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Implications

1. The margin of safety between typical glyphosate and AMPA exposure levels and the maximum allowed human exposures has narrowed substantially in the last decade. The margin may well have disappeared for heavily exposed segments of the population in some countries, especially where glyphosate and AMPA are present in drinking water. In addition, farmworkers and rural residents may incur relatively high dermal absorption and/or exposures via drinking water. We conclude that existing toxicological data and risk assessments are not sufficient to infer that GBHs, as currently used, are safe.
2. GBH-product formulations are more potent, or toxic, than glyphosate alone to a wide array of non-target organisms including mammals [42, 43], aquatic insects, and fish [44]. As a result, risk assessments of GBHs that are based on studies quantifying the impacts of glyphosate alone underestimate both toxicity and exposure, and thus risk. This all-too-common shortcoming has repeatedly led regulators to set inappropriately high exposure thresholds (cRfDs, ADIs).
3. The toxicological data supporting current GBH regulatory risk assessments are out-of-date and insufficient to judge the impacts of contemporary glyphosate and AMPA exposure levels on the developing mammalian fetus, the liver and kidneys, and reproductive outcomes in humans and a variety of other animals [3, 25].
4. Most toxicological studies using advanced, modern tools and experimental designs within molecular genetics, reproductive, developmental, endocrinological, immunological and other disciplines have been undertaken in academic and research institute laboratories, and results have been published in peer-reviewed journals. Regulators have not incorporated, formally or indirectly, such research into their risk assessments. Rather, they rely on unpublished, non-peer reviewed data generated by the registrants. They have largely ignored published research because it often uses standards and procedures to assess quality that are different from those codified in regulatory agency data requirements, which largely focus on avoiding fraud [79]. Additionally, endocrine-disruption study protocols have not been codified by regulators[8].
5. While the German Federal Institute for Risk Assessment, rapporteur for the European Food Safety Authority's current reassessment of glyphosate, claimed to have examined more than 900 scientific studies published in peer-reviewed journals, most of the studies were deemed of limited value, and hence had little influence on the outcome of their assessment. Studies were classified of 'limited value' based on degree of adherence to traditional, toxicology protocols and 'validated' endpoints, rather than scientific rigor and relevance in understanding the mechanisms leading to adverse health outcomes. Had the German Institute used scientific quality and relevance in identifying useful studies, instead of relying on similarity to outdated methodologies and/or controversial evaluation criteria [80] (such as the Klimisch score), we are nearly certain that they would have concluded that published studies collectively provide strong evidence in support of at least a three-fold reduction in the glyphosate E.U. ADI and consequently a 15-fold reduction in the U.S. cRfD [3, 21, 25, 26].

Conclusions

GBH use has increased approximately 100-fold since the first decade of its use in the 1970s. It is now the world's most heavily applied herbicide. Major increases in its use resulted from widespread adoption of Roundup Ready crops that were genetically engineered to be tolerant to glyphosate. Applications of GBHs have also expanded in aquatic, estuarine, rangeland, and forest habitats.

Initial risk assessments of glyphosate assumed a limited hazard to vertebrates because its stated herbicidal mechanism of action targeted a plant enzyme not present in vertebrates. In addition, because GBHs kill nearly all actively growing plants, farmers had to apply GBHs early in the year, before crop germination or post-harvest, and so it seemed unlikely that there would be residues in harvested crops and the food supply. However, these assumptions ignored the possibility that glyphosate and its metabolites might act via other pathways, including those present in vertebrates, as well as the profound consequences of major increases in the

area treated and volume applied, coupled with changes in how and when GBHs are used by farmers (e.g., on GE, herbicide-tolerant crops, and as a pre-harvest desiccant to accelerate harvest).

Evidence has accumulated over the past two decades, especially, that several vertebrate pathways are likely targets of action, including hepatorenal damage, effects on nutrient balance through glyphosate chelating action and endocrine disruption. Other early assumptions about glyphosate, for example that it is not persistent in the environment, have also been called into question, depending upon soil type. In addition, the prediction that glyphosate would never be present widely in surface water, rainfall, or groundwater has also been shown to be inaccurate.

Existing data, while not systematic, indicate GBHs and metabolites are widely present in the global soybean system and that human exposures to GBHs are clearly rising. Tolerable daily intakes for glyphosate in the U.S. and Germany are based upon outdated science.

Taken together, these conclusions all indicate that a fresh and independent examination of GBH toxicity should be undertaken, and that this re-examination be accompanied by systematic efforts by relevant agencies to monitor GBH levels in people and in the food supply, none of which are occurring today. The U.S. National Toxicology Program should prioritize a thorough toxicological assessment of the multiple pathways now identified as potentially vulnerable to GBHs. The urgency of such work was reinforced in March 2015 when the IARC concluded glyphosate is a probable human carcinogen.

