



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF CHEMICAL SAFETY AND
POLLUTION PREVENTION

MEMORANDUM

DATE: 25-JUL-2012

SUBJECT: Glufosinate Ammonium. Updated Human Health Risk Assessment for the Proposed New Use of Glufosinate Ammonium in/on Citrus Fruit (Crop Group 10), Pome Fruit (Crop Group 11), Stone Fruit (Crop Group 12), Olives and Sweet Corn.

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Petition No.: NA

Risk Assessment Type: Single chemical aggregate

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DP Barcode: D387413

Registration No.: NA

Regulatory Action: Section 3 Registration

Case No.: NA

CAS No.: 60168-88-9

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FROM: William Donovan, Ph.D., Chemist
John Doherty, Ph.D., Toxicologist
Ana Rivera-Lupiañez, Chemist
Risk Assessment Branch V (RABV)
Health Effects Division (7509P)

William H. Donovan
John Doherty
William H. Donovan for

THROUGH: Michael S. Metzger, Branch Chief
Risk Assessment Branch V (RABV)
Health Effects Division (7509P)

Michael S. Metzger

TO: Kathryn Montague, RM 23
Sidney Jackson/Barbara Madden, RM 05
Registration Division (7505P)

The most recent human-health risk assessment for glufosinate ammonium identified potential risk concerns resulting from the established rice use (D372623, W. Donovan *et al.*, 15-DEC-2010). This document updates and supersedes previous assessments, providing updated toxicological, dietary (food and drinking water), residential (handler and post-application), residue chemistry, occupational, and aggregate assessments. The primary changes include the following:

- Revised residue of concern definition for drinking water.
- Revised chronic EDWC based on rice uses and PFAM modeling.
- Revised dietary exposure analysis using updated EDWC with the NHANES/WWEIA food consumption survey data, 2003-2008.
- Revised ORE assessment according to updated SOPs.

1.0. EXECUTIVE SUMMARY

Bayer Crop Science requested a Section 3 registration for application of glufosinate ammonium (butanoic acid, 2-amino-4-(hydroxymethylphosphinyl)-, monoammonium salt) to citrus fruit, pome fruit, stone fruit, and olives; and the Interregional Research Project Number 4 (IR-4) requested a Section 3 registration for application of glufosinate ammonium to sweet corn. A summary of the human-health risk estimates resulting from the requested and registered uses of glufosinate ammonium is provided in this document.

Technical glufosinate ammonium is a racemic mixture of the D and L enantiomers; with the L enantiomer being responsible for its herbicidal activity. The compound is a non-selective herbicide and acts as an inhibitor of glutamine synthetase which leads to poisoning of the plant by ammonia. Glufosinate ammonium is currently registered for use on both transgenic and nontransgenic crops. The transgenic plants currently registered (rice, cotton, canola, sugar beet, corn, soybean) have been genetically engineered to express phosphiothrion-acetyltransferase (PAT) which enables the plant to metabolize glufosinate ammonium into N-acetylglufosinate.

Toxicology and Dose-Response

The toxicology database for glufosinate ammonium is complete for current risk assessment purposes, including the assessment of potential increased susceptibility of infants and children.

For acute exposure durations, glufosinate ammonium is not highly toxic: it was classified into toxicity category III or IV for all acute toxicity studies, including dermal and eye irritation. It is not a dermal sensitizer.

In the subchronic rat study, inhibition of glutamate synthetase was evident and inhibition of this enzyme in the brain is considered a possible mode of action resulting in neurotoxicity. However, inhibition of this enzyme in the liver and kidney is considered adaptive. In the chronic studies in the rat, increased mortality, increased occurrence of retinal atrophy, and inhibition of brain glutamine synthetase were observed, as were increased liver and kidney weights. In the mouse, increased mortality was observed, as were changes in glucose levels consistent with changes in glutathione levels. Increased mortality and electrocardiogram (EKG) alterations were observed in dogs.

Although there was no evidence of neurotoxicity in two *acute* neurotoxicity studies at doses up to 500 mg/kg/day, clinical signs of neurotoxicity were seen in several studies, including the subchronic, developmental, and chronic studies in rats and dogs. In addition to a variety of clinical signs, retinal atrophy was also observed. The rat developmental neurotoxicity study demonstrated altered brain morphometrics at dose levels that did not cause maternal toxicity.

Since increased fetal mortality was observed in the presence of maternal toxicity in the rabbit developmental study, there is evidence of *qualitative* increased susceptibility in fetuses. The reproductive toxicity study in rats indicated postnatal developmental toxicity at the highest dose tested (HDT) in the form of decrease in viable pups. No parental toxicity was seen at the HDT. Since pup mortality was observed in the absence of parental toxicity, there is evidence of *quantitative* increased susceptibility in offspring. The rat developmental toxicity study demonstrated dilated pelvis and/or hydroureter in the offspring at the same doses that the dams demonstrated hyperactivity and vaginal bleeding.

An acceptable 28-day inhalation toxicity study demonstrated toxicity at the lowest dose tested as indicated by lung and bronchial effects and slight behavioral changes.

There is no concern for mutagenic activity based on the results of several mutagenicity studies.

Glufosinate ammonium was classified as “not likely to be a human carcinogen.” There was no evidence of a treatment-related increase in tumors in either rats or mice.

Additional testing was conducted with the L-isomer of glufosinate ammonium (HOE 058192), and degradates HOE 061517 (MPP) and HOE 099730 (NAG). These compounds, tested in subchronic rat, mouse, and dog studies, and in developmental toxicity studies in rat and rabbit, are generally less toxic than the parent compound. However, HOE 058192 was found to be slightly more toxic than the racemic parent compound. This finding is not a concern since this isomer is included in the toxicity testing of the parent compound at the levels in the technical material.

Endpoints for risk assessment were selected for dietary, dermal, and inhalation scenarios. The FQPA Safety Factor was reduced to 1× for acute dietary exposure only; thus a standard uncertainty factor of 100× was used for the acute dietary risk assessment. An additional uncertainty factor of 10× was used for other exposure scenarios (total uncertainty factor of 1000×) because the studies selected for endpoint setting used LOAELs (the developmental neurotoxicity and the 28-day inhalation studies) and did not establish NOAELs.

Residue Chemistry

The nature of the residue in plants and livestock is understood. The residues of concern in plant and livestock commodities are parent, glufosinate propanoic acid, and N-acetylglufosinate.

The registration requirements for magnitude of the residue in plants have been evaluated and deemed fulfilled. The field trials on citrus fruits, pome fruits, stone fruits, olives, and sweet corn are adequate. An adequate number of trials were conducted reflecting the proposed use patterns in the appropriate geographic regions, and the appropriate commodities were collected at the proposed PHIs. However, the proposed use directions should specify the maximum application rate, the maximum number of applications, and the retreatment intervals. Samples were analyzed using validated analytical methods. Processing studies were conducted on the appropriate commodities and these results were taken into consideration in the recommended tolerance levels.

Acceptable analytical methods are available for enforcement of residue tolerances of glufosinate ammonium and metabolites in/on plant and livestock commodities.

No increase in dietary burden results from the proposed uses; established tolerance levels in meat, milk, poultry and egg commodities remain appropriate.

Dietary Risk (Food and Drinking Water)

The Environmental Fate and Effects Division (EFED) provided a drinking water assessment for glufosinate ammonium. The assessment evaluated the maximum use patterns of the established and proposed new uses of glufosinate ammonium. Based on maximum use patterns, the peak and 1-in-10-year annual mean estimated drinking water concentrations (EDWCs) are 390 and 95 ppb, respectively, driven by the rice use. The drinking water exposure estimates are lower for all other uses.

An acute dietary exposure assessment for glufosinate ammonium was conducted for the only relevant population subgroup, females 13-49 years old. A screening level assessment made use of tolerance level residues, 100% crop treated assumptions, default processing factors and an acute EDWC of 390 ppb. This acute dietary exposure estimate gave results below HED's level of concern (LOC), with the females 13-49 year old population subgroup at 39% aPAD.

The chronic dietary exposure assessment for glufosinate ammonium is refined using anticipated residues based on average residue levels from crop field trials. Percent crop treated (%CT) information and processing factors, where available, were used in the assessment. The chronic dietary risk assessment for glufosinate ammonium at an EDWC of 95 ppb showed that chronic dietary risk estimates are below HED's LOC (<100% cPAD) for infants (<1 year old), the highest exposed population subgroup (98% of the cPAD), as well as for the general US population (39% of the cPAD).

Non-Occupational and Residential Risk

The current petition for glufosinate ammonium results in no non-occupational/residential exposures. Post-application short-term dermal and inhalation exposures to adult homeowners are possible based on the existing spot treatments to turf; however, these exposures are expected to be low and current Agency practice does not routinely require these assessments. In contrast, residential handler assessments are needed for spot treatments to turf. The inhalation margins of exposure (MOEs) for all potential exposure scenarios are greater than 300, the inhalation LOC. Similarly, the dermal MOEs for all potential exposure scenarios related to turf spot treatments are greater than 1000, the dermal LOC.

Aggregate Risk

An aggregate exposure risk assessment was conducted by incorporating drinking water directly into the dietary exposure assessment for the following scenarios: acute, chronic, and short-term aggregate exposure. The short-term aggregate exposure risk assessment also included residential exposure estimates. Intermediate- and long-term residential exposures are not anticipated; therefore intermediate-/long-term aggregate risk assessments were not performed. Cancer aggregate-risk assessments were not performed because glufosinate ammonium is not likely carcinogenic.

Acute aggregate risk estimates are identical to the acute dietary risk estimates and do not exceed HED's LOC. Chronic aggregate risk estimates are identical to the chronic dietary risk estimates and do not exceed HED's LOC.

Using average food and water exposures together with residential exposures from turf spot treatments, the short-term aggregate MOE is estimated as 1800, which is greater than the target MOE of 1000. For residential exposures, only the dermal route of exposure was included in the aggregate analysis since potential dermal exposures are higher than potential inhalation exposures; it is not appropriate to aggregate the dermal and inhalation exposures since the toxicity endpoints are different. Therefore, the short-term aggregate risk exposure estimate is not of concern to the Agency as it does not exceed HED's LOC (MOEs less than or equal to 1000).

Occupational Exposure and Risk

Results from the assessment of all occupational handler scenarios, with the exception of mixer/loader/applicator (MLAP) spot treatments scenarios with mechanically pressurized handgun, indicate that short- and intermediate-term dermal and inhalation risks are not of concern to HED. Short- and intermediate-term risks for spot treatment MLAP scenarios with mechanically pressurized handgun are of concern to HED even with the addition of personal protection and/or engineering controls.

Post Occupational Exposure and Risk

Results of the glufosinate ammonium post-application exposure and dermal risk assessment for sweet corn indicate that an MOE of 1,050 is not achieved until Day 4 for irrigation of mature/high foliage plants; therefore, this post-application activity is of potential concern to the Agency at shorter re-entry intervals (REIs). The proposed Liberty® Herbicide label indicates an REI of 12 hrs. This timing needs revision to achieve an MOE above the target of 1000 based upon the post-application exposure estimates obtained for sweet corn.

Review of Human Research

This risk assessment relies in part on data from studies in which adult human subjects were intentionally exposed to a pesticide or other chemical. These data, which include studies from the Pesticide Handlers Exposure Database Version 1.1 (PHED 1.1); the Agricultural Handler Exposure Task Force (AHETF) database; the Outdoor Residential Exposure Task Force (ORETF) database; and the Agricultural Re-entry Task Force (ARTF) database; are subject to ethics review pursuant to 40 CFR 26, have received that review, and are compliant with applicable ethics requirements. For certain studies that review may have included review by the Human Studies Review Board. Descriptions of data sources as well as guidance on their use can be found at <http://www.epa.gov/pesticides/science/handler-exposure-data.html> and <http://www.epa.gov/pesticides/science/post-app-exposure-data.html>

2.0 HED Recommendations

HED recommends for a registration and tolerances for the use of glufosinate ammonium in/on citrus fruit, pome fruit, stone fruit, olives, and sweet corn. The specific tolerance recommendations are provided in Section 2.2, and label modifications are listed in Section 2.3.

2.1 Data Deficiencies

- None

2.2 Tolerance Considerations

2.2.1 Enforcement Analytical Method

There are adequate residue analytical methods for tolerance enforcement. Also, the methods used for data collection were adequate based on the method recoveries, precision and the range of levels tested.

Two analytical methods have been validated by the Analytical Chemistry Branch (ACB) for enforcement of the currently established tolerances: (1) method HRAV-5A was validated by ACB for the determination of glufosinate ammonium and glufosinate propanoic acid in/on apple, grape, almond, soybean seed, corn grain, and corn forage (PP# 8F3607, J. Garbus, 14-Sep-1989) and (2) method BK/01/99 was validated by ACB for determination of glufosinate ammonium, N-acetyl-glufosinate, and glufosinate propanoic acid in/on canola seed and sugar beet root (D258420, T. Bloem, 19-Aug-2000). Both methods involve extraction with water, anion-exchange chromatography, derivatization with trimethylorthoacetate, silica-gel column clean-up, and quantification via gas chromatography with flame photometric detection (residues expressed as glufosinate free acid equivalents). Method BK/01/99 includes a cation ion-exchange column prior to derivatization which fractionates glufosinate ammonium and N-acetyl-glufosinate and allows for speciation of these compounds (both compounds are derivatized to the same compound). This step can be eliminated if separation of these two compounds is unnecessary. The methods do not distinguish between the D and L enantiomers of glufosinate ammonium and N-acetyl-glufosinate.

Based on the similarity in the two methods and the results from the petition method validations (PMVs), HED concludes that adequate enforcement methods are available for sweet corn, stone fruit, pome fruit, citrus fruit and olive.

2.2.2 Recommended Tolerances

The tolerance expression under 40 CFR §180.473 should be revised to read as follows:

Tolerances are established for residues of glufosinate ammonium, including its metabolites and degradates, in or on the commodities in the table below. Compliance with the tolerance levels specified below is to be determined by measuring the sum of glufosinate ammonium (butanoic acid, 2-amino-4-(hydroxymethylphosphinyl) monoammonium salt) and its metabolites, 2-(acetyl-amino)-4-(hydroxymethyl phosphinyl) butanoic acid, and 3-(hydroxymethylphosphinyl) propanoic acid, expressed as 2-amino-4-(hydroxymethylphosphinyl)butanoic acid equivalents:

Commodity	Proposed Tolerance (ppm)	Recommended Tolerance (ppm)	Comments
Citrus (CG 10)	0.05	0.15	Fruit, citrus, group 10-10
Pome Fruit (CG 11)	0.10	0.25	Fruit, pome, group 11-10
Stone Fruit (CG 12)	0.10	0.25	Fruit, stone, group 12-12
Olives	0.05	0.15	
Corn, sweet, kernels plus cob with husks removed	0.2	0.30	
Corn, sweet, forage	4.0	1.5	
Corn, sweet, stover	6.0	6.0	
Plum, prune, dried	0.20	None	Covered by the Stone Fruit tolerance.

