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Herbicide Tolerant Genes, Part 2
Giddy 'bout Glyphosate

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Over the past eight months, I've been using these pages to examine the science behind transgenic crops in light of public perception of the dangers of these technologies. In March, April, May, and June's issues, I looked at insecticidal genes, namely, the *Bacillus thuringiensis* (Bt) genes that have been incorporated into certain production crops. In September, I turned to focus on crops that have been engineered to contain herbicide-tolerant genes—products like Roundup Ready (RR) corn, soybeans, cotton, and canola. In that essay, I laid out the scientific principles behind the transgenic technology, and addressed three concerns about herbicide-tolerant crops, namely:

- whether engineered RR genes have unintended effects on other plant genes or traits;
- whether plant metabolism is sufficiently affected to produce new toxic proteins or allergens; and
- whether RR crops are nutritionally equivalent to traditionally bred crops.

These three concerns are similar to those expressed regarding the insect-resistant Bt transgenic crops. But herbicide-tolerant crops face another hurdle for public acceptance. If acres of corn and soybeans are bullet-proof to glyphosate (Roundup), won't wholesale aerial spraying ensue? And won't that make us all sick? Some claim we just don't know enough about glyphosate. When industry advocates claim we do, the frequent retort is, "Yeah, that's what they said about DDT."

I'm willing to bet most people who cite Rachel Carson's *Silent Spring* (3) and its landmark indictment of DDT never actually read the book; if they had, they would have seen its references to scientific articles about DDT's hazards dating back to the late 1940s and 1950s, at least 15 years before *Silent Spring*'s publication. Similarly, an incredible amount of information has been collected about glyphosate over the last 20 years (7, 9, 14,15, 17).

Of course critical analyses of the scientific literature have never stopped scary pronouncements about doom on certain websites. The following concerns about glyphosate use have repeatedly appeared on a number of environmental advocacy group (EAG) websites. The concerns seem to be a recycling of a lot of information in a pesticide factsheet that appeared in the Northwest Coalition for Alternatives to Pesticides' (NCAP's) *Journal of Pesticide Reform* (4).

- Glyphosate and its formulation, Roundup, cause systemic toxicity.
- Glyphosate is hazardous to workers.
- Glyphosate causes mutations and cancer.
- Glyphosate adversely affects reproduction.
- Glyphosate poses ecological hazards to soil microorganisms, invertebrates, fish, and wildlife.
- Hazard will increase significantly due to widespread use of glyphosate on transgenic crops.
- The data used by the EPA to determine glyphosate's hazards can't be trusted because it comes from industry.

Let's take a look at these concerns in light of the volumes of available data.

Framework for Assessing Safety

The above laundry list of concerns could apply to any chemical used at work or at home and released into the environment. Some of them are true. For example, at some doses glyphosate does cause systemic toxicity (i.e., adverse effects on internal organs and physiological systems). But knowing that tells us nothing about the probability of real-life adverse effects from using glyphosate and from inadvertent exposures like spray drift. To determine the validity of EAG concerns and aid a decision about safety, glyphosate and its formulation Roundup must be judged in the context of a risk assessment procedure.

Risk assessment consists of four basic information-gathering activities:

1. characterization of hazards;
2. determination of the relationship between dose and response;
3. assessment of exposure; and
4. integration of the above information to characterize risk (i.e., probability of an adverse effect).

As will be shown, many of the concerns expressed by EAGs over pesticide use stem from myopic attention to

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hazard characterization without integrating dose-response relationships for specific biological effects and real-world exposures. Determining whether exposure to a pesticide poses a high or low risk under specific conditions is as much dependent on what regulatory agencies "feel" is an acceptable risk (a social decision) as it does on the magnitude of exposure (measured or otherwise estimated).

