

Review

Unintended Effects of the Intended Herbicides on Transgenic Herbicide-Resistant Crops

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Abstract

The herbicides used with crops that have been made resistant to them with transgenes are assumed to have no significant effects on these crops. Crops made resistant to glyphosate, glufosinate, dicamba, 2,4-D, mesotrione, and isoxaflutole are discussed in this paper. Most of the literature on this topic has been on glyphosate-resistant crops, as these have been the most successful of all herbicide-resistant crops. Reports of adverse effects, such as phytotoxicity symptoms, disrupted mineral nutrition, and reduced yield, caused by these herbicides on these crops are reviewed and critiqued herein. These reports are often conflicting, however, and there is no consistent evidence of any major adverse effects of these herbicides on these crops. Literature on the accumulation of residues of the intended herbicides in the parts of the plants that are used as food is also discussed. Reports of potential unintended beneficial effects, such as effects on crop pests and stimulation of crop growth and development are also critiqued.

Keywords: dicamba; glufosinate; glyphosate; herbicide-resistant crop; herbicide-tolerant crop; hormesis; isoxaflutole; mesotrione; topramezone; transgenic crops; 2,4-D



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1. Introduction

During the first few decades during which synthetic herbicides were used for weed management in crops, efforts were made to develop ‘selective’ herbicides that could be used with major crops without causing unacceptable damage to those crops. However, some excellent herbicides, such as glyphosate [1], could not be used with any crop after planting because of crop damage. Since 1995, crops have been commercialized that have been made resistant to certain herbicides through the use of transgenes for herbicide resistance (termed ‘tolerance’ by much of the biotechnology industry). These herbicide-resistant (HR) crops, especially those made glyphosate-resistant (GR), have been widely adopted where they are available because they have made weed management more effective and economical and less complicated. Transgenic GR crops are now available that are resistant to glyphosate, glufosinate, dicamba, 2,4-D, and two herbicides that inhibit hydroxyphenylpyruvate dioxygenase (HPPD), often with multiple transgenes (stacked genes) for resistance to up to four of these herbicides. These HR crops give farmers more options for dealing with the growing problem of herbicide-resistant weeds.

One might assume that there would be no significant effects on these crops caused by the herbicides to which the crops have been made resistant. However, reports of such

effects have been published. The most successful of these crops have been GR crops, and accordingly, there have been numerous studies on the effects of glyphosate on GR crops. Relatively few such studies have been published on the effects of other herbicides on crops that have been made resistant to them. For example, we are unaware of good dose–response relationship studies on the level of resistance of any of these crops other than GR crops.

Because of a fear of the unintended effects of the transgenes that impart herbicide resistance to the physiological and chemical composition of HR crops, regulatory authorities have required rigorous studies on whether or not these transgenes cause undesirable effects. Some of these studies have been published by the companies developing HR crops (e.g., [2,3]). However, few studies have come from the companies commercializing HR crops regarding the effects of herbicides used with HR crops when used at the highest recommended application rates, at the latest-allowed crop growth stage for treatment, and under a range of environmental conditions. The parameters measured are generally only related to the seed composition of the HR crop, whether or not it has been sprayed with the intended herbicide (e.g., [4,5]); although, such an industry study was performed on the forage quality of GR alfalfa plants sprayed with glyphosate [6]. Most of the studies on HR crops sprayed with their intended herbicide have been published by university and government scientists (e.g., [7,8]).

If HR crops are highly resistant to the herbicides intended to be used with them, one would expect no significant effects at the recommended application rates. Despite the importance of HR crops, there are relatively few published studies on the effects of their intended herbicides. This is probably, at least in part, because ‘no effect’ studies are often rejected by journals as “so what?” papers. We will minimize discussion of the effects of higher-than-recommended application rates of these herbicides, as HR crops are not intended to be exposed to such herbicide doses. Insufficient resistance-imparting transgene expression in some crop tissues or indirect effects on the crop through the effects of the herbicide on beneficial endophytes (e.g., nitrogen-fixing bacteria) could have unintended effects, even at recommended application rates. Examples of the effects caused by both of these mechanisms are discussed.

Reviews and books on HR crops are available (e.g., [9–13]), but none of these are focused on the effects of the accompanying herbicide on these crops. Some of what we cover in this review is mentioned in earlier reviews (e.g., [14,15]), but this review provides an update on the recent literature and further interpretation of the older literature. We also expand the topic to all commercial HR crops. However, the emphasis is on GR crops because most of the literature on this topic is on GR crops.

2. Glyphosate-Resistant Crops

Glyphosate is the most used herbicide worldwide [16,17], partly because of its use with most HR crops in North and South America, as well as Australia and other countries. Commercial GR crops include soybean, maize, canola, sugarbeet, cotton, and alfalfa. GR crops were introduced in 1996, the year after glufosinate-resistant (GluR) crops were introduced. GR crops were much more successful than GluR crops, in many cases gaining more than 90% of the crop market share where they were available. From the earliest days of their introduction, there were reports of phytotoxicity and other unintended effects of glyphosate on GR crops. These reports included effects on growth, yield, chlorophyll content, mineral nutrition, production of secondary metabolites, and crop disease. Despite such reports, field studies with numerous GR cultivars have found no effects of glyphosate on GR crop yields (e.g., [18]), the crop parameter of most interest to farmers. Previous reviews have dealt in part with the unintended effects of glyphosate on GR crops [14,15].

