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Glyphosate: North America's toxic food crops and the enduring legacy of disease

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Glyphosate-based herbicides are the most widely used agricultural chemicals globally and have been widely regarded as safe due to their low acute toxicity. However, this safety paradigm has been shaped by regulatory frameworks that prioritize short-term endpoints and may overlook slower, systemic pathways to disease. In this perspective, we revisit glyphosate through a public health lens, drawing parallels with the historical trajectory of dichlorodiphenyltrichloroethane (DDT), where early assurances of safety preceded recognition of long-term ecological and human health harms. We highlight emerging evidence linking glyphosate exposure to metabolic, inflammatory, and neurological conditions, alongside ecological disruptions that may indirectly affect human health by altering soil systems, biodiversity, and food quality. We examine how safety assumptions have been challenged by reassessment of early toxicological data, raising concerns about biases in regulatory evidence selection and the exclusion of independent academic studies. Mechanistic insights from microbiome science, epigenetics, and non-monotonic dose response relationships suggest that glyphosate's effects may manifest across generations, pathways not captured in conventional risk assessment. Glyphosate exposure is not evenly distributed where populations with close connections to land-based food systems, including Indigenous communities, face disproportionate risks, highlighting an underexamined environmental injustice. We argue that current regulatory approaches are insufficient to address the chronic disease drivers embedded in modern food systems. A systems-based framework is needed, one that incorporates microbiome and epigenetic endpoints, prioritizes long-term studies, and evaluates cumulative effects. Reframing glyphosate within this broader context is essential to inform policy, protect population health, and prevent the repetition of historical failures in environmental health governance.

KEYWORDS
environmental health, glyphosate-based herbicides, pseudo-persistent pollutants, dietary exposome, toxicology, gut microbiome, chronic disease, regulatory risk policy

Introduction

Joni Mitchell's "*Big Yellow Taxi*" (1970) captured a turning point in environmental consciousness, released just two years before the U.S. banned DDT for agricultural use. Initially hailed as a miracle of public health and agriculture, DDT's legacy soon darkened: a chemical that seemed harmless in the moment left a toxic inheritance through persistence and accumulation. More than half a century later, its legacy remains embedded in ecosystems worldwide (1).

As DDT's era ended, glyphosate-based herbicides (GBHs) quietly emerged onto the agricultural stage. Marketed as biodegradable and even "safe enough to drink," glyphosate was embraced as a symbol of progress rather than caution (2, 3). Glyphosate was not DDT's toxicological equivalent, but rather its historical echo; another compound celebrated as safe based largely on the absence of acute toxicity, before broader ecological and chronic health concerns emerged. Consequently, GBHs have been woven into everyday practice, and unleashed with little restraint. And, as with DDT, the absence of acute toxicity lulled regulators, industry, and the public into mistaking short-term safety for long-term harmlessness. Praised for its efficiency and versatility in weed control, glyphosate's use skyrocketed. Amplified by the development of glyphosate-resistant crops, its use exploded, surpassing 100 million kilograms annually in the U.S. by 2010 (Figure 1A) (4). To put that number into perspective, at its peak, DDT use in America reached "only" 30 million kilograms annually (5). Yet, even at these lower volumes, DDT's persistence and bioaccumulation proved catastrophic.

Glyphosate was hailed for its apparent environmental safety as it was said to break down into glycine and phosphate without accumulation. That promise has not held. Research shows that glyphosate's persistence in the environment ranges from a few days to several months, depending on environmental and soil conditions. Factors such as microbial composition and activity, soil compaction, and soil organic matter all influence the rates of retention and degradation in soil (6, 7). In addition to its use in agriculture, glyphosate is used extensively in forestry. Here, glyphosate is applied aerially to reduce the growth of broadleaf plants, shrubs, and grasses that compete with commercially valuable conifer trees. Recent studies have shown that in forest soils, glyphosate can accumulate, remain biologically active, and be recovered from plant material for durations ranging from 1 year to more than a decade (6, 8–9). Even more concerning, its breakdown product, aminomethylphosphonic acid (AMPA), persists in nearly 93% of croplands and may pose an even greater long-term risk, demanding urgent study of ecotoxicity and biodegradation (10). Concerningly, decades of research have demonstrated glyphosate bioaccumulates in mammals, fish and birds with high levels detected in the liver, intestines, kidney and can even be detected in the brain (11).

Notably, glyphosate's widespread adoption coincided with rising rates of chronic inflammatory conditions in regions adopting Western industrialized agricultural practices (Figure 1B) (12). While many factors have changed in this period, glyphosate exposure adds a concerning and underexamined layer to this broader public health puzzle, one that conventional toxicology was not equipped to detect at the time.