Dose Makes the Poison

EPA characterizes risk of adverse health effects by comparing estimated pesticide exposures to its Reference Dose (RfD). The RfD, which is expressed as milligrams of pesticide per kilogram of body weight per day (mg/kg/day), is defined as an exposure with reasonable certainty of no harmful effects after a single (acute) or lifetime daily (chronic) exposure. The hazard characterization process is very important to development of the RfD. Knowing the dose at which an effect occurs is as important as characterizing the effect itself. EAGs are fond of pointing out that glyphosate and other pesticides cause illness with symptoms like nausea, vomiting, and depressed blood enzymes. What they don't tell you are the doses that cause no harm.

When a compound is of very low toxicity, like glyphosate, a lot of it can be fed to rodents before they keel over and die. Short of death, however, some serious injury can occur. For example, glyphosate causes death to 50% of rats tested at an oral dose above 5000 mg/kg (17) (Table 1, oral LD_{50}). To put that exposure into perspective, consider that vitamin A has a body-weight-adjusted LD_{50} of nearly 2000 mg/kg, table salt (sodium chloride) has an LD_{50} of about 3500 mg/kg, and the caffeine in coffee and soft drinks has an LD_{50} of about 200 mg/kg. In short, glyphosate is not a very potent toxin, whether exposure occurs by ingestion or by skin contact (Table 1, dermal LD_{50}).

One of the tricks that EAGs use to make glyphosate and other pesticides look very hazardous is to recite the litany of hazards from Material Safety Data Sheets (MSDS). The MSDS is meant to provide workers with information about the potential hazards when handling chemicals in comparatively pure or highly concentrated forms. Workers face the greatest risk of being excessively exposed to concentrated pesticide formulations. The MSDS is misused when its stated hazards are used to characterize biological effects from exposure to environmental levels of pesticide residues (5).

How High Can You Go?

The information in the MSDS comes from the manufacturers' databases of toxicity studies. Glyphosate testing for systemic toxicity is an excellent example of the cliché "at some dose everything is a poison." In a subchronic toxicity study, rats were fed daily for three months a diet containing 0, 1000, 5000, or 20000 ppm of glyphosate. At a concentration of 20000 ppm, glyphosate would constitute 2% of the total weight of the diet! Based on the amount of food the rats ate each day and their body weights, the average dose to both males and females was 0, 74, 361, and 1445 mg/kg/day.

In subchronic toxicity tests, just about every organ system and physiological parameter you can imagine are examined for changes relative to a non-dosed group of rodents or dogs. At the highest dose tested (1445 mg/kg) in the glyphosate subchronic test some males, but not females, had pancreatic lesions. Also, levels of blood urea nitrogen and an enzyme called serum alkaline phosphatase were elevated compared to non-dosed animals. Serum phosphorus and potassium were elevated in all dose groups and glucose was elevated in the mid- and high-dose groups.

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TABLE 1

Acute toxicity and potential for eye and skin irritation of glyphosate, some of its formulated products, and the surfactant POEA (modified from 17).

Test Material	Oral LD50 (mg/kg)	Dermal LD50 (mg/kg)	Inhalation LC50 (mg/L)	Eye Irritation	Skin Irritation
glyphosate	4392	5600	not tested	mild	slight
Roundup (41% glyphosate + 15% POEA)	>5000	>5000	3.18	severe	slight
Roundup T/O (18% glyphosate + 7% POEA)	>5000	>5000	>5.7	moderate	nil
Ready to Use (1% glyphosate + 0.4% POEA)	>5000	>5000	>8.9	slight	nil
POEA	1200	1260	not tested	corrosive	severe

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The ion and glucose elevations are not necessarily an adverse toxicological effect; rather, they could be related to the chemical properties of the diet when an organic acid like glyphosate is added at such a high concentration. Such effects of diet composition were hypothesized to be responsible for salivary gland lesions in rats fed doses between 200 and 3300 mg/kg (17). Only the parts of the salivary gland responsible for secretions stimulated by acid foods (think citrus products) were affected, suggesting that the high concentrations of glyphosate substantially changed the pH of the food. Such an effect is best described as a physical irritation rather than a toxicological effect, especially when no other systemic effects were observed (17).