2.1. Phytotoxicity

We are aware of only two papers that have detailed dose–response studies of glyphosate’s effects on GR crops. Nandula et al. [19] found that the resistance factors (I_{50} of the HR plant divided by that of the isogenic plant) for glyphosate on GR soybeans and canola were both approximately 50-fold, and Hetherington et al. [20] reported an approximately 100-fold resistance factor for GR maize. We have found no such published studies regarding GR cotton, GR sugar beet, or GR alfalfa. Duke [21] proposed that the higher resistance factor for GR maize than for GR soybeans or GR canola could be due to the finding that there is very little metabolism of glyphosate to aminomethylphosphonic acid (AMPA) in maize, whereas there is considerable metabolism of glyphosate to AMPA in cultivars of soybean and the canola cultivar used in the Nandula et al. [19] paper. AMPA is mildly phytotoxic to plants [22]. At high levels of glyphosate in GR soybean, AMPA can accumulate to phytotoxic levels under some environmental conditions [23]. This apparently accounts for chlorosis (termed ‘yellow flash’, e.g., [24]) that occurs in GR soybeans treated with glyphosate under some environmental conditions or at high application rates, such as those that occur when spray patterns overlap. The first GR canola varieties, such as the one used in the Nandula et al. [19] paper, had a transgene for a glyphosate oxidoreductase (GOX) that generates AMPA, as well as a transgene for a GR 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS), the target enzyme of glyphosate [13]. High levels of AMPA were found in such varieties that were sprayed with glyphosate [25]. At present, GR canola varieties with only a GR *epsps* transgene or with only a glyphosate acyltransferase (GAT) transgene (*gat*) are available [26]. Canola is the only GR crop to use the *gat* transgene. GAT converts glyphosate to *N*-acetylglyphosate which has no phytotoxicity [27]. We cannot find any resistance factor data for commercial *gat*-containing or *epsps* alone-containing GR canola, but we would expect that these resistance factors might be higher than those of early varieties containing both *epsps* and *gox* transgenes, due to the phytotoxicity of AMPA. The *gat*-containing canola is advertised to have higher glyphosate resistance than the GR *epsps* canola varieties. Hardly any or no AMPA is produced in GR maize treated with glyphosate [21,28]. Thus, at least part of the lower resistance factor is probably due to the phytotoxicity of high AMPA levels in GR soybeans [7] and early canola varieties [25]. However, the relative phytotoxicity of any herbicide will vary with environmental conditions, growth stage, formulation additives, and other factors. Also, to determine the impact of the gene(s) causing resistance on the level of resistance, an isogenic or near isogenic variety of the crop should be used for comparison. This was the case in the two studies mentioned above, but obtaining the susceptible, isogenic lines may be difficult for other GR crops, especially for scientists unaffiliated with the company producing the GR crops. Also, care must be taken in such studies to rule out effects of formulation ingredients.

Despite the very high reported resistance factor of GR maize for glyphosate, mild phytotoxicity occurred in GR maize treated with approximately two- to nine-fold the maximum recommended application rates (up to 8.64 kg ae ha^{−1}) of glyphosate at the V4 growth stage, but there was no reduction in yield, even at the highest application rate [29]. Use of a recommended glyphosate application rate (0.8 kg ae ha^{−1} at the V5 growth stage) had no effect on yield of five GR maize cultivars in a 3-year study [30].

At present, most GR crops are made resistant to glyphosate through the use of a transgene encoding a microbial or plant GR form of EPSPS [13]. The high level of resistance of GR crops to glyphosate, especially in maize, which produces little AMPA, indicates that this is the only molecular target of glyphosate at concentrations to which plants are exposed in the field. Nevertheless, there are numerous reports of the phytotoxicity of glyphosate to GR crops, especially in the early years of their cultivation. In the case of phytotoxic effects observed with glyphosate on the reproductive tissues of GR cotton (e.g., [31,32]), the effect

was due to an incomplete expression of the EPSPS transgene for glyphosate resistance in reproductive tissues, a problem that was overcome with a better transgene promoter [33]. Whether any other reported phytotoxicity of glyphosate to GR crops is due to incomplete gene expression is unclear, but the only next generation of GR crops that claimed to have increased crop safety has been GR cotton [13].

The only well-proven cases of significant phytotoxic effects of glyphosate on GR crops at recommended application rates are those mentioned above of GR soybeans and cotton, each with a different mechanism—AMPA toxicity in soybeans and poor gene expression in cotton. Other cases are not as clear, and reexamination has often resulted in contradictory findings. Furthermore, overall yields of crops per hectare that are now about 90% GR in the USA (soybean, cotton, and maize) have generally improved since GR crops were introduced [17] and have continued to improve from 1996 to 2024 for GR maize, cotton, and soybeans (e.g., soybean data are shown in Figure 1), something that would not occur if there was significant phytotoxicity of glyphosate to these GR crops. Therefore, we will limit ourselves to reviewing only those cases that have plausible mechanisms.

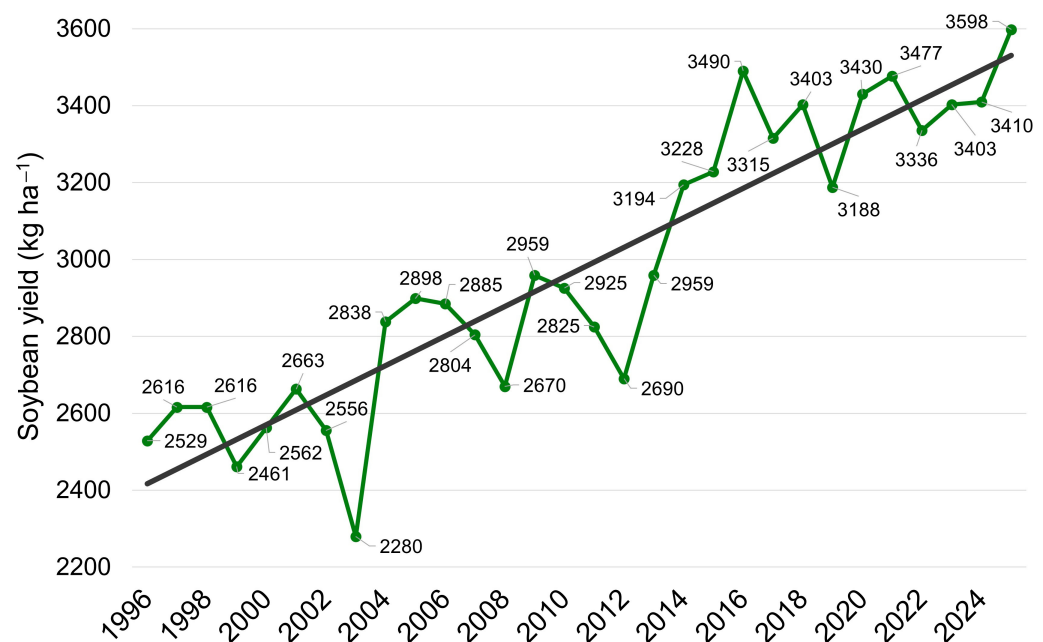


Figure 1. Yield of soybeans in the US since GR soybeans were introduced (United States Department of Agriculture, National Agricultural Statistics Service, 12 September 2025. https://www.nass.usda.gov/Charts_and_Maps/Field_Crops/soyld.php (accessed on 3 October 2025). Most of the variation reflects year-to-year weather variation.