Unveiling the hidden health impacts

Glyphosate inhibits the Shikimate pathway, which plants and bacteria use to synthesize aromatic amino acids. Since humans lack the Shikimate pathway and all its enzymes, glyphosate was thought to pose no risk to human health. Monsanto promoted Roundup® as exceptionally safe, cultivating a culture of complacency that encouraged careless handling and repeated exposure. Glyphosate was often sprayed without gloves or protective clothing, and applicators walked through treated fields moments later. The absence of acute health effects was precisely what made glyphosate so dangerous. As with DDT, a chemical once marketed as safe, such assurances fostered repeated, high-level exposures that only decades later revealed links to cancer, including non-Hodgkin's lymphoma, DNA damage, and reproductive harm (13, 14). As with DDT, this cavalier attitude, fueled by misinformation including safety data that has since been retracted, led to the failure to anticipate long-term consequences and contributed to serious health outcomes.

Today, the hidden human costs are coming into view. In the U.S., cross-sectional and longitudinal studies link environmental glyphosate exposure to type 2 diabetes and metabolic syndrome in both adults (15) and children (16). Occupational exposure has been associated with coronary artery disease (17), cognitive decline (18), and neurological symptoms resembling Parkinson's disease (19–21). Rodent studies have shown that oral exposure to glyphosate (in the form of Roundup® Maxload) exacerbated dopaminergic neurotoxicity in an MPTP mouse model of Parkinson's (22). In parts of Europe, including France and Germany, national programs exist recognizing Parkinson's as an occupational disease for agricultural workers, providing lifelong compensation (23, 24). Despite growing health concerns, the European Union does not recognize the link between pesticide exposure and Parkinson's, and the European Commission reauthorized glyphosate for another 10 years (effective until 2033) (25).

These direct health effects represent only part of glyphosate's potential impact. More troubling are the emerging signs that, like DDT, glyphosate's influence extends beyond the exposed individual to affect subsequent generations.

Beyond direct toxicity: the generational effects of glyphosate

The parallels with DDT extend beyond immediate toxicity to what is passed between generations. Animal studies reveal that maternal glyphosate exposure can lead to behavioural changes in offspring, including increased depression, anxiety-like behaviours, and social deficits (26). In murine models of asthma, maternal exposure resulted in immune impairments in first-generation offspring (27). Our own recent research shows that prenatal glyphosate exposure, even at doses of 0.01 mg/kg/day, well below EPA safety limits, disrupts host-microbe-metabolite relationships across multiple generations. These effects included reduced goblet cells, impaired glucose tolerance, dysfunctional metabolism, including reduced incretin (GLP-1) signalling, cytokine dysregulation, and behavioural deficits linked to the gut–brain axis, suggesting microbiome-mediated effects with potential for transgenerational compounding effects (28).

Epigenetic studies further strengthen this concern. Glyphosate exposure has been associated with differential DNA methylation patterns in rats across the F1, F2, and F3 generations, leading to a variety of disease phenotypes, including obesity, prostate, kidney, and ovarian disorders (29). In addition, perinatal exposure has been shown to alter the expression of more than 600 circular RNAs in the murine hippocampus, pointing to potential impacts on neurodevelopment (30).