TABLE 2

No Observable Adverse Effect levels (NOAELs) for glyphosate, its metabolite AMPA, and the Roundup surfactant POEA (15, 17).

Toxicity Endpoint	No Observable Adverse Effect Level (NOAEL, mg/kg/day)		
	Glyphosate	AMPA	POEA
Subchronic Systemic	209	263	36
Chronic Systemic	409	>2.8	-
Developmental	175	400	300
Reproductive	694	>4.2	-
Carcinogenicity[1]	Negative	Negative	Negative
Endocrine Disruption[2]	Negative	Negative	Negative
Neurotoxicity	Negative	Negative	Negative
EPA Reference Dose[3]	2	-	-

[1]EPA has classified glyphosate as class E, non-carcinogenic for humans.

[2]Glyphosate was tested in an in-vitro assay and found negative for the ability to interact with estrogen receptors; the findings for AMPA and POEA are based on the lack of any endocrine modulation effects in developmental studies and two- or three-generation reproduction studies.

[3]The reference dose (RfD) was set by EPA based on an NOAEL of 175 mg/kg for maternal toxicity in a developmental toxicity study; no effects on fetal development were noted at doses of 1000 mg/kg.

Ironically, the effects noted in the subchronic toxicity studies were not observed in the same species of rat fed for two years doses of 0, 101, 410, and 1062 mg/kg. In this chronic toxicity study, effects were seen only at the high dose, and they consisted of a comparative decrease in body weight, increased incidence of cataracts and lens abnormalities (males only), decreased urinary pH, and increased liver weight (17). Although the high dose didn't kill the rats, it was a substantial percentage of the LD_{50}.

Together with information from tests for eye and skin irritancy of glyphosate, the observations from the subchronic and chronic toxicity studies would be incorporated into an MSDS. But neither the MSDS nor the NCAP article mention there was a dose at which no effect occurred (called the NOAEL or No Observable Adverse Effect Level, Table 2). To put the magnitude of the 409 mg/kg chronic toxicity NOAEL in perspective, if glyphosate was pelleted as is regular strength aspirin (350 mg per tablet), then 82 tablets could be consumed each day without effect. Obviously, that is not something you want to try at home.

Other types of effects, including neurotoxicity and developmental and reproductive toxicity, are also studied at extremely high doses administered to rats daily for long periods of times. Always bear in mind that the doses are chosen to be below lethal levels yet to be high enough to cause a definitive effect. For regulatory purposes, at least one dose should be low enough to not cause any effect.

The NOAELs from a variety of glyphosate toxicity tests are shown in Table 2. Despite the high doses fed to rodents, glyphosate was not neurotoxic nor did it adversely affect fetal development or reproductive performance. These latter two tests are probably the most sensitive way to test for effects on the endocrine system because they are geared to detecting subtle changes in hormonal modulation and a variety of endocrine-sensitive endpoints (2). If glyphosate were a so-called "endocrine disrupter," then reproductive physiology and fetal development would likely have been affected.

What about Worker Poisonings?

Several interesting tidbits regarding human glyphosate exposures have been repeated on EAG websites. Citing statistics from California, the authors have ranked glyphosate third in number of worker exposures reported to health authorities. However, when normalized for the number and amount of applications, glyphosate incidences fall out of the top ten. Furthermore, complaints were often recorded in one of several categories of

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likelihood of cause and effect. Many recorded cases fell into the category of suspected illness, but little evidence was gathered to confirm such classification and whether glyphosate exposure had actually occurred (17).

The vast majority of the complaints about glyphosate relate to skin and eye irritation (15). As discussed beginning on page 10, the surfactants in any kind of product can be irritating, but such an effect is a physical injury, not systemic toxicity.

