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POLICY PERSPECTIVE

Glyphosate-Based Herbicides and Public Health: Making Sense of the Science

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Abstract

The controversy over glyphosate-based herbicides (GBHs), where there is extreme divergences in health and environmental assessments, is rooted in several methodological and normative factors. Foremost among them are the differences found in testing pure glyphosate compared to the testing of glyphosate formulations. The adjuvant chemicals found in formulations can be more toxic than the so-called "active ingredient." Other factors can also account for why scientists reach different conclusions on the toxicological effects of GBH including the preconceptions and methodological choices they bring into the study. Lack of consensus on the science can be problematic for policymakers. The paper argues that the toxicological science behind the GBH assessments is embedded in a normative substratum, which must be considered in policy decisions.

Keywords Glyphosate · Glyphosate-based herbicides · Roundup™ · Meta-analysis · IARC

Introduction

The human health and environmental effects of glyphosate-based herbicides (GBHs), the most widely used herbicides in the world, have been widely debated. The scientific literature is anything but consistent and unambiguous on almost all human health and environmental evaluations of GBHs. This essay reveals the complexity of the glyphosate controversy, the science behind it, and the normative context of the science.

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History

Glyphosate was synthesized in 1950 by a Swiss chemist in a small pharmaceutical company who was investigating a new derivative of the amino acid glycine. Not finding any pharmaceutical applications, the new synthetic chemical was kept in storage until it was eventually passed on to Monsanto and studied as a potential chelating agent for water softening. In 1971 a Monsanto chemist John E. Franz learned that glyphosate was effective as a broad spectrum phytotoxicant against weeds. Monsanto received a patent for a formulation of glyphosate in 1974, which the company trademarked as Roundup. Other formulations of glyphosate by Monsanto include Ranger Pro, Garlon 3A, Rodeo Aquatic Herbicide and Vinegar.

Early Agricultural Uses

Roundup received a license for weed control based on early studies that found it to be a relatively safe herbicide. In 1992 a paper published in Weed Technology by authors working for Monsanto wrote: "Glyphosate has favorable environmental features such as rapid soil inactivation and degradation to natural products, little or no toxicity to non-plant life forms, and minimum soil mobility." (Kishore et al., 1992).

GBHs were considered to have had a safety edge against other herbicides because the chemical pathway (shikimate) in which glyphosate disrupted plants and bacteria is not found in mammals. Herrman and Weaver (1999, 479) wrote: "The shikimate pathway is found only in microorganisms and plants, never in animals (Herrman & Weaver, 1999). However, as noted by Gandhi et al. (2021), "glyphosate also influences other pathways, which are based on humans and animals." (Gandhi et al., 2021) And since microbes are in the human gut, the human microbiome has been viewed as a potential target of GBHs (Mesnage & Antoniou, 2020).

Between the 1970s and 1980s hundreds of products were marketed in the United States and throughout the world for agriculture, lawn care, gardens, and forestry. Landrigan and Belpoggi (2018) estimated that 750 products containing glyphosate were sold in 2018 (Landrigan & Belpoggi, 2018). In 1985 an EPA panel classified glyphosate as a Class C chemical (suggestive evidence of carcinogenic potential) based on kidney tumors in male mice (U.S. Environmental Protection Agency, 1985). Six years later the EPA reclassified glyphosate as a Class E chemical (evidence of non-carcinogenic in humans). Roundup was a popular garden herbicide found in many chain stores including pharmacies giving glyphosate the imprimatur of being a safe and effective product. It took another 20 years after EPA's reclassification for the illusion of the safety of GBHs to be shattered.

