Relative Toxicity of Herbicide Use in the United States 1990 to 2015

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Abstract. Herbicide use is among the most criticized aspects of modern farming operations, especially in response to widespread adoption of genetically-engineered (GE) herbicide-resistant crops. Many previous analyses of herbicide use have relied on flawed metrics in an attempt to evaluate trends in herbicide intensity and toxicity. Here, it is shown that herbicide use intensity has increased over the last 25 years in corn, cotton, rice, and wheat. Although GE glyphosate-resistant crops have been previously blamed for increasing herbicide use, herbicide use increased more rapidly in the non-GE crops rice and wheat. Even as herbicide use has increased, the chronic toxicity hazard associated with herbicide use decreased in 3 out of 6 crops, while acute toxicity hazard decreased in 5 out of 6 crops. In GE glyphosate-resistant crops, glyphosate accounted for 26% of corn, 43% of soybean, and 45% of cotton herbicide applications. However, due to it's relatively low chronic toxicity, glyphosate contributed only 0.1%, 0.3%, and 3.5% of the chronic toxicity hazard in these same crops, respectively.

1 Introduction

Herbicides are a powerful weed control tool for many farming operations around the world. In the United States, herbicides accounted for 65% of all pesticide expenditures, costing farmers roughly $5.1 billion in 2007 (Nehring 2012). A perceived over-reliance on herbicides for weed control has sparked much debate on how to best incorporate herbicides into sustainable weed management systems (Benbrook 2012; Harker et al. 2012; Mortensen et al. 2012). Robert Zimdahl once wrote: "Whether one likes [herbicides] or deplores them, they cannot be ignored. To ignore them is to be unaware of the opportunities and problems of modern weed management." (Zimdahl 2013) Certainly, herbicides should not be ignored. Even many of the most ardent critics of pesticides recognize their importance. If we are to have an informed discussion on the future of herbicide use, it is important to understand how herbicides are currently being used. Herbicide use trends of the past can help inform weed management decisions in the future. This analysis will present herbicide use changes for 6 major crops grown in the United States over the last 25 years.

A variety of risk assessment methods can be used to compare herbicide programs, including the risk cup method used by U.S. EPA and other regulatory bodies. Regulatory agencies typically consider a wide variety of environmental and human health endpoints in their risk analysis processes. Risk analysis is complex even when considering only a single active ingredient since multiple endpoints must be considered (applicator health, aquatic organisms, birds, insects, etc.). Risk analysis becomes exponentially more complex when looking at multiple herbicides used across multiple crops. Several previous researchers have attempted to quantify overall pesticide impacts using simplified measures; however, the two most commonly used metrics (weight of herbicides applied and the environmental impact quotient) lead to misleading or incorect conclusions.

The total amount of herbicide applied per unit area has been reported in several recent publications (Benbrook et al. 2012; Perry et al. 2016). However, when a variety of different pesticides are applied, each with different use rates and toxicity profiles, simply reporting the weight of pesticide applied is dubious at best, and misleading at worst. A recent National Academy of Sciences report recommended that "Researchers should be discouraged from publishing data that simply compares total kilograms of herbicide used per hectare per year because such data can mislead readers." (NAS 2016, pg 87) Heeding this recommendation, total herbicide applied in kg of active ingredient per hectare will not be discussed in this report. In addition to being misleading from a risk analysis perspective, focusing exclusively on the weight of active ingredient

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applied per unit area is unhelpful in a practical sense. Herbicide use rates range from grams to kilograms per hectare and depend on many factors, including the effectiveness of the active ingredient and the environment where it is applied. A large increase in the weight of herbicide applied could simply be due to a switch from a herbicide which is active at low doses to a less bioactive herbicide. Likewise, a reduction in the total weight of herbicide applied may not actually be indicative of reduced herbicide use, as a single herbicide may be replaced by many different herbicides with lower use rates, and could have greater risk to applicators and the environment even though the total amount of active ingredient has been reduced.

The environmental impact quotient (EIQ) developed by Kovach et al. (1992) is another common metric, and has been used regularly to provide an overall assessment of risk from various herbicide programs (for example, Beckie et al. 2014; Brookes and Barfoot 2012; Green 2012; Perry et al. 2016). The EIQ combines risk factors for both applicator toxicity and environmental health, and thus is perceived as a simple tool for comparing herbicide programs. However, the EIQ is a poor indicator of risk, especially for herbicides (Dushoff et al. 1994; Kniss and Coburn 2015). Due to the way toxicity data is scaled in calculation of the EIQ, it readily leads to nonsensical conclusions. For example, according to the EIQ, the water used to dilute and apply pesticides will nearly always have a greater negative environmental impact than the pesticide itself (Dushoff et al. 1994; Kniss 2015). Additionally, a single proxy for exposure (application timing) can explain over 25% of the variability in herbicide EIQ, even though application timing has no consistent effect on actual risk (Kniss and Coburn 2015).

Acute herbicide toxicity is relatively simple to quantify and interpret, since the endpoint of interest in acute toxicity testing is mortality. To put it bluntly, it is simple to determine whether a rat is dead or alive. The dose of a herbicide resulting in death of 50% of test animals (LD_{50}) is a standard measure of acute toxicity, and is required as part of a standard set of pesticide safety studies to obtain regulatory approval. Chronic toxicity is much more difficult to quantify and standardize, as the endpoint of interest can vary widely; liver deformations, cancers, reduced body weight, or any other departure from a healthy test population can indicate chronic toxicity issues. Chronic studies also have greater variation in study design, test species, duration, and endpoints measured, adding to the complexity. When making pesticide registration decisions, a variety of chronic studies conducted on a variety of test organisms are evaluated in an attempt to determine the most relevant endpoints and to set residue tolerances, acceptable use rates, and acceptable daily intakes. This makes it somewhat difficult to make comparisons between herbicides with respect to chronic toxicity comparisons. Of the chronic toxicity data that is readily available for herbicides, the no observable effect level (NOEL) from 24-month chronic rat studies is the most consistent, and was therefore chosen to compare the chronic toxicity of herbicides in this analysis. This choice has the benefit of allowing an "apples to apples" comparison of various herbicide active ingredients, since the chronic studies were conducted on the same test species for the same amount of time. However, rat NOEL values do not necessarily relate directly to human health risk. For some chronic effects, the rat is not an ideal test model for humans, and rabbit or dog studies may have provided results more relevant to applicator health risks. Selecting different test species for different herbicides would be a potential source of bias in this analysis, though, so the same test organism (rat) was used for all active ingredients.

2 Methods

2.1 Data soucres

Data for herbicide use and crop planted area were downloaded from USDA-NASS (quickstats.nass.usda.gov) for all available years between 1990 and 2015. For each herbicide active ingredient included in the NASS data, the herbicide site of action (by WSSA code), the acute rat LD_{50}, and the chronic 24 month rat NOEL was recorded. Site of action and toxicity data were collected from the Herbicide Handbook (Shaner et al. 2014) if available, otherwise US EPA registration documents were searched to find the information.

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2.2 Area-treatments

An area-treatment can be roughly defined as the number of times one herbicide was applied to one field. The total amount (*Amount*) of each herbicide active ingredient applied per crop per year was divided by the average application rate (*Rate*) within each crop for each year, then further divided by the number of planted acres (*acres*) of that crop in that year to obtain area-treatments (*AT*).

$$AT = \frac{Amount/Rate}{acres}$$

All area-treatments were then added together for each herbicide *ai* to determine the total number of area-treatments applied in each year to each crop. It is possible (common, in fact) for the total number of area-treatments to exceed 1 (or 100% of total crop area). For example, a value of 2 area-treatments could be obtained in several ways; either by applying two herbicides at full rates in a tank-mixture to the same field (1 + 1 = 2), or by applying the same herbicide to the same field twice (1 * 2 = 2), or even by applying four different herbicides at half of their average application rates to the same field (0.5 + 0.5 + 0.5 + 0.5 = 2).

2.3 Relative toxicity

A hazard quotient approach (Stoner and Eitzer 2013) was used to evaluate the relative toxicity of herbicides being used in each crop over time. While the term 'hazard quotient' is relatively new and may not be familiar to many scientists, this approach has been used previously in the literature, though not always called by that name, to compare the relative toxicity of accumulated pesticides, herbicide programs, and other toxins (Nelson and Bullock 2003; Gardner and Nelson 2007; Mesnage et al. 2015). For this analysis, the hazard quotient (*HQ*) is defined as the sum of the amount of each herbicide applied per hectare divided by the toxicity of each herbicide:

$$HQ = \sum_{ai=1}^{N} \frac{Amount_{ai}}{Toxicity_{ai}}$$

where N is the total number of herbicide active ingredients applied to a crop in a year, *Amount* is the total weight of each active ingredient (*ai*) applied in mg/ha, and *Toxicity* is either the chronic or acute toxicity value for each *ai*. For the chronic hazard quotient, *Toxicity* is the 24 month rat NOEL expressed in mg/kg body weight/d. For the acute hazard quotient, *Toxicity* is the acute rat LD_{50} expressed in mg/kg. The hazard quotient has a direct interpretation as the number of LD_{50} or NOEL values applied per hectare. High hazard quotient values indicate a relatively more toxic combination of herbicides.