We are aware of current limits on, and demands for, public funding for research. In the absence of government funds to support essential GBH research, we recommend that a system be put in place through which manufacturers of GBHs provide funds to the appropriate regulatory body as part of routine registration actions and fees. Such funds should then be transferred to appropriate government research institutes, or to an agency experienced in the award of competitive grants. In either case, funds would be made available to independent scientists to conduct the appropriate long-term (minimum 2 years) safety studies in recognized animal model systems. A thorough and modern assessment of GBH toxicity will encompass potential endocrine disruption, impacts on the gut microbiome, carcinogenicity, and multigenerational effects looking at reproductive capability and frequency of birth defects.

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Abbreviations

2,4-D	2,4-Dichlorophenoxyacetic acid
ADI	Acceptable daily intake
AMPA	Aminomethylphosphonic acid
Bt	Bacillus thuringiensis
cPAD	Chronic Population Adjusted Dose
cRfD	Chronic reference dose
EPSPS	5-enolpyruvylshikimate-3-phosphate synthase
EU	European Union
FQPA	US food quality protection act of 1996
GBHs	Glyphosate-based herbicides
IARC	International Agency for Research on Cancer

LOAEL Lowest Observed Adverse Effect Level
NOAEL No Observed Adverse Effect Level
US EPA United States Environmental Protection Agency
Go to:

Footnotes

[1]The E.U. ADI was calculated based on observed kidney (hepatorenal) effects in rat chronic toxicity studies. The "No Observable Adverse Effect Level" (NOAEL) was 31 mg/kg/day, and the "Lowest Observable Adverse Effect Level" (LOAEL) occurred at a dose of 60 mg/kg/day (determined then to be the LOAEL). A standard 100-fold safety factor was applied in converting the E.U.-set NOAEL to the ADI of 0.3 mg/kg/day. The new ADI recommended by the German regulators of 0.5 mg/kg/day is based on teratogenic effects in rabbits. The NOAEL was considered to be 50 mg/kg/day. Independent scientists argue that the 2002 determination was not based on the most sensitive species or dataset, as is required by regulatory authorities. See ref 14. Antoniou M, Habib MEM, Howard CV, Jennings RC, Leifert C, Nodari RO, Robinson CJ, Fagan J: Teratogenic effects of glyphosate-based herbicides: divergence of regulatory decisions from scientific evidence. J Environ Anal Toxicol 2012, S4:006.

[2]The EPA issued an updated registration review of GBHs in 1993. Studies dating from the early 1970s through mid-1980s dominated the reference list accompanying the chapter setting forth the EPA's estimate of GBH human health risks.

[3]Table B.7.3-8 in the document "Renewal Assessment Report, Glyphosate Residue Data" (Vol. 3, Annex B.7, Dec. 18, 2013, RMS: Germany, Co-RMS-Slovakia) provides an overview of the levels of glyphosate and AMPA measured in the meat, milk, and eggs from several livestock species, as well as in the fat, meat, kidney, and livers of the animals. In most cases the levels reported in liver and kidney exceed those in other tissues by several-fold, and the levels in kidney exceed those in liver by 3-fold to over 10-fold.

[4]Retinoic acid signaling plays a key role in guiding embryonic development, affecting the expression of multiple genes in a variety of cell types. Altered retinoic acid activity causes birth defects (see 58. Duester G: Retinoic acid synthesis and signaling during early organogenesis. Cell 2008, 134(6):921-931.

[5]Glutamate is a common vertebrate neurotransmitter released by neurons into the synapse, and is important for learning and memory (for a review, see 59. Meldrum BS: Glutamate as a neurotransmitter in the brain: review of physiology and pathology. J Nutr 2000, 130(4S Suppl):1007s-1015s. Glyphosate's structural similarity to glutamate creates the potential for interfering with this key signaling process.

[6]"Environmentally relevant" exposures to GBHs are those that fall within the documented exposure levels arising from the way GBHs are typically used.

[7]Pharmacokinetic studies project and monitor the levels of a chemical absorbed by an organism (via ingestion, inhalation, dermal absorption, or some other route of exposure), how the chemical is distributed throughout the body to specific tissues (measuring the concentrations in different organs and in the blood), how the chemical is metabolized (including which metabolites are produced, and whether the presence of these metabolites and their relative abundance is dependent on route of exposure), and finally, how a compound is excreted (e.g., in feces or urine). Pharmacokinetic studies provide a valuable link between estimates of exposure, toxicity studies, and estimates of human risk.

[8]The process of establishing testing protocols for endocrine-mediated impacts has been underway in the U.S. since 1997, in response to a mandate in the 1996 Food Quality Protection Act to consider such effects in

assuring a "reasonable certainty of no harm" for pregnant women, infants, and children. Seventeen years later, the EPA remains years away from codifying a new battery of tests capable of identifying the risk of low-dose, endocrine-disruption driven effects.

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