Pharmacokinetics—The Secret Behind Glyphosate's Low Toxicity

The secret to understanding why dose makes the poison is pharmacokinetics, a fancy word for describing what happens to a chemical and how fast it happens after we are exposed. To understand the toxicity of a pesticide, we need to understand its basic chemistry and answer the following questions:

- How much is absorbed via skin and intestines?
- How fast is the chemical distributed in the body?
- How quickly is the chemical detoxified?
- How quickly is it eliminated from the body?
- What is the nature of the metabolic products?

EAG websites like to portray glyphosate as an organophosphate (OP) compound (it has one phosphorous atom in it) because this links it to the controversial OP insecticides infamous for their effects on the nervous system. The formal chemical name of glyphosate, N-phosphono methylglycine tells the real story—glyphosate is actually an amino acid related chemically to glycine, one of the amino acids synthesized by our body. One of the known environmental breakdown products of glyphosate is AMPA (aminomethyl phosphonic acid), which is eventually broken down by microbes into glycine. Because AMPA residues may be in food, we also need to understand how the body processes this metabolite.

Glyphosate and AMPA are poorly absorbed by the skin and intestine. Studies with human skin preparations and live monkeys indicate that at most 2% of a dermal dose actually enters the body (16). The lack of glyphosate penetration of the skin allows it to be easily washed off with soap and water. After oral exposure, the intestine can absorb less than 35% of the glyphosate dose.

Of the dose of glyphosate or AMPA that makes it into the blood, nearly 99% of it is excreted in the urine within 24 hours (16). For oral doses, most of the elimination is in the feces, largely because glyphosate is so poorly absorbed across the intestines.

Although plants and soil microorganisms have the ability to degrade glyphosate to AMPA, mammals don't. Thus far, no one has been able to find any biotransformation products of glyphosate in mammalian tissue. At reasonable exposure levels, glyphosate seems not to be capable of interacting with any mammalian enzymes or physiological receptors. In short, animals lack the EPSPS enzyme that glyphosate inhibits in plants (6), disrupting their ability to make aromatic amino acids.

TABLE 3

Aggregate exposure (mg/kg/day) of children (1 – 6 years old) to glyphosate, the metabolite AMPA, and the Roundup surfactant POEA (modified from 17).

Type of Exposure	Glyphosate		AMPA		POEA	
	Acute	Chronic	Acute	Chronic	Acute	Chronic
Dietary	0.058	0.058	0.01	0.01	0.026	0.026
Drinking Water	0.001	0.001	0.001	0.001	0.001	0.001
Application	0	0	0	0	0	0
Re-entry	0.026	0	0	0	0.065	0
Spray Drift	0.538	0	0	0	0.9	0
Aggregate (sum of all types)	0.623	0.059	0.011	0.011	0.992	0.027
Aggregate[1] (10X dietary exposure)	1.145	58.1	0.105	0.105	1.226	0.261

[1]To account for increased usage of glyphosate products on Roundup Ready crops, this aggregate exposure was also calculated, assuming a very conservative 10x more dietary exposure.

Cranky Concerning Cancer

"It causes cancer" is an old battle cry applied by EAGs in opposition to the use of nearly

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every pesticide. The EPA wrangled for many years about how to classify the carcinogenic potential of glyphosate. In two-year rodent dietary exposure studies with daily doses ranging to well over 1000 mg/kg/day, occasional tumors would be found, but they were not dose related. In other words, animals at the lower doses would have tumors that animals at the higher doses did not. When an effect is noted without a relationship to dose, toxicologists usually dismiss it as random chance. After all, animals not fed glyphosate also develop tumors occasionally. After an independent panel under the auspices of the EPA's Scientific Advisory Panel reviewed one of the more perplexing studies, EPA finally classified glyphosate in Group E: "Evidence of non-carcinogenicity for humans" (15).