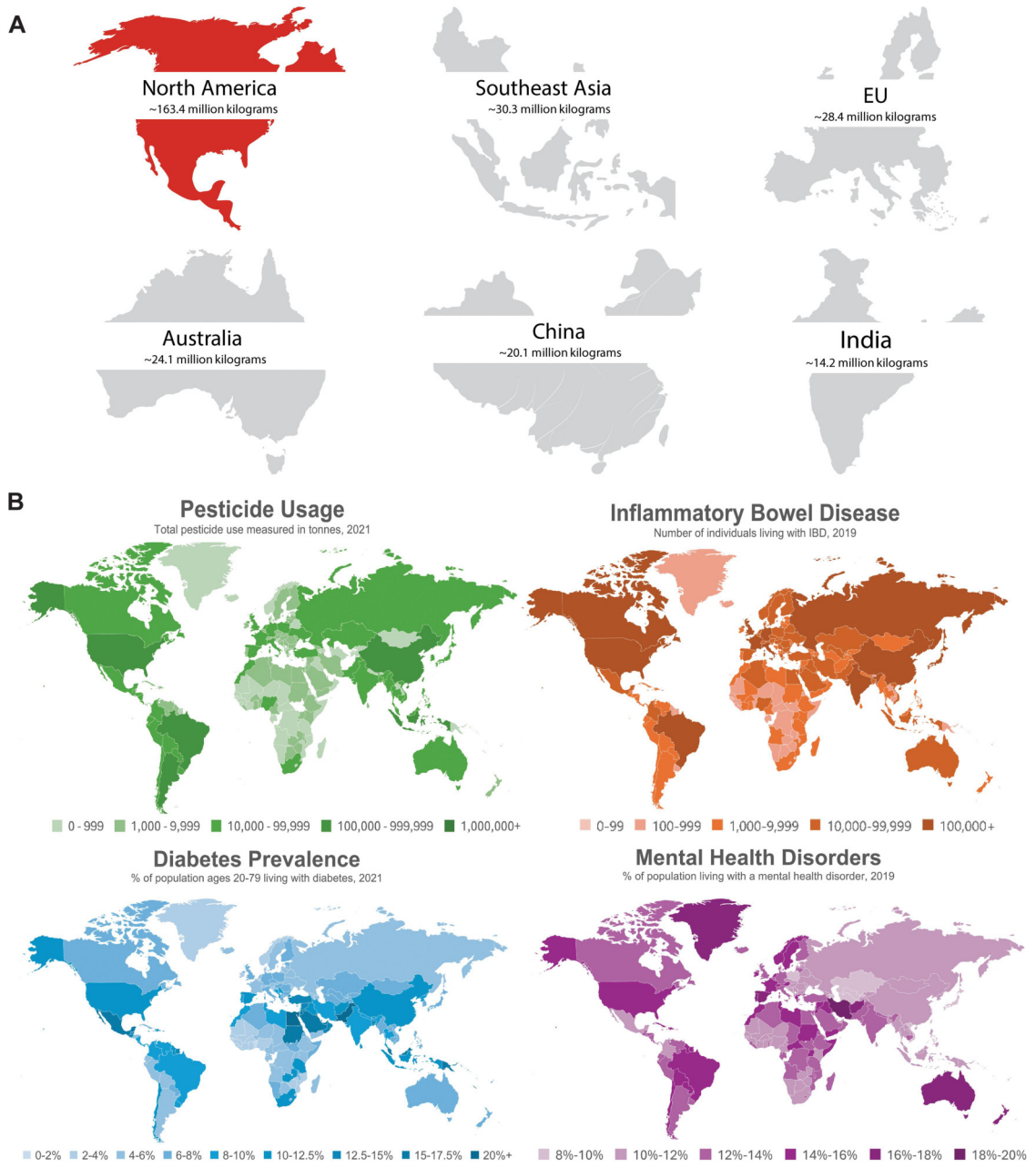

FIGURE 1
The global rise in pesticide usage is paralleled by an increase in chronic physical and mental health conditions. **(A)** North America is the highest consumer of glyphosate worldwide. **(B)** Regions adopting Western-industrialized pesticide practices exhibit corresponding increase in chronic diseases including IBD, diabetes and mental health disorders underscoring the potential link between widespread pesticide use and the proliferation of chronic health conditions, globally. Data used to generate this image were reported https://ourworldindata.org/grapher/pesticide-use-tonnes.

Given that glyphosate targets a pathway not found in mammals, how could such effects persist across generations? Animal and mechanistic studies point to two complementary routes. Disruption of the gut microbiome, the most plausible molecular initiating event, given glyphosate's documented inhibition of the Shikimate pathway enzyme EPSPS in microbial taxa, may propagate downstream effects through altered metabolite production, immune signalling, and oxidative stress. These perturbations, in turn, may induce epigenetic reprogramming, including differential DNA methylation and altered circular RNA expression, providing a heritable substrate through which disease phenotypes persist across lineages. The gut microbiome, now recognized as central to immune, metabolic, and neurological health, offers a mechanism for heritable vulnerability, given its transmission during birth and lactation. Glyphosate exposure can disrupt the gut microbiome, which plays a central role in gut-related conditions such as inflammatory bowel disease, metabolic regulation, and mental health via the gut–brain axis. Such disruptions may therefore contribute to a broad spectrum of chronic conditions. Although still speculative, we hypothesize that pesticide exposure, including GBHs, may be linked to the sharp rise in chronic diseases observed today (Figure 1). Correlations

between Roundup® use and the prevalence of inflammatory bowel disease, metabolic disorders, and mental health conditions in the U.S. have been reported (31). These insights underscore a critical blind spot in toxicology: glyphosate's risks stem not from acute toxicity, but from slow, insidious effects that unfold through the microbiome and epigenome across years and generations.

Leave us the birds and the bees (*please*): ecological consequences across the food web

Glyphosate's pervasive use across agriculture, forestry and residential landscaping reverberates far beyond weed control, with consequences that ripple through ecosystems and ultimately circle back to human health. At the base of the soil food web, glyphosate alters soil microbiota including bacterial and fungal community structures, disrupting nutrient cycling and leaving plants more vulnerable to pathogens. Exposure to glyphosate-based herbicides like Roundup® disrupts two keystone soil organisms: earthworms and arbuscular mycorrhizal fungi, both essential for soil fertility and crop productivity (32).

Pollinators, too, are not spared. Changes in soil nutrient availability reduce plant quality, indirectly limiting nectar and pollen resources. Directly, glyphosate alters the bee microbiome, making them more susceptible to opportunistic infections (33). Developing larvae are also susceptible to glyphosate-induced dysbiosis, resulting in slow larval growth, delayed moulting, and developmental teratogenic effects and elevated post-emergence mortality (34). Beyond microbiome disruption, glyphosate exposure influences multiple neurological processes in bees, including sleep (35), locomotion, colour discrimination and foraging behaviour (36). Interestingly, young nurse bees can learn to avoid glyphosate-contaminated pollen, reducing consumption by up to 23% following malaise-associated experiences with contaminated food (37). However, this avoidance response is not without cost. Reduced pollen intake compromises nutrition, and in agricultural landscapes where nearly all available resources are potentially contaminated, the adaptive value of avoidance is limited. These disruptions cascade up the food chain, where reduced arthropod populations mean less prey for higher trophic levels, while residues from exposed birds are passed into eggs, impairing embryonic development and survival (38).