2.2.3 Revisions to Petitioned-For Tolerances

HED recommends higher tolerance levels for citrus, pome fruit, stone fruit, and olives due to summation of the full LOQ for each of the three residues of concern in situations where <LOQ

residue levels were found. The recommended sweet corn tolerances are based on the OECD tolerance calculation procedures. No separate prune tolerance was recommended as residues in this processed commodity are covered by the stone fruit group tolerance.

2.2.4 International Harmonization

The Codex Alimentarius Commission has not established maximum residue limits (MRLs) for glufosinate ammonium in/on olives and sweet corn commodities. However, for glufosinate ammonium in/on citrus fruit, pome fruit and stone fruit, Codex has set MRLs of 0.1, 0.05, and 0.05 ppm, respectively. The recommended US tolerances for citrus fruit, pome fruit, and stone fruit, are 0.15, 0.25 and 0.25 ppm, respectively. The US tolerance values for these commodities is higher than the Codex MRL; therefore, harmonization of the US tolerances with the Codex MRLs is not possible with this petition because MRLs could be exceeded with the proposed uses. There are no MRLs established in Mexico or Canada for the use of glufosinate ammonium on the commodities under the current petition.

2.3 Label Recommendations

2.3.1 Recommendations from Residue Reviews

- Crop use pattern information should include maximum single and seasonal use rates, re-treatment intervals (RTIs), and pre-harvest intervals (PHIs).

2.3.2 Recommendations from Occupational Assessment

- Short- and intermediate-term risks for mixer/loader/applicator (MLAP) scenarios with mechanically pressurized handgun for spot treatments on olives, citrus, pome, and stone fruit are of concern to the Agency even with the addition of personal protection to mitigate exposure such as: extra layer of clothing (coveralls), gloves and PF10R respirator.
- The proposed Liberty® Herbicide label indicates an REI of 12 hrs; however, this timing needs revision to 4 days based upon the post-application exposure estimates obtained for sweet corn in order to achieve an MOE which exceeds the target MOE = 1000.

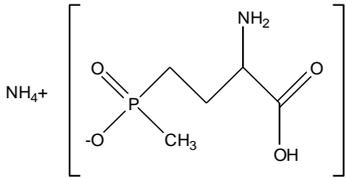
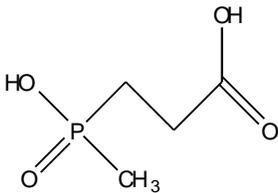
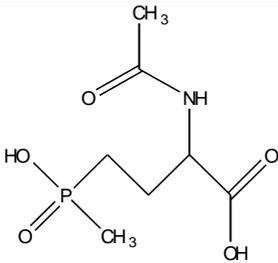
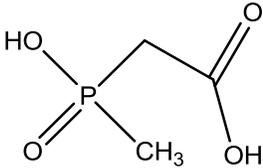
2.3.3 Recommendations from Residential Assessment

- Since glufosinate ammonium is not a restricted use pesticide it is recommended that all labels that could potentially be used by homeowners clearly restrict lawn/turf uses to spot treatments only. The maximum application rate should not exceed 4 fl ounces per gallon of water per 1,000 square feet.

3.0. Introduction

The nomenclature and physicochemical properties of glufosinate ammonium are presented below in Tables 3.1 and 3.2.

3.1. Chemical Identity

TABLE 3.1. Test Compound Nomenclature.	
Compound	
Common name	Glufosinate ammonium
Company experimental name	AE F039866, HOE 039866
IUPAC name	ammonium (2 <i>RS</i>)-2-amino-4-(methylphosphinato)butyric acid
CAS name	2-amino-4-(hydroxymethylphosphinyl)butanoic acid monoammonium salt
CAS registry number	77182-82-2
End-use product (EP)	RELY 200 SC
Compound	
Common name	Glu-PPA, AE F061517, Glufosinate propanoic acid, HOE 061517, MPP
Chemical name	3-methylphosphinico-propionic acid or 3-(hydroxymethylphosphinyl) propanoic acid
Compound	
Common name	Glu-NAG, AE F085355, N-acetylglufosinate, HOE 099730, NAG
Chemical name	2-(acetylamino)-4-(hydroxymethyl phosphinyl) butanoic acid
Compound	
Common name	HOE 064619, Glufosinate acetic acid, MPA
Chemical name	2-methyl phosphinico acetic acid

3.2. Physical/Chemical Characteristics

Table 3.2. Physicochemical Properties of the Technical Grade Test Compound.	
Parameter	Value ¹
Melting point/range (°C)	215-218
pH	4.7
Density (g/cm ³)	1.32
Water solubility (g/L at pH 5)	1370
Solvent solubility at room temp (g/L)	methanol: 5.73; DMSO: 0.049; polyethylene glycol: 0.047; acetonitrile, toluene, acetone, ethyl acetate, and hexane: <0.00025

Parameter	Value ¹
Vapor pressure at 25 °C (mPa)	0.031
Dissociation constant, pK _a	9.15
Octanol/water partition coefficient (Log K _{OW})	-4.01
UV/visible absorption spectrum (nm)	>190

¹Source: 46573701.der.doc, T. Bloem, 2005

Based on its physical-chemical properties, glufosinate ammonium is highly water soluble, non-volatile, and a weak acid.

3.3 Pesticide Use Pattern

Table 3.3 summarizes the proposed use patterns for the new crop uses requested in the current petitions.

Applic. Timing, Type, and Equip.	Applic. Rate (lb ai/A)	Retreat. Interval (days)	Max. No. Applic. Per Season	Max. Seasonal Applic. Rate (lb ai/A)	PHI (days)	Adjuvant	Use Directions and Limitations
Broadcast, banded or spot treatment	Stone Fruit						Do not apply this product through any type of irrigation system.
	1.5	28	2	3.0	14	non-ionic antifoam ²	
	Pome Fruit						Do not apply this product aerially.
	1.5	14	3	4.5	14	non-ionic antifoam ²	
	Citrus						Do not graze, harvest, and/or feed treated orchard cover crops to livestock.
	1.5	14	3	4.5	14	non-ionic antifoam ²	
Olives						Do not make spot spray applications to suckers.	
1.5	14	3	4.5	14	non-ionic antifoam ²		
Foliar treatment	Transgenic Sweet Corn						Must be applied with ammonium sulfate.
	0.365	14	2	0.73	50	None	

¹ The listings in bold are not in the label but correspond to the field trial use patterns.

² A non-ionic antifoam adjuvant may be added.

See Section 2.3 for recommended modifications to the proposed label.

3.4 Anticipated Exposure Pathways

The Registration Division has requested an assessment of human health risk to support the proposed new uses of glufosinate ammonium in/on a variety of crops. Humans may be exposed to glufosinate ammonium in food and drinking water, since it may be applied directly to growing crops and application may result in glufosinate ammonium reaching surface and ground water sources of drinking water. There are registered residential turf uses (limited to spot treatments)

that may lead to homeowner exposures. In an occupational setting, applicators may be exposed while handling the pesticide prior to application, as well as during application. There is a potential for post-application exposure for workers re-entering treated fields.

Risk assessments have been previously conducted for glufosinate ammonium and the only new toxicity study received since the last risk assessment was the immunotoxicity study, which demonstrated effects only at higher doses than selected as points of departure in the earlier risk assessments. A detailed description of the toxicity data and metabolism information may be found in the risk assessment dated 12/15/2010 (W. Donovan *et. al.*, D372623). The current risk assessment considers all of the aforementioned exposure pathways based on the proposed new uses of glufosinate ammonium, but also considers the existing new uses as well, particularly for the dietary and residential exposure assessments.

3.5 Consideration of Environmental Justice

Potential areas of environmental justice concerns, to the extent possible, were considered in this human-health risk assessment, in accordance with U.S. Executive Order 12898, "Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations," (<http://homer.ornl.gov/nuclearsafety/nsea/oepa/guidance/justice/eo12898.pdf>).

As a part of every pesticide risk assessment, OPP considers a large variety of consumer subgroups according to well-established procedures. In line with OPP policy, HED estimates risks to population subgroups from pesticide exposures that are based on patterns of that subgroup's food and water consumption, and activities in and around the home that involve pesticide use in a residential setting. Extensive data on food consumption patterns are compiled by the USDA under the National Health and Nutrition Examination Survey/"What We Eat in America" (NHANES/WWEIA), and are used in pesticide risk assessments for all registered food uses of a pesticide. These data are analyzed and categorized by subgroups based on age, season of the year, ethnic group, and region of the country. Additionally, OPP is able to assess dietary exposure to smaller, specialized subgroups and exposure assessments are performed when conditions or circumstances warrant. Whenever appropriate, non-dietary exposures based on home use of pesticide products and associated risks for adult applicators and for toddlers, youths, and adults entering or playing on treated areas post-application are evaluated. Further considerations are currently in development as OPP has committed resources and expertise to the development of specialized software and models that consider exposure to bystanders and farm workers as well as lifestyle and traditional dietary patterns among specific subgroups.

4.0 HAZARD CHARACTERIZATION

The existing toxicological database for glufosinate ammonium is adequate to support the existing and proposed uses. The registrant recently submitted an acceptable immunotoxicity study that previously had been identified as a data gap (D390963, J. Doherty, 28-OCT-2011). The following information includes summaries of prior assessments (D372623, W. Donovan *et. al.*, 15-DEC-2010).

4.1 Summary of Toxicological Effects

There are several pharmacokinetic studies with glufosinate and they render similar results. Glufosinate is eliminated via the excreta with 95-98% in the first 24 hours. The feces is the major route of excretion with 88% in males and 84% in females of the administered dose recovered in

the feces. Parent compound (~88% in males and ~74% in females) was the principle compound found in the excreta indicated that glufosinate is poorly absorbed from the gastro intestinal tract. The only metabolite found at > 1% of the administered dose was HOE 061517 (representing < 2% of the dose) in both the urine and feces. Tissue retention of label resulting from administration of radio labeled glufosinate was not remarkable in the kidneys, liver, or gonads with residual values not much above background level following a single dose; more was found following repeated dosing.

For subchronic toxicity in rats, inhibition of glutamate synthetase was noted at the LOAEL. The HED Hazard Identification Assessment Review Committee (HIARC, HED Document TXR No.: 0051833, April 17, 2003) concluded that the changes in *brain* glutamine synthetase activity are of significant concern for possible neurotoxicity and/or expression of clinical signs. However, the alterations in liver and kidney glutamate synthetase are considered an adaptive response. The primary effects in the mouse subchronic study were increased liver and kidney weights with increases in serum aspartate amino transferase and alkaline phosphatase.

In the chronic studies in the rat, inhibition of brain glutamine synthetase, increased mortality, and increased occurrence of retinal atrophy were noted, as were increased liver and kidney weights. In the mouse, increased mortality was noted, as were changes in glucose levels consistent with changes in glutathione levels. Increased mortality and electrocardiogram (EKG) alterations were observed in dogs. There was no evidence of a treatment-related increase in tumors in the rat or mouse carcinogenicity studies.

The developmental toxicity study in the rat produced dilated renal pelvis and/or hydroureter in the fetuses at levels that produced significant increases in hyperactivity and vaginal bleeding in dams. In the rabbit, decreased fetal body weight and increased mortality were observed at 20 mg/kg/day, while in rabbit dams, decreased food consumption, body weight, and body weight gain were observed at 20 mg/kg/day. Since increased fetal mortality was observed in the presence of less severe maternal toxicity in the rabbit developmental study, there is evidence of *qualitative* increased susceptibility in fetuses.

The reproductive toxicity study in rats indicated postnatal developmental toxicity at the highest dose tested (HDT) in the form of decrease in viable pups. No parental toxicity was seen at the HDT. Since pup mortality was observed in the absence of parental toxicity, there is evidence of *quantitative* increased susceptibility in offspring. However, the susceptibility was only observed at the highest dose tested, which is 3.3 times higher than the point of departure used for the risk assessment.

There were indications of neurotoxicity in several studies. Of particular concern is that the developmental neurotoxicity study demonstrated alterations in brain morphometrics in the adult offspring exposed *in utero* or during lactation at dose levels not associated with maternal toxicity. Retinal atrophy was observed in the rat oral subchronic study. In the 90-day dietary neurotoxicity study, increases in the incidence of decreased exploratory activity, decreased alertness, and decreased startle response, increased incidence of fearfulness, increased pain response and meiosis were reported. The subchronic dermal toxicity study indicated aggressive behavior, a high startle response and piloerection. The 28-day subchronic inhalation study demonstrated tono-clonic convulsions at the high dose in at least some males. However, in a 37-day dietary neurotoxicity study, there was no evidence of neurotoxicity at doses up to 143.3 mg/kg/day. There was no evidence of neurotoxicity in two acute neurotoxicity studies at doses up to 500 mg/kg/day. Also, there was no evidence of neurotoxicity in White Leghorn hens following an

acute dose of up to 10000 mg/kg. Changes in glutamine synthetase levels were observed in liver, kidney, and brain in rats. The altered electrocardiograms seen in the dog studies imply a possible neuromuscular effect.

A 28-day inhalation toxicity study with glufosinate ammonium generated from a powder technical grade product is available but it is classified as unacceptable due to the particle size exceeding guideline criteria. This study indicates a concern for exposure via the inhalation route because it suggested that animals are more sensitive to effects by the inhalation route. A later second 28-day inhalation toxicity study was provided but this study assessed an aerosol generated from an aqueous technical product. The study was determined to have atmospheric particles within the guideline criteria and was classified as Acceptable/Non-Guideline. It was considered non-guideline because it was for only 28 and not 90 days duration. The LOAEL was unexpectedly greater than the previous study with the unacceptable particle size. HED considers that differences in pH of each test material *may contribute* to the differences in expression of toxicity since the first study assessed a powder with near neutral pH and the second study assessed an aqueous preparation with pH of 4.9. The pH conditions of the test material regulate the conversion of ammonium to ammonia; further, the potential for glufosinate to inhibit ammonia clearance may amplify the toxicity of inhaled ammonia.

There is no concern for mutagenic activity in several studies including: Salmonella E. Coli, *in vitro* mammalian cell gene mutation assays, mammalian cell chromosome aberration assays, *in vivo* mouse bone marrow micronucleus assays, and unscheduled DNA synthesis assays.

There is also no concern for immunotoxicity based on review of the series 870.7800 immunotoxicity study.

Consistent with the 2003 risk assessment (D290086, T. Bloem *et. al.*, 07-AUG-2003) and ToxSAC recommendation (J. Kidwell, 29-JUN-2010), a dermal absorption factor of 9% was assumed, based on a dermal absorption study in male rats (D289836, B. Daiss, 17-JUN-2003).

4.2 Safety Factor for Infants and Children (FQPA Safety Factor)

When the DNT study is used as an endpoint for risk assessment, an extra 10× database uncertainty (UF_L) factor is applied because the DNT study did not demonstrate a NOAEL for altered brain morphometrics. This additional uncertainty factor will also account for indications of increased qualitative or quantitative sensitivity evident in the rat and rabbit developmental studies, and the rat multigenerational reproduction study. Similarly, a UF_L was applied to the inhalation exposure scenarios because the 28-day inhalation study in the rat did not identify a NOAEL. The toxicity and exposure databases for glufosinate ammonium are otherwise complete. Acute, subchronic, and developmental neurotoxicity studies are available, and all endpoints used in this risk assessment are protective of neurotoxic effects when the extra 10× UF_L is applied. The dietary, occupational, and residential assessments are based on reliable data and will not underestimate exposure.