Nevertheless, making the circuit around EAG websites this past year was the proclamation that new evidence showed glyphosate causes cancer. The "new" evidence was an epidemiological study in Sweden linking increased risk for non-Hodgkin's lymphoma (NHL) to glyphosate use (8). A negative critique of the study has already been published (1). The data and conclusions of the Swedish study bear examination to illustrate how easy it is to mischaracterize the results of epidemiological studies with pesticides and their general unreliability for risk assessment.

First, the Swedish study's data showing an association between NHL and glyphosate was based on self-reporting of pesticide use among the study population. The subjects, who developed NHL during 1987-1990, were interviewed during 1993-1995 about pesticide use that may have occurred as long as 40 years ago. The vast majority of interviewees had used the subject pesticides between the 1970s and 1980s. These types of subject surveys are common but they depend on recall of activities perhaps a decade or more earlier. When the subjects were deceased, their next of kin were requested to provide the exposure history.

A second problem with the Swedish study is that the conclusions ignored the fact that the association between glyphosate use and incidence of NHL was not even statistically significant. The key word is association, because epidemiology studies of chemicals cannot tell us anything about cause and effect.

Finally, the Swedish study ignores the very low potential of glyphosate to penetrate the skin even if a worker was exposed (11, 16). Furthermore, glyphosate has failed to produce dose-related tumors in experimental animals, and numerous studies of its mutagenic potential have failed to even prove it is a mutagen or can cause chromosomal aberrations (14, 15, 17). In short, the Swedish study made faulty conclusions that were not supported by the available data.

Rabid Regarding Reproduction

Until recently, epidemiology studies like the one from Sweden have focused almost solely on linking pesticides with cancer. Today, however, endocrine disrupters are demanding equal attention. Epidemiological studies of pregnancy outcome (for example, miscarriages, preterm births) and chemical exposure are being increasingly reported. Despite glyphosate not showing any evidence of effects on fetal development nor reproduction over three generations in rodent studies, one epidemiology study of reproductive outcome among couples living on farms in Ontario, Canada, has been invoked as the "smoking gun" for causing pregnancy problems (4, 13).

This Canadian reproduction study suffers from the same problems as the attempts to link glyphosate use with NHL—namely subject recall of distant exposures and failure to consider the low absorption potential of glyphosate. The results depicted in the NCAP glyphosate fact sheet that showed an increased rate of miscarriages in association with glyphosate exposure were misinterpreted (4). In fact, the correct risk parameter to examine, the odds ratio, was ignored, probably because it leads to the proper conclusion that an association between pregnancy outcome and glyphosate exposure was not statistically significant.

Silly Over Surfactants

One of the ironies of a compound with toxicity as low as exhibited by glyphosate is that more attention is paid to its formulation. In fact, organizations like NCAP have been screaming for a long time about the toxicity of inert ingredients in pesticide formulations. Mammalian and ecotoxicology studies of Roundup formulations and some of their more prominent inerts have been comparatively well studied. The most common inert in

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Roundup is a surfactant called POEA (polyethoxylated tallow amine, a.k.a. polyoxyethyleneamine). POEA is added to Roundup formulations at a concentration of approximately one-third that of glyphosate.

Surfactants are added to pesticide formulations both to help solubilize the active ingredient in water as well as to help "spread" the spray droplets across a leaf surface for better coverage. Surfactants come in all sizes, shapes, and chemistries but all of them have several properties in common. For example, they all reduce the surface tension of water and they can disrupt the lipid layer of biological membranes. We are exposed to surfactants everyday, unless you refrain from hand washing, hair shampooing, and dealing with dirty dishes.

As is true of any substance, surfactants at high enough doses can cause some nasty effects. However, POEA seems to be nearly as innocuous to mammals as glyphosate itself. The acute oral LD_{50} has been estimated to be as low as 1200 mg/kg (17). One source applied a mathematical technique to the toxicity data for Roundup itself and estimated the acute oral LD_{50} may be 40,000 mg/kg (14)!