2.2. Mineral Nutrition

There have been several claims of effects of glyphosate on the mineral nutrition of GR crops. Glyphosate chelates metal cations, especially divalent metal cations such as Fe^{2+} and Mg^{2+} [34,35]. Therefore, even after the molecular target of glyphosate was found to be EPSPS in Amrhein's lab [36], some scientists theorized that the metal cation-binding properties of glyphosate might be responsible for at least part of its phytotoxicity. For example, Duke et al. [37] found glyphosate to have strong effects on the translocation of Ca^{2+} from roots to shoots of non-GR soybean seedlings. This was concluded to be a secondary effect of another mechanism of action, but later findings showing that glyphosate caused drastic effects on the intracellular distribution of Ca^{2+} and Mg^{2+} in non-GR soybean seedling cells led these researchers to hypothesize that this effect was direct, through metal ion chelation, and could partly explain the mode of action of glyphosate [38]. This

conclusion was made highly unlikely when the level of glyphosate resistance in GR crops made resistant with only a GR *EPSPS* transgene was found to be very high.

Nevertheless, for several years, papers appeared that reported that glyphosate causes reduction in minerals that are found in the divalent form in GR soybean, particularly Fe, Mg, and Mn (e.g., [39–43]). Some argued that this effect caused susceptibility to plant pathogens (e.g., [44,45]). Such results would have been incompatible with the high level for glyphosate resistance in this crop imparted with a GR *EPSPS* transgene. More recent papers examining the effects of glyphosate on the mineral content of GR maize and soybeans at several different locations with different environmental conditions have found no effects [28,29,46–51]. Duke et al. [17] pointed out that due to the stoichiometry between the glyphosate and metal cations in the treated GR crops, glyphosate would be unlikely to bind a sufficient fraction of available metal ions in the plant to have a significant effect. Furthermore, Harris et al. [52] calculated that over 90% of the glyphosate in a GR crop's phloem after treatment would not be chelated. These considerations make it difficult to invoke chelation of metal ions by glyphosate as a factor in significantly influencing metal element content of GR crops. Nevertheless, the reason(s) for the inconsistencies between these reports remain(s) unclear. Any influence of glyphosate on the canopy of GR soybeans might indirectly influence the mineral content of the seeds, as the canopy position of the filling pods can profoundly influence mineral content [53]. Reports of effects of glyphosate on the mineral nutrition of GR crops have attenuated over the past decade.

2.3. Secondary Metabolites

Glyphosate's effects on non-GR plants at recommended application rates are assumed to be due entirely to the inhibition of EPSPS, an enzyme of the shikimate pathway. In addition to producing aromatic amino acids (phenylalanine, tyrosine, and tryptophan), this pathway produces many phenolic secondary compounds. Inhibition of this enzyme results in rapid accumulation of shikimic acid which is routinely used as a very sensitive indicator of the effects of glyphosate on plants (e.g., [54]). Relatively few studies have determined the effect of glyphosate on shikimate levels in GR crops. Pline et al. [32] found that 5 mM glyphosate did not cause shikimate accumulation in the leaves of an early GR cotton variety. Although Lappé et al. [55] reported reduced estrogenic isoflavone (a shikimate pathway product) content of GR soybean seeds in comparison to non-GR soybeans, they did not determine whether those effects were caused by glyphosate. Their study was invalid because their GR and non-GR cultivars were not isogenic. In a properly performed similar study, no effects of the EPSPS resistance gene (*C4 EPSPS*) were found on isoflavone content [2]. Taylor et al. [4], Duke et al. [7], and Bohm et al. [56,57] found no effects of recommended rates of glyphosate on the isoflavone content of GR soybeans.

2.4. Crop Disease

Part of the reason that some have claimed that glyphosate application to GR crops results in more crop disease is that blockage of the shikimate pathway by glyphosate inhibition of EPSPS results in reductions in some phytoalexins and lignin that are required for defense of plants from various pathogens [44]. This clearly occurs in non-GR plants [18], and increased pathogen damage to weeds sprayed with glyphosate greatly enhances the herbicidal efficacy of glyphosate in many cases (e.g., [58]). However, we are unaware of reductions in phenolic compounds involved in disease resistance by glyphosate in GR crops. Furthermore, studies have shown no increases in disease in glyphosate-treated GR maize [48], GR soybeans [59], GR sugar beet [60], GR cotton [61], and non-commercialized GR wheat [62]. Powell and Swanton [63] concluded that there is no evidence that glyphosate use in GR crops increases *Fusarium* spp. diseases in those crops. Involvement of plant

diseases in the death of glyphosate-treated weeds could increase the level of inoculum of diseases to which crops are susceptible, causing a secondary effect of the herbicide on crop disease [64].

Some herbicides are also toxic to plant pathogens by the same modes of action as those of their herbicide modes [65]. Glyphosate is one of these, as it is toxic to both bacteria and fungi that have a glyphosate-sensitive EPSPS [18]. Thus, glyphosate application to GR crops can be effective in crop disease control, provided the microbe is glyphosate-sensitive and the application timing is appropriate for fungicide use. For example, glyphosate is effective against rust diseases in GR alfalfa [66], soybeans [67–70], and wheat [67,71] (Figure 2). *Rhizoctonia solani* can be controlled by glyphosate in GR cotton [72]. Applied at the right time, glyphosate can reduce downy mildew [73] infections in GR canola and charcoal rot in GR soybeans [74]. Although there is no commercial GR rice, glyphosate has been shown to control rice blast as well as a commercial fungicide in GR rice [75].