Round-Up Ready Crops

Throughout the 1980s, plant geneticists were using the newly developed recombinant DNA molecule techniques to develop a new plant breeding technology called molecular breeding. By introducing foreign genes into the seeds, the crops were

endowed with new traits designed to increase productivity. In 1996 Roundup-ready herbicide tolerant genetically modified soybeans, maize, and cotton, developed by Monsanto. Were approved for use in the U.S. agricultural system. These herbicide-tolerant seeds were marketed so that their introduction into large scale farms were premised on the use of the post-emergent, broad spectrum glyphosate-based herbicides. In 1995 a total of 40 million pounds of glyphosate was applied in all applications. By 2000 that figure rose to 98.5 million pounds. And by 2014, the total glyphosate use in the United States was 276 million pounds. From the period 1985–1994, prior to the introduction of glyphosate tolerant crops, to the period 1995–2004 the use of glyphosate increased by 356%. Between the period 1995–2004 to the period 2005–2014 the rise in glyphosate use was 617% (Benbrook 2016).

Other than the development of herbicide-tolerant crops, another factor responsible for the increase in the use of glyphosate-based herbicides (GBH) was the termination of patent protection on glyphosate. Once the patent expired, around thirty companies began manufacturing GBHs. By 2010, glyphosate was certified in 130 countries. Figure 1 shows the rise in glyphosate studies in Web of Science during the period that herbicide-tolerant crops were introduced into agriculture.

IARC's Finding

The International Agency for Research on Cancer (IARC) is an independent research group established in 1965, within the World Health Organization. IARC issues periodic reports on the carcinogenic risks to humans of industrial chemicals. In 2017 it released one of its monographs in which it concluded that glyphosate was a "probable human carcinogen (International Agency for Research on Cancer (IARC), IARC Working Group on the Evaluation of Carcinoenic Risks to Humans, Some Organophosphate Insecticides and Herbicides, 2017). This was the first health agency that declared a strong correlation between glyphosate and human cancer. In 1985 EPA tentatively classified glyphosate as a possible human carcinogen (Group C chemical) based on kidney tumors in mice (U.S. EPA., 1985). IARC's finding brought a

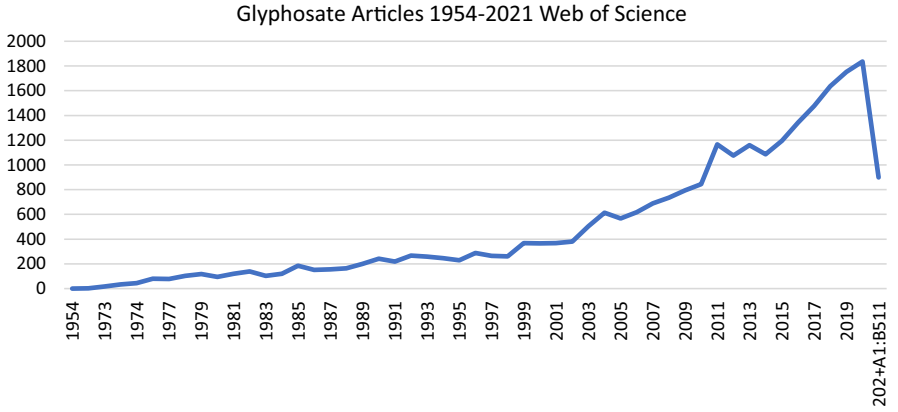

Fig. 1 Glyphosate articles 1954–2021 web of science

spate of litigation from people exposed to glyphosate-based herbicides who attributed their non-Hodgkin's cancer to the chemical.

In 2019, Krimsky wrote: "It should be understood that the controversy over the toxicity of glyphosate or Roundup™ is not a debate over neutral sectors of the scientific community at odds over the intricacies of toxicological methods. The issues are replete with political overtones." (Krimsky, 2019a) Lawyers on both sides of the litigation brought their experts to make the case about the science behind the human health assessment of glyphosate. The plaintiff's quoted IARC's findings, while the defense attorneys cited the EPA's reports that glyphosate was not carcinogenic to humans. Eventually, the jury decided in which experts it believed. Several plaintiffs won multi-million-dollar compensation awards (Stokstad, 2019).

Glyphosate-Based Herbicides

Glyphosate is a laboratory manufactured organophosphate compound with the chemical name N-(phosphomethyl) glycine. It results from a coupling of the methyl group of methyl phosphoric acid with glycine, an amino acid. The chemical symbol for glyphosate is: C3H8N05P.