4.2.1 Completeness of the Toxicology Data Base

The toxicity data base for the evaluation sensitivity/susceptibility to infants and children is complete. No additional studies are required at this time. The toxicity data base consists of acute

and subchronic neurotoxicity screen studies, a developmental neurotoxicity study, rat and rabbit developmental studies, a rat multi-generation reproduction study and an immunotoxicity study.

4.2.2 Evidence of Neurotoxicity

A critical indication of neurotoxicity was evident in the developmental neurotoxicity study where alterations in brain morphometrics in the adult offspring were demonstrated.

The clinical signs seen in rat or dog studies were varied and included hyperactivity, aggressive behavior, tonic-clonic convulsion, piloerection, high startle response, and retinal atrophy. In a 90-day dietary neurotoxicity study, increases in the incidence of decreased exploratory activity, decreased alertness, decreased startle response, increased incidence of fearfulness, increased pain response and meiosis were observed.

4.2.3 Evidence of Sensitivity/Susceptibility in the Developing Young Animal

In the DNT, alterations in brain morphometrics in adults following *in utero* exposure was evident at the lowest dose tested and at a dose level where there was no maternal toxicity. Thus, there is evidence of *quantitative* increased sensitivity in the rat.

In the multi-generational reproductive toxicity study in rats, pup mortality was observed in the absence of parental toxicity. Thus, there is further evidence of *quantitative* increased susceptibility in offspring in rats. However, the susceptibility was only observed at the highest dose tested, which is 3.3× higher than the point of departure used for the risk assessment.

In the rabbit, decreased fetal body weight and increased mortality were observed at 20 mg/kg/day, while in rabbit dams, decreased food consumption, body weight, and body weight gain were observed at 20 mg/kg/day. Since increased fetal mortality is more severe than the responses in the dams, there is evidence of *qualitative* increased susceptibility in the rabbit offspring.

The developmental toxicity study in the rat produced dilated renal pelvis and/or hydronephrosis in the offspring at levels that produced significant increases in hyperactivity and vaginal bleeding in dams. Since effects occurred in the pups and dams at the same dose, this study is not considered to demonstrate either increased qualitative or quantitative sensitivity in the rat.

4.2.4 Residual Uncertainty in the Exposure Database.

There are no residual uncertainties in the exposure database. Although the chronic dietary exposure estimates are partially refined, HED does not believe that the exposure estimates are underestimated. These assumptions and refinements are detailed in Section 5.4.3. With limited monitoring data available, upper-bound assumptions were used to determine exposure through drinking water sources.

4.3 Toxicity Endpoint and Point of Departure Selections

There have been no changes to the prior dose-response assessment, recommendations for combining routes of exposure, or the cancer classification. Table 4.3.1 presents a summary of the toxicological doses and endpoints used in the dietary, residential, and occupational risk assessments. A 10X uncertainty factor, attributable to the DNT study not demonstrating a NOAEL, should be applied to chronic dietary and residential dermal assessments. For

occupational and residential inhalation exposure assessments, a 10X database uncertainty factor should be applied due to the inhalation study not demonstrating a NOAEL.

Table 4.3.1 Summary of Toxicological Doses and Endpoints for Glufosinate Ammonium for Use in Dietary and Non-Occupational and Occupational Human Health Risk Assessments				
Exposure/ Scenario	Point of Departure	Uncertainty/FQPA Safety Factors	RfD, PAD, Level of Concern for Risk Assessment	Study and Toxicological Effects
Acute Dietary (General Population, including Infants and Children)	An endpoint attributable to a single exposure was not available from the toxicity studies, including the developmental toxicity and developmental neurotoxicity studies.			
Acute Dietary (Females 13-49 years of age)	NOAEL = 6.3 mg/kg/day	UF _A =10× UF _H =10× FQPA SF= 1× Total UF = 100×	Acute RfD = 0.063 mg/kg/day aPAD=0.063 mg/kg/day	Developmental Toxicity Study in Rabbits LOAEL = 20 mg/kg/day based on increased fetal deaths.
Chronic Dietary (All Populations)	NOAEL= 6 mg/kg/day	UF _A =10× UF _H =10× FQPA SF = UF _L = 10× Total UF = 1000×	Chronic RfD = 0.006 mg/kg/day cPAD = 0.006 mg/kg/day	“Weight of evidence” approach from four studies. Rat subchronic and chronic studies with the LOAEL based on inhibition of brain glutamate synthetase. A dog chronic study with the LOAEL based on altered electrocardiogram and mortality. The rat developmental neurotoxicity study with a LOAEL (without a NOAEL, basis for UF _L) based on altered morphometrics in the offspring as adults.
Incidental Oral Short-Term (1-30 days) and Intermediate term (1-6 months)	LOAEL= 14 mg/kg/day (LDT)	UF _A =10× UF _H =10× FQPA SF= UF _L = 10× Total UF = 1000×	Residential LOC for MOE = 1000	Developmental Neurotoxicity Study in Rats LOAEL = 14 mg/kg/day based on brain morphometric changes at PND 72. No NOAEL identified.
Dermal Short-Term (1-30 days), and Intermediate-Term (1-6 months)	LOAEL= 14 mg/kg/day (LDT)	UF _A =10× UF _H =10× FQPA SF=UF _L = 10× Total UF = 1000×	Residential and Occupational LOC for MOE = 1000 for short and intermediate-term exposures	Developmental Neurotoxicity Study in Rats LOAEL = 14 mg/kg/day based on brain morphometric changes at PND 72. No NOAEL identified.

Table 4.3.1 Summary of Toxicological Doses and Endpoints for Glufosinate Ammonium for Use in Dietary and Non-Occupational and Occupational Human Health Risk Assessments

Exposure/ Scenario	Point of Departure	Uncertainty/FQPA Safety Factors	RfD, PAD, Level of Concern for Risk Assessment	Study and Toxicological Effects
Inhalation Acute, Short-Term (1-30 days), intermediate (1-6 months)	LOAEL=12.5 mg/kg/day (56 mg/m ³)	UF _A = 3× UF _H =10× FQPA SF=UF _L = 10× Total UF = 300×	Residential and Occupational LOC for MOE = 300 for short and intermediate term	28-day Inhalation Study (MRID 47058101) 2007 LOAEL = 12.5 mg/kg/day based on lung/bronchial congestion and increased lung/bronchi weight in female rats and increased kidney and liver weights.
Cancer (oral, dermal, inhalation)	Classification: “Not likely to be Carcinogenic to Humans” based on the absence of significant tumor increases in two adequate rodent carcinogenicity studies.			

Point of Departure (POD) = A data point or an estimated point that is derived from observed dose-response data and used to mark the beginning of extrapolation to determine risk associated with lower environmentally relevant human exposures. NOAEL = no observed adverse effect level. LOAEL = lowest observed adverse effect level. UF = uncertainty factor. UF_A = extrapolation from animal to human (interspecies). UF_H = potential variation in sensitivity among members of the human population (intraspecies). UF_L = use of a LOAEL to extrapolate a NOAEL. FQPA SF = FQPA Safety Factor. PAD = population adjusted dose (a = acute, c = chronic). RfD = reference dose. MOE = margin of exposure. LOC = level of concern. N/A = not applicable.

5.0 Dietary Exposure and Risk Assessment

5.1 Residues of Concern Summary and Rationale

No plant or animal metabolism data were submitted with the subject petitions. The nature of the residue of glufosinate ammonium in livestock and plants has been adequately delineated. HED has previously reviewed metabolism studies conducted with nontransgenic (corn, soybean, apple, and lettuce; 8F3607, J. Garbus, 14-Oct-1988 & 8-Aug-1990) and transgenic (corn, soybean, sugar beet, canola, and rice; D227386, M. Rodriguez, 7-Mar-1996; D257629, T. Bloem, 9-Jul-1999; 45204405.der.wpd) crops. The transgenic corn, soybean, sugar beet, canola, and rice investigated in the metabolism studies were engineered to express PAT which acetylates glufosinate (herbicidally active) to form N-acetyl-glufosinate (not herbicidally active).

Based on the metabolism and magnitude of the residue studies, the Metabolism Assessment Review Committee (MARC) concluded that the residues of concern in plants and livestock, for tolerance expression and risk assessment purposes, are glufosinate ammonium, N-acetyl-glufosinate, and glufosinate propanoic acid (D282757, T. Bloem, 9-May-2002). HED concludes that the results from the currently available metabolism studies may be translated to citrus fruit, pome fruit, stone fruit, olive, and sweet corn.

The residue of concern in drinking water was considered at a meeting of the Residue of Concern Knowledgebase Subcommittee (ROCKS) (D397644, I. Negrón-Encarnación, 29-MAR-2012). The ROCKS noted that MPP may have different toxicity than the parent and is likely to have a lower toxicity, but not so low that it could be definitely excluded from consideration. After the

ROCKS meeting, the team consulted with Health Effects Division Toxicology Science Advisory Council (ToxSAC) regarding the toxicity of MPP (J. Kidwell, 02/21/2012). The consensus of the ToxSAC was that glufosinate ammonium and MPP show different toxicities, such that they should not be aggregated. Because MPP is less toxic than glufosinate ammonium and should not be aggregated with it, if MPP EDWCs are not more than 4-5× greater than those for glufosinate, the risk assessment for the parent will be protective of any toxicity associated with exposure to MPP in drinking water. Indeed, the Environmental Fate and Effects Division has indicated that the acute and chronic concentrations of MPP are not likely to be more than twice the corresponding levels of glufosinate ammonium in drinking water (see Table 5.1). Therefore, a quantitative risk assessment for MPP in drinking water is not needed. Table 4.2.1 summarizes the nature of the residue decisions for glufosinate ammonium.

Table 5.1. Summary of Metabolites and Degradates to be included in the Risk Assessment and Tolerance Expression			
Matrix		Residues included in Risk Assessment	Residues included in Tolerance Expression
Plants	Primary and Rotational Crops	Glufosinate ammonium, glufosinate propanoic acid, and N-acetylglufosinate	Glufosinate ammonium, glufosinate propanoic acid, and N-acetylglufosinate
	Ruminant	Glufosinate ammonium, glufosinate propanoic acid, and N-acetylglufosinate	Glufosinate ammonium, glufosinate propanoic acid, and N-acetylglufosinate
Swine			
Poultry			
Drinking Water		Glufosinate ammonium ¹	Not applicable

¹ Since MPP has a different toxicity profile than the parent compound, it should not be aggregated with the parent. Because MPP EDWCs are not significantly greater than those for glufosinate, the risk assessment for the parent is protective of any toxicity associated with exposure to MPP in drinking water, and a quantitative risk assessment for MPP is not required.

5.2 Food Residue Profile

A review of the residue chemistry data submitted in conjunction with the current petition is provided in D372625 (I. Negrón-Encarnación, 31-AUG-2010). Specific information regarding the magnitude of the residue data and tolerance derivations is provided in Appendix B.

No adjuvant effects were discernible in any of the field trials, which are supported by adequate storage stability data. The trials were adequate with respect to number and location.

Processing studies were performed for orange, plum and olive commodities. Details concerning the processing studies are in the following reviews: orange (47915706.der.wpd), plum (47915707.der.wpd) and olive (47915708.der.wpd). These studies are considered acceptable and demonstrated no concentration in any processed commodity except for prune, where concentration factors for GA, MPP, and NAG were <1.0×, 2.5×, and <1.0×, respectively. Multiplying the field trial HAFT values for plum by the concentration factors for prune gives: <0.05 ppm × <1.0 + 0.0655 ppm × 2.5 + <0.05 ppm × <1.0 = <0.26 ppm. Because the residue concentration in prunes is covered by the plum RAC tolerance level of 0.25 ppm, no separate prune tolerance is needed.

Except for sweet corn, there are no livestock feedstuffs associated with the proposed new use crops. Inclusion of sweet corn does not result in an increase in the dietary burden from what was previously determined (D271110, T. Bloem, 20-June-2002). Accordingly, the established tolerances for livestock commodities remain adequate and no revisions to these values are needed.

Sweet corn is the only proposed crop to which plant back intervals (PBIs) apply. The Liberty Herbicide label indicates that plant rotation to other crops after treatment of sweet corn can occur with a PBI of 120 days with the exception of small cereal grains, for which a PBI of 70 days is allowed. Based on the results from the confined and field rotational studies, HED concludes that the proposed rotational crop restrictions are appropriate for sweet corn.

5.3 Water Residue Profile

The following information was provided by EFED (D372624, C. Peck, 22-JUN-2010; D368799, C. Peck, 07-JUL-2010; D381992, C. Peck, 20-OCT-2010; and D387412, C. Peck, 30-MAY-2012).

Environmental Fate Assessment: Environmental fate studies indicate glufosinate-ammonium is relatively stable and is very mobile ($K_d = 1.5$; $K_{oc} = 173$; water solubility 1370 g/liter). It dissipated with a first order half-life ranging from 4.3 – 10.3 days on bare ground and 8 – 30 days on cropped fields following a single application. The main degradation pathway in water and soil is via microbial action, metabolizing primarily to CO₂, HOE 061517 [MPP], 2-methylphosphinico acetic acid (HOE 064619), and 2-acetamido-4-methylphosphinico-butanoic acid. Aerobic soil metabolism produced a half-life of approximately 4 – 23 days; metabolite concentrations peaked at 3 weeks and then began to decline. Anaerobic soil metabolism produced a half-life of 56 days. The aerobic aquatic metabolism half-life was 64 days in gravel pit water sand sediment.

Glufosinate may leach to ground water under certain conditions (such as in areas of sandy soils with high permeability and shallow ground water). Degradates HOE 061517 ($K_d = 0.7$ and $K_{oc} = 84$) and N-acetyl-glufosinate ($K_d = 0.8$) are more mobile than the parent, and may also be expected to leach to ground water. However, the potential for degradate HOE 064619 to leach to ground water is much lower because of its higher adsorption coefficient ($K_d = 24$).

Ground and Surface Water Estimated Drinking Water Concentrations (EDWCs): EFED estimated acute EDWCs for glufosinate-ammonium and MPP using the refined Tier I Rice Model and Pesticide Flooded Application Model (PFAM) [version 0.70] without the index reservoir. To estimate chronic EDWCs, the acute concentrations from PFAM without the index reservoir were assumed to degrade over a 365-day period, using aerobic aquatic degradation half-lives; thus allowing calculation of average concentrations over a one-year period. This method results in chronic values approximately 76% and 3% lower than the acute values for glufosinate-ammonium and MPP, respectively.

Previous analyses from EFED demonstrated that the maximum acute and chronic EDWCs for glufosinate ammonium arise from the rice uses; these values being nearly an order of magnitude higher than the values from any other crop use of glufosinate ammonium (DP 372624, C. Peck, 6/22/2010 & DP 381992, C. Peck, 10/20/2010). Thus, EFED conducted a comprehensive refinement of the drinking water assessment for the rice use of glufosinate ammonium, with the expectation that the resulting values should be protective of other uses.