An oral dose to rats of 324 mg/kg body weight (4500 ppm in diet) caused intestinal irritation, decreased food consumption, weight gain, and some alteration in serum hematological parameters (17). However, a dose of 36 mg/kg was without adverse reactions. The adverse response at the highest dose is typical for any surfactant because these types of chemicals can irritate tissues by disrupting membranes. Relative to the potential for exposure (whether dietary or from spray drift), even the NOAEL dose is unrealistically extreme, especially considering the exposure was given daily for 90 days. Assuming that POEA made it into the body via oral exposure or by skin absorption, its chemical nature indicates it would be metabolized into short-chain carboxylic acids (17), smaller molecules that would enter into the body's normal respiratory metabolism pathways. Thus, it is not surprising that POEA has exhibited no reproductive, developmental, neurotoxic, or endocrine system toxicity in subchronic feeding studies (17).

The ability of surfactants to irritate tissue is well illustrated by comparing glyphosate's classification as an eye or skin irritant with that of its formulations containing the surfactant POEA (Table 1, page 7). Note that the acute oral and dermal toxicity of glyphosate and its formulated products are similar, but each has a different potential for eye and skin irritation. Glyphosate itself causes mild to slight irritation of eye and skin tissue. In contrast, POEA is extremely irritating to dermal tissues. Consequently, formulated glyphosate is also irritating, but the severity declines as the concentration of POEA decreases.

Irritating properties of Roundup have been compared to baby shampoo, dishwashing detergent, and household liquid cleaner. Roundup and the baby shampoo were similar in irritation potential, and each was less irritating than the detergent and cleaner (12).

The Missing Link— Exposure Assessment

A key element lacking in every EAG website on pesticides that I visit is exposure assessment. If hazard characterization and dose-response relationships definitively show there are NOAELs for any effect, then logically we would ask, "How much are we exposed to in the real world?"

Before the Food Quality Protection Act (FQPA), EPA only estimated our total exposure to pesticide residues in the diet. Now, EPA must also consider exposure to residues in water and from home use. EPA's findings about glyphosate were issued in 1993 in a Re-registration Eligibility Decision Document (RED) (15). At that time, EPA assumed all the residues in food were at the level of the tolerance. Tolerances, although they are legal limits for maximum residues, grossly overestimate food residues. Nevertheless, EPA found that exposure to glyphosate at the time was at maximum 2% of the RfD. Anything under 100% of the RfD makes the EPA happy, and the agency has no problems with renewing a pesticide's registration.

Recently, an aggregate exposure assessment was conducted for glyphosate that essentially reached the same conclusion as the EPA's 1993 RED (17). Elements of this aggregate assessment for acute and chronic exposure are shown in Table 3. Note that in the aggregate assessment water and occupational exposures

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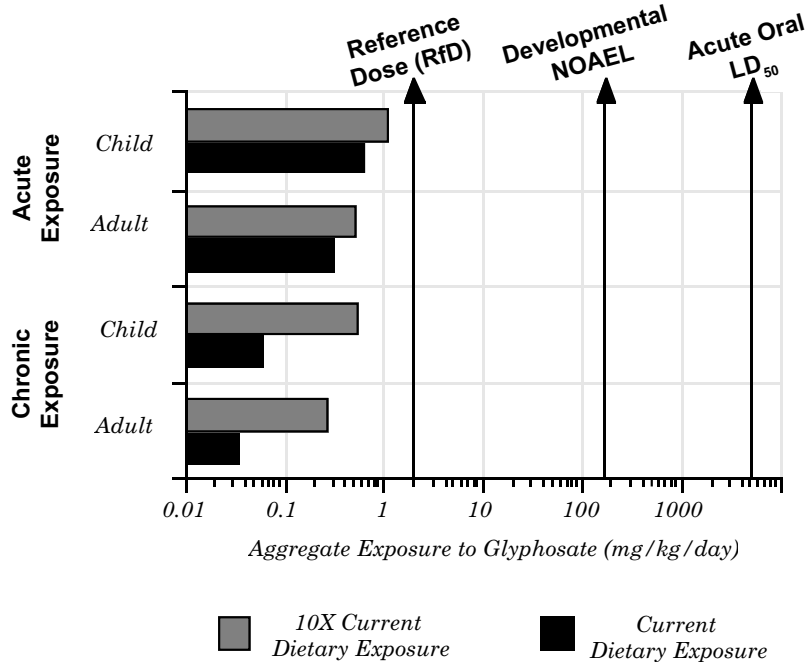
were included. Also, the exposure assessment covered the only relevant plant metabolite of glyphosate, AMPA, and the surfactant, POEA. The surfactant was assumed to be present as a residue in the same proportion that it occurred in the Roundup formulation.