Glyphosate-based herbicides (GBHs) contain other chemicals aside from glyphosate Called adjuvants. The formulation of GBHs contain a list of all the adjuvants. The toxicological evaluation of a GBH may only examine the so-called active ingredient, namely, pure glyphosate. But the adjuvants play a significant role in the functionality of the herbicide and its toxicology. Chemicals in the GBH formulation are considered inactive because they are not directly responsible for herbicidal activity (Defarge et al., 2018). "An important aspect of glyphosate toxicity is due to its formulations. The glyphosate-based herbicides containing formulations and surfactants makes it difficult to establish toxicity due to glyphosate only, as the components contribute to the overall toxicity." (Defarge et al., 2018) Because the studies of glyphosate-based pesticides are based on different formulations, a comparison of studies or undertaking a weight of evidence analysis can be problematic since one is comparing apples and oranges.

Divergences Behind the Science of GBHs

When scientific studies reach different conclusions, policy makers and the general public are left little on which to base their decisions. In such circumstances "confirmation bias" plays an important role (Rollwage et al., 2020). People choose the scientific results that support their pre-existing beliefs. When authoritative sources produce diametrically opposite conclusions on health and safety of popular consumer products there is more at stake. Public confidence in science is threatened.

In the case of GBHs, scientific conclusions are all over the map. GBHs are found and they are not found to be carcinogenic to humans. They are found and not found to be safe for the environment. They do and do not represent a hazard on food residues. They are safe and not safe for herbicide applicators who follow the manufacturers' guidelines. In many of these contradictory outcomes, the reasons for the

divergence can be ascertained by undertaking an in-depth analysis of the scientific methodology. Here are some of the factors that account for different outcomes.

- Studies are done on different species of mice or rats.
- Short term studies and long-term studies exhibit different results.
- Studies on pure glyphosate yield different outcomes compared with studies on glyphosate formulations, which include adjuvants such as surfactants (Song et al., 2012).
- In vivo studies yield different outcomes than in vitro studies.
- Meta-analyses may differ on the selection criteria for studies.
- Mammalian studies yield different results than non-mammalian studies.
- Weight of evidence analyses may differ on the criterion for selecting studies, i.e., only refereed studies versus all studies, refereed and non-refereed.
- Some GBH assessment studies may be based on average exposure while others may use the high exposures of applicators.

These factors make the science seem divergent and even contradictory. In his study of why IARC and the EPA reached diametrically opposite conclusions on whether glyphosate was a human carcinogen, Charles Benbrook reached these findings (Benbrook, 2019).

1. The EPA's assessment was primarily based on company-commissioned unpublished Regulatory reports, 97% of which showed glyphosate was safe. IARC selected mostly peer-reviewed studies of which 70% were positive.
2. The EPA's evaluation of glyphosate health effects was based on the pure chemical glyphosate, whereas IARC reviewed results of glyphosate-based formulations including the degradation product AMPA (aminomethylphosphonic acid) and the surfactant POEA (polyoxyethylene tallow amine).
3. The EPA's evaluation was premised on typical, general dietary exposure to glyphosate and neglected higher occupational exposures, which IARC considered.

The IARC and the EPA assessments of glyphosate's carcinogenicity, both published in 2017 were analyzed and compared by their references (Krimsky, 2019b). The EPA cited 114 references in its analysis of which 30 were unpublished articles. IARC's report cited 269 references of which none were unpublished articles. From the differences in these studies and the many factors that distinguish toxicological methods, the divergence in the science should not be surprising.

Glyphosate Versus Other Herbicides

Among the arguments in favor of glyphosate-based herbicides is that the active ingredient is less toxic than other herbicides. One way to document toxicity is by oral LD_{50} values, which is the amount of the chemical required to give a lethal dose to 50% of the animals tested, usually mice. LD_{50} is measured in mg. of a chemical

administered per kg of body weight. An oral LD_{50} of 100 means 100 mg of a chemical was found to be lethal to 50% of 1 kg subjects.