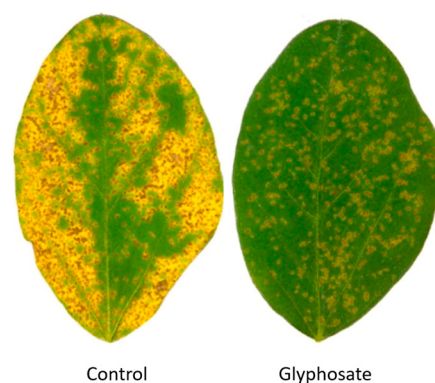


Figure 2. Effects of glyphosate ($0.96 \text{ kg ae ha}^{-1}$) applied 2 days before the inoculation of GR soybeans with soybean rust (*Phakospora pachyrhizi*). The adaxial leaf surfaces are shown 15 days after inoculation. Adapted from Einhardt et al. [69] with permission.

Not all fungi are significantly affected by recommended glyphosate application rates in GR crops. A 4-year field study of the effects of glyphosate on aflatoxin levels produced by *Aspergillus flavus* in harvested GR maize seed found no effects [76]. In a 5-year study at three locations, there were no differences between levels of the fumonisins (highly toxic mycotoxins) produced by *Fusarium* spp. in harvested maize grain between a non-GR and a GR cultivar [77]. The authors gave no information about the glyphosate treatments used with the GR cultivar, and the variation in the content of fumonisins was large. Similarly, in a 3-year study, Bruns and Abbas [30] found no effect of glyphosate applied at $0.8 \text{ kg ae ha}^{-1}$ on either the aflatoxin or fumonisin content of the grain of five GR maize cultivars compared to levels in a GluR and an atrazine-tolerant cultivar. A lack of effect is not surprising, as extremely little of the glyphosate taken up by GR maize is translocated to the seed [50], where *A. flavus* and *Fusarium* spp. grow and produce aflatoxin and fumonisin, respectively.

Glyphosate apparently increases the virulence of white leaf spot disease (*Neopseudocercoporella capsellae*) in GR canola by increasing blastospore formation by the microbe [78,79]. Whether inhibition of the EPSPS of the pathogen causes such an effect is unknown. Findings indicate that the effect is greatest when glyphosate is applied before inoculation, which suggests that the effect is caused by an unknown effect of glyphosate on the GR crop, rather than direct effects on the microbe.

2.5. Nodulation and Nitrogen Fixation

Both soybeans and alfalfa benefit from nitrogen-fixing bacteria that reside in the root nodules of these crops. Four years before GR soybeans were commercialized, Moorman et al. [80] found *Bradyrhizobium japonicum* to be sensitive to glyphosate, and they

speculated that this could be a problem for GR soybeans, as glyphosate is preferentially translocated to metabolic sinks such as nitrogen-fixing nodules. They found glyphosate to strongly stimulate shikimate production by the microbe, indicating that it acts on this bacterium through inhibition of EPSPS. After GR soybeans became available, research showed that glyphosate inhibits nodulation (reductions in nodule number and biomass) in GR soybeans at a high application rate ($2.24 \text{ kg ae ha}^{-1}$) but not at lower rates [81]. Similarly, King et al. [82] found that two applications of $1.68 \text{ kg ae ha}^{-1}$ of glyphosate inhibited nitrogen fixation, but the effect varied with location and cultivar. Reddy and Zablotowicz [83] reported that a recommended glyphosate application rate (0.84 kg ha^{-1}) in GR soybeans reduced nodule biomass by about 25%; however, the crop recovered from this effect. Bohm et al. [56] found no effect of two applications of 0.96 kg ha^{-1} of glyphosate on the nodule weight, nitrogen fixation, or yield of a GR cultivar in Brazil. In an extensive study over three years, Zablotowicz and Reddy [84] found variable effects of glyphosate on nitrogen fixation in GR soybean. They concluded that at recommended use rates there was only a slight effect on nitrogen fixation, but that there was a significant effect at higher use rates, particularly when soil moisture stress occurred after application. Yield was only affected in one year out of three at the high application rates. There is thus a clearly established effect of high application rates of glyphosate in GR soybeans, so this effect should be considered when using such rates. Nevertheless, the significantly increased yields of U.S. soybeans since 1996 (Figure 1), after which 90% or more were GR soybeans, indicates that overall effects of glyphosate on nodulation and nitrogen fixation have not significantly affected yields.

2.6. Effects on the Crop Through Effects on Insects

Glyphosate has been reported to have both adverse and stimulatory (hormesis) effects on insects that are pests of GR crops. In a laboratory experiment, Quesada et al. [85] found formulated glyphosate to be toxic to the lepidopteran insects *Spodoptera frugiperda* and *Chrysodeixias includens*, which are pests of maize and soybeans, when applied at the recommended application rate. A 20-fold dilution of the application rate was stimulatory to the larvae of *C. includens*. Their study could not differentiate between the effects of glyphosate and the formulation ingredients of the commercially formulated glyphosate that they used. There are no known molecular targets of glyphosate in insects at the concentrations used in weed management, so how such effects could occur is unknown. If such effects occur under field conditions, the toxicity of glyphosate to insect pests could be an additional benefit to GR crops sprayed with recommended rates of glyphosate.

2.7. Hormesis

Hormesis is the stimulatory effect of a subtoxic dose of a toxin. Stimulatory effects can be either beneficial or detrimental. Herbicide hormesis is well documented, and glyphosate-caused hormesis is the most frequently reported, more reproduceable, and the most pronounced example of herbicide hormesis [86]. In the field, hormetic effects are difficult to reproduce, because the hormetic dose and the hormetic amplitude are strongly influenced by environmental and plant development parameters [87]. That hormesis is an adaptive response to mild stress that is manifested as a form of overcompensation is generally accepted [88], but, with glyphosate, this may only be part of the explanation [86].