Table 5.3 summarizes the PFAM EDWCs for glufosinate ammonium and MPP based on the rice

use patterns, as provided by EFED. Most of the application scenarios result in lower values for MPP than for glufosinate ammonium, although the maximum chronic EDWC for MPP is approximately 2x higher than the corresponding value for glufosinate ammonium: 177 and 95 ppb, respectively. Because glufosinate ammonium is considerably more toxic than MPP, and these compounds have sufficient differences in toxicity that precludes aggregation, glufosinate ammonium EDWCs are protective of MPP EDWCs. Accordingly, for purposes of acute and chronic dietary analyses, the recommended glufosinate ammonium EDWCs are 390 and 95 ppb, respectively.

Table 5.3. EDWCs for glufosinate-ammonium and MPP from rice use derived using PFAM (maximum values appear in bold).				
Use rate, Number of apps, retreatment interval, water holding period, application timing	Acute EDWC (µg/L)		Chronic EDWC¹ (µg/L)	
	4"	8"	4"	8"
Glufosinate-ammonium				
1.46 lbs ai/acre, 1, NA, 7 days, dry	173	86	42	21
0.73 lbs ai/acre, 2, 10, 30, dry and flooded ²	NA	320	NA	78
0.73 lbs ai/acre, 2, 10, 55, dry and flooded ²	390	NA	95	NA
0.89 lbs ai/acre, 1, NA, 7 days, dry	106	53	26	13
0.66 lbs ai/acre, 1, NA, 7 days, dry	74	38	18	9
0.44 lbs ai/acre, 2, 10 days, 7 days, dry	88	45	22	11
0.44 lbs ai/acre, 2, 10 days, 7 days, flooded	NA	375	NA	92
MPP				
1.46 lbs ai/acre, 1, NA, 7 days, dry	5.7	2.9	5.5	2.8
0.73 lbs ai/acre, 2, 10, 30, dry and flooded ²	NA	88	NA	85
0.73 lbs ai/acre, 2, 10, 55, dry and flooded ²	183	NA	177	NA
0.89 lbs ai/acre, 1, NA, 7 days, dry	3.5	1.8	3.4	1.7
0.66 lbs ai/acre, 1, NA, 7 days, dry	2.2	1.1	2.1	1.1
0.44 lbs ai/acre, 2, 10 days, 7 days, dry	3.0	1.5	2.9	1.5
0.44 lbs ai/acre, 2, 10 days, 7 days, flooded	NA	45	NA	44

1. Chronic EDWC calculated by taking acute EDWC and allowing it to degrade for 365 days, using the aerobic aquatic degradation half-life, and then taking the average value over the 365-day period.

2. For this scenario the first application is made to a dry field, while the second application is made to a flooded field. NA – not allowed.

5.4. Dietary Risk Assessment

Acute and chronic dietary (food + drinking water) exposure analyses for glufosinate ammonium were conducted by HED (D402741, W. Donovan, 19-JUN-2012). Glufosinate ammonium acute and chronic dietary exposure assessments were conducted using the Dietary Exposure Evaluation Model software with the Food Commodity Intake Database DEEM-FCID™, Version 3.10, which incorporates consumption data from USDA’s National Health and Nutrition Examination Survey/“What We Eat in America” (NHANES/WWEIA) dietary survey conducted in 2003-2008. The 2003-2008 data are based on the reported consumption of individuals over two non-consecutive survey days.

5.4.1 Acute Dietary Exposure/Risk

Residue Data Used for Acute Assessment: Established and recommended tolerance level residues were used for the acute analysis, along with default processing factors, and 100% crop treated assumptions (unrefined analysis).

Results of Acute Dietary Exposure Analysis: An unrefined acute dietary risk assessment for glufosinate ammonium at the EDWC of 390 ppb showed that **acute dietary risk estimates are below HED’s level of concern (i.e. <100% aPAD)** for the relevant population subgroup, females 13-49 years old (39% aPAD).

5.4.2 Chronic Dietary Exposure/Risk

The chronic dietary exposure assessment for glufosinate ammonium is refined using anticipated residues based on average residue levels from field trial studies. Average %CT information and processing factors, where available, were used in the assessment. There were no PDP monitoring data available for glufosinate ammonium.

Results of Chronic Dietary Exposure Analysis: The chronic dietary risk assessment for glufosinate ammonium at an EDWC of 95 ppb showed that **chronic dietary risk estimates are below HED’s level of concern (i.e. <100% cPAD)** for all population subgroups, with the highest exposed population subgroup [all infants (<1 year old)] at 98% of the cPAD, and the U.S. Population at 39% of the cPAD.

The results of the dietary exposure analyses are reported in Tables 5.4.2.

Population Subgroup ¹	DEEM Acute Dietary Analysis, 95 th Percentile		DEEM Chronic Dietary Analysis	
	Exposure (mg/kg/day)	% aPAD	Exposure (mg/kg/day)	% cPAD
General U.S. Population	NA ²	NA ²	0.002324	39
All Infants (< 1 year old)			0.005887	98
Children 1-2 years old			0.004387	73
Children 3-5 years old			0.003447	57
Children 6-12 years old			0.002261	38
Youth 13-19 years old			0.001706	28
Adults 20-49 years old			0.002203	37
Adults 50-99 years old			0.002202	37
Females 13-49 years old			0.024401	39

¹ Values for the population with the highest risk for each type of risk assessment are **bolded**.

² NA = Not Applicable

5.4.3 Anticipated Residue and Percent Crop Treated (%CT) Information

The DEEM-FCID™ chronic analysis was performed using anticipated residues (ARs) from field

trial data, processing factors and updated %CT information. The DEEM default processing factors were used for all commodities except apple juice, pear juice, grape juice, and raisins, for which factors derived from the processing studies were used.

Percent Crop Treated: One hundred percent crop treated values were used for all proposed new uses. The following average percent crop treated estimates (BEAD SLUA, 19-MAR-2012) were used in the chronic dietary analysis for crops that are currently registered for glufosinate ammonium: almond: 15%; blueberry: 5%; field corn, 5%; grape, 15%; pecan, 1%; potato, 10%; soybean, 1%; walnut, 10%; canola, 25%; cotton, 5%; filbert, 10%; pistachio, 20%; and rice, 1%.

6.0 Residential (Non-Occupational) Exposure/Risk Characterization

Glufosinate ammonium is not a restricted use pesticide (RUP). There are no proposed residential uses associated with this petition ; however, there are existing residential turf uses that have been reassessed in this document to reflect updates to HED's 2012 Residential SOPs along with policy changes for body weight assumptions. The revision of residential exposures will impact the human health aggregate risk assessment for glufosinate ammonium.

The quantitative exposure/risk assessment developed for residential handlers is based on the following exposure scenarios:

1. mixing/loading/applying liquids with a manually pressurized handgun,
2. mixing/loading/applying liquids with a hose-end sprayer,
3. mixing/loading/applying liquids with a backpack sprayer, and
4. mixing/loading/applying liquids with a sprinkler can.

Data and Assumptions for Residential Handler Exposure Scenarios

Unit Exposures and Area/Amount Treated

Unit exposure values and estimates for area treated were taken from HED's 2012 Residential SOPs: Lawns/Turf. It was assumed that residential handlers would treat a maximum of 1000 ft² (0.023 acres).

Application Rate

A maximum single application rate of 1.36 lb ai/Acre (4 fl. oz./ 1000 ft²) was used for all spot treatment scenarios (EPA Reg. No. 432-1229).

Body Weight

The average female adult body weight of 69 kg was used for estimating short-term dermal dose because the selected toxicological POD is based on developmental effects; the average male adult body weight of 80 kg was used for estimating short-term inhalation dose.

Absorption Factors

- A dermal absorption factor of 9% is used to estimate short-term dermal exposure since the point of departure is based on an oral study.
- A route-specific study was used for the inhalation assessment.

6.1 Residential Handler Exposure

Residential handler exposure is expected to be short-term. Intermediate-term exposures are not likely because of the intermittent nature of applications by homeowners. Summaries of the short-term dermal and inhalation risk estimates for residential handlers are included in Table 4.5. The maximum application rate for each exposure scenario is presented as the worst case scenario.

All dermal scenarios for residential handlers performing spot treatment applications utilizing hose-end-sprayer and sprinkler can, resulted in MOEs greater than the LOC (i.e., MOEs \geq 1,000) and, therefore are not of concern.

All inhalation scenarios for residential handlers resulted in MOEs greater than the LOC (i.e., MOEs \geq 300) and, therefore are not of concern. In this assessment, since the PODs selected for both dermal and inhalation routes of exposure are based on different endpoints; the MOEs for the dermal and inhalation exposure routes were not combined.

Table 6.1 Updated Summary of Short-Term Residential Handler Exposures and Risks Estimates for Application of Glufosinate-Ammonium on Residential Turf.								
Exposure Scenario	Application Rate ^a	Area Treated Daily ^b	Unit Exposure ^c		Dose ^d		MOE ^e	
			Dermal	Inhalation	Dermal	Inhalation	Dermal	Inhalation
	<i>lb ai per 1000 ft²</i>	<i>mg/lb ai</i>	<i>mg/kg/day</i>	Dermal	Inhalation			
Spot Treatments								
Hose-end Sprayer	0.0312	1000 ft ²	13.4	0.022	0.0005	0.000009	26000	1,500,000
Manually-pressurized handwand	0.0312	1000 ft ²	63	0.018	0.0026	0.000007	5400	1,800,000
Sprinkler can	0.0312	1000 ft ²	13.4	0.022	0.0005	0.000009	26000	1,500,000
Backpack Sprayer	0.0312	1000 ft ²	130	0.14	0.0053^f	0.0001	2600	230,000

a Application Rates based on maximum application rates of registered residential turf uses for glufosinate ammonium 4 fl oz per gallon of water to treat to treat 1000 ft².

b Based on HED's SOPs: Lawns/Turf (January 2012). Area treated daily: 0.023 A = 1000 ft².

c Residential Handler Attire: no gloves, short pants, short-sleeved shirt, no respirator.

d Dose (mg/kg/day) = daily unit exposure (mg/lb ai) × application rate (lb ai/A) × area treated (Acres/day) × absorption factor (%) ÷ body weight (69 kg for dermal; 80 kg for inhalation). Dermal absorption factor = 9%.

e Dermal MOE = LOAEL (14 mg/kg/day) / dermal daily dose (mg/kg/day), where LOC = 1000

Inhalation MOE = LOAEL (12.5 mg/kg/day) / inhalation daily dose (mg/kg/day), where LOC = 300

f Residential exposure estimate recommended for use in aggregate assessment.

6.2 Residential Post-Application Exposure

Exposure is possible during post-application activities on treated turf. Children may experience exposure via incidental non-dietary ingestion (i.e., hand-to-mouth, object-to-mouth (turfgrass), and soil ingestion) during post-application activities on treated turf. Based on the Agency's current practices, post-application dermal assessments are not performed for spot treatment uses (HED's SOPs: Lawns/Turf; January 2012). These types of uses can result in residues on turf but residential exposure is expected to be low.

Likewise, post-application inhalation exposure while engaged in activities on or around previously treated turf is generally not assessed. The combination of low vapor pressure for chemicals typically used as active ingredients in outdoor residential pesticide products and

dilution in outdoor air is likely to result in minimal inhalation exposure. Therefore, a quantitative post-application inhalation exposure assessment was not performed for glufosinate ammonium at this time primarily because it has very low vapor pressure (vapor pressure less than 1×10^{-8} mmHg). However, volatilization of pesticides may be a potential source of post-application inhalation exposure to individuals nearby to pesticide applications. The Agency sought expert advice and input on issues related to volatilization of pesticides from its Federal Insecticide, Fungicide, and Rodenticide Act Scientific Advisory Panel (SAP) in December 2009. The Agency received the SAP's final report on March 2, 2010 (<http://www.epa.gov/scipoly/SAP/meetings/2009/120109meeting.html>) and is in the process of evaluating the SAP report. The Agency may, as appropriate, develop policies and procedures to identify the need for and, subsequently, the way to incorporate post-application inhalation exposure into the Agency's risk assessments. If new policies or procedures are put into place, the Agency may revisit the need for a quantitative post-application inhalation exposure assessment for glufosinate ammonium. However, it should be noted that residential handler inhalation exposures result in high MOEs, ranging from approximately 230,000 to 1,800,000.

6.3 Spray Drift

Spray drift is always a potential source of exposure to residents nearby to spraying operations. This is particularly the case with aerial application but, to a lesser extent, could also be a potential source of exposure from the groundboom application methods additionally employed for glufosinate ammonium. The Agency has been working with the Spray Drift Task Force, EPA Regional Offices, and State Lead Agencies for pesticide regulation and other parties to develop the best spray drift management practices. The Agency is now requiring interim mitigation measures for aerial applications that must be placed on product labels/labeling. The Agency has completed its evaluation of the new data base submitted by the Spray Drift Task Force, a membership of U.S. pesticide registrants, and is developing a policy on how to appropriately apply the data and the AgDRIFT computer model to its risk assessments for pesticides applied by air, orchard airblast, and ground hydraulic methods. After the policy is in place, the Agency may impose further refinements in spray drift management practices to reduce off-target drift and risk estimates associated with aerial as well as other application types where appropriate.

Although a quantitative residential post-application inhalation exposure assessment was not performed as a result of pesticide drift from neighboring treated agricultural fields, an inhalation exposure assessment was conducted for residential handlers use on turf. This exposure scenario is representative of a worse case inhalation (drift) exposure and may be considered protective of most outdoor agricultural and commercial post-application inhalation exposure scenarios.

7.0 Aggregate Exposure/Risk Characterization

In accordance with the FQPA, HED must consider and aggregate glufosinate ammonium pesticide exposures and risks from three major sources: food, drinking water, and residential exposures. In an aggregate assessment, exposures from relevant sources are added together and compared to quantitative estimates of hazard (e.g., a NOAEL or PAD), or the risks themselves can be aggregated. When aggregating exposures and risks from various sources, HED has considered both the route and duration of exposure.

7.1 Acute Aggregate Risk

The acute dietary assessment included food and water exposures for females 13-49 years old and represents the acute aggregate risk assessment for glufosinate ammonium (see Section 5.4.1).

7.2 Short-Term Aggregate Risk

The short-term aggregate risk assessment takes into account average exposure estimates from dietary consumption of glufosinate ammonium (food and drinking water) and residential/non-occupational exposures. HED uses average (chronic) food and water exposure estimates when conducting short-term aggregate exposure assessments. Short-term exposure has been defined as from 1- 30 days and HED has concluded that average exposures to food and water will more accurately reflect actual exposure over these time periods than will high end exposures. The refined chronic dietary assessment incorporated percent crop treated information for several crops and assumed average residues levels from field trials. For livestock commodities, anticipated residues were also assumed. The residential handler dermal and inhalation exposure estimates were conducted using residential SOPs and ORETF data. The resulting exposures were less than HED’s level of concern (see Table 4.5) for all scenarios, with the backpack sprayer scenario giving the highest potential exposure.