These ecological disruptions disproportionately affect communities with deep connections to the land, creating overlapping environmental and health injustices. In Canada, Indigenous communities are disproportionately exposed to forestry spraying, which exceeds 10,000 hectares annually in British Columbia. Such practices erode biodiversity and threaten cultural practices like wild food harvesting, while rates of chronic disease, including diabetes, remain three to five times higher in First Nations communities compared to non-Indigenous populations (39). These disparities underscore an urgent need to examine whether forestry herbicide use compounds existing health inequities.

In New Brunswick, reports of unexplained neurological symptoms in coastal communities have further raised concerns. Neurologist Alier Marrero, who has publicly suggested that glyphosate runoff could be a potential driver, points to an underexplored mechanism (40). Glyphosate acts as a phosphorus source for cyanobacteria, which can produce the neurotoxin β-Methylamino-L-alanine (BMAA). BMAA misincorporates into proteins, leading to misfolding and aggregation, a hallmark of neurodegenerative diseases such as Parkinson's and Alzheimer's (41). While this mechanism remains speculative and causality unproven, the possibility highlights the need for vigilance in communities reliant on aquatic ecosystems.

As with the broader ecosystem, glyphosate's consequences for humans appear twofold: direct exposure influences health outcomes, while indirect effects, through reduced biodiversity, disrupted soil fertility, and altered nutritional quality of food, may exacerbate the burden of chronic diseases already weighing heavily on society (Figure 2).

The battle over glyphosate regulation

Despite mounting evidence of harm, efforts to ban or restrict glyphosate repeatedly fail. Several countries have announced prohibitions, only to reverse course under political or agricultural pressure. In Canada, a proposal to raise glyphosate residue limits on imported foods sparked such public backlash that regulators were forced to delay the decision indefinitely (42).

Why is it so difficult to acknowledge that glyphosate might follow the troubling legacy of previously used chemicals like DDT? Part of the answer lies in the perception of inconsistency across studies. Depending on the metric, whether soil health, biodiversity, cancer risk, or neurological disease is examined, findings appear mixed, fueling uncertainty and mistrust. Yet this variability often reflects not the absence of signal, but differences in methodology and, critically, in which studies regulators choose to privilege.

As Novotny has noted, regulatory risk assessments have historically relied heavily on industry-sponsored studies conducted under Good Laboratory Practice (GLP) standards, which have overwhelmingly reported no harm. In contrast, many independent, peer-reviewed academic studies, often excluded from risk assessments for failing to meet GLP protocols, find evidence of genotoxicity, microbiome disruption, and links to chronic disease (43). The result is a systematic skew: regulators conclude glyphosate is safe, while academic literature points to the opposite. This imbalance is compounded by documented corporate influence over the glyphosate evidence base, shaping both the research landscape and public discourse.

The scientific complexity of glyphosate adds another layer of challenge. Commercial GBHs vary widely: Roundup®, Glyphogan®, Accord®, and others contain different co-formulants that alter toxicity. Moreover, research has documented that glyphosate's dose-response relationship is non-monotonic, similar to PFAS (per and polyfluoroalkyl substances), meaning effects can appear at low concentrations, vanish at intermediate doses, and re-emerge at higher exposures. Such patterns complicate conventional toxicological interpretation and regulatory testing paradigms, often designed around assumptions that toxicity increases linearly with dose. In fact, negative studies may simply have evaluated the "wrong" dose to see an effect.