I decided to modify the published aggregate assessment by assuming in the acute exposure scenario that a person would be accidentally exposed directly to the pesticide spray at a maximum rate of application (4 kg glyphosate per hectare). To make things interesting (and, arguably, more conservative), I assumed the person was naked and his or her whole body was under the spray boom.

To further spice up the exposure assessment and address concerns that increased use of glyphosate on Roundup Ready corn and beans would significantly increase pesticide residues (10), I increased dietary exposure by tenfold. The dietary exposure was also changed to reflect EPA's assumptions that all crops have glyphosate residues at the level of the tolerance. In contrast to the popular EAG claim that Monsanto requested an increase in the soybean glyphosate tolerance to accommodate its Roundup Ready technology, the tolerance was 20 ppm long before commercialization of Roundup Ready crops and remains so today (15). Also, the idea that residues in our diet would increase tenfold is kind of crazy considering that most of the increased use of glyphosate would be on crops that are largely fed to livestock before it makes its way to our tables. Given the rapid excretion of glyphosate and lack of storage in tissues (pharmacokinetics!), the possibility of exposure via residues in meat is very remote.

The maximum exposure on a bad day when a child playing next to a cornfield would be accidentally oversprayed with glyphosate was estimated to be 0.6 mg/kg (Table 3, page 9). Daily (chronic) exposures would be far less. POEA exposures would be a little higher, but nothing to worry about as most surfactants have similar toxicological properties and we use them every day at home. Note that assuming dietary exposure is tenfold higher due to an increase in glyphosate residues on RR crops raises acute aggregate exposure slightly less than twofold but chronic exposure by tenfold. This big difference is because the imaginary spray drift incident represents the largest proportion of the acute exposure.

FIGURE 1



Graphical characterization of risk from glyphosate exposure. The exposure assumes the adult works as a glyphosate applicator, and both the adult and child may be acutely exposed directly to spray drift as bystanders. The 10X dietary exposure assumes that increased use of glyphosate on RR crops will increase dietary residues. Note that all exposure bars are below the EPA RfD of 2 mg/kg/day, indicative of a reasonable certainty of no harm from glyphosate exposure.

But Is It Safe?

Now for the risk assessment finale—how do you characterize the probability that an adverse effect might occur from these estimated glyphosate exposures? At this point, the hazards of glyphosate, as represented by the NOAELs, are integrated with the estimated exposures. If exposure were substantially below the NOAELs, preferably by a factor of at least 100, then toxicologists world-