Short-term aggregate exposure to glufosinate ammonium is provided in Table 7.2. For average dietary exposure, the General U.S. Population was selected as it is protective for all other population subgroups except for infants and young children, who will not be applying glufosinate ammonium. For residential/non-occupational exposures, only the dermal route of exposure was included in the aggregate analysis since potential dermal exposures are higher than potential inhalation exposures (see Table 6.1); it is not appropriate to aggregate the dermal and inhalation exposures since the toxicity endpoints are different. The backpack sprayer residential exposure scenario was chosen for aggregate analysis because it is protective for the other residential exposure scenarios. The point of departure (POD) selected for short-term aggregate exposure to glufosinate ammonium is 14 mg/kg/day. Based on these assumptions, the estimated short-term aggregate MOE of 1800 (LOC = 1000) is not of concern to HED.

Population	LOAEL (mg/kg/day)	Food + Drinking Water Exposure (mg/kg/day)	Dermal Dose (mg/kg/day)	Total Exposure (mg/kg/day)	LOC	MOE _{Agg}
General U.S. Population	14	0.002324	0.0053	0.00762	1000	1800

LOC=Level of Concern

MOE= LOAEL/Exposure

MOE_{Aggregate}= LOAEL/(Exposure_{food and water} + Exposure_{dermal})

Food + Drinking Water exposure: See Table 5.4.2.

Dermal exposure: See Table 6.1.

7.3 Intermediate/Long-Term Aggregate Risk

Intermediate/long-term exposures are not anticipated. Therefore an intermediate/long-term aggregate risk assessment was not performed.

7.4 Chronic Aggregate Risk

The chronic dietary assessment included food and water exposures and represents the chronic aggregate risk assessment for glufosinate ammonium (see Section 5.4.2).

8.0 Cumulative Exposure/Risk Characterization

Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, EPA has not made a common mechanism of toxicity finding as to glufosinate ammonium and any other substances, and glufosinate ammonium does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that glufosinate ammonium has a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the policy statements released by EPA's Office of Pesticide Programs concerning common mechanism determinations and procedures for cumulating effects from substances found to have a common mechanism on EPA's website at <http://www.epa.gov/pesticides/cumulative/>.

9.0 Occupational Exposure/Risk Characterization

9.1 Occupational Handler

Occupational exposure to glufosinate ammonium is anticipated for handlers who apply the formulated products to the proposed new use sites. Based on application rate and label information, dermal and inhalation exposure is expected to occur for short- and intermediate-term durations. Chronic exposure is not expected for the proposed use patterns. Potential occupational exposure scenarios include:

- 1) Mixer/Loader using open pouring of liquids in support of aerial, groundboom, and spot/directed spray application operations;
- 2) Aerial Applicators (enclosed cockpit);
- 3) Applicators using open-cab ground boom equipment;
- 4) Flagger in support of aerial applications; and
- 5) Mixer/Loader/ Applicator (MLAP) for mechanically pressurized handgun spray applications.
- 6) Mixer/Loader/ Applicator (MLAP) for Backpack sprayer applications.

Chemical-specific data were not submitted to the Agency in support of this Section 3 registration. It is the policy of HED to use the best available data to assess handler exposure. Sources of generic handler data, used as surrogate data in the absence of chemical-specific data, include the Pesticide Handlers Exposure Database Version 1.1 (PHED 1.1), the Agricultural Handler Exposure Task Force (AHETF) database, or other registrant-submitted occupational exposure studies. Some of these data are proprietary (*e.g.*, AHETF data), and subject to the data protection provisions of FIFRA. The standard values recommended for use in predicting handler exposure that are used in this assessment, known as "unit exposures", are outlined in the "Occupational

Pesticide Handler Unit Exposure Surrogate Reference Table”

(<http://www.epa.gov/opp00001/science/handler-exposure-table.pdf>), which, along with additional information on HED policy on use of surrogate data, including descriptions of the various sources, can be found at <http://www.epa.gov/pesticides/science/handler-exposure-data.html>.

The average adult weight of 80 kg was used for estimating inhalation exposure. For dermal risk assessments, a 69 kg body weight was used to calculate exposure to glufosinate ammonium since the point of departure was selected from a developmental toxicity study. A dermal absorption factor of 9% was applied during the conduct of the short- and intermediate-term dermal exposure assessments, based on a dermal penetration study (D289836, B. Daiss, 17-JUN-2003). For inhalation risk assessment, a route specific study was used.

In this assessment, since the Points of Departure (PODs) selected for both short- and intermediate-term dermal and inhalation routes of exposure are based on different endpoints and the toxicological effects are different; the MOEs for the dermal and inhalation exposure routes were not combined.

Daily dermal or inhalation handler exposures are estimated for each applicable handler task with the application rate, the area treated in a day, and the applicable dermal or inhalation unit exposure using the following formula:

$$\text{Daily Exposure (mg ai/day)} = \text{Unit Exposure (mg ai/lb ai handled)} \times \text{Application Rate (lbs ai/area)} \times \text{Daily Area Treated (area/day)}$$

Where:

Daily Exposure	=	Amount (mg ai/day) deposited on the surface of the skin that is available for dermal absorption or amount inhaled that is available for inhalation absorption;
Unit Exposure	=	Unit exposure value (mg ai/lb ai)
Application Rate	=	Normalized application rate based on a logical unit treatment, such as acres; and
Daily Area Treated	=	Normalized application area based on a logical unit treatment such as acres (A/day).

The daily dermal or inhalation dose is calculated by normalizing the daily exposure by body weight and adjusting, if necessary, with an appropriate dermal or inhalation absorption factor using the following formula:

$$\text{Average Daily Dose (mg/kg/day)} = \text{Daily Exposure (mg ai/day)} \times (\text{Absorption Factor (\%/100)}) / \text{Body Weight (kg)}$$

Where:

Average Daily Dose	=	Absorbed dose received from exposure to a pesticide in a given scenario (mg ai/kg body weight/day);
Daily Exposure	=	Amount (mg ai/day) deposited on the surface of the skin that is available for dermal absorption or amount inhaled that is available for inhalation absorption;
Absorption Factor	=	A measure of the amount of chemical that crosses a

biological boundary such as the skin or lungs (% of the total available absorbed); and
Body Weight = Body weight determined to represent the population of interest in a risk assessment (kg).

Non-cancer dermal and inhalation risks for each applicable handler scenario are calculated using a MOE, which is a ratio of the point of departure (POD) to the daily dose. All MOE values were calculated using the formula below:

$$\text{MOE} = \text{POD (mg/kg/day)} / \text{Average Daily Dose (mg/kg/day)}$$

See **Tables 9.1a - 9.1c** for a summary of estimated exposures and risks. Results from the assessment of all occupational handler scenarios, with the exception of mixer/loader/applicator (MLAP) spot treatments scenarios with mechanically pressurized handgun, indicate that short- and intermediate-term dermal risks are not of concern to the Agency (i.e., MOEs \geq 1000) at level of personal protection recommended on the label (i.e., gloves). Short- and intermediate-term dermal risks MLAP scenarios with mechanically pressurized handgun for spot treatments on olives, citrus, pome, and stone fruit are of potential concern to the Agency (**MOE= 160**) even with the addition of personal protection to mitigate exposure such as an extra layer of clothing (coveralls) and gloves.

Likewise, results from the assessment of all occupational handler scenarios, with the exception of MLAP spot treatments scenarios with mechanically pressurized handgun, indicate that short- and intermediate-term inhalation risks are not of concern to the Agency (i.e., MOEs \geq 300) at baseline level of personal protection (i.e. no respirator). MLAP scenarios, for spot treatments on olives, citrus, pome, and stone fruit, result in short- and intermediate-term dermal risks of potential concern to the Agency (**MOE= 82**) even with the addition of a PF10R respirator.

The formulated end use products labels involved in this assessment indicate user restrictions and state: “*for retail sale to and use only by certified applicators or persons under their supervision and only for the uses covered by the certified applicator’s certification*”. The proposed glufosinate ammonium labels direct applicators and other handlers who might be exposed to the diluted chemical and anything that has been treated, such as plants, soil, or water to wear specific personal protection equipment (PPE). Handlers mixing, loading, and cleaning equipment are instructed to wear a chemical resistant apron. Applicators and other handlers must wear coveralls; chemical resistant gloves and footwear, plus socks, and protective eyewear (goggles, face shield or safety glasses). Mixers and loaders supporting aerial applications must wear a dust mist filtering respirator (MSHA/NIOSH approval number prefix TG21 C), or a NIOSH approved respirator with any N, R, P or HE filter. PPE required for early entry to treated areas that is permitted under the Worker Protection Standard must be used.

The minimum level of PPE for handlers is based on acute toxicity for the end-use product. The Registration Division (RD) is responsible for ensuring that PPE listed on the label is in compliance with the Worker Protection Standard (WPS) for agricultural pesticides.

Table 9.1a Short-/Intermediate Term Agricultural Handler Exposure and Risk Estimates for Glufosinate Ammonium												
Exposure Scenario	Crop or Target	App Rate ^a (lb ai/A)	Acres Treated Daily ^b	Unit Exposure ^c			Dose (mg/kg/Day)			MOEs (Dermal LOC = 1000 Inhalation LOC = 300)		
				Baseline Dermal (mg/lb ai)	PPE-Dermal (mg/lb ai)	Inhalation (ug/lb ai)	Baseline Dermal ^{d,h}	PPE Dermal ⁱ	Inhalation ^e	Baseline Dermal ^f	PPE-Dermal	Inhalation ^{g,i}
Mixer/Loader												
Mixing/Loading to Support Aerial treatments (PHED)	Sweet Corn	0.365	350	0.220	0.0376 (SL/G)	0.219	0.0367	0.00626 (SL/G) 0.00485 (DL/G)	0.00035	380	2,200 (SL/G)	36,000
Mixing/Loading to Support Groundboom Applications (PHED)	Sweet Corn	0.365	80	0.220	0.0376 (SL/G)	0.219	0.00837	0.00143 (SL/G)	0.000079	1,700	9,800 (SL/G)	160,000
	Citrus Fruit, Pome Fruit, Stone Fruit, Olives	1.50	80	0.220	0.0376 (SL/G)	0.219	0.0344	0.00588 (SL/G)	0.000329	410	2,400 (SL/G)	38,000

- a) Application Rates based on proposed uses on label for Glufosinate -ammonium formulations Liberty Herbicide (EPA Reg. No. 264-660) and Liberty 280 SL Herbicide (EPA Reg. No. 264-829). Units expressed in lb ai/Acre unless specified otherwise.
- b) Exposure Science Advisory Council Policy # 9.1
- c) Unit Exposures based on Unit Exposures based on “Occupational Pesticide Handler Unit Exposure Surrogate Reference Table” (PHED) (<http://www.epa.gov/pesticides/science/handler-exposure-table.pdf>), dated September 2011. Engineering control unit exposure for applying sprays via aerial equipment = enclosed cockpit.
- d) Dermal Dose (mg/kg/day) = daily unit exposure (mg/lb ai) x application rate (lb ai/acre) x acres treated x **dermal absorption factor (9%)** / body weight (69 kg adult female).
- e) Inhalation Dose (mg/kg/day) = daily unit exposure (ug/lb ai) x conversion factor (1 mg/1,000 ug) x application rate (lb ai/acre) x acres treated/ body weight (80 kg).
- f) Dermal MOE = LOAEL (14 mg/kg/day) / dermal daily dose (mg/kg/day). Level of concern = 1000.
- g) Inhalation MOE = LOAEL (12.5 mg/kg/day) / inhalation daily dose (mg/kg/day). Level of concern = 300.
- h) Baseline Dermal: Long-sleeve shirt, long pants, and no gloves;
- i) Baseline Inhalation: no respirator
- j) PPE: G (gloves), SL (Single Layer), DL (Double Layer), PF10R (respirator), E.C. (Engineering Control; Closed System).

Table 9.1b Short-/Intermediate Term Agricultural Handler Exposure and Risk Estimates for Glufosinate Ammonium												
Exposure Scenario	Crop or Target	App Rate ^a (lb ai/A)	Acres Treated Daily ^b	Unit Exposure ^c			Dose (mg/kg/Day)			MOEs (Dermal LOC = 1000 Inhalation LOC = 300)		
				Baseline Dermal ^h (mg/lb ai)	PPE Dermal ^{i, g} (mg/lb ai)	Inhalation ^e (ug/lb ai)	Baseline Dermal ^{d,h}	PPE-G Dermal	Inhalation ^e	Baseline Dermal ^{f, h}	PPE-G Dermal	Inhalation ⁱ
Applicator												
Applying Sprays via Aerial Equipment – Enclosed Cockpit (PHED)	Sweet Corn	0.365	350	No Data	0.005 (EC)	0.068 (EC)	No Data	0.000833 (EC)	0.000109 (EC)	No Data	17,000 (EC)	110,000 (EC)
Applying Sprays via Groundboom Equipment Open Cab	Sweet Corn	0.365	80	0.0786	0.0161 (SL/G)	0.34	0.003	0.000613 (SL/G)	0.000124	4,700	23,000 (G)	100,00
	Citrus Fruit, Pome Fruit, Stone Fruit, Olives	1.5	80	0.0786	0.0161 (SL/G)	0.34	0.0123	0.00252 (SL/G)	0.00057	1,100	5,600 (G)	25,000
Flagger												
Flagging for Aerial Sprays	Sweet Corn	0.365	350	0.011	0.012	0.35	.00183	0.0020	0.00064	7,600	7,000 (G)	19,000

- a) Application Rates based on proposed uses on label for Glufosinate -ammonium formulations Liberty Herbicide (EPA Reg. No. 264-660) and Liberty 280 SL Herbicide (EPA Reg. No. 264-829). Units expressed in lb ai/Acre unless specified otherwise.
- b) Exposure Science Advisory Council Policy # 9.1
- c) Unit Exposures based on Unit Exposures based on “Occupational Pesticide Handler Unit Exposure Surrogate Reference Table” (PHED) (<http://www.epa.gov/pesticides/science/handler-exposure-table.pdf>), dated September 2011. Engineering control unit exposure for applying sprays via aerial equipment = enclosed cockpit.
- d) Dermal Dose (mg/kg/day) = daily unit exposure (mg/lb ai) x application rate (lb ai/acre) x acres treated x **dermal absorption factor (9%)** / body weight (69 kg adult female).
- e) Inhalation Dose (mg/kg/day) = daily unit exposure (ug/lb ai) x conversion factor (1 mg/1,000 ug) x application rate (lb ai/acre) x acres treated/ body weight (80 kg).
- f) Dermal MOE = LOAEL (14 mg/kg/day) / dermal daily dose (mg/kg/day). Level of concern = 1000.
- g) Inhalation MOE = LOAEL (12.5 mg/kg/day) / inhalation daily dose (mg/kg/day). Level of concern = 300.
- h) Baseline Dermal: Long-sleeve shirt, long pants, and no gloves;
- i) Baseline Inhalation: no respirator
- j) PPE: G (gloves), SL (Single Layer), DL (Double Layer), PF10R (respirator), E.C. (Engineering Control; Closed System).