3 Results

There were a total of 159 unique entries in the USDA-NASS data set, but many of these entries were various salts of the same herbicide active ingredient. For example, there were 8 different salts of 2,4-D and 7 different salts of glyphosate. Combining different formulations, there were 118 unique herbicide active ingredients in the full data set; 75 were used in maize, 54 in cotton, 57 in soybean, 34 in rice, 44 in spring wheat, and 56 in winter wheat.

A steady, linear trend for increasing number of herbicide area-treatments ($P<0.001$, Figure 1) over the last 25 years was observed for all crops except soybean. The linear trend was not statistically significant for soybean ($P=0.271$); it was instead characterized by a sustained decrease in the number of herbicide area-treatments between 1994 and 2005, followed by a marked increase betwen 2005 and 2015. Of the 5 crops characterized by a linear trend, herbicide use increased faster in rice (slope = 0.07), spring wheat (slope = 0.09) and winter wheat (slope = 0.06) compared with crops where glyphosate-resistant cultivars are widely planted (maize and cotton, slope = 0.05).

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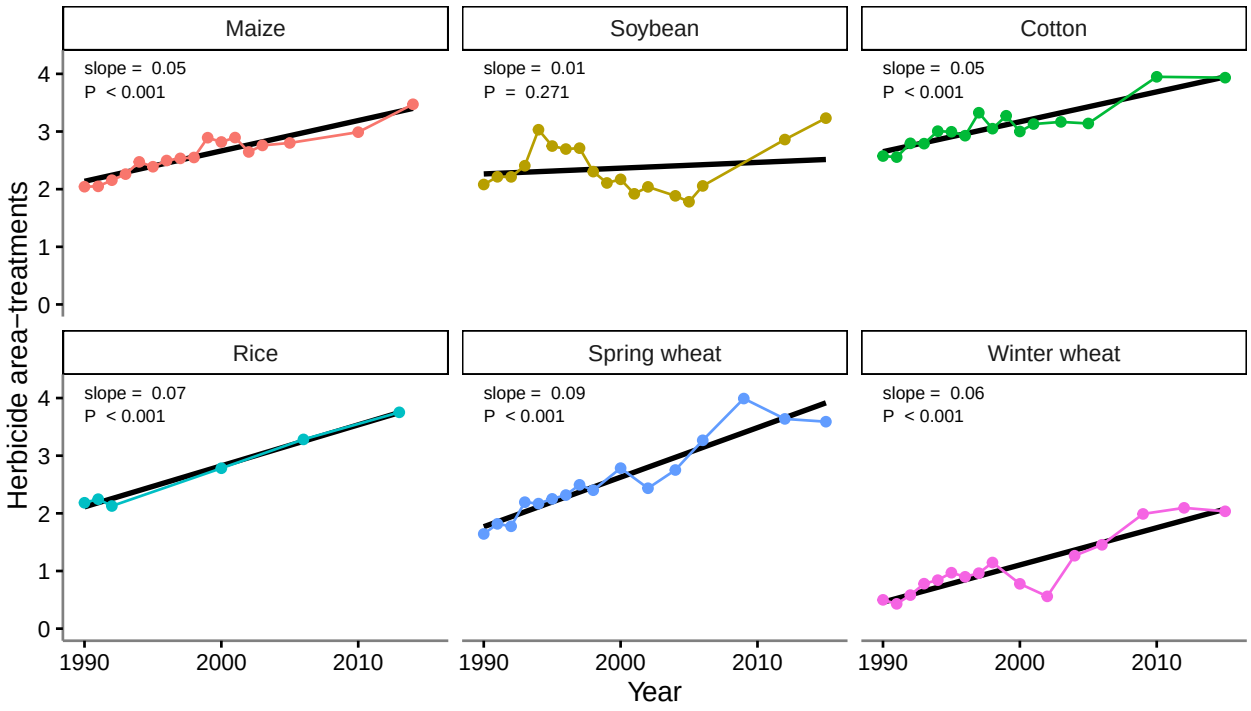

Figure 1: **Herbicide area-treatments for six crops in the United States, 1990 to 2015.**

Chronic and acute toxicity values ranged widely between herbicides (Figure 2). Chronic NOEL values ranged from 0.03 to 20000 mg/kg/d, and acute LD_{50} values ranged from 112 to 9000 mg/kg. For acute toxicity, many herbicide active ingredients had LD_{50} values listed as >5000 mg/kg. EPA registration requirements state that pesticides with acute oral LD_{50} values greater than 5000 mg/kg are considered Category IV, which is the least toxic category (40 CFR Ch I 156.62). Therefore toxicity tests to identify LD_{50} values greater than this upper limit are unwarranted from the registrants' perspective. This censoring of acute toxicity values may result in slight bias in the acute toxicity data, since 5000 mg/kg was used as a conservative estimate in the hazard quotient estimates for any herbicide where the LD_{50} was listed as >5000 mg/kg.

Pesticide toxicity is often discussed in a very general sense (*e.g.* using "more toxic herbicides"), but there is not necessarily a strong relationship between acute and chronic toxicity, and therefore, the distinction between these two measures of toxicity is important. For the 118 active ingredients in this data set, the correlation between chronic and acute toxicity values was not statistically significant ($r = 0.096$, $P = 0.31$).

3.1 Maize

The chronic hazard quotient has increased over time in maize, from 1.57 million in 1990 to 1.68 million in 2014, though it has trended downward slightly in recent years (Figure 3). Throughout the 1990's, atrazine was responsible for a large majority of the chronic hazard quotient in maize (Figure S5). In 2014, just two herbicides (atrazine and mesotrione) were responsible for 88% of the chronic hazard quotient. Acute herbicide toxicity has decreased 88% in maize, from an acute hazard quotient of 7016 in 1990 to 819 in 2014 (Figure 4). Much of the reduction in acute toxicity was due to phasing out of alachlor and cyanazine from the maize market. In 1990, these two herbicides accounted for 85% of the total acute hazard quotient (FIgure S5).

3.2 Soybean

Chronic and acute herbicide toxicity in soybean has decreased 78 and 68%, respectively, between 1990 and 2015 (Figures 3 & 4). Most of the reduction in the chronic hazard quotient has been due to reduction in

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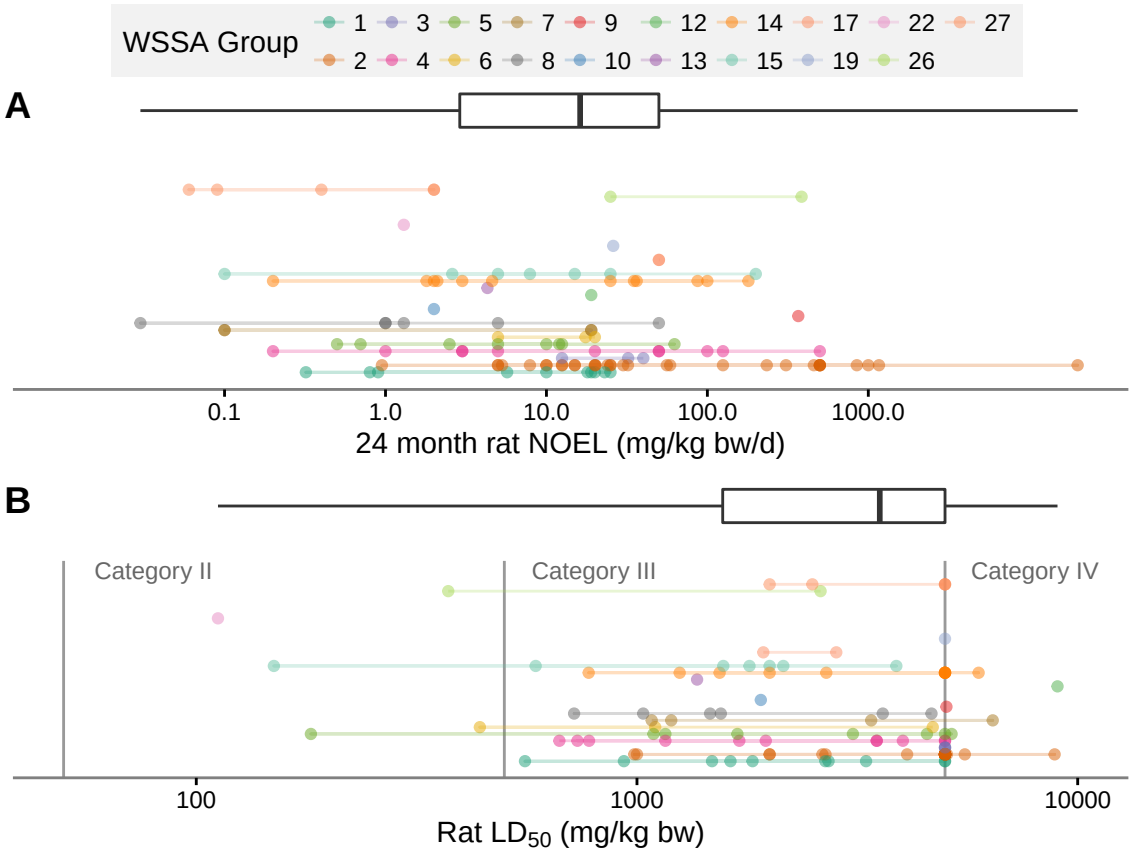