With a resistance factor for GR crops of 50- to 100-fold, the concentration or dose of glyphosate that would cause hormesis in a GR crop is unclear, as is whether the exceptional hormesis in non-GR plants is related to glyphosate's unique mode of action (EPSPS inhibition) [86]. We do not know if the phytotoxicity of high doses of glyphosate on GR crops is related to an effect on EPSPS or is due to a different mechanism. In GR soybeans and the

first commercial GR canola varieties, the phytotoxicity at high glyphosate doses is probably mostly due to AMPA created from glyphosate in the crop, perhaps decreasing the potential strong hormetic effect of a stress-inducing dose of glyphosate. Nevertheless, foliar, seed, and combined foliar and seed glyphosate treatments of the same rates increased yields of some GR soybean cultivars at some geographic locations [89] (Figure 3). Results of this extensive study of hormesis in several GR soybean varieties at different locations suggest that glyphosate hormesis may sometimes be an additional benefit of glyphosate in GR crops, other than the benefit of weed management. A patent exists that claims that yield increases can be obtained through optimal doses of glyphosate used on GR crops [90]. This topic needs further study to determine whether hormesis is sometimes occurring in the field with GR crops and whether hormesis in GR crops is due to effects on EPSPS.

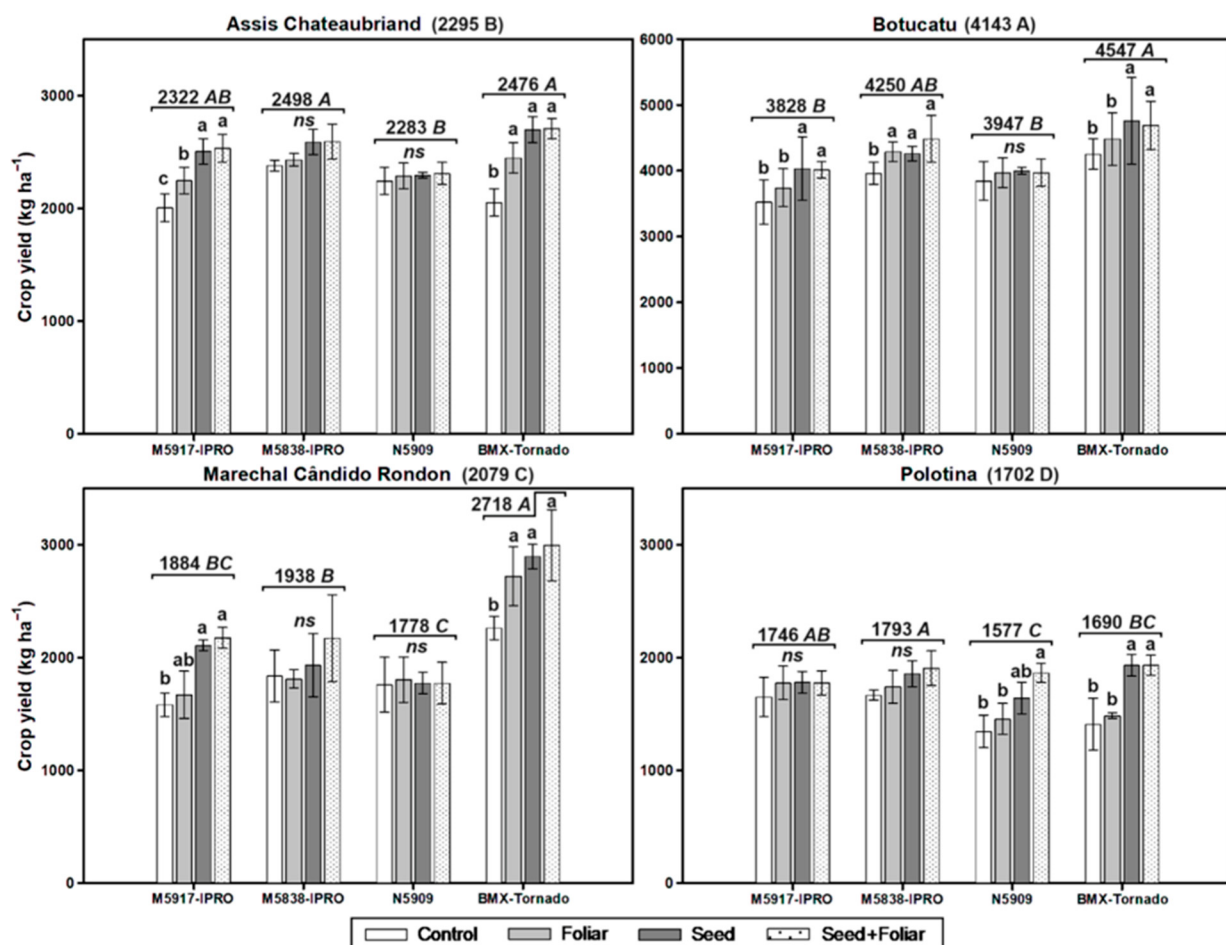


Figure 3. Effects of foliar (90 g ae h⁻¹), seed (equivalent of 90 g ae h⁻¹), and foliar plus seed (180 g ha⁻¹) treatments with glyphosate on the yield of four different GR soybean cultivars at four different locations in Brazil. The seeds were coated with a commercial glyphosate formulation along with an adjuvant to provide the equivalent of 90 g ae ha⁻¹ of glyphosate at the seeding rates used. Capital letters between locations, italic capital letters between cultivars, and lowercase letters between treatments within a cultivar did not differ from each other using the Tukey test ($p \leq 0.05$). ns—not significant. From Krenchinski et al. [89] with permission.

2.8. Glyphosate Accumulation in Harvested Food Products

Accumulation of the herbicide(s) used with a HR crop in the edible parts of the crop is an unintended effect of using the herbicide(s) for weed management. Because glyphosate is highly mobile in the phloem, it readily transports to metabolic sinks, such as developing seeds (e.g., soybeans) and roots (e.g., sugarbeets) from sprayed leaves. In