Table 9.1c Short-/Intermediate Term Agricultural Handler Exposure and Risk Estimates for Glufosinate Ammonium (Spot/Directed Spray Applications)

Exposure Scenario	App Rate ^a (lb ai/gal)	Acres Treated Daily ^b	Unit Exposure ^{c,j}			Dose (mg/kg/Day)			MOEs (Dermal LOC = 1000 Inhalation LOC = 300)		
			Baseline Dermal ^h (mg/lb ai)	PPE Dermal ^j (mg/lb ai)	Inhalation (ug/lb ai)	Baseline Dermal ^{d,h}	PPE Dermal ^j	Inhalation ^e	Baseline Dermal ^{f,h}	PPE Dermal ^j	Inhalation ^{g,i}
Citrus Fruit, Pome Fruit, Stone Fruit, Olives											
Mixer/Loader/Applicator											
Mixing/Loading/Applying (MLAP) with Mechanically Pressurized Handgun (spot/ directed spray)	1.7 fl oz/gal 0.031 lbai/gal	1,000 gallons	4.310	2.160 (DL/G)	3931 (No respirator) 393.1 (PF10R)	0.175	0.0874 (DL/G)	0.0004 0.153 (PF10R)	80	160 (DL/G)	8.2 82 (PF10R)
Mixing/Loading/Applying (MLAP) with Backpack Sprayer (spot/ directed spray)		40 gallons	8.260	8.260	2.58	0.0133	0.0133	0.0004	1,100	1,100 (SL/G)	310,000

- a) Application Rates based on proposed uses on label for Glufosinate -ammonium formulations Liberty Herbicide (EPA Reg. No. 264-660) and Liberty 280 SL Herbicide (EPA Reg. No. 264-829). Units expressed in lb ai/Acre unless specified otherwise.
- b) Exposure Science Advisory Council Policy # 9.1
- c) Unit Exposures based on Unit Exposures based on “Occupational Pesticide Handler Unit Exposure Surrogate Reference Table” (PHED) (<http://www.epa.gov/pesticides/science/handler-exposure-table.pdf>), dated September 2011. Engineering control unit exposure for applying sprays via aerial equipment = enclosed cockpit.
- d) Dermal Dose (mg/kg/day) = daily unit exposure (mg/lb ai) x application rate (lb ai/acre) x acres treated x **dermal absorption factor (9%)** / body weight (69 kg adult female).
- e) Inhalation Dose (mg/kg/day) = daily unit exposure (ug/lb ai) x conversion factor (1 mg/1,000 ug) x application rate (lb ai/acre) x acres treated/ body weight (80 kg).
- f) Dermal MOE = LOAEL (14 mg/kg/day) / dermal daily dose (mg/kg/day). Level of concern = 1000.
- g) Inhalation MOE = LOAEL (12.5 mg/kg/day) / inhalation daily dose (mg/kg/day). Level of concern = 300.
- h) Baseline Dermal: Long-sleeve shirt, long pants, and no gloves;
- i) Baseline Inhalation: no respirator
- j) PPE: G (gloves), SL (Single Layer), DL (Double Layer), PF10R (respirator), E.C. (Engineering Control; Closed System).

9.2 Occupational Post-Application Risk

Inhalation Post-Application Risk

Based on the Agency's current practices, a quantitative occupational post-application inhalation exposure assessment was not performed for glufosinate ammonium. However, there are multiple potential sources of post-application inhalation exposure to individuals performing post-application activities in previously treated fields. These potential sources include volatilization of pesticides and resuspension of dusts and/or particulates that contain pesticides. The Agency sought expert advice and input on issues related to volatilization of pesticides from its Federal Insecticide, Fungicide, and Rodenticide Act Scientific Advisory Panel (SAP) in December 2009. The Agency received the SAP's final report on March 2, 2010 (<http://www.epa.gov/scipoly/SAP/meetings/2009/120109meeting.html>). The Agency is in the process of evaluating the SAP report as well as available post-application inhalation exposure data generated by the Agricultural Reentry Task Force and may, as appropriate, develop policies and procedures, to identify the need for and, subsequently, the way to incorporate occupational post-application inhalation exposure into the Agency's risk assessments. If new policies or procedures are put into place, the Agency may revisit the need for a quantitative occupational post-application inhalation exposure assessment for glufosinate ammonium. Although a quantitative occupational post-application inhalation exposure assessment was not performed, an inhalation exposure assessment was performed for occupational/commercial handlers. Handler exposure resulting from mixing/loading pesticides is likely to result in higher exposure than post-application exposure. Thus, handler inhalation exposure estimates would be protective of most occupational post-application inhalation exposure scenarios.

Dermal Post-Application Risk

A quantitative post-application dermal exposure assessment was not performed for glufosinate ammonium uses on citrus fruit, pome fruit, stone fruit, and olives. There is a low potential for occupational post-application exposure associated with the use of ground and spot-directed application of herbicides. Most of the proposed uses for glufosinate ammonium are ground-directed uses where crop foliage treatment should be avoided. Currently, HED has no transfer coefficients (TCs) or other data to assess post-application dermal exposures to soil by occupational workers. Therefore, for the proposed soil-directed uses, post-application exposures and risks to occupational workers were not quantitatively assessed for these crops. In general, such exposures are considered to be minimal.

Even though the proposed uses for glufosinate ammonium are for post-emergent applications when crop foliage is present, dislodgeable foliar residues are expected to be very low during post-application activities since the proposed label advises applicators to avoid contact of Liberty® 280 SL Herbicide with foliage or parts of trees, other than mature brown bark as serious injury may occur. The label specifies a preharvest interval of 14 days for citrus fruit, pome fruit, stone fruit, and olive crops.

There is potential for post-application dermal exposure to glufosinate ammonium following the proposed use on sweet corn. Occupational re-entry workers may experience short-/intermediate-term exposure to glufosinate ammonium while performing post-application activities such as scouting and irrigation of sweet corn. Post-application exposure resulting from detasseling activities was not assessed since it is estimated that this activity would occur too late after the last application permitted for the herbicide (ExpoSAC meeting August 5, 2010). Therefore, post-application dermal exposure scenarios for the proposed new uses of glufosinate ammonium on sweet corn were assessed for scouting and irrigation activities only. The post-application activity scenarios along with respective TCs and risk estimates for short-/intermediate-term MOEs are summarized in **Table 9.2**.

It is the policy of HED to use the best available data to assess post-application exposure. Since no chemical-specific dislodgeable foliar residue (DFR) data were submitted in support of this registration action, dermal transfer coefficients (TCs) were used to relate foliage residue values to activity patterns (e.g., scouting) to estimate potential human exposure. Sources of generic post-application data, used as surrogate data in the absence of chemical-specific data, are derived from Agricultural Reentry Task Force (ARTF) exposure monitoring studies, and, as proprietary data, are subject to the data protection provisions of FIFRA. The standard values recommended for use in predicting post-application exposure that are used in this assessment, known as “transfer coefficients”, are presented in the “Science Advisory Council for Exposure (ExpoSAC) Policy 3” (http://www.epa.gov/pesticides/science/exposac_policy3.pdf), which, along with additional information about the ARTF data, can be found at <http://www.epa.gov/pesticides/science/post-app-exposure-data.html>. It is also assumed that a fraction of ai (25% of the rate of application) is available as dislodgeable foliar residue on day zero after initial treatment with a continued dissipation rate of 10% per day on following days.

The following equations were used to calculate risks for workers performing post-application activities:

Dislodgeable Foliar Residue

$$DFR_t (\mu\text{g}/\text{cm}^2) = AR (\text{lb ai}/\text{acre}) \times F \times (1-D)^t \times 4.54\text{E}8 \mu\text{g}/\text{lb} \times 2.47\text{E}-8 \text{ acre}/\text{cm}^2$$

Where:

- DFR_t = dislodgeable foliage residue on day "t" (μg/cm²),
- AR = application rate (lb ai/acre),
- F = fraction of ai retained on foliage (unitless), and
- D = fraction of residue that dissipates daily (unitless).

Daily Dose

Daily dose (dermal) is calculated by normalizing the daily exposure value by body weight and accounting for absorption factors:

$$DD_t (\text{mg}/\text{kg}\text{-day}) = \frac{DFR_t (\mu\text{g}/\text{cm}^2) \times 1\text{E}-3 \text{ mg}/\mu\text{g} \times TC (\text{cm}^2/\text{hr}) \times DA (9\%) \times ET (\text{hrs})}{BW (\text{kg})}$$

Where:

- DD_t = daily dermal dose on day “t,”
- t = number of days after application day (days),
- DFR_t = dislodgeable foliage residue on day "t" (μg/cm²),
- TC = transfer coefficient (cm²/hr),
- DA = dermal absorption factor (unitless),
- ET = exposure time (hr/day), and
- BW = body weight (kg).

Margin of Exposure (MOE)

The daily dermal dose received by occupational handlers was compared to the appropriate PoD (i.e. LOAEL) to assess the risk to occupational handlers. All MOE values were calculated using the following formula:

$$MOE = \frac{LOAEL (\text{mg}/\text{kg}/\text{day})}{ADD (\text{mg}/\text{kg}/\text{day})}$$

Where:

- MOE = Margin Of Exposure (unitless),
- ADD = Average Daily Dose (mg/kg/day), and
- LOAEL = Lowest Observed Adverse Effect Level (mg/kg/day)

The results of the glufosinate ammonium post-application exposure and dermal risk assessment for sweet corn indicate that an MOE of 1,200 is achieved on Day 0 for scouting mature/high foliage plants. However, for irrigation activities, an acceptable MOE of 1,050 is not achieved until Day 4; therefore, this post-application activity is of potential concern to the Agency. The proposed Liberty® Herbicide label indicates an REI of 12 hrs; thus, this timing needs revision based upon the post-application exposure estimates obtained for sweet corn to achieve an MOE \geq 1000. The proposed label states a preharvest interval of 50 days for sweet corn. The post-application activity scenarios along with respective TCs and risk estimates for short-/intermediate-term MOEs are summarized in Table 9.2.

Crop	Maximum Application Rate^a	DAT^b	DFR^c	TC^d	Activity^d	Short-/Int-Term MOE^e
Sweet corn	0.365	0	1.024	1,100	Scouting mature/high foliage plants	1,200
		0	1.024	1,900	Irrigation mature/high foliage plants	690
		4	0.672	1,900	Irrigation mature/high foliage plants	1,050

- a) Maximum application rate (lb ai/A) indicated on proposed labels Reg.No.279-3313, 279-3108.
- b) DAT = Days after treatment needed to reach the LOC of 1000; DAT 0 = the day of treatment, after sprays have dried; assumed to be approximately 12 hours.
- c) DFR ($\mu\text{g}/\text{cm}^2$) = Application rate (lb ai/A) \times Fraction of ai retained on foliage on day zero (25%) \times (1 - fraction of residue that dissipates daily 10%)^t \times 4.54E8 $\mu\text{g}/\text{lb}$ \times 2.47E-8 acre/ cm^2 .
- d) TC (cm^2/hr) = Transfer coefficients and associated activities from ExpoSAC Policy 3” (http://www.epa.gov/pesticides/science/exposac_policy3.pdf)
- e) MOE = MOE on the corresponding DAT. MOE = LOAEL / Daily Dose.
Daily Dose = [DFR ($\mu\text{g}/\text{cm}^2$) \times Transfer Coefficient \times 0.001 mg/ μg \times 8 hrs/day \times dermal absorption 9 %] \div body weight (69 kg adult).

The toxicity categories of the active ingredient for acute dermal, eye irritation, and skin irritation potential are used to determine the interim REI (Restricted-entry Intervals). The glufosinate ammonium technical material has been classified in Toxicity Category III for acute dermal and primary eye irritation. It is not a dermal irritant (toxicity category IV) nor is it a dermal sensitizer. Per the Worker Protection Standard (WPS), a 12-hr restricted entry interval (REI) is required for chemicals classified under Toxicity Category III or IV. Both labels indicate an REI of 12 hrs, which is in compliance with the WPS for most post-application activities. However, an REI of 4 days is needed based upon the post-application exposure estimates obtained for sweet corn to achieve the target MOE \geq 1000.

10.0 References

Endpoint Selection Documents

Glufosinate ammonium – 3rd Report of the Hazard Identification Assessment Review Committee; B. Tarplee, 4/17/2003.

Glufosinate ammonium – Review of Developmental Neurotoxicity Study in Rats (MRID 46455701); DP#: 312684, R.J. Mitkus, 8/24/2005.

Glufosinate ammonium – Review of 28-Day Inhalation Toxicity Study (MRID 47058101); DP#: 337298, L. Austin, 9/6/2007.

Glufosinate ammonium – Request to Waive Requirement for Glutamine Synthetase Measurements and Other Data Requirements; DP#: 328229, L. Austin, TXR No. 0054838, 4/8/2008.

Glufosinate ammonium – ToxSAC Meeting on June 10, 2010; J. Kidwell, 6/29/2010.

Risk Assessments

Glufosinate Ammonium (PC Code 128850). Section 3 Registrations for Transgenic Cotton and Cotton (ID# - 0F06140), Transgenic Rice (ID# - 0F06210), and Bushberry (ID# - 2E06404). Human Health Risk Assessment. DP#: 290086; T. Bloem, PV Shah, and M. Dow, 8/7/2003.

Glufosinate Ammonium. Human Health Risk Assessment in Support for the Proposed New Use of Glufosinate Ammonium in/on Citrus Fruits (Crop Group 10), Pome Fruits (Crop Group 11), Stone Fruits (Crop Group 12), Olives, and Sweet Corn; DP#: 372623, W. Donovan et. al., 12/15/2010.

Dietary Exposure Memoranda

Glufosinate Ammonium. Update of the Revised Acute and Chronic Aggregate Dietary (Food and Drinking Water) Exposure Assessment in Support of the Petition Proposing Tolerances for Residues of Glufosinate Ammonium in/on Citrus Fruits (Crop Group 10), Pome Fruits (Crop Group 11), Stone Fruits (Crop Group 12), Olives, and Sweet Corn; DP#: 402741, W. Donovan, 6/19/2012.

Drinking Water Memoranda

Tier II Drinking Water Assessment for Glufosinate-ammonium on Stone Fruit, Citrus, Pome Fruit, and Olives; DP#: 372624; C. Peck; 6/22/2010.

Tier II Drinking Water Assessment for Glufosinate-ammonium on Sweet Corn; DP#: 368799; C. Peck; 7/7/2010.

Refined Drinking Water Assessment for Glufosinate-ammonium Use on Rice; DP#: 387412; C. Peck; 5/30/2012.

Residue Chemistry Data Review

Glufosinate Ammonium. Petitions for the Establishment of Permanent Tolerances in/on Citrus Fruits (Crop Group 10), Pome Fruits (Crop Group 11), Stone Fruits (Crop Group 12), Olives and Sweet Corn. Summary of Analytical Chemistry and Residue Data. DP#: 372625; I. Negrón-Encarnación; 8/31/2010.

Occupational and Residential Exposure Assessment

Glufosinate-Ammonium; Occupational and Residential Risk Assessment to Support Request for Section 3 Registrations on Sweet Corn, Citrus Fruit, Pome Fruit, Stone Fruit, and Olives. DP#: 368798, A. Rivera-Lupiáñez, 8/31/2010.

Appendix A

A.1 Toxicology Data Requirements

The requirements (40 CFR 158.340) for food uses of glufosinate ammonium are in Table 1. Use of the new guideline numbers does not imply that the new (1998) guideline protocols were used.