Figure 2: **Chronic (A) and acute (B) toxicity values for herbicides used in maize, cotton, soybean, rice, and wheat between 1990 and 2015 in the United States.** Toxicity is presented on a $\log_{10}$ scale. Bold line represents median toxicity value, box encloses the inter-quartile range, and lines extend from minimum to maximum values. Each point represents a unique herbicide active ingredient, color coded by WSSA site of action group number. Acute toxicity categories are those defined by US EPA.

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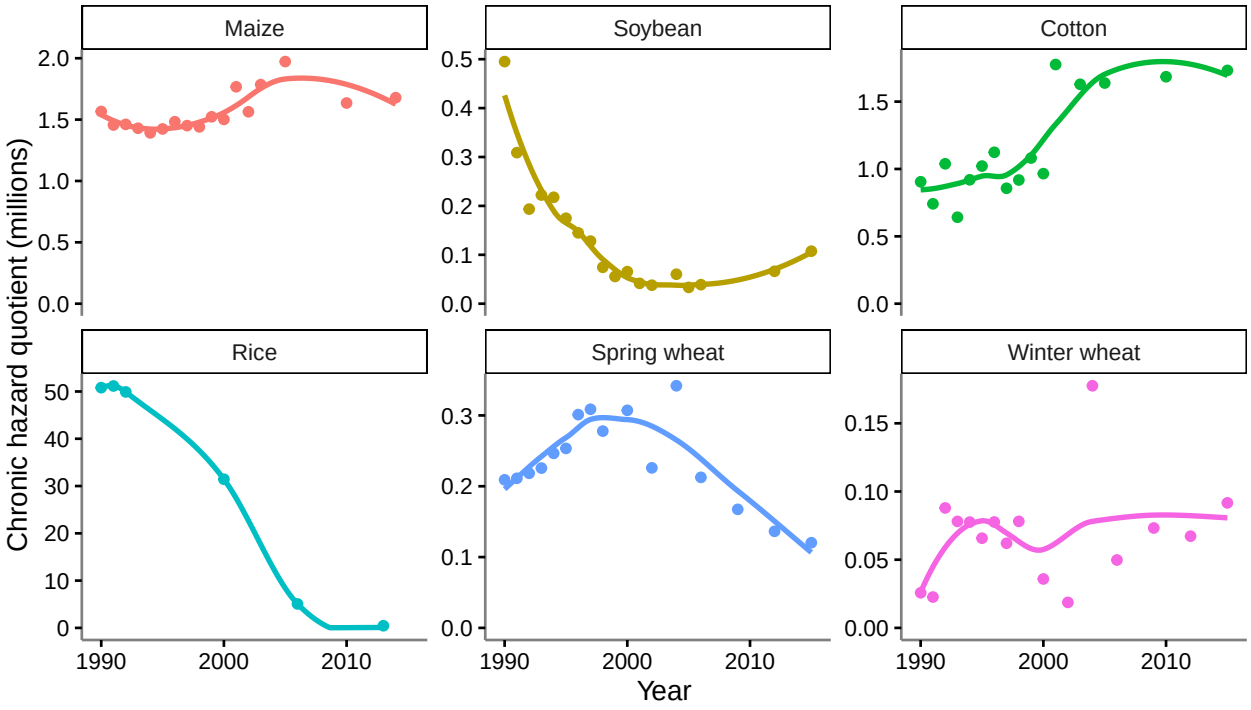

Figure 3: **Herbicide chronic hazard quotients for six crops in the United States, 1990 to 2015.**

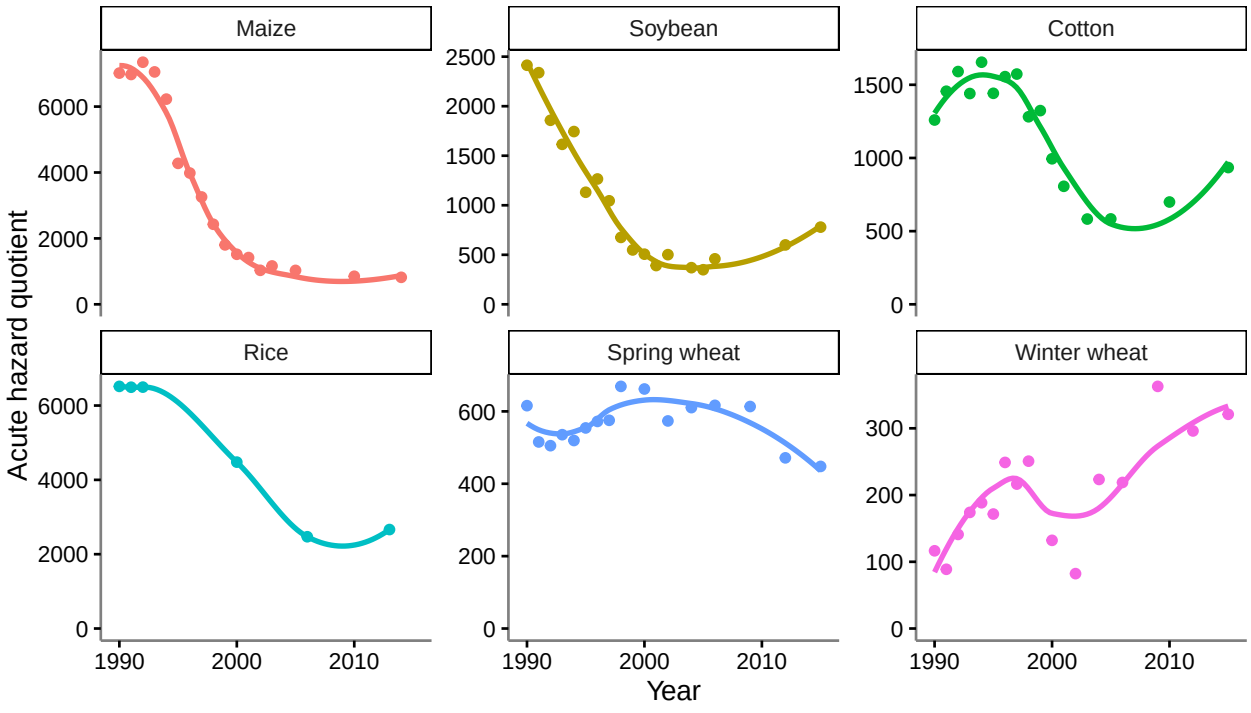

Figure 4: **Herbicide acute hazard quotients for six crops in the United States, 1990 to 2015.**

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linuron use, while most of the acute hazard quotient reduction was due to reduction in alachlor use (Figure S6). In 1990, linuron was responsible for 80% of the chronic hazard quotient, and alachlor was responsible for 79% of the acute hazard quotient in soybean. In 2015, paraquat was responsible for 25% of the acute hazard quotient in soybean. In 2005, which was the peak of glyphosate dominance in the soybean market in this USDA-NASS data set, glyphosate represented 76% of all area-treatments, but was responsible for 10 and 75% of chronic and acute toxicity, respectively.

3.3 Cotton

The chronic hazard quotient for cotton increased between 1990 and 2015 (Figure 3), although the increase has been driven almost completely by a single herbicide. In 2015, diuron was responsible for 89% of the chronic hazard quotient in cotton (Figure S7). The acute hazard quotient has decreased from a peak of 1654 in 1994 to a low of 583 in 2003. After 2004, acute toxicity increased to 934 by 2015, but that was still substantially lower than any acute hazard quotient value observed at any time before the year 2001. Much of the reduction in cotton acute hazard quotient was due to phasing out of the herbicide cyanazine, which made up 60 to 70% of the acute hazard quotient between 1990 and 1998. Similar to soybean, the peak of glyphosate dominance in the cotton market occurred in 2005 in this USDA-NASS data set, when glyphosate represented 54% of all area-treatments. Even with this high reliance on glyphosate for weed control, this herbicide was responsible for only 0.2% of the chronic hazard quotient. Glyphosate's contribution to the acute hazard quotient was similar to it's contribution to total area-treatments, at 52% of acute toxicity.