glyphosate-sensitive plants, phytotoxicity will limit translocation, but this will not occur in GR crops, making movement of glyphosate to harvested seeds or roots more likely. Numerous studies have shown significant accumulation of both glyphosate and AMPA in the seeds of GR soybeans sprayed with glyphosate in the field (e.g., [7,56]), but only trace amounts of glyphosate and AMPA have been found in GR maize in field studies (e.g., [49,50]). In glyphosate-treated GR soybeans, the levels of AMPA found in the seed at harvest are generally higher than those of glyphosate (e.g., [7]). Whether the AMPA is from the degradation of glyphosate in the seed or transported to the seed from degradation elsewhere in the plant or even the soil is unknown. The seeds of soybeans sprayed with the recommended glyphosate levels for weed control appear to have the highest reported levels of glyphosate of any food from a GR crop. The allowable content (maximum residue level, MRL; called tolerances in the USA) varies between countries. The glyphosate content of harvested soybean samples from agricultural fields in U.S. state of Iowa in 2009 were all below (averaging less than 5 mg kg^{-1}) the United States MRL for glyphosate in soybeans (40 mg kg^{-1}), whereas levels found in Argentinian soybeans grown in 2013 exceeded the 20 mg kg^{-1} MRL of Europe in three samples of eleven [91]. This unintended issue with GR soybeans has raised concerns, especially because of the many claims of adverse effects of glyphosate on human health (e.g., [92]) despite evidence that concentrations of glyphosate in food are generally below MRL limits (e.g., [93,94]) and that human consumption of glyphosate is far below levels considered unsafe by regulatory agencies [95].

Information on the glyphosate content of food products from other GR crops is more limited. Because glyphosate is a highly water-soluble compound, it should not be found in the oil from GR canola or GR cotton, the main food products from these crops. However, cotton seed meal is used for animal feed, and we have found no publications on the glyphosate content of this seed component in GR cotton production. Sucrose, as a very water-soluble molecule in GR sugar beets, might be expected to be contaminated with glyphosate. However, Barker and Dayan [96] reported that most of the glyphosate translocated to the roots is exuded from the roots into the soil before harvest and that the remaining glyphosate is eliminated by extensive processing procedures to produce pure crystalline sucrose. Glyphosate has long been known to be exuded from roots of foliar-treated plants (reviewed by Ghanizadeh and Harrington [97]), but what fraction of the glyphosate intercepted by GR crops reaches the soil by this mechanism is unknown. Glyphosate levels in GR alfalfa treated with glyphosate for weed management were 0.25 mg kg^{-1} [98]. In this study, there was no difference between feed intake, milk composition, and milk production of dairy cattle fed this GR alfalfa compared to those fed non-GR alfalfa not treated with glyphosate.

3. Glufosinate-Resistant Crops

Glufosinate is a chemically synthesized racemic mixture of D- and L-phosphinothricin. Only L-phosphinothricin, a natural compound from *Streptomyces hygroscopicus* and *S. viridochromogenes*, is phytotoxic. Glufosinate kills weeds by inhibition of the enzyme glutamine synthetase (GS), an enzyme involved in nitrogen assimilation and the second most abundant protein in green plant tissues [99]. Glufosinate is a non-selective herbicide used as a foliar spray like glyphosate, but unlike glyphosate, it is poorly translocated in plant tissues [100]. Glufosinate-resistant (GluR) canola, commercialized in 1995, was the first transgenic HR crop. Commercialized GluR crops (maize, soybeans, canola, and cotton) have been made resistant to glufosinate through the use of either a transgene from *S. hygroscopicus* (the *bar* gene) or *S. viridochromogenes* (the *pat* gene) that encodes an enzyme that detoxifies glufosinate by acylating it to a herbicidally inactive molecule [10,101]. The *pat* and *bar* genes have also been used as selectable markers in the production of some

transgenic crops other than GluR crops (e.g., crops with transgenes encoding peptide toxins that kill insects). Knowing that such crops have some level of glufosinate resistance, some farmers have used glufosinate on these crops for weed management, but the level of glufosinate resistance is generally not as high as that of crops sold as GluR crops [102,103]. Krenchinski et al. [104] found the level of *pat* marker gene expression positively correlated with the level of glufosinate resistance in six commercial maize hybrids not sold as GluR maize. Thus, farmers can risk crop damage from glufosinate through this practice. Because such crops are not sold as GluR crops, we will not consider them further in this review.

We have found no published dose–response studies comparing the effects of glufosinate on commercial GluR and isogenic, non-GluR crops. However, non-commercial wheat with the *bar* transgene was reported to have a 15-fold higher ED₅₀ value for glufosinate than a non-GluR line derived from the same cultivar [105]. This resistance level is lower than those reported for GR crops, but commercial GluR crops may have higher levels of resistance. Unlike GR crops, there have been relatively few reports of the unintended effects of glufosinate on GluR crops. GluR rice was reported to be injured by both early- and late-season application of glufosinate [106], but GluR rice has not been commercialized. Mild phytotoxicity occurred in GluR maize treated with approximately 2.5- to 10-fold the maximum recommended application rate (up to 6 kg ae ha^{−1}) of glufosinate at the V4 growth stage, but there was no reduction in yield, even at the highest application rate [29]. In a 3-year field study, Bruns and Abbas [30] found no effects of 3.65 kg ha^{−1} of glufosinate ammonium on the yield of a GluR maize cultivar. Reddy et al. [107] reported no effects of the recommended application rate of glufosinate on the nitrogen nutrition, growth, and yield of GluR soybeans, but they claimed there were minor effects on the seed protein and oil of the harvested soybeans. However, we question the reliability of some of their analytical methods, as extraction, isolation, and chemical analysis of the chemical components were not used. Mundt et al. [108] found no effects of the recommended glufosinate application rates applied at the V4 growth stage on growth, chlorophyll content, and the nodule number and size of two commercial GluR soybean varieties. However, higher rates (above 1.24 kg ha^{−1}) caused growth reductions. In a two growing season study, Krenchinski et al. [109] found no effects of glufosinate on GluR maize that was also GR, but in both years found the combined use of glyphosate and glufosinate as well as atrazine (to which maize is naturally tolerant) reduced yield. There was no effect of using glyphosate with glufosinate, glyphosate with atrazine, or glufosinate with atrazine. There were no effects of any herbicide regime on the germination or vigor of the harvested seed. Albrecht et al. [110] published similar results. In neither study could the yield effects of the three herbicides used together be attributed to any one of the three herbicides. Regarding growth effects and yield, the preponderance of evidence indicates no adverse effects of glufosinate on GluR crops when used at recommended application rates.