Scientists at the Institute of Food and Agricultural Sciences at the University of Florida compared the oral LD_{50} of several commonly used herbicides. The lower the lethal dose, the higher the toxicity. Is glyphosate the safest broad-spectrum herbicide one could use for HT crops and is that safe enough? The LD_{50} of the leading herbicides are: Paraquat ~ 100; Triclopyr: 630; 2,4 D: 666; Pendimethalin: 1050; Atrazine: 3090; Glyphosate: 4900; Imazaquin: > 5000. On the LD_{50} criteria, glyphosate comes out fairly well (Fishel et al., 2021). But LD_{50} is only one criterion with which to judge the toxicity of an herbicide.

Other toxicological criteria include whether the substance is a neurotoxin, mutagen or endocrine disruptor, tests not ordinarily required by EPA. Also, to be considered are the formulations for glyphosate, which includes adjuvants that amplify permeability and increase toxicity. A 2009 study confirms that the adjuvants for Roundup (a trade variety of glyphosate) can sometimes be more toxic to organisms that the active ingredient:

> …adjuvants in Roundup formulations are not inert. Moreover, the proprietary mixtures available on the market could cause cell damage and even death around residual levels to be expected, especially in food and feed derived from R [Roundup] formulation-treated crops [i.e. POEA]. (Benachour & Séralini, 2009)

Gilles-Eric Séralini, a professor of molecular biology at the University of Caen in France, undertook several studies on pure glyphosate and glyphosate-based herbicides and showed that the glyphosate formulations induced cell death and necrosis in human umbilical, embryonic, and placental cells, and alters aromatase levels in testes and sperm (increases estrogen) and impairs pregnancies. He reported that toxic effects were not detected with the so-called active ingredient glyphosate alone. The effects were related to the formulations of the herbicide and its adjuvants (Mesnage et al., 2010). Additional studies on the toxicity of the adjuvants of GBHs confirmed the findings of Séralini on the toxicity of the GBH adjuvants (Hao et al., 2020; Mesnage & Antoniou, 2018; Vincent & Davidson, 2015). Some of these studies clearly demonstrate that glyphosate alone can be harmful to human cells, rodents and non-target species (Table 1). Pure glyphosate has been found to be an endocrine disrupter inducing the growth of human breast cancer cells (Thongprakaisang et al., 2013), lipid metabolism disruption in the offspring of mice (Ren et al., 2019), and estuarine crab reproduction (Avigliano et al., 2014).

Conclusion: Glyphosate and Meta-analyses

Given the diversity of GBH studies, the endpoints tested, and the health and Environmental outcomes, decisionmakers can avail themselves of meta-analyses and systematic reviews to determine whether there is a convergence of viewpoints. From Web of Science and Pub Med I found 14 meta-analyses and systematic reviews for glyphosate. Table 1 summarizes these findings. Of the 14 systematic reviews and

Table 1 Divergences among meta-analyses and systematic reviews of glyphosate-based herbicides on health and environmental effects