3.4 Rice

Herbicide use in rice was only surveyed six times over the last 25 years, but since the surveys were conducted near the beginning and end of the period they still provide valuable information on the trend in herbicide use. In 1990, the chronic hazard quotient for rice (50.8) was far greater than any other crop in this analysis (Figure 3), but was almost completely driven by a single herbicide. Molinate made up 99% of the chronic hazard quotient in 1990 (Figure S8). Although molinate use (and the associated chronic hazard quotient) declined dramatically between 1990 and 2006, molinate still accounted for 93% of the chronic hazard quotient in 2006. US registration of molinate herbicide was cancelled in 2008, and further use of molinate was prohibited after 2009 (EPA 2008). In 2013, after molinate use was discontinued, thiobencarb and propanil made up 56 and 25% of the chronic hazard quotient in rice, respectively.

Molinate was also a substantial contributor to the acute hazard quotient, accounting for 32% of herbicide acute toxicity in 1990 (Figure S8). Discontinuation of molinate, therefore, also had a beneficial impact on acute toxicity of rice herbicide programs. Propanil has been the largest contributor to the acute hazard quotient over time in rice, accounting for 58 and 75% of the acute hazard quotient in 1990 and 2013, respectively.

3.5 Spring wheat

The chronic hazard quotient in spring wheat has decreased from 210000 in 1990 to 120000 in 2015 (Figure 3). MCPA and 2,4-D accounted for 56 and 20% of the chronic hazard quotient in 1990, respectively, compared to 60 and 11% in 2015. The acute hazard quotient has remained relatively steady in spring wheat, although a decreasing trend is apparent since 1998 (Figure 4). Bromoxynil accounted for the greatest proportion of the acute hazard quotient in 2015 at 46% of the total.

3.6 Winter wheat

The chronic hazard quotient for winter wheat has remained relatively flat (Figure 3), with the exception of peaks resulting from diuron (in 1992 followed by a decline through 2004) and dichlorprop which was present only in a single year in 2004 (Figure S10). Dichlorprop was only observed in the NASS data set once

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(in 2004 in both spring and winter wheat). The acute hazard quotient has increased rather steadily from 116, to 321 in 2015 (Figure 4). 2,4-D has been the most consistent contributor to acute hazard quotient throughout the last 25 years, though both bromoxynil and glyphosate have increased in recent years (Figure S10). Although acute toxicity increased in winter wheat, both chronic and acute hazard quotients were generally lower for winter wheat than all other crops in this analysis, primarily becuase winter wheat also had the fewest area-treatments applied (Figure 1).

4 Discussion

Previous analyses have attempted to quantify the environmental and health impacts of herbicide use over time, especially as it relates to adoption of genetically-engineered (GE) herbicide-resistant crops. Unfortunately, many of those efforts relied on fundamentally flawed metrics. In particular, the summed weight of herbicides applied with no regard for their relative toxicity is uninformative at best and misleading at worst (NAS 2016). This analysis corrects this deficiency of previous works, by using area-treatments as a more informative indicator of herbicde intensity.

An upward trend in herbicide area-treatments was observed in all six crops that were analyzed, although the upward trend was preceeded by a downward trend in soybean. This result is consistent with the "herbicide treadmill" criticism that U.S. crop production has become increasingly dependent on herbicides for weed control. No causal relationships can be determined from this data, however, and there are many factors that may have driven increased herbicide use over time. Use of tillage in the US has steadily decreased in most crops since 1996, though the rate of tillage reduction depends on the crop and growing region (USDA-ERS 2016). Whether or not tillage is used explicitly for weed control, most tillage operations will provide weed control benefits like killing emerged seedlings and burying weed seed. When tillage is reduced, farmers become more reliant on other weed control practices, including herbicides. It is possible that some of the widespread increase in herbicide use is attributable to adoption of conservation tillage practices. In addition, although no new herbicide sites of action have been commercialized in the last 25 years, many new herbicide products have entered the market. Many of these new products contain multiple active ingredients. Increased marketing and use of these multi-ingredient products would increase the number of herbicide area-treatments, though this data set did not provide commercial formulation information so it is unclear whether this was the case.

Some researchers have blamed glyphosate-resistant crops and the resulting evolution of glyphosate-resistant weeds for increasing herbicide use in maize, soybean, and cotton (Benbrook 2012; Perry et al. 2016). While this explanation is plausible for these three glyphosate-resistant crops, it can not explain the similar trends for increasing herbicide intensity in rice and wheat, since no glyphosate-resistant cultivars are commercially available for those crops. In fact, herbicide area-treatments increased at a faster rate in rice and wheat compared to the glyphosate-resistant crops, so the claim that glyphosate-resistant crops are the primary driver of increasing herbicide use is at odds with the emprical data. The broader problem of herbicide-resistant weeds (rather than the artificially narrow focus on glyphosate) may certainly have played a role in increasing herbicide use for all of the crops in this analysis. The most likely explanation, though, is probably a combination of inter-related factors and is far more complex than any single scapegoat.

The EIQ commonly used in previous analyses of herbicide use over time suffers from severe methodological flaws (Dushoff et al. 1994) that are even more pronounced when comparing herbicides (Kniss and Coburn 2015). The hazard quotient approach used here, while certainly not perfect, is a far more defensible metric with which to compare herbicide toxicity and relative impacts of herbicide use changes, albeit for a small subset of potential toxicity endpoints. This analysis was limited to mammalian toxicity, and therefore is most relevant to chronic and acute risks faced by pesticide applicators, and to a much lesser extent, consumers. This analysis should not be extrapolated to draw conclusions about non-mammalian systems, and should be interpreted with caution even for human health risks. The acute mammalian toxicity of herbicide programs used in the U.S. has decreased over the last 20 to 25 years for 5 out of 6 crops, and chronic toxicity has decreased for 3 of the 6 crops. The largest decreases in the hazard quotient were a result of discontinuation of several products with relatively high toxicity including alachlor, cyanazine, and molinate. In this regard,

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the EPA's decisions to discontinue these products appears to have had a beneficial effect on applicator health risks.

Because adoption of genetically-engineered (GE) herbicide-resistant crops was so rapid and so widespread, the temporal component confounds the ability to define causal relationships between adoption of GE crops and herbicide use trends. Brookes and Barfoot (2012) convincingly explain that extrapolating recent non-GE herbicide usage to represent what all non-GE crop growers would be doing in the absence of GE technology is problematic for several reasons. The minority of growers not using GE technology today are probably not representative of all growers, and therefore their pesticide use is almost certainly not an accurate way to compare overall pesticide use between GE and conventional crops. For example, one reason a farmer might not adopt glyphosate-resistant crops is because they don't have major weed pressure to begin with. Or they may have weeds that are not resistant to other herbicides already, as weed biotypes resistant to other sites of action can be found in every crop production region in the US. Herbicide use is likely to be lower for non-adopters regardless of which technology they chose. Farmers who had fields with high weed pressure or weeds resistant to other herbicides were presumably more likely to adopt GE herbicide resistant crop technology, thereby biasing results toward higher herbicide use in GE crops if these groups are compared directly.

Although the USDA data set does not allow direct comparison between herbicide use in glyphosate-resistant versus conventional varieties, some general conclusions can be drawn in this regard. Glyphosate has an approximate acute LD_{50} of 5037 mg/kg, with some variation depending on which salt is applied. This makes glyphosate less acutely toxic than 94% of the herbicides in this data set. Although glyphosate is considered a relatively safe herbicide with respect to acute toxicity, it is not an outlier in this regard. The median acute LD_{50} in this analysis was 3556 mg/kg, and only 5 herbicides had acute LD_{50} of less than 500 mg/kg, placing them in EPA's toxicity Category II (Figure 2). Therefore, the contribution of glyphosate to acute toxicity was similar relative to its contribution to herbicide use as measured by area-treatments.

Chronic toxicity was a different story, however. Glyphosate has a lower chronic toxicity than 90% of all herbicides in this analysis, but it falls much further from the median chronic toxicity value compared to acute toxicity. Therefore, displacement of glyphosate by other herbicides is more likely to have a negative impact on chronic toxicity. In the last year of survey data for each crop, glyphosate made up 26% of corn, 43% of soybean, and 45% of cotton area-treatments, but only contributed 0.1, 0.3, and 3.5% of the total chronic hazard quotients in those crops, respectively. So although the chronic hazard quotient increased in 2 of 3 glyphosate-resistant crops, if glyphosate were not used the chronic hazard quotient would almost certainly be even greater since other herbicides with greater chronic toxicity would have been used instead.