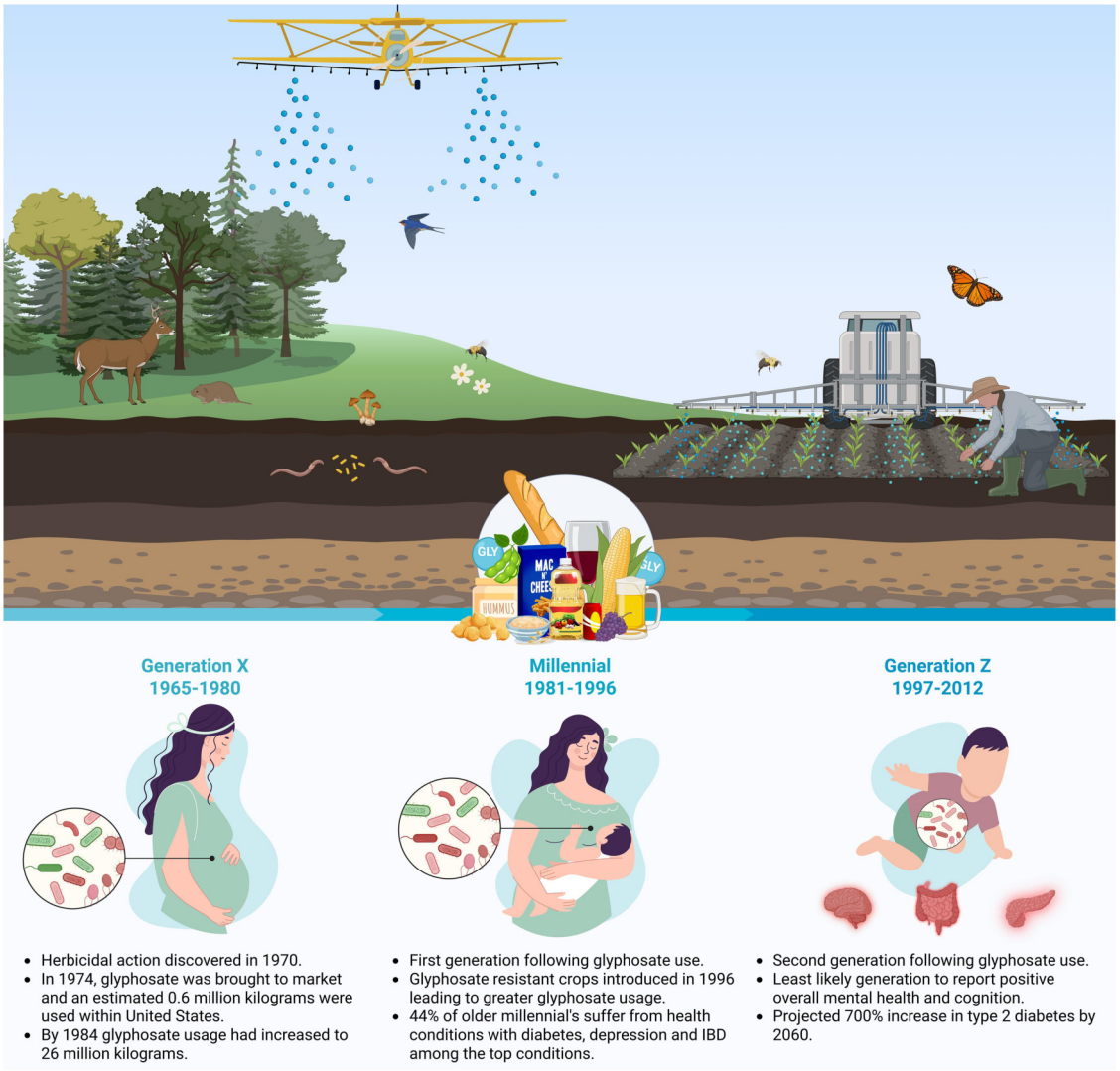

FIGURE 2
Hypothesis of the potential impact of glyphosate on the food web and generational health. The extensive use of glyphosate in forestry and agriculture has widespread effects across the food web. The dispersal of glyphosate through ecosystems compromises forest animals, soil microbiomes, and key pollinators such as bees, butterflies, and birds. This disruption extends to affect primary, secondary, and tertiary consumers, including humans. Introduced during Generation X, glyphosate's environmental presence coincides with an escalation in chronic diseases such as diabetes, depression, and inflammatory bowel disease IBD observed in Millennials and Generation Z. This trend underscores the potential transgenerational consequences of glyphosate exposure, highlighting its dual threat: ecological imbalance and the persistent decline of human health across generations.

Meanwhile, glyphosate's efficiency and cost-effectiveness make it difficult to dislodge. Farmers, foresters, and land managers view GBHs as indispensable tools, especially given the absence of comparably effective alternatives. Yet the tragedy, echoing DDT, is not that glyphosate has no safe uses, but that its promotion as "harmless" fostered cavalier practices, blanket spraying, lack of protective gear, and repeated exposures that could have been avoided. Used sparingly and with strict precautions, the risk-benefit trade-off might look very different.

Some regions have begun experimenting with more cautious approaches. Quebec, for example, abandoned glyphosate in forestry in 2001, turning instead to manual vegetation management and selective planting (44). Belgium, France, the Netherlands, and Germany have restricted GBH applications in public areas. Vietnam is the only country to have imposed a full ban (2021), though data on its agricultural or health impacts of this ban remain absent. Elsewhere, prohibitions have been reversed before evidence could be collected. These fragmented policies highlight both the urgent need and the political difficulty of testing whether reduced glyphosate use translates into tangible ecosystem or health benefits.

More than spots on apples: current policies fail to ensure glyphosate-free organics

Unlike nicotine or alcohol, exposure to agricultural chemicals is not a matter of personal choice. These chemicals are woven into food systems, water, and air, affecting populations

indiscriminately. Even health-promoting diets, such as the Mediterranean pattern, inadvertently increase glyphosate and other pesticide exposures through residues on otherwise wholesome foods (45). This makes agricultural chemicals unique among public health risks; they bypass individual agency and disproportionately burden vulnerable groups, from children to Indigenous communities. When considering organic produce as a "healthier" choice, this may not necessarily be glyphosate-free. While organic farming standards prohibit glyphosate application, emerging evidence suggesting prolonged environmental persistence of glyphosate and AMPA raises questions about whether current transition timelines fully account for residual contamination from prior land use. For example, when a conventional farm converts to certified organic status, a three-year transition period is required during which no substances prohibited under organic standards can be applied to the crops. After this three-year waiting period, harvested commodities can be sold as organic following certification. However, we now know glyphosate can persist for more than a decade after its last application, indicating that a three-year washout period may be insufficient.