Author	Effects studied	Results
Acquavella et al. (2016)	NHL; MM	No support in the epidemiological literature for a causal connection between glyphosate and NHL or MM
Andreotti et al. (2017)	NHL	In this cohort study of 44,932 glyphosate applicators, no association was apparent between glyphosate and any solid tumors or lymphoid malignancies, including NHL. Some evidence of risk of AML
Battisti et al. (2021)	Bees	The exposure of bees to glyphosate-based formulations might cause lethal effects on bees
Boffetta et al. (2021)	NHL; DLBCL	Lack of association between exposure to glyphosate and risk of NHL; association found with DLBCL cannot be ruled out. Small studies showing NHL risk ruled out in larger better designed studies
Cai et al. (2017)	Rodents' reproduction	Glyphosate is toxic to male rodents' reproduction; decreased sperm in rats and mice
Chang and Delzell (2016)	LC; NHL; MM	Our meta-analysis yielded borderline significant RRs [risk ratios] of 1.3 and 1.4 between glyphosate and the risk of NHL and MM respectively and no significant association with risk of HL or leukemia. No causal relationship established between glyphosate exposure and risk of any typo of LHC
De Araujo et al. (2016)	Pregnancy	Current epidemiological evidence (4 studies) does not lend support that glyphosate might pose developmental risks to the unborn child. These negative findings cannot be taken as definitive evidence that glyphosate poses no risks for human development and reproduction
De Ghisi et al. (2016)	MN frequency	Exposure to glyphosate and its formulations increases the frequency of MN formation-a measure of genotoxicity. Higher MN formation found in mixtures than pure glyphosate
Donato et al. (2020)	NHL; MM	No consistent indication of an association between exposure to glyphosate and risks of NHL or MM. Data for risk of NHL came from small studies that suffered from publication and other forms of bias
Kabat et al. (2021)	NHL	The strength of evidence for an association between glyphosate and NHL was greater when estimates from AHS, based on assumptions of long latency, were selected. One cannot say definitively that any particular meta-analysis is closer to the truth; each meta-analysis (including this one) should be interpreted with great caution
Leon et al. (2019)	NHL; MHR; DLBCL	No observed association between risk of NHL and overall use of glyphosate. Glyphosate associated with elevated MHR for DLBCL
Nagy et al. (2020)	Cytotoxicity	Glyphosate formulations have more pronounced cytotoxic effects compared to pure glyphosate
Schinasi and Leon (2014)	BCL; NHL	B-cell lymphoma was positively associated with glyphosate. Strongest RR estimates associated with subtypes of NHL
Zhang et al. (2019)	NHL	With evidence from experimental animals and mechanistic studies our meta-analysis suggests a compelling link between exposures to GBHs and increase risk of NHL

AML acute myeloid leukemia, *AHS* Agricultural Health Study, *BCL* B-cell lymphoma, *DLBCL* diffuse large B-cell lymphoma, *GBH* glyphosate-based herbicide, *HL* Hodgkin's lymphoma, *LC or LHC* lymphohematopoietic cancers, *MM* multiple myeloma, *MN* micronuclei, *NHL* non-Hodgkin's lymphoma

meta-analyses, most looked at cancer endpoints (lymphomas). Some were negative, some positive and some mixed. Some saw strong associations with specific cancers such as MM and LC. Most saw no association between glyphosate and NHL, one study saw compelling evidence, while others saw a weak or marginal association. The study on pregnancy could not rule out an association but could not make a definitive claim. On genotoxicity and cytotoxicity to mammalian cells, two reviews had positive conclusions.

Most of the reviews were hesitant to draw causal relationships between GBHs and human Diseases. The exception were the controlled animal studies. Some studies pointed to the paucity of good data from which to draw conclusions. An important principle in public health should be recalled. 'No (or insufficient) evidence of harm' is not a basis for claiming there is 'evidence of no harm.' The latter conclusion requires good evidence, not a paucity of evidence.

Among the factors that can explain outcome divergencies is bias through conflict of interest. In such instances, toxicologists adopt discretionary assumptions and statistical methods that weigh against a positive finding of health risk. In one review the authors (Acquavella et al., 2016) declare an association with Monsanto. From the systematic reviews and meta-analyses of refereed studies we are left with divergent conclusions regarding the health effect of GBHs ranging from "no human health risks," "compelling human health risks," and "possible or probable human health risks." Without a consensus on the criteria used in the studies, convergence on the outcome of health and environmental effects of GBHs will be problematic.

The normative substructure of applied toxicological research are the discretionary decisions that are made throughout the process. Do we do short term or long-term studies? Do we test glyphosate exclusively or do we test the entire formulation of the GBH with the adjuvants? When we engage in weight of evidence, do we only choose refereed articles, or do we use unpublished research of the manufacturer? Do we accept studies where the author(s) have a financial interest in the outcome? Which kinds of animals do we test on? Should they be young or old? Should we test on pregnant animals? What endpoints do we look for? These questions cannot be answered by science alone. They must be answered by policy makers consulting with scientists.

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