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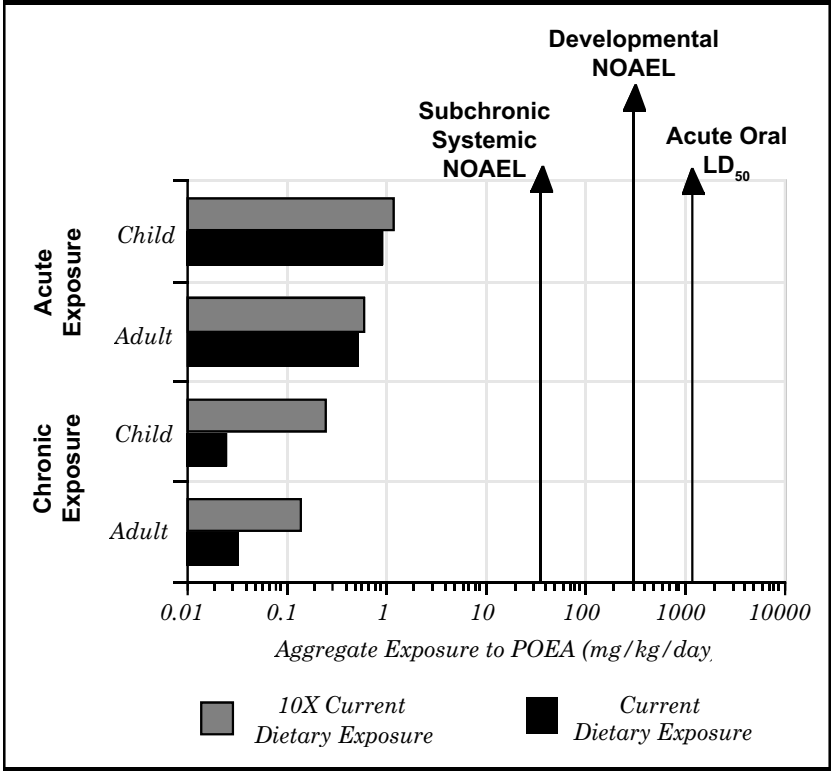
FIGURE 2



Graphical characterization of risk from exposure to the Roundup surfactant POEA. A reference dose (RfD) has not been developed for POEA, but note that most exposures are from 15 to 100 times lower than the NOAEL for the most sensitive toxicological endpoint, subchronic systemic toxicity.

wide would agree that there is a reasonable certainty of no harm from glyphosate exposure. To put it more bluntly, the stuff should be considered safe!

In Figures 1 and 2, I've integrated the hazard characterization for glyphosate and POEA with their estimated levels of exposure. Note that for glyphosate, all exposures to a subject child and adult population are comfortably below the RfD that is already 100-fold lower than the most sensitive toxicological endpoint (i.e., the one with the lowest NOAEL). No RfD has been established for POEA, but my extreme estimates of exposures are at least fifteenfold below the NOAELs.

Beware of Ad Hominem Attacks

After perusal of a number of EAG websites I concluded that glyphosate, given its incredibly low toxicity, its inability to be absorbed by the skin, its rapid elimination, and absence of any toxicologically significant long-term effects, was a particular challenge to critique. So, when you can't fight the argument on the data, it's time to pull out the ad hominem attacks. EAGs masterfully impugn the credibility of the toxicological data on chronic toxicity by suggesting to readers that industry data is all that's out there and it's not trustworthy (4). NCAP goes one step further by deriding the quality of the glyphosate data with a historical recitation about two contract companies in the late 1970s and early 1980s that were accused and convicted of falsifying data about a number of pesticides, including glyphosate. (By the way, all of the studies were redone and resubmitted for EPA review.)

The EAGs somehow forget to inform readers about the Good Laboratory Practices (GLP) standards promulgated into statutory law back in the early 1980s under the auspices of FIFRA (Federal Insecticide, Fungicide, and Rodenticide Act). Under GLP, every bit of data collected by a company doing research to support pesticide registration requirement is subject to auditing by the EPA.

Next...Ecological Concerns

Despite the plethora of data attesting to safety, I suspect attacks on the integrity of researchers (even university faculty!) will continue. In upcoming issues of *AENews*, I will risk my good reputation as I continue to examine concerns about genetically engineered herbicide tolerant plants, including their impact on non-target organisms (e.g., wildlife) and their potential to "leak genes" to other plants, thereby creating "superweeds."

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