Future considerations for evidence-driven stewardship of chemicals in food systems

Nearly five decades after Mitchell's words rang out, we stand at another crossroads in environmental awareness. Chronic inflammatory diseases spanning the gut, metabolism, and mental health now affect millions. These conditions arise from a complex interplay of genetic, microbial, immunological, and environmental factors, with early-life disruptions carrying consequences that extend across generations. The lesson from DDT parallels the dangers of assuming that the absence of acute toxicity equates to safety. Glyphosate may yet prove to be another example where short-term convenience overshadows long-term cost. What is needed is a fundamental shift in how we evaluate and deploy agricultural chemicals. This includes: (1) incorporating microbiome and epigenetic endpoints into standard toxicological assessments; (2) requiring independent, long-term studies before approving widespread use; (3) implementing precautionary restrictions for vulnerable populations and ecosystems; and (4) investigating sustainable alternatives that reduce chemical dependence.

The first of these represents a particularly urgent regulatory gap. Regulatory agencies and industry sponsors rely on Organization for Economic Cooperation and Development (OECD)-standardized toxicological test guidelines, which carry significant institutional weight, but whose consensus-driven approval process can span a decade or more. The gut microbiome is now a clinically meaningful, well-validated indicator of chemical perturbation and disease risk, yet it carries no formal weight in the risk assessments that determine regulatory thresholds and acceptable daily intakes. The European Food Safety Authority (EFSA) has itself acknowledged this, noting that further developments are needed to understand the importance of the microbiota in risk assessment and to identify specific strategies and methodologies accordingly (46). The practical consequence is that a chemical can satisfy the full suite of required toxicological tests while producing precisely the class of low-dose, microbiome-mediated, multigenerational harm described here.

The remaining priorities are equally interdependent. Independent long-term studies with robust, transparent methodology are essential to resolving current uncertainties in dose-response relationships and risk to vulnerable populations. Precautionary restrictions, particularly for pregnant women, infants, and agricultural workers with the highest exposure, are warranted given the multigenerational effects described here. Finally, a meaningful reduction in chemical dependence will require coordinated investment in agroecological alternatives alongside the regulatory and economic frameworks that support their adoption at scale. Moving beyond reactive regulation toward precautionary, evidence-driven stewardship of chemicals in food systems is not just an environmental imperative–it is a public health necessity.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary Material, further inquiries can be directed to the corresponding author.

Author contributions

DG: Validation, Project administration, Supervision, Funding acquisition, Conceptualization, Resources, Investigation, Visualization, Writing – review & editing, Formal analysis. JB: Writing – original draft, Formal analysis, Visualization, Methodology, Investigation, Validation, Conceptualization.

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References

1. Vasseghian Y, Hosseinzadeh S, Khataee A, Dragoi EN. The concentration of persistent organic pollutants in water resources: a global systematic review, meta-analysis and probabilistic risk assessment. *Sci Total Environ*. (2021) 796:149000. doi: 10.1016/j.scitotenv.2021.149000

2. Giesy JP, Dobson S, Solomon KR. Ecotoxicological risk assessment for roundup herbicide. In: Ware GW, editor, *Reviews of Environmental Contamination and Toxicology*. Vol. 167. New York, NY: Springer (2000). p. 35–120.

3. Regan H. Watch a GMO advocate claim a weed killer is safe to drink but then refuse to drink it. Time (2015). Available online at: https://time.com/3761053/monsanto-weed-killer-drink-patrick-moore/ (Accessed March 26, 2015).

4. Benbrook CM. Trends in glyphosate herbicide use in the United States and globally. *Environ Sci Eur*. (2016) 28(1):3. doi: 10.1186/s12302-016-0070-0

5. United States Environmental Protection Agency. DDT: A review of scientific and economic aspects of the decision to ban its use as a pesticide: Prepared for committee on appropriations, U.S. house of representatives. U.S. Environmental Protection Agency; for sale by National Technical Information Service, 1975 (1975).

6. Edge CB, Brown MI, Heartz S, Thompson D, Ritter L, Ramadoss M. The persistence of glyphosate in vegetation one year after application. *Forests*. (2021) 12(5):601. doi: 10.3390/f12050601

7. Mohy-Ud-Din W, Bashir S, Akhtar MJ, Asghar HMN, Ghafoor U, Hussain MM, et al. Glyphosate in the environment: interactions and fate in complex soil and water settings, and (phyto) remediation strategies. *Int J Phytoremediation*. (2024) 26(6):816–37. doi: 10.1080/15226514.2023.2282720

8. Wood LJ. The presence of glyphosate in forest plants with different life strategies one year after application. *Can J for Res*. (2019) 49(6):586–94. doi: 10.1139/cjfr-2018-0331

9. Botten N, Wood LJ, Werner JR. Glyphosate remains in forest plant tissues for a decade or more. *For Ecol Manag*. (2021) 493:119259. doi: 10.1016/j.foreco.2021.119259

10. Maggi F, la Cecilia D, Tang FHM, McBratney A. The global environmental hazard of glyphosate use. *Sci Total Environ*. (2020) 717:137167. doi: 10.1016/j.scitotenv.2020.137167

11. https://osf.io/p93su/overview (Accessed March 5, 2026).