Fungi also assimilate ammonia with GS, and this fungal enzyme is also inhibited by glufosinate [65]. Thus, glufosinate has the potential to add to its value as a pesticide in GluR crops by acting as a fungicide against crop fungal pathogens. Glufosinate is fungicidal to a number of plant pathogens at concentrations similar to the application rates for weeds [111]. Although there is no commercial GluR rice, bialaphos (a tripeptide that is converted to the active enantiomer of glufosinate in planta) and glufosinate were found to act as fungicides in experimental GluR rice [112–114]. Similarly, glufosinate was found to be an effective fungicide on plant disease in non-commercial GluR bent grasses (*Agrostis* spp.) [115]. Thus, glufosinate could be expected to prevent or ameliorate fungal infections of the foliar tissues of GluR crops if applied at the right time. Unfortunately, there are no published data that can be used to determine whether this is occurring in the field with glufosinate-treated GluR crops.

Glufosinate inhibits production of the fungal mycotoxin aflatoxin by *Aspergillus flavus* [116]. In a 3-year field study, Bruns and Abbas [117] reported no effects of glufosinate use on the levels of aflatoxin or fumonisin produced in GluR maize inoculated with *Aspergillus flavus*. The fumonisins were produced by naturally occurring *Fusarium verticilloides*. In another 3-year study, Bruns and Abbas [30] reported no effect of the recommended glufosinate application on aflatoxin or fumonisin contamination from natural fungal infestations of GluR maize. Because glufosinate is poorly translocated [99], the concentrations of glufosinate accumulating in the seed would be expected to be very low (below fungitoxic concentrations), compared to those in the leaves after spraying before seeds develop. Considering the early work on the fungicidal effects of glufosinate on foliar fungal diseases of GluR rice and bentgrasses, more research should be conducted on the effects of recommended rates of glufosinate on the foliar diseases of commercial GluR crops. Some potential detrimental influences of glufosinate are its fungitoxic effects on the beneficial fungi in GluR crops. De Novais et al. [118] found glufosinate to strongly inhibit the growth and development of the arbuscular mycorrhizal fungus *Funneliformis mosseae*.

Hormesis also occurs with glufosinate [119]. A patent exists that claims that yield increases can be obtained with optimal doses of glufosinate used on GluR crops [120], but this has not been confirmed by peer-reviewed research publications. Like glyphosate effects on GR crops, glufosinate may sometimes cause hormesis in GluR crops, depending on the application rate, crop developmental stage when sprayed, and environmental conditions. Garcia et al. [121] reported that glufosinate stimulated the longitudinal growth of shoot and roots and dry weight of the roots of seedlings of a soybean cultivar made resistant to glufosinate, glyphosate, and 2,4-D.

4. Dicamba-Resistant Crops

Dicamba has herbicidal effects on dicotyledonous plants by interfering with the functions of auxin in the regulation of growth and development [122]. Soybeans and cotton have been made resistant to dicamba through the use of a transgene from a microbe that encodes an enzyme that acts as a dicamba monooxygenase, degrading dicamba to an inactive molecule. Dicamba-resistant (DR) cotton and soybeans were made available in the USA in 2016 and 2017, respectively. Dicamba is a highly volatile molecule, contributing to problems with the aerial movement of sprayed dicamba to non-target crops, on which it has caused considerable crop damage [123]. Although dicamba and 2,4-D have the same mode of action [122], DR crops are not resistant to 2,4-D [124]. There is considerable literature on the effects of dicamba on non-DR cotton [125] and soybeans [126], but this review only deals with the effects on the two commercialized DR crops.

Compared to GR crops, relatively little has been published on the effect of the intended herbicide on commercialized DR crops. For example, we have found no published dose-response studies on the effect of dicamba on any GR crop. The company that introduced DR soybeans showed that the resistance transgene has no compositional effects on harvested soybean seeds [3], but they did not publish studies on whether the herbicide would have any effects on DR soybeans at the recommended use rates or higher. Soltani et al. [127] reported transient symptoms of crop injury in soybeans that were both GR and DR when sprayed with recommended application rates of both herbicides and a 6% reduction in yield at a higher (2.4 kg ha⁻¹ glyphosate/1.2 kg ha⁻¹ dicamba)-than-recommended rate applied at the V4/V5 growth stage. Whether the effect was due to glyphosate, dicamba, or both was not determined. Dicamba has been found to accumulate to levels below the MRL (10 mg kg⁻¹) in the seeds of non-DR soybeans when sprayed with sub-lethal rates of dicamba during the pod-filling stage [128,129]. The monooxygenase that degrades dicamba apparently prevents dicamba from accumulating in the seeds of DR soybean.

Some have speculated that dicamba might cause hormesis in non-DR plants [122], but Kniss [130], Castner et al. [131], and Milosevic et al. [132] found no evidence of hormesis at ultralow doses of dicamba in non-DR soybeans. Sperry et al. [133] reported a hormetic effect of dicamba on the shoot height of non-DR soybeans treated with the lowest dose tested ($0.002 \text{ g ae ha}^{-1}$). Because there is no dose–response curve published for dicamba used on DR soybeans, the dose required for such an effect, if it occurs, is unknown. Other potential effects of dicamba on DR crops have not been reported. For example, dicamba can have direct adverse effects on some beneficial and non-beneficial insects [134,135]. Whether the recommended application rates of dicamba directly influence any insect pests of DR soybeans is unknown. We are unaware of any studies examining the influence of dicamba on crop diseases in DR crops. However, Kataria and Gisi [136] reported dicamba to be fungitoxic to *Rhizoctonia crealis* on wheat, and de Novais et al. [118] found dicamba to be fungitoxic to the arbuscular mycorrhizal fungus *Funnelformis mosseae*. Further studies of possible beneficial and harmful effects of dicamba on the microbial pathogens and beneficial microbes, respectively, of DR crops are needed.

