerations articulated here, this Court should uphold the decision below.

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