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6 Supplementary Information

6.1 Supplementary Figures

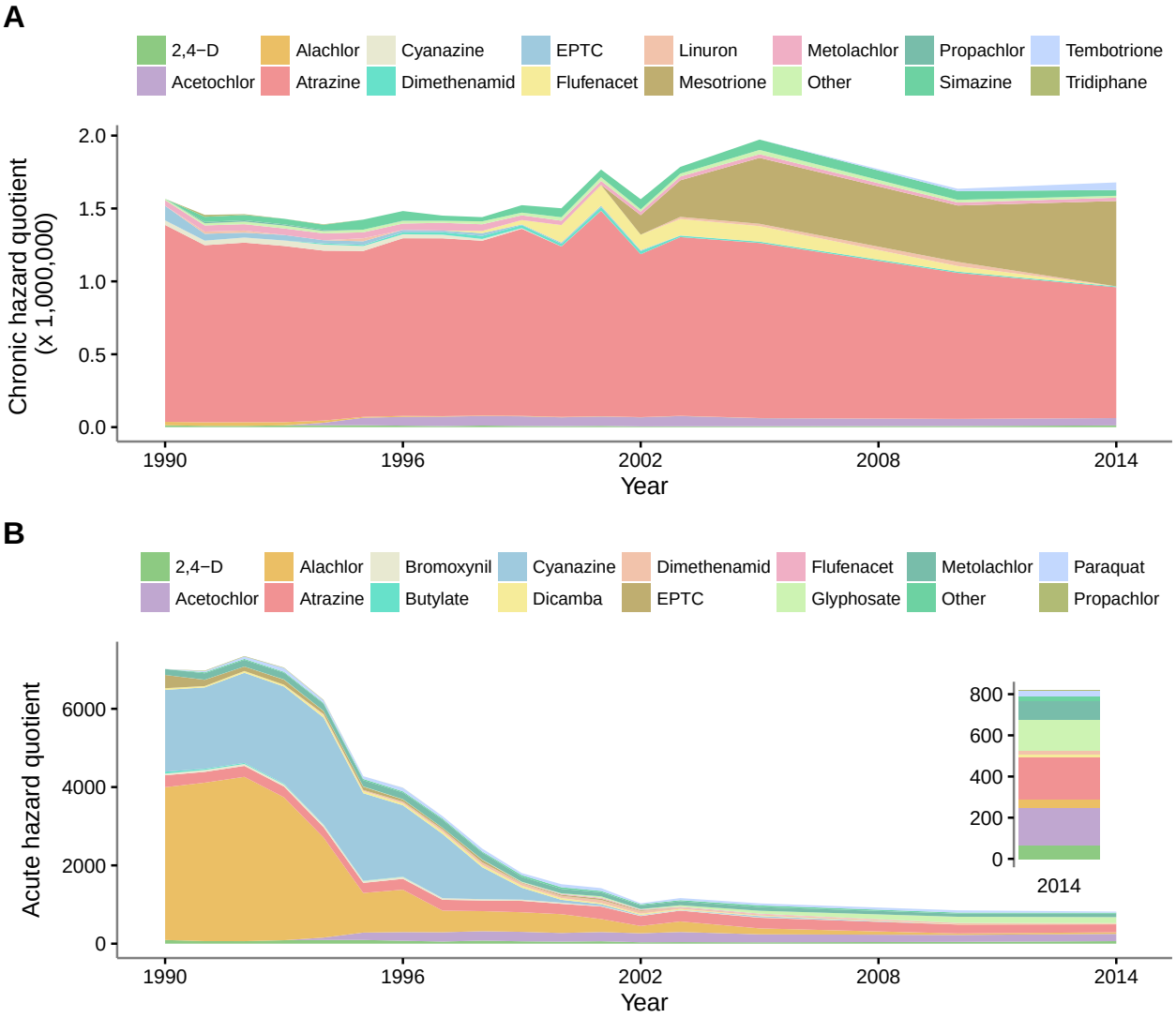

Figure 5: **Chronic and acute toxicity from maize herbicide use in the United States, 1990 to 2014.** Acute mammalian toxicity of herbicides applied, LD_{50}/acre. (A): Chronic (24 month rat) toxicity of herbicides applied, NOEL/acre (B).

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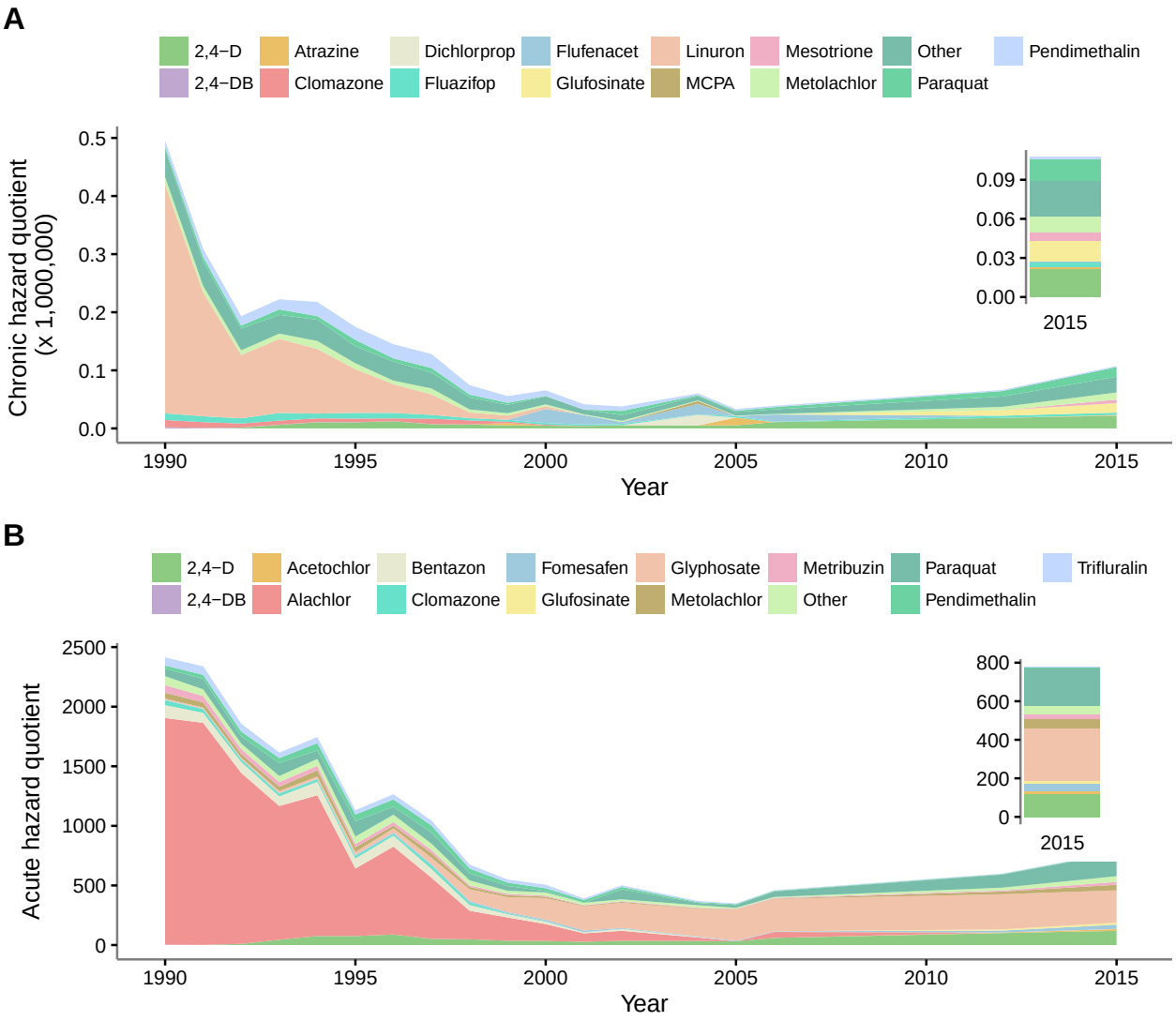

Figure 6: **Chronic and acute toxicity from soybean herbicide use in the United States, 1990 to 2014.** Acute mammalian toxicity of herbicides applied, LD_{50}/acre. (A): Chronic (24 month rat) toxicity of herbicides applied, NOEL/acre (B).