Study	Technical	
	Required	Satisfied
870.1100 Acute Oral Toxicity.....	yes	yes
870.1200 Acute Dermal Toxicity.....	yes	yes
870.1300 Acute Inhalation Toxicity.....	yes	yes
870.2400 Acute Eye Irritation.....	yes	yes
870.2500 Acute Dermal Irritation	yes	yes
870.2600 Skin Sensitization	yes	yes
870.3100 90-Day Oral Toxicity in Rodents	Yes	Yes
870.3150 90-Day Oral Toxicity in Nonrodents.....	Yes	See Chronic
870.3200 21/28-Day Dermal Toxicity	yes	Yes
870.3250 90-Day Dermal Toxicity	No	--
870.3465 90 -Day Inhalation Toxicity (28 days)	Yes	Yes
870.3700a Prenatal Developmental Toxicity (rodent)	Yes	Yes
870.3700b Prenatal Developmental Toxicity (nonrodent)	Yes	Yes
870.3800 Reproduction and Fertility Effects	yes	Yes
870.4100a Chronic Toxicity (rodent).....	Yes.	See combined.
870.4100b Chronic Toxicity (nonrodent).....	Yes	Yes
870.4200a Carcinogenicity (rat).....	Yes	Yes
870.4200b Carcinogenicity (mouse)	Yes	Yes
870.4300 Combined Chronic Toxicity/Carcinogenicity	Yes	Yes
870.5100 Mutagenicity—Bacterial Reverse Mutation Test.....	Yes	Yes
870.5300 Mutagenicity—Mammalian Cell Gene Mutation Test..	Yes	Yes
870.5395 Mutagenicity—Structural Chromosomal Aberrations ..	Yes	Yes ¹
870.5395 Mutagenicity – in vivo mammalian cytogenetics	Yes	Yes
870.5500 Mutagenicity—bacterial DNA damage/repair test	No	Yes
870.5550 Mutagenicity –unscheduled DNA synthesis	No	Yes
870.6200a Acute Neurotoxicity Screening Battery (rat)	Yes	Yes
870.6200b 90-Day Neurotoxicity Screening Battery (rat)	Yes	Yes ²
870.6300 Developmental Neurotoxicity	Yes	Yes
870.7485 Metabolism and Pharmacokinetics.....	Yes	Yes
870.7600 Dermal Penetration.....	Yes	Yes
870.7800 Immunotoxicity	Yes	Yes

1 Although the structural chromosome aberration study is listed as being required, it is no longer considered necessary for glufosinate ammonium because there are two additional studies (bacterial DNA damage/repair test and unscheduled DNA synthesis) and because glufosinate is classified as "not likely" to be a carcinogen in humans.

2 The submitted study was classified as unacceptable/guideline but demonstrated neurotoxicity at high doses. A developmental neurotoxicity study classified as acceptable set the LOAEL at a low level; thus, a repeat of the subchronic neurotoxicity screening study is unnecessary.

A.2 Toxicology Profile

Table 1: Acute Toxicity of Glufosinate ammonium Technical

Guideline No.	Study Type	MRID No.	Results	Toxicity Category
81-1	Acute Oral	00142430, 00142431, 00142432	LD ₅₀ = 4010 mg/kg in males LD ₅₀ = 3030 mg/kg in females	III
81-2	Acute Dermal	00142436, 00142437	LD ₅₀ = >2000 mg/kg in males & females	III
81-3	Acute Inhalation	00151496, 00151497	LC ₅₀ = 4.42 m/liter estimated in males & females	III
81-4	Primary Eye Irritation	00142438	eye irritant, corneal opacity reversible within 72 hours	III
81-5	Primary Skin Irritation	00142438	not a dermal irritant	IV
81-6	Dermal Sensitization	00142439	not a dermal sensitizer	N/A

Table 2: Toxicity Profile of Glufosinate Ammonium Technical

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
870.3100 90-Day oral toxicity rodents-rat (range-finding study)	45179103 (2000) Acceptable/nonguideline 0, 100 or 1000 ppm, Glufosinate 0, 6.2-8.8, or 64-90 mg/kg/day (males only) 0, 1000 or 10,000 ppm, N.acetyl-L-glufosinate 0, 65-90, or 657-935 mg/kg/day (males only)	<u>Glufosinate ammonium</u> NOAEL = 6.2-8.8 mg/kg/day in males LOAEL = 64-90 mg/kg/day in males, based on glutamine synthetase inhibition in the brains <u>N-acetyl-L-glufosinate disodium</u> NOAEL = 65-90 mg/kg/day in males LOAEL = 657-935 mg/kg/day in males, based on glutamine synthetase inhibition in the brains
870.3100 90-Day oral toxicity rodents-mouse	40345609 (1986) Acceptable/guideline 0, 80, 320 or 1,280 ppm; 0, 12, 48 or 192 mg/kg/day	NOAEL = 48 mg/kg/day in males, 192 mg/kg/day in females (HDT) LOAEL = 192 mg/kg/day in males, not achieved in females; based on the changes in clinical biochemistry and liver weights in males
870.3200 Repeated Dose Dermal Toxicity-rat	40345605 (1985) Acceptable/guideline 0, 100, 300 or 1000 mg/kg/day	NOAEL= 100 mg/kg/day LOAEL= 300 mg/kg/day based on clinical observations (aggressive behavior, piloerection, and a high startle response)
870.3465 Subchronic inhalation- 28-day repeat dosing - rat	40345606 (1985) Supplementary 0, 8, 20 or 46 mg/m ³ (estimated 0, 2.2, 5.5 or 12.6 mg/kg/day).	NOAEL = 8 mg/m ³ (estimated 2.2 mg/kg/day) LOAEL = 20 mg/m ³ (estimated 5.5 mg/kg/day) based on clinical signs, decreased lung weight, increased body weight (female), tono-clonic convulsions (2 males). At the highest dose, 4 deaths.
879.3465 Subchronic inhalation- 28-day repeat dosing - rat	47058101 (2007) Acceptable/Non-guideline 0, 56 or 105 mg/m ³ , or an estimated 0, 12.5 or 25 mg/kg/day	LOAEL = 0.56 mg/m ³ (estimated 12.5 mg/kg/day) – lung/bronchial congestion in females, some clinical signs (isolated and transient). Liver and kidney weights.
870.3700a Prenatal developmental in rodents- rat	00142445, 00142446 (1982) 0, 0.50, 2.24 or 10 mg/kg/day 00151499, 00151500 (1982) 0, 0.50, 2.24 or 10 mg/kg/day 0, 10, 50 or 250 mg/kg/day 40345610 (1986) 0, 0.5, 2.24 or 10.0 mg/kg/day All three studies combined Acceptable/guideline	<u>Maternal</u> : NOAEL = 10 mg/kg/day LOAEL = 50 mg/kg/day based on vaginal bleeding and hyperactivity <u>Developmental</u> : NOAEL = 50 mg/kg/day LOAEL =250 mg/kg/day based on dilated renal pelvis
870.3700b Prenatal developmental in nonrodents- rabbit	40345611, 41144703 (1984) Acceptable/guideline 0, 2.0, 6.3 or 20.0 mg/kg/day	<u>Maternal</u> : NOAEL = 6.3 mg/kg/day LOAEL = 20.0 mg/kg/day based on reduced food consumption, body weight and weight gains <u>Developmental</u> : NOAEL = 6.3 mg/kg/day LOAEL = 20.0 mg/kg/day based on decreased body weights and fetal death
870.3800 Reproduction and fertility effects- rat	40345612 (1988) Acceptable/guideline 0, 40, 120 or 360 ppm 0, 2.0, 6.0, or 18.0 mg/kg/day	Parental/Systemic NOAEL = 18.0 mg/kg/day (HDT). LOAEL = not established Reproductive NOAEL = 6.0 mg/kg/day LOAEL = 18.0 mg/kg/day based on decreased number of viable pups

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
		Offspring NOAEL = 6.0 mg/kg/day LOAEL = 18.0 mg/kg/day based on decreased number of viable pups
870.3700b Prenatal developmental in nonrodents- rabbit	40345611, 41144703 (1984) Acceptable/guideline 0, 2.0, 6.3 or 20.0 mg/kg/day	Maternal: NOAEL = 6.3 mg/kg/day LOAEL = 20.0 mg/kg/day based on reduced food consumption, body weight and weight gains Developmental: NOAEL = 6.3 mg/kg/day LOAEL = 20.0 mg/kg/day based on decreased body weights and fetal death
870.3800 Reproduction and fertility effects- rat	40345612 (1988) Acceptable/guideline 0, 40, 120 or 360 ppm 0, 2.0, 6.0, or 18.0 mg/kg/day	Parental/Systemic NOAEL = 18.0 mg/kg/day (HDT). LOAEL = not established Reproductive NOAEL = 6.0 mg/kg/day LOAEL = 18.0 mg/kg/day based on decreased number of viable pups Offspring NOAEL = 6.0 mg/kg/day LOAEL = 18.0 mg/kg/day based on decreased number of viable pups
870.4100b Chronic toxicity- dog	40345608 (1984) Acceptable/guideline 0, 2.0, 5.0 or 8.5 mg/kg/day	NOAEL = 5.0 mg/kg/day LOAEL = 8.5 mg/kg/day based on mortality (week 2) and alterations in the electrocardiogram at 6 months
870.4200 Carcinogenicity- rat	44539501 (1989) Acceptable/guideline 0, 1000, 5000 or 10000 ppm 0/0, 45.4/57.1, 228.9/281.5, or 466.3/579.3 mg/kg/day, M/F	NOAEL = 45.4 mg/kg/day in males, 57.1 mg/kg/day in females LOAEL = 228.9 mg/kg/day in males and 281.5 based on increased incidences of retinal atrophy No evidence of carcinogenicity.
870.4300 Chronic/ Carcinogenicity- rat	40345607, 41144701 (1986) Acceptable/guideline 0, 40, 140 or 500 ppm 0/0, 1.9/2.4, 6.8/8.2, or 24.4/28.7 mg/kg/day, M/F	NOAEL = 24.4 mg/kg/day in males, 8.2 mg/kg/day in females LOAEL = not achieved in males and 28.7 based on inhibition of brain glutamate synthetase in females at 130 weeks No evidence of carcinogenicity.
870.4300 Carcinogenicity- mice	40345609, 41144702 (1986) Acceptable/guideline 0, 20, 80, 160 (males only) or 320 (females only) ppm 0/0, 2.83/4.23, 10.82/16.19 or 22.60/66.96 mg/kg/day, M/F	NOAEL = 10.82 mg/kg/day in males, 16.19 mg/kg/day in females LOAEL = 22.60 mg/kg/day in males, 63.96 mg/kg/day in females based on increased mortality and glucose levels and consistent changes in glutathione levels in males, increased glucose levels and decreased albumin and total proteins No evidence of carcinogenicity.
870.5265 Reverse Mutation Assay	Accession No. 072962 (1984) Acceptable/guideline 0, 5, 10, 50, 100, 500, and 1000 µg/plate	In a bacterial cell gene reverse mutation assay Salmonella typhimurium strains TA98, TA100, TA1535 and TA1537 were exposed to glufosinate ammonium (92.1% a.i.) at concentrations of 0, 5, 10, 50, 100, 500, and 1000 µg/plate in the presence and absence of mammalian metabolic activation (S9-mix). No increases in mutation frequencies, with or without metabolic activation, were noted in any of the test strains

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
		at any of the doses tested. Virtually total inhibition of growth was noted in all strains at the highest dose, 1000 µg/plate. Therefore, the requirement that chemicals be tested to the limits of cytotoxicity was satisfied. The positive controls, 2-aminoanthracene, AF-2, 1-ethyl-2-nitro-3-nitroso-guanidine, 9-amino-acridine, and 2-nitro-fluorine, induced the appropriate responses. Therefore the test systems were sensitive to agents that induce gene mutation. Under the conditions of the test, glufosinate-ammonium failed to cause reverse mutations in bacteria with and without metabolic activation.
870.5300 Detection of gene mutations in somatic cells in culture	40445616 (1988) Acceptable/guideline 50 to 5000 µg/mL 300 to 5000 µg/mL (S9-activated doses).	In a mouse lymphoma L5179Y forward mutation assay, HOE 039866 was tested at seven nonactivated doses of 50 to 5000 µg/mL or at six S9-activated doses of 300 to 5000 µg/mL. HOE 39866 did not increase the mutation frequency at the thymidine kinase locus. The solvent controls gave acceptable values and the positive controls ethylmethanesulfonate (nonactivated) and 3-methylcholanthrene (S9-activated) provided evidence that the assay had adequate sensitivity for detecting mutagenicity.
870.5395 In vivo mammalian cytogenetic tests	41144704 (1986) Acceptable/guideline 100, 200, and 350 mg/kg by gavage	In a mouse micronucleus assay 13 groups of mice (5/sex/dose) received a single administration of HOE 039866 at dose levels of 100, 200, and 350 mg/kg by gavage. A positive control group received 50 mg/kg of cyclophosphamide. After dosing, the animals were sacrificed at 24, 48, and 72 hrs., and the erythrocytes from the bone marrows were sampled at these times. The results indicated the test agent had no effect on micronucleus formation.
870.5500 Bacterial DNA damage or repair test	Accession No. 072962 (1984) Acceptable/guideline 0, 50, 100, 500, 1000, 5000 or 10,000 µg/plate.	In a DNA damage/repair assay, glufosinate ammonium was exposed overnight to B. subtilis that lacks the capacity for repair (H45) at concentrations of 0, 50, 100, 500, 1000, 5000 or 10,000 µg/plate. Glufosinate ammonium was also exposed, at the same dose levels, to an isogenic sister strain which has the capacity for DNA repair (H17). Under the conditions of the study, no difference in the inhibition of growth between these two strains was noted at any of the doses tested. Since the test measures the inhibition of growth in response to the test article, the requirement that chemicals be tested to the limits of cytotoxicity was satisfied. The positive controls, 2-(2-furyl)-3-(5-nitro-2-furyl)acrlamide (AF-2), caused a differential growth inhibition, whereas the negative controls (NaOH, HCL, and Kanamycin) produced no significant difference in growth inhibition. The test system was therefore sensitive to agents that damage DNA. Under the conditions of the test, the test article failed to cause damage to DNA that could be detected by this repair assay.
870.5550 Unschedule DNA synthesis in	40345614 (1984) Acceptable/guideline 0.1 to 5240 µg/mL	In an unscheduled DNA synthesis assay (MRID 40345614), primary rat hepatocyte cultures were exposed to HOE 039886 in deionized water at 15