12. Roser M, Ritchie H, Ortiz-Ospina E. Internet. Our World in Data (2020). Available online at: https://ourworldindata.org/

13. Weisenburger DD. A review and update with perspective of evidence that the herbicide glyphosate (roundup) is a cause of non-hodgkin lymphoma. *Clin Lymphoma Myeloma Leuk*. (2021 Sep) 21(9):621–30. doi: 10.1016/j.clml.2021.04.009

14. Milesi MM, Lorenz V, Durando M, Rossetti MF, Varayoud J. Glyphosate herbicide: reproductive outcomes and multigenerational effects. *Front Endocrinol (Lausanne)*. (2021) 12:672532. doi: 10.3389/fendo.2021.672532

15. Qi X, Huang Q, Chen X, Qiu L, Wang S, Ouyang K, et al. Associations between urinary glyphosate and diabetes mellitus in the US general adult: a cross-sectional study from NHANES 2013–2016. *Environ Sci Pollut Res Int*. (2023) 30(59):124195–203. doi: 10.1007/s11356-023-31015-4

16. Eskenazi B, Gunier RB, Rauch S, Kogut K, Perito ER, Mendez X, et al. Association of lifetime exposure to glyphosate and aminomethylphosphonic acid (AMPA) with liver inflammation and metabolic syndrome at young adulthood: findings from the CHAMACOS study. *Environ Health Perspect*. (2023) 131(3):37001. doi: 10.1289/EHP11721

17. Pan L, Xu M, Yang D, Wang B, Zhao Q, Ding E, et al. The association between coronary artery disease and glyphosate exposure found in pesticide factory workers. *Public Health Emerg*. (2016) 1:9. doi: 10.21037/phe.2016.06.07

18. Fuhrimann S, Farnham A, Staudacher P, Atuhaire A, Manfioletti T, Niwagaba CB, et al. Exposure to multiple pesticides and neurobehavioral outcomes among smallholder farmers in Uganda. *Environ Int*. (2021) 152:106477. doi: 10.1016/j.envint.2021.106477

19. Eriguchi M, Iida K, Ikeda S, Osoegawa M, Nishioka K, Hattori N, et al. Parkinsonism relating to intoxication with glyphosate. *Intern Med (Tokyo, Japan)*. (2019) 58(13):1935–8. doi: 10.2169/internalmedicine.2028-18

20. Barbosa ER, Leiros da Costa MD, Bacheschi LA, Scaff M, Leite CC. Parkinsonism after glycine-derivate exposure. *Mov Disord*. (2001) 16(3):565–8. doi: 10.1002/mds.1105

21. Zheng Q, Yin J, Zhu L, Jiao L, Xu Z. Reversible parkinsonism induced by acute exposure glyphosate. *Park Relat Disord*. (2018) 50:121. doi: 10.1016/j.parkreldis.2018.01.021

22. Pu Y, Chang L, Qu Y, Wang S, Tan Y, Wang X, et al. Glyphosate exposure exacerbates the dopaminergic neurotoxicity in the mouse brain after repeated administration of MPTP. *Neurosci Lett*. (2020) 730:135032. doi: 10.1016/j.neulet.2020.135032

23. Höllerhage M. Pesticides and Parkinson's disease: causal relationship at the population and individual level? *J Neural Transm*. (2026) 133(2):229–64. doi: 10.1007/s00702-025-03054-3

24. Euronews. France to continue compensating farmers with Parkinson's disease linked to glyphosate use. Euronews (2023). Available online at: https://www.euronews.com/health/2023/11/17/france-to-continue-compensating-farmers-with-parkinsons-disease-linked-to-glyphosate-use (Accessed November 17, 2023).

25. European Commission, Directorate-General for Health and Food Safety. Glyphosate. Food Safety—European Commission (n.d.). Available online at: https://food.ec.europa.eu/plants/pesticides/approval-active-substances-safeners-and-synergists/renewal-approval/glyphosate_en (Accessed May 18, 2026).

26. Buchenauer L, Haange S, Bauer M, Rolle-Kampczyk UE, Wagner M, Stucke J, et al. Maternal exposure of mice to glyphosate induces depression- and anxiety-like behavior in the offspring via alterations of the gut-brain axis. *Sci Total Environ*. (2023) 905:167034. doi: 10.1016/j.scitotenv.2023.167034

27. Buchenauer L, Junge KM, Haange SB, Simon JC, von Bergen M, Hoh AL, et al. Glyphosate differentially affects the allergic immune response across generations in mice. *Sci Total Environ*. (2022) 850:157973. doi: 10.1016/j.scitotenv.2022.157973

28. Barnett JA, Josephson JK, Yuzbashian E, Haskey N, Hart MM, Soma KK, et al. Prenatal exposure to dietary levels of glyphosate disrupts metabolic, immune, and behavioral markers across generations in mice. *Sci Total Environ*. (2025) 1002:180437. doi: 10.1016/j.scitotenv.2025.180437