5. 2,4-D-Resistant Crops

Like dicamba, 2,4-D has herbicidal effects on dicotyledonous plants by interfering with the function of auxin in the regulation of growth and development [122]. Commercial crops have been made resistant to 2,4-D through the use of transgenes from a microbe that encodes aryloxyalkanoate dioxygenases (AAD) that degrade 2,4-D into two non-phytotoxic molecules, glyoxylate and 2,4-dichlorophenol [137]. The AAD encoded by the transgene used for 2,4-D resistance also degrades the grass-active aryloxyalkanoate herbicide quizalofop through the same mechanism [138]. Quizalofop-resistant maize with this gene is now sold, but there are no publications on any unintended effects of this herbicide on this crop, so we will not discuss it. 2,4-dichlorophenol is rapidly conjugated with glucose [139]. 2,4-D-resistant (2,4-DR) soybeans and maize became available to farmers in 2016, and 2,4-DR cotton was commercialized in 2019 in the USA. Volatility issues with 2,4-D were solved by the introduction of the choline salt of 2,4-D, but, like dicamba, some crops are very sensitive to 2,4-D, and the use of 2,4-D in 2,4-DR crops has resulted in a drift problem with sensitive crops [122]. As a result of this problem, most of the literature on the effects of 2,4-D on soybean, cotton, and maize has been on its effects on 2,4-D-sensitive crop cultivars, which is not the topic of this review. Also, works on non-commercialized 2,4-DR crops are not discussed because the level of resistance of these crops may not have been sufficient for commercialization. For example, Charles et al. [140] found a transformation of cotton with a 2,4-D detoxification gene to provide some level of protection from 2,4-D drift, but not enough to use 2,4-D for weed management in cotton.

Hormesis caused by 2,4-D has been reported in both non-2,4-DR and 2,4-DR crops. In a greenhouse study, Marques et al. [141] reported hormetic effects of 2,4-D on non-2,4-DR cotton that were dependent on the phenological stage when treated. Garcia et al. [121] reported that the choline salt of 2,4-D stimulated the longitudinal growth of shoots and roots and the dry weight of the roots of seedlings of a HR soybean variety made resistant to glufosinate, glyphosate, and 2,4-D.

There are few data about how much 2,4-D or its degradation product 2,4-dichlorophenol has been found in the harvested seeds of 2,4DR crops. No 2,4-D has been detected in most grains of 2,4-DR maize treated with 2,4-D, and no glucosylated 2,4-dichlorophenol has been found in any samples [139]. However, both 2,4-D and 2,4-dichlorophenol have been found at levels up to 3 and 3.9 mg kg^{-1} in forage from the crop. Concern has been expressed regarding 2,4-dichlorophenol in these crops because of the general cytotoxicity of this compound [142].

6. HPPD Inhibitor-Resistant Crops

Hydroxyphenylpyruvate dioxygenase (HPPD)-inhibiting herbicides comprise the last major herbicide mode-of-action category introduced in this paper [143]. Inhibition of HPPD blocks the synthesis of plastoquinone, which is required for both electron transport of photosystem II and the functioning of phytoene desaturase in carotenoid synthesis. Transgenic soybeans sold by two different companies are resistant to two different HPPD inhibitor herbicides, mesotrione and isoxaflutole. These cultivars were introduced in 2019 and 2020, respectively. Cotton varieties resistant to the HPPD inhibitors isoxaflutole and topramezone (stacked with genes for resistance to glyphosate, glufosinate, and dicamba) were commercialized in 2024. These HPPD herbicide-resistant (HPPDR) crops contain transgenes encoding mutant forms of HPPD that are resistant to the HPPD herbicides used with the specific transformant cultivar. A paper on crop injury was published on experiments performed in 2012 and 2013 with a HPPDR soybean cultivar [144]. Some minor crop injury symptoms occurred from both isoxaflutole and mesotrione applied in particular ways and at certain growth stages. We have not found a similar paper using commercialized HPPDR soybean cultivars. Minor injury to HPPDR cotton was reported with both isoxaflutole and topramezone [145].

As discussed by Duke et al. [65], some HPPD inhibitors are fungitoxic, but no reports of effects of commercial HPPD inhibitor herbicides on fungal diseases of crops have been reported in field situations. This may be because no one has looked for such an effect.

7. Summary

Crop injury with the herbicides intended for specific HR crops when the herbicide is used at the recommended application rates and at the required crop developmental stage is, at most, minor and usually nil. Early reports of significant adverse effects (e.g., disruption of mineral nutrition of GR crops by glyphosate) of the intended herbicides of HR crops have largely been discounted. There is some evidence that recommended application rates may sometimes be hormetic, enhancing crop yield, although this possibility needs further study. However, as with non-HR crops, the predictable use of herbicide hormesis to enhance crop productivity has not been successful because of the complex interplay of environmental and plant development parameters that influence this process. Thus, such a benefit may be only an infrequent benefit. Another possible unintended beneficial effect is through the antimicrobial effects of the intended herbicides on plant pathogens in HR crops. This has been clearly shown in glyphosate used on GR crops. This benefit probably occurs rarely in field situations, because the needed application timing for weed control and for pathogen management would rarely coincide. Still, little has been done to determine whether this phenomenon is significant in the field. There are potentially negative effects of the herbicides used on HR crops on beneficial microbes associated with these crops, such as toxic effects on nitrogen-fixing bacteria and mycorrhizal fungi. Inhibition of nitrogen fixation by glyphosate translocated to nodules of GR soybeans is a minor problem, but more research is needed to determine if such problems exist with other HR crops and their herbicides. Because the yields of HR crops, including soybeans, are rarely affected by the recommended application rates, such effects, if any, are expected to be minor. Finally, little has been done to look for unintended effects of the use of multiple herbicides on HR crops made resistant to more than one herbicide. With some transgenic HR crops now having resistance to as many as four herbicides with four different modes of action, the potential for unintended interactions has grown. There is potential for both antagonistic and synergistic interactions of these herbicides on the crops made resistant to them, providing many potential avenues for future research.

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Abbreviations

The following abbreviations are used in this manuscript:

HR	herbicide-resistant
GR	glyphosate-resistant
HPPD	the enzyme hydroxyphenylpyruvate dioxygenase
GluR	glufosinate-resistant
AMPA	aminomethylphosphonic acid
GOX	the enzyme glyphosate oxidoreductase
EPSPS	the enzyme 5-enolpyruvylshikimate-3-phosphate synthase
MRL	maximum residue level
GS	glutamine synthetase
DR	dicamba-resistant
2,4-DR	2,4-D-resistant
HPPDR	HPPD herbicide-resistant

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