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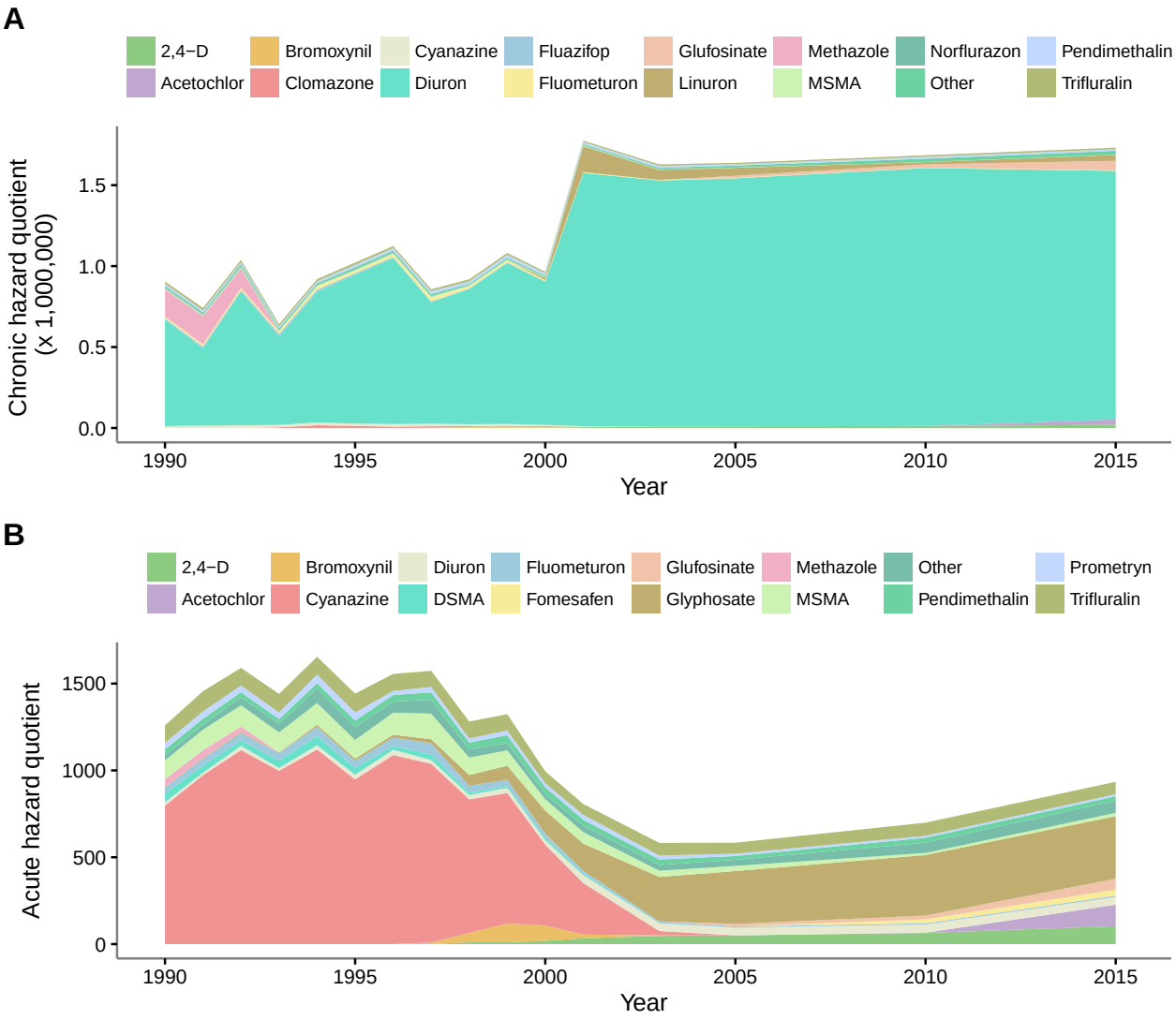

Figure 7: **Chronic and acute toxicity from cotton herbicide use in the United States, 1990 to 2015.** Acute mammalian toxicity of herbicides applied, LD_{50}/acre. (A): Chronic (24 month rat) toxicity of herbicides applied, NOEL/acre (B).

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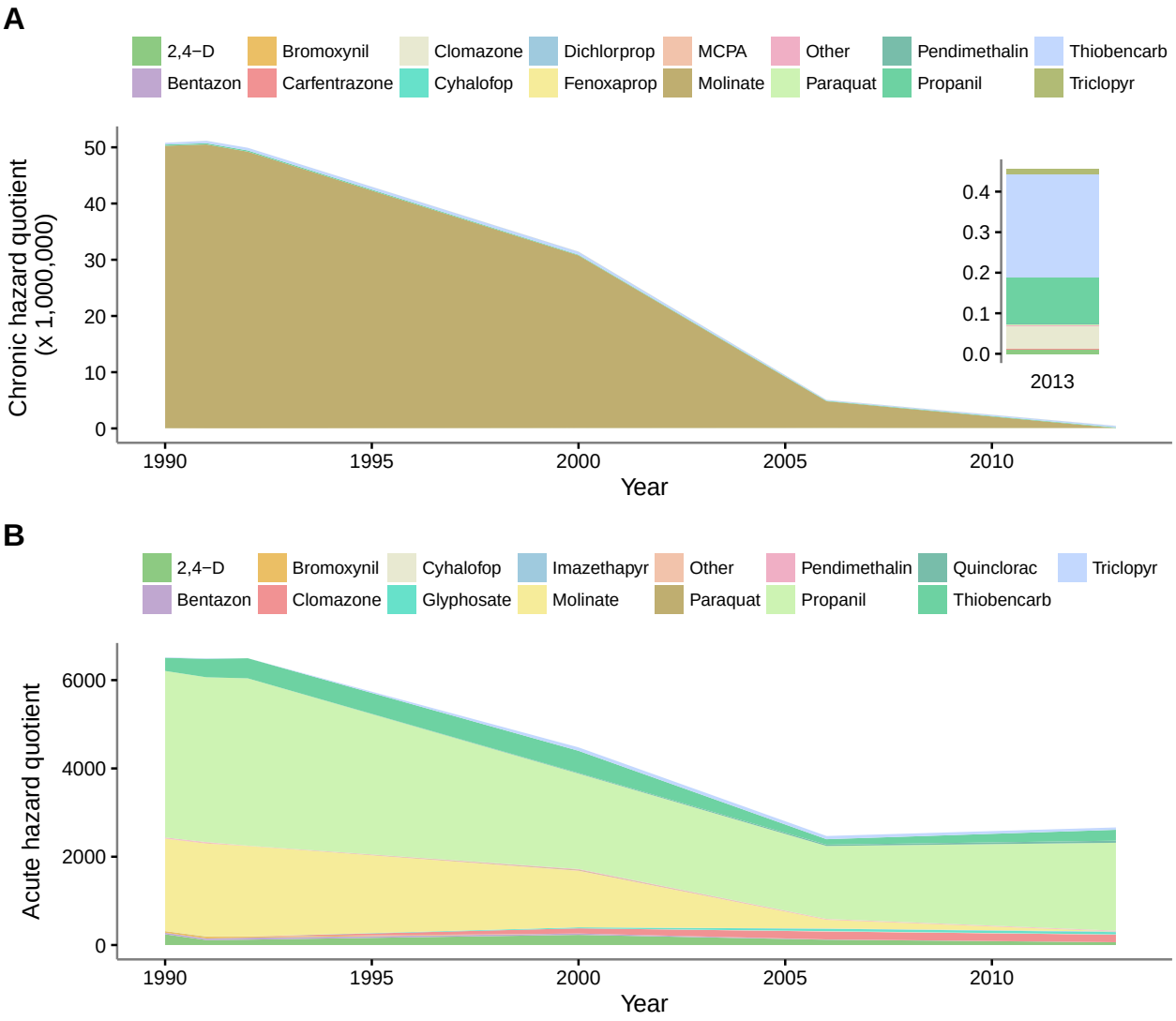

Figure 8: **Chronic and acute toxicity from rice herbicide use in the United States, 1990 to 2014.** Acute mammalian toxicity of herbicides applied, LD_{50}/acre. (A): Chronic (24 month rat) toxicity of herbicides applied, NOEL/acre (B).