29. Kubsad D, Nilsson EE, King SE, Sadler-Riggleman I, Beck D, Skinner MK. Assessment of glyphosate induced epigenetic transgenerational inheritance of pathologies and sperm epimutations: generational toxicology. *Sci Rep*. (2019) 9(1):6372. doi: 10.1038/s41598-019-42860-0

30. Yu N, Tong Y, Zhang D, Zhao S, Fan X, Wu L, et al. Circular RNA expression profiles in hippocampus from mice with perinatal glyphosate exposure. *Biochem Biophys Res Commun*. (2018) 501(4):838–45. doi: 10.1016/j.bbrc.2018.04.200

31. Swanson NL, Leu A, Abrahamson J, Wallet B. Genetically engineered crops, glyphosate and the deterioration of health in the United States of America. *J Org Syst*. (2014) 9(2):6–37.

32. Zaller JG, Heigl F, Ruess L, Grabmaier A. Glyphosate herbicide affects belowground interactions between earthworms and symbiotic mycorrhizal fungi in a model ecosystem. *Sci Rep*. (2014) 4:5634. doi: 10.1038/srep05634

33. Motta EVS, Raymann K, Moran NA. Glyphosate perturbs the gut microbiota of honey bees. *Proc Natl Acad Sci USA*. (2018) 115(41):10305–10. doi: 10.1073/pnas.1803880115

34. Vázquez DE, Villegas Martínez LE, Medina V, Latorre-Estivalis JM, Zavala JA, Farina WM. Glyphosate affects larval gut microbiota and metamorphosis of honey bees with differences between rearing procedures. *Environ Pollut (1987)*. (2023) 334:122200. doi: 10.1016/j.envpol.2023.122200

35. Vázquez DE, Balbuena MS, Chaves F, Gora J, Menzel R, Farina WM. Sleep in honey bees is affected by the herbicide glyphosate. *Sci Rep*. (2020) 10(1):10516. doi: 10.1038/s41598-020-67477-6

36. Helander M, Lehtonen TK, Saikkonen K, Despains L, Nyckees D, Antinoja A, et al. Field-realistic acute exposure to glyphosate-based herbicide impairs fine-color discrimination in bumblebees. *Sci Total Environ*. (2023) 857(Pt 1):159298. doi: 10.1016/j.scitotenv.2022.159298

37. Hunkeler C, Lajad R, Farina WM, Arenas A. Young honey bees apis mellifera learn to avoid pollen contaminated with glyphosate or imidacloprid. *Sci Rep*. (2026) 16(1):5601. doi: 10.1038/s41598-026-35416-6

38. Ruuskanen S, Rainio MJ, Uusitalo M, Saikkonen K, Helander M. Effects of parental exposure to glyphosate-based herbicides on embryonic development and oxidative status: a long-term experiment in a bird model. *Sci Rep*. (2020) 10:6349. doi: 10.1038/s41598-020-63365-1

39. Turin TC, Saad N, Jun M, Tonelli M, Ma Z, Barnabe CCM, et al. Lifetime risk of diabetes among first nations and non-first nations people. *Can Med Assoc J*. (2016) 188(16):1147–53. doi: 10.1503/cmaj.150787

40. The Guardian. Canadian government urged to test sick patients for herbicide (2023). Available online at: https://www.theguardian.com/environment/2023/mar/01/canadian-government-urged-to-test-sick-patients-for-herbicide (Accessed March 1, 2023).

41. Caller T, Henegan P, Stommel E. The potential role of BMAA in neurodegeneration. *Neurotox Res*. (2018) 33(1):222–6. doi: 10.1007/s12640-017-9752-7

42. Government of Canada. Government of Canada moves forward on federal pesticide commitments. Health Canada (2023). Available online at: https://www.canada.ca/en/health-canada/news/2023/06/government-of-canada-moves-forward-on-federal-pesticide-commitments.html (Accessed June 2023).

43. Novotny E. Glyphosate, roundup and the failures of regulatory assessment. *Toxics (Basel)*. (2022) 10(6):321. doi: 10.3390/toxics10060321

44. Québec. P-9.3, r. 1 — Code de gestion des pesticides (Code de gestion des pesticides). Légis Québec (2025). Available online at: https://www.legisquebec.gouv.qc.ca/fr/document/rc/p-9.3,%20r.%201 (Accessed February 1, 2026).

45. Rempelos L, Wang J, Barański M, Watson A, Volakakis N, Hoppe H, et al. Diet and food type affect urinary pesticide residue excretion profiles in healthy individuals: results of a randomized controlled dietary intervention trial. *Am J Clin Nutr*. (2022) 115(2):364–77. doi: 10.1093/ajcn/nqab308

46. Merten C, Schoonjans R, Di Gioia D, Peláez C, Sanz Y, Maurici D, et al. Editorial: exploring the need to include microbiomes into EFSA's scientific assessments. *EFSA J*. (2020) 18(6):e18061. doi: 10.2903/j.efsa.2020.e18061