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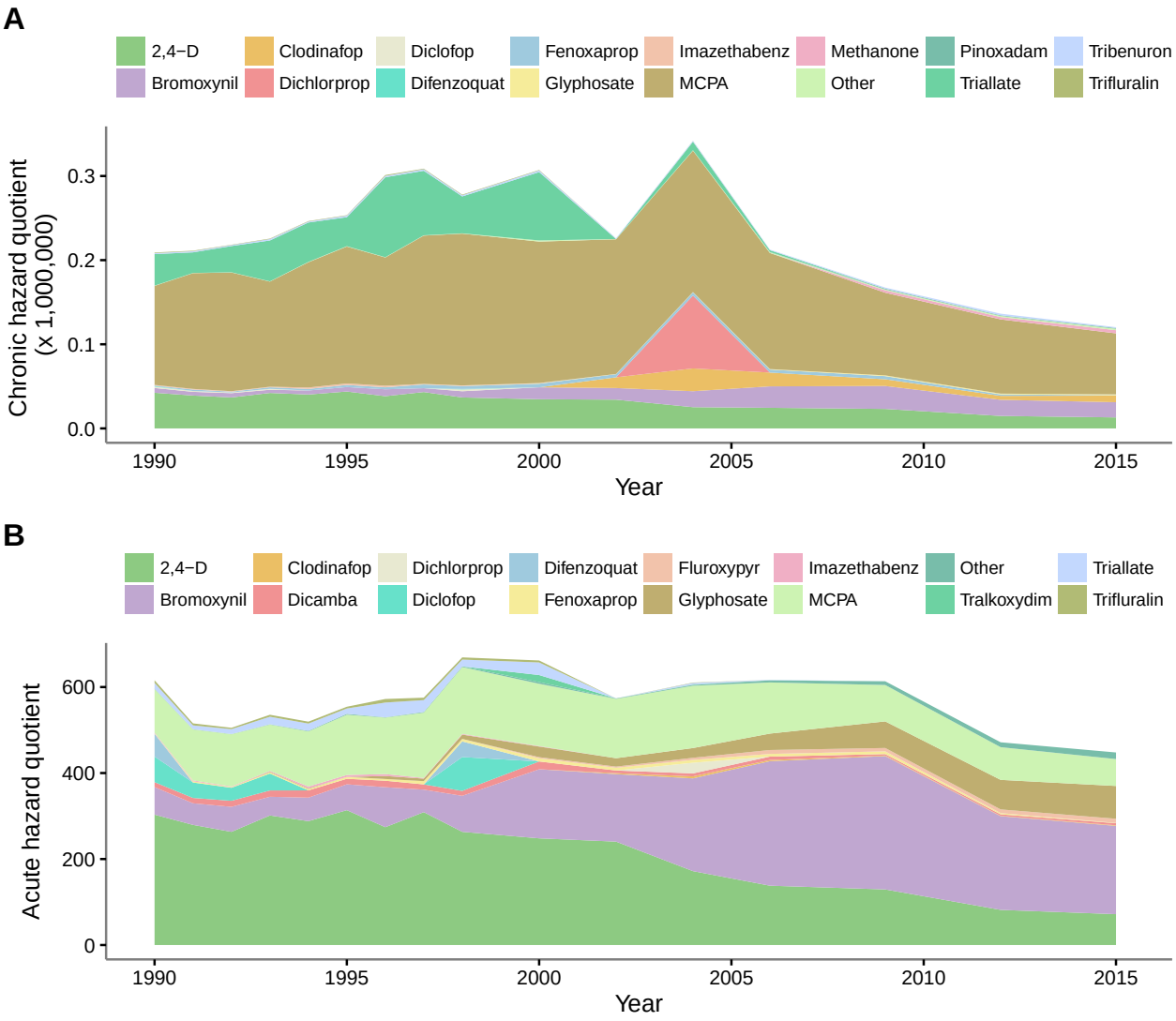

Figure 9: **Chronic and acute toxicity from spring wheat herbicide use in the United States, 1990 to 2014.** Acute mammalian toxicity of herbicides applied, LD_{50}/acre. (A): Chronic (24 month rat) toxicity of herbicides applied, NOEL/acre (B).

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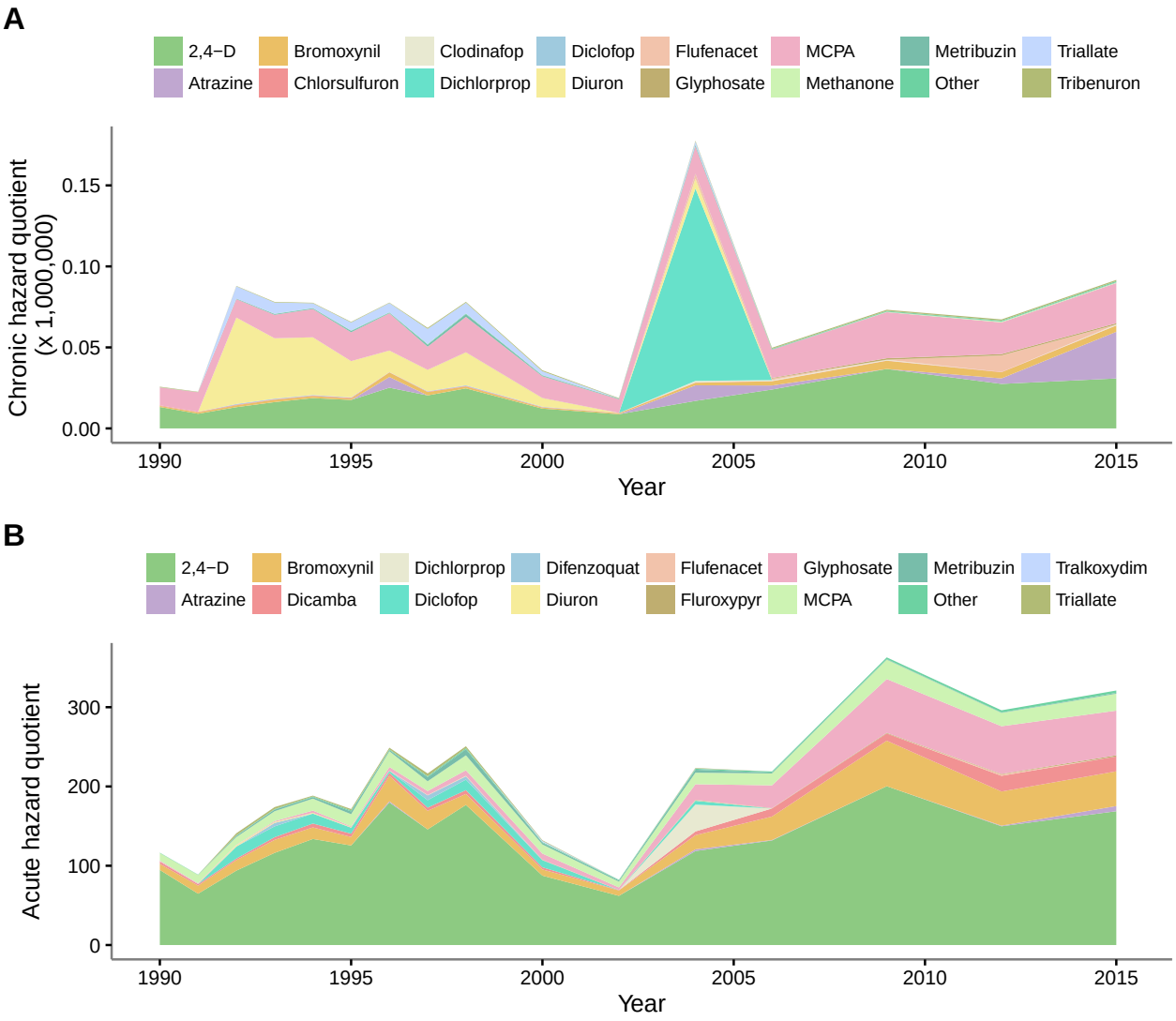

Figure 10: **Chronic and acute toxicity from winter wheat herbicide use in the United States, 1990 to 2014.** Acute mammalian toxicity of herbicides applied, LD_{50}/acre. (A): Chronic (24 month rat) toxicity of herbicides applied, NOEL/acre (B).

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6.2 Supplementary Tables

Herbicide site of action group, chronic and acute toxicity values used in the analysis.

NASS herbicide designation	WSSA group	Acute LD50	Chronic NOEL
(2,4-D, 2-EHE = 30063)	4	896	5
(2,4-D = 30001)	4	699	5
(2,4-DB = 30801)	4	1960	3
(2,4-DB, DIMETH. SALT = 30819)	4	1960	3
(2,4-D, BEE = 30053)	4	699	5
(2,4-D, CHOLINE SALT = 51505)	4	699	5
(2,4-D, DIETH. SALT = 30016)	4	949	5
(2,4-D, DIMETH. SALT = 30019)	4	949	5
(2,4-D, ISOPROP. SALT = 30025)	4	699	5
(2,4-DP, DIMETH. SALT = 31419)	4	699	0
(2,4-D, TRIISO. SALT = 30035)	4	699	5
(ACETOCHLOR = 121601)	15	2148	8
(ACIFLUORFEN, SODIUM = 114402)	14	1540	180
(ALACHLOR = 90501)	15	150	25
(AMETRYN = 80801)	5	1160	2
(AMINOPYRALID = 5100)	4	5000	50
(AMMONIUM CHLORAMBEN = 29902)	4	3500	500
(ATRAZINE = 80803)	5	3090	1
(BENSULFURON-METHYL = 128820)	2	5000	308
(BENTAZON = 103901)	6	1100	18
(BISPYRIBAC-SODIUM = 78906)	2	2635	20
(BROMACIL = 12301)	5	5175	12
(BROMOXYNIL = 35301)	6	440	5
(BROMOXYNIL HEPTAN. = 128920)	6	440	5
(BROMOXYNIL OCTANOATE = 35302)	6	440	5
(BUTOXYETHYL TRICLOPY = 116004)	4	440	3
(BUTYLATE = 41405)	8	4659	50
(CARFENTRAZONE-ETHYL = 128712)	14	5000	3
(CHLORIMURON-ETHYL = 128901)	2	4102	12
(CHLORSULFURON = 118601)	2	5545	5
(CLETHODIM = 121011)	1	1630	19
(CLODINAFOP-PROPARGIL = 125203)	1	1829	0
(CLOMAZONE = 125401)	13	1369	4
(CLOPYRALID = 117403)	4	5000	50
(CLOPYRALID MONO SALT = 117401)	4	5000	50
(CLOPYRALID POTASSIUM = 117423)	4	5000	50
(CLORANSULAM-METHYL = 129116)	2	5000	10
(CYANAZINE = 100101)	5	182	12
(CYHALOFOP-BUTYL = 82583)	1	5000	25
(DIALLATE = 78801)	8	NaN	125
(DICAMBA = 29801)	4	1707	125
(DICAMBA, DIGLY. SALT = 128931)	4	1707	125
(DICAMBA, DIMET. SALT = 29802)	4	1707	125
(DICAMBA, POT. SALT = 129043)	4	1707	125
(DICAMBA, SODIUM SALT = 29806)	4	1707	125
(DICHLORPROP = 31401)	4	800	0
(DICLOFOP-METHYL = 110902)	1	557	20
(DIFENZOQUAT = 106401)	26	373	25
(DIFLUFENZOPYR-SODIUM = 5107)	19	5000	26

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NASS herbicide designation	WSSA group	Acute LD50	Chronic NOEL
(DIMETHENAMID = 129051)	15	1570	5
(DIMETHENAMID-P = 120051)	15	1570	5
(DIURON = 35505)	7	3400	0
(DSMA = 13802)	17	1935	50
(EPTC = 41401)	8	1465	5
(ETHALFLURALIN = 113101)	3	5000	32
(FENOXAPROP = 128701)	1	3310	6
(FENOXAPROP-P-ETHYL = 129092)	1	3310	6
(FLUAZIFOP-P-BUTYL = 122809)	1	2721	1
(FLUCARBAZONE-SODIUM = 114009)	2	5000	125
(FLUFENACET = 121903)	15	589	0
(FLUMETSULAM = 129016)	2	5000	500
(FLUMICLORAC-PENTYL = 128724)	14	5000	35
(FLUMIOXAZIN = 129034)	14	5000	2
(FLUOMETURON = 35503)	7	6416	19
(FLUROXYPYR = 128959)	4	2000	100
(FLUROXYPYR 1-MHE = 128968)	4	5000	100
(FLUTHIACET-METHYL = 108803)	14	5000	2
(FOMESAFEN = 123803)	14	1250	100
(FOMESAFEN SODIUM = 123802)	14	1250	100
(FORAMSULFURON = 122020)	2	5000	849
(GLUFOSINATE-AMMONIUM = 128850)	10	1910	2
(GLYPHOSATE = 417300)	9	5600	400
(GLYPHOSATE AMM. SALT = 103604)	9	5600	400
(GLYPHOSATE DIA. SALT = 103607)	9	5600	400
(GLYPHOSATE DIM. SALT = 103608)	9	5600	400
(GLYPHOSATE ISO. SALT = 103601)	9	5600	400
(GLYPHOSATE POT. SALT = 103613)	9	5600	400
(HALOSULFURON = 128721)	2	8866	56
(HEXAZINONE = 107201)	5	1690	10
(IMAZAMETHABENZ = 128842)	2	5000	12
(IMAZAMOX = 129171)	2	5000	1165
(IMAZAPYR = 128821)	2	5000	500
(IMAZAPYR, ISO. SALT = 128829)	2	5000	500
(IMAZAQUIN = 128848)	2	5000	500
(IMAZAQUIN, MON. SALT = 128840)	2	5000	500
(IMAZAQUIN, SOD. SALT = 129023)	2	5000	500
(IMAZETHAPYR = 128922)	2	5000	500
(IMAZETHAPYR, AMMON. = 128923)	2	5000	500
(IMAZOSULFURON = 118602)	2	5000	5
(IODOSULFURON-MET-SOD = 122021)	2	2678	30
(ISOXAFLUTOLE = 123000)	27	5000	2
(KANTOR = 129108)	2	5000	10
(LACTOFEN = 128888)	14	5960	25
(LINURON = 35506)	7	1196	0
(MCPA, 2-ETHYLHEXYL = 30564)	4	1160	1
(MCPA = 30501)	4	1160	1
(MCPA, DIMETHYL. SALT = 30516)	4	1160	1
(MCPA, ISOOCTYL ESTER = 30563)	4	1160	1
(MCPA, SODIUM SALT = 30502)	4	1160	1
(MESOSULFURON-METHYL = 122009)	2	5000	25
(MESOTRIONE = 122990)	27	5000	0

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NASS herbicide designation	WSSA group	Acute LD50	Chronic NOEL
(METHANONE = 692)	27	5000	2
(METHAZOLE = 106001)	14	777	0
(METOLACHLOR = 108801)	15	3877	15
(METRIBUZIN = 101101)	5	1090	5
(METSULFURON-METHYL = 122010)	2	5000	25
(MOLINATE = 41402)	8	720	0
(MSMA = 13803)	17	2833	50
(NICOSULFURON = 129008)	2	5000	20000
(NORFLURAZON = 105801)	12	9000	19
(ORTHOSULFAMURON = 108209)	2	NaN	NaN
(OXYFLUORFEN = 111601)	14	5000	2
(PARAQUAT = 61601)	22	112	1
(PENDIMETHALIN = 108501)	3	5000	12
(PENOXSULAM = 119031)	2	5000	5
(PICLORAM, K SALT = 5104)	4	4012	20
(PICLORAM, TRIISOPROP = 5102)	4	4012	20
(PINOXADEN = 147500)	1	5000	10
(PRIMISULFURON = 128973)	2	5050	15
(PROMETRYN = 80805)	5	4550	62
(PROPACHLOR = 19101)	15	1800	3
(PROPANIL = 28201)	7	1080	19
(PROPANOIC ACID = 129046)	NA	NaN	NaN
(PROPAZINE = 80808)	5	NaN	5
(PROPOXYCARBAZONE-SOD = 122019)	2	5000	459
(PROSULFURON = 129031)	2	986	8
(PYRAFLUFEN-ETHYL = 30090)	14	5000	87
(PYRIDATE = 128834)	6	4690	20
(PYRITHIOBAC-SODIUM = 78905)	2	1000	59
(PYROXASULFONE = 90099)	15	2000	200
(PYROXSULAM = 108702)	2	2000	1000
(QUINCLORAC = 128974)	26	2610	385
(QUINCLORAC DIMETHYLAMINE SALT = 28974)	26	2610	385
(QUIZALOFOP-ETHYL = 128711)	1	1480	1
(QUIZALOFOP-P-ETHYL = 128709)	1	1480	1
(RIMSULFURON = 129009)	2	5000	15
(SAFLUFENACIL = 118203)	14	2000	5
(SETHOXYDIM = 121001)	1	2676	18
(SIMAZINE = 80807)	5	5000	0
(S-METOLACHLOR = 108800)	15	3877	15
(SULFENTRAZONE = 129081)	14	2689	36
(SULFOSATE = 128501)	9	748	118
(SULFOSULFURON = 85601)	2	5000	24
(TEMBOTRIONE = 12801)	27	2500	0
(THIENCARBAZONE-METHY = 15804)	2	2000	234
(THIFENSULFURON = 128845)	2	5000	20
(THIOBENCARB = 108401)	8	1033	1
(TOPRAMEZONE = 123009)	27	2000	0
(TRALKOXYDIM = 121000)	1	934	23
(TRIALLATE = 78802)	8	3612	1
(TRIASULFURON = 128969)	2	5000	32
(TRIBENURON-METHYL = 128887)	2	5000	1
(TRICLOPYR = 116001)	4	712	3

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NASS herbicide designation	WSSA group	Acute LD50	Chronic NOEL
(TRIDIPHANE = 123901)	NA	1743	0
(TRIETHYLAMINE TRICLO = 116002)	4	712	3
(TRIFLOXYSULFURON-SOD = 119009)	2	5000	20
(TRIFLURALIN = 36101)	3	5000	40
(TRIISOPROPANOLAMINE = 5209)	NA	NaN	NaN
(VERNOLATE = 41404)	8	1550	1

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