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
mammalian cells in culture		concentrations ranging from 0.1 to 5240 µg/mL for 18 - 19 hours. HOE 039866 was tested up to cytotoxic concentrations as evidenced by decreased survival rate as low as 34% There was no evidence that unscheduled DNA synthesis was induced by the test material.
870.6200 Acute Neurotoxicity -rat	45190704 (1999) Acceptable/nonguideline 0, 10, 100 or 500 mg/kg	NOAEL= ≥500 mg/kg in males and females LOAEL= Not established in both sexes
870.6200 Acute Neurotoxicity -rat	45190703 (1999) Acceptable/nonguideline 0, 10, 100 or 500 mg/kg	NOAEL= ≥500 mg/kg in males and females LOAEL= Not established in both sexes
870.6200b Repeat Dose Neurotoxicity-rat (37-day study)	45179101, 45179102, 45297001 (2000) Acceptable/nonguideline 0, 20, 200 or 2000 ppm 0/0, 1.5/1.8, 14.9/17.1 or 143.3/161.5 mg/kg/day, M/F	NOAEL= 1.5 mg/kg/day in males, 1.8 mg/kg/day in females LOAEL= 14.9 mg/kg/day in males, 17.1 mg/kg/day in females, based on the inhibition of glutamate synthetase in the brain
870.6200b Repeat Dose Neurotoxicity-rat (90-day study)	42768201 (1993) Unacceptable/guideline 0, 7500, 10000 or 20000 ppm 0/0, 521.45/573.79, 685.95/740.57 or 1351.09/1442.64 mg/kg/day, M/F	NOAEL= Not established LOAEL= 521.45 mg/kg/day in males, 573.79 mg/kg/day in females based on increases in the incidence of decreased exploratory activity, decreased alertness, decreased startle response and meiosis
870.6300. Developmental Neurotoxicity study – rats.	46455701 (2004) Acceptable/Non-guideline 0, 14, 69 or 292 mg/kg/day (during gestation)	Maternal: NOAEL = 69 mg/kg/day. LOAEL = 292 mg/kg/day based on decreased body weight, body weight gain and food consumption during gestation and lactation. Developmental: NOAEL – Not established. LOAEL = 14 mg/kg/day based on altered brain morphogenics.
870.7485 Metabolism and pharmacokinetics - rat	43766913 (1993) Acceptable/nonguideline 2.0 mg/kg single dose	In a metabolism study (85-1), groups of Wistar rats (5/sex) received a single dose (2 mg/kg) of 14C-Hoe 039866 (glufosinate ammonium) by gavage. The majority of the radioactivity (95-98% of the dose) was eliminated during the first 24 hrs after dosing. The parent compound, Hoe 039866, accounted for most of the eliminated radioactivity in the urine and feces of both males (80% of the dose) and females (73% of the dose). The metabolite, Hoe 061517, was consistently found in both urine and feces of both sexes. Hoe 099730 (7-8% of the dose) and Hoe 042231 (≈3% of the dose) were found in the feces of both male and female rats and none in the urine.
870.7485 Metabolism and pharmacokinetics - rat	43766914, 43778402 (1995) Acceptable/nonguideline 500 mg/kg single dose	In a metabolism study, groups of Wistar rats (5/sex or 2/sex) received a single dose (500 mg/kg) of 14C-Hoe 039866 (glufosinate ammonium) by gavage. Animals were sacrificed at various times (2, 6, 24, and 96 hrs) after dosing. The majority of the radioactivity was eliminated during the first 24 to 48 hrs after dosing. The parent compound, Hoe 039866, accounted for the majority of the radioactivity eliminated in the excreta of both males (≈80% of the dose) and females (88% of the dose). This finding is consistent with the results of a

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
		previous metabolism studies (MRID No. 40345638 and MRID No. 43766913). The metabolite, Hoe 061517, was consistently found in both urine (0.22-1.20% of the dose) and feces (0.44-1.36% of the dose) of both sexes. Hoe 099730 was found in feces (0.28-1.72% of the dose) of both male and female rats and barely above or at the level of the detection in the urine of both sexes (0.02-0.04% of the dose). Hoe 042231 was mainly found in the feces of both male and females (\approx 0.2-0.28% of the dose). Very little if any of administered Hoe 039866 was sequestered in any tissues examined.
870.7485 Metabolism and pharmacokinetics - rat	40345640 (1985) Acceptable/nonguideline 30 mg/kg single dose	Groups of Wistar rats (5/sex) were orally administered a single nominal dose (30 mg/kg) of 14C-HOE 039866. Rapid elimination during the first 24 hr for both males and females was observed. The major route of excretion was via feces (88% and 84% of the administered radioactivity for males and females, respectively). Within seven days of post dosing, greater than 94% of the dose was eliminated. Kinetics analysis indicated that the process of excretion was a two-phase process. The tissue radioactivity level for kidneys, liver and gonads was just above the background level.
870.7485 Metabolism and pharmacokinetics - rat	43766913 (1993) Acceptable/nonguideline 2.0 mg/kg single dose	In a metabolism study (85-1), groups of Wistar rats (5/sex) received a single dose (2 mg/kg) of 14C-Hoe 039866 (glufosinate ammonium) by gavage. The majority of the radioactivity (95-98% of the dose) was eliminated during the first 24 hrs after dosing. The parent compound, Hoe 039866, accounted for most of the eliminated radioactivity in the urine and feces of both males (80% of the dose) and females (73% of the dose). The metabolite, Hoe 061517, was consistently found in both urine and feces of both sexes. Hoe 099730 (7-8% of the dose) and Hoe 042231 (\approx 3% of the dose) were found in the feces of both male and female rats and none in the urine.
870.7485 Metabolism and pharmacokinetics - rat	40345642 (1985) Acceptable/nonguideline 2.0 mg/kg/day (repeat dose 14 days)	Groups of Wistar rats (6/sex) were orally administered (gavage) unlabeled HOE 039866 for 14 days and 14C-HOE039866 at the 15th day at a nominal dose of 2 mg/kg. The majority of the radioactivity was excreted within 24 hr after the last dose. The major route of elimination was via feces. There was also a two-phased elimination process. More radioactivity was found in the tissues of animals dosed repeatedly than that of animals receiving a single dose.
870.7600 Dermal Penetration- rat	40345620 (1986) Acceptable/guideline 0, 0.1, 1.0 or 10.0 mg/6cm ²	The results indicate that at the low dose (0.1 mg) 42.5 to 50.8% of the applied radioactivity was absorbed whereas at the high dose (10 mg) 26% was absorbed. After removal and washing of the treated skin a substantial amount of the radioactivity still remained in the skin, and it was gradually absorbed and eliminated. Radioactivity was found in both feces and urine samples, but the majority of HOE 039866 was eliminated in the urine. In all organs/tissues examined, radioactivity was found to reach a maximum level either at four or 10 hr after exposure. Subsequently, the radioactivity dropped rapidly. The amount of radioactivity found in the brain was very minimal relative to that of kidneys and liver.

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
870.7600. Dermal penetration – rat	45922103 (1995) Acceptable/Guideline 12, 116 or 1218 µg/cm ² .	A dermal absorption factor of 9% can be supported based on similar total absorbed and remaining on the skin at 10 and 24 hours
HOE 061517 Metabolite		
870.3100 90-Day oral toxicity rodents-rat	44076206 (1988) Acceptable/guideline) 0, 400, 1600 or 6400 ppm 0/0, 30/32, 102/113 or 420/439 mg/kg/day M/F	NOAEL = 102 mg/kg/day in males, 113 mg/kg/day in females LOAEL = 420 mg/kg/day in males, 439 mg/kg/day in females based on increased in reticulocytes and increased in absolute and relative liver weights in males
870.3100 90-Day oral toxicity rodents-mice	44076207 (1989) Acceptable/guideline) 0, 320, 1600, 3200 or 8000 ppm 0/0, 46/47, 209/220, 496/561 or 1121/1340 mg/kg/day M/F	NOAEL = 1121 mg/kg/day in males, 1340 mg/kg/day in females LOAEL = Not established
870.3700a Prenatal developmental in rodents- rat	44076209 (1994) Acceptable/nonguideline 0, 100, 300 or 900 mg/kg/day	<u>Maternal</u> : NOAEL = 300 mg/kg/day LOAEL = 900 mg/kg/day based on one death and clinical findings (persistent piloerection and/or increased urinary output) <u>Developmental</u> : NOAEL = 300 mg/kg/day LOAEL =900 mg/kg/day based on increases in the incidences of total litter loss and in the fetal and litter incidences of wavy and/or thickened ribs
870.3700b Prenatal developmental in nonrodents- rabbit	44076210 (1994) Unacceptable/guideline 0, 50, 100 or 200 mg/kg/day	Maternal: NOAEL = 50 mg/kg/day LOAEL = 100 mg/kg/day based on increased abortions, mortality, and reductions in food and water consumption, body weight gain, and fecal output Developmental: NOAEL = 200 mg/kg/day LOAEL = Not observed
HOE 099730 metabolite		
870.3100 90-Day oral toxicity rodents-rat	44076201 (1994) Acceptable/guideline 0, 400, 2000 or 10,000 ppm 0/0, 29/32, 147/162 or 738/800 mg/kg/day M/F	NOAEL = 147 mg/kg/day in males, 162 mg/kg/day in females LOAEL = 738 mg/kg/day in males, 800 mg/kg/day in females based on glutamine synthetase inhibition in the brain
870.3100 90-Day oral toxicity rodents-mice	44076202 (1994) Acceptable/guideline 0, 500, 2000 or 8000 ppm 0/0, 83/110, 324/436 or 1296/1743 mg/kg/day M/F	NOAEL = Not established for males, 110 mg/kg/day in females LOAEL = 83 mg/kg/day in males, 436 mg/kg/day in females based on glutamine synthetase inhibition in the brain
870.3150 Subchronic Nonrodent Oral Toxicity-dog	44076203 (1994) Acceptable/guideline 0, 500, 2000 or 8000 ppm 0/, 19/21, 72/79 or 289/300 mg/kg/day M/F	NOAEL = 19 mg/kg/day in males, 21 mg/kg/day in females LOAEL = 72 mg/kg/day in males, 79 mg/kg/day in females based on glutamine synthetase inhibition in the brain
870.3700a Prenatal developmental in rodents- rat	44076204 (1993) Acceptable/guideline 0 or 1000 mg/kg/day	<u>Maternal</u> : NOAEL = 1000 mg/kg/day LOAEL = Not observed <u>Developmental</u> : NOAEL = 1000 mg/kg/day LOAEL = Not observed
870.3700b Prenatal developmental in nonrodents- rabbit	44076205 (1995) Acceptable/guideline 0, 64, 160 or 400 mg/kg/day	<u>Maternal</u> : NOAEL = 64 mg/kg/day LOAEL = 160 mg/kg/day based on reduced feed consumption <u>Developmental</u> : NOAEL = 64 mg/kg/day LOAEL = 160 based on uni- or bilateral extra at the 13 th

Guideline No./ Study Type	MRID No. (year)/ Classification /Doses	Results
		thoracic vertebra
870.6200 Acute Neurotoxicity -rat	45190702 (1999) Acceptable/nonguideline 0, 100, 1000 or 2000 mg/kg	NOAEL= 1000 mg/kg in males and females LOAEL= 2000 mg/kg in males and females based on clinical signs of toxicity including sedation, ruffled fur, and diarrhea
870.6200 Acute Neurotoxicity -rat	45190701 (1999) Acceptable/nonguideline 0, 100, 1000 or 2000 mg/kg	NOAEL= 100 mg/kg in males and females LOAEL= 1000 mg/kg in males and females based on decreased body weight gain
870.6200b Repeat Dose Neurotoxicity- rat	45179101, 45179102, 45297001 (2000); Acceptable/nonguideline 0, 20, 200 or 2000 ppm 0/0, 1.6/1.75, 15.5/17.7 or 158.9/179.4 mg/kg/day, M/F	NOAEL= 158.9 mg/kg/day in males, 179.4 mg/kg/day in females LOAEL= Not established in males and females
HOE 058192 (L-isomer of glufosinate ammonium)		
870.3100 90-Day oral toxicity rodents-rat	44068501 (1989) Acceptable/guideline 0, 25, 250, 1250 or 2500 ppm 0/0, 1.9/1.9, 18.5/19.8, 91.8/100.3 or 186.4/194.3 mg/kg/day M/F	NOAEL = 18.5 mg/kg/day in males, 19.8 mg/kg/day in females LOAEL = 91.8 mg/kg/day in males, 100.3 mg/kg/day in females based on increased ammonia levels in the plasma and urine and slight kidney weight increases
870.3150 Subchronic Nonrodent Oral Toxicity-dog	44068502 (1989) Acceptable/guideline 0, 2, 5 or 8.5 mg/kg/day	NOAEL = 2 mg/kg/day LOAEL = 5 mg/kg/day based on increased plasma and kidney ammonia levels
870.3700b Prenatal developmental in nonrodents- rabbit	43829405 (1992) Acceptable/guideline 0, 1.25, 2.50, or 5.00 mg/kg/day	<u>Maternal</u> : NOAEL = 1.25 mg/kg/day ; LOAEL = 2.50 mg/kg/day based on decrease in body weight gains and food consumption, neurotoxic signs and abortions <u>Developmental</u> : NOAEL = 1.25 mg/kg/day; LOAEL = 2.50 mg/kg/day based on an increase in post implantation loss (fetal resorptions)

Appendix B. Magnitude of the Residue Considerations

Citrus Fruit

For citrus fruits, a total of twenty three magnitude of the residue field trials were conducted for orange, lemon, and grapefruit. A summary of the residue data is presented in Table B.1. Eight of the 23 trials used a non-ionic antifoam adjuvant. Following three broadcast applications at a target rate of 1.50 lb ai/A/application, with a 14-day RTI and a 14-day PHI, residues of glufosinate ammonium (GA) and its two metabolites, glufosinate propanoic acid (MPP) and N-acetylglufosinate (NAG) were not detected above the limit of quantitation (LOQ) (0.05 ppm) in/on oranges, lemons, or grapefruit. Residue decline studies performed on each of the three representative crop types at PHIs of 7, 11, 14, 17, and 21 days showed residues below the LOQ for all residues of concern. Accordingly, the appropriate tolerance level for citrus fruit is 0.15 ppm [computed as the sum of the LOQs for each of the three residues of concern].

Stone Fruit

For peach, nine crop field trials were conducted, all without adjuvant. Following two broadcast applications at a target rate of 1.50 lb ai/A/application, with a 28-day RTI and a 14-day PHI, there were no residues of GA, MPP or NAG detected above the LOQ (0.05 ppm) in/on peach samples. There were no residue decline studies performed for peach.

For plum and cherry, six crop field trials each were conducted. Five of the 12 trials for plum and cherries used a non-ionic antifoam adjuvant. Following two broadcast applications at a target rate of 1.50 lb ai/A/application, with a 28-day RTI and a 14-day PHI, residues of GA and NAG were not detected above the LOQ (0.05 ppm). One cherry trial and one plum trial showed MPP residues above the LOQ. Thus, for cherries, the highest average field trial (HAFT) was 0.083 ppm with a maximum residue of 0.098 ppm MPP. For total glufosinate ammonium, the cherry HAFT was 0.183 ppm with a maximum residue of 0.198 ppm. For plums, the HAFT was 0.0655 ppm with a maximum residue of 0.066 ppm MPP. For total glufosinate ammonium, the plum HAFT was 0.1655 ppm with a maximum residue of 0.166 ppm. Results from the residue decline studies performed on both crops at PHIs of 7, 11, 14, 17, and 21 days indicated that total glufosinate ammonium residues in cherry and plum were found to be near or below the LOQ at all PHIs; therefore, a trend was not observed. The recommended stone fruit tolerance level was based on the cherry data; the maximum total glufosinate ammonium residue of 0.198 ppm indicates a stone fruit tolerance level of 0.25 ppm is appropriate.

Pome Fruit

For pear, six crop field trials were conducted. In all trials, ammonium sulfate fertilizer was added to each tank mixture. Two sites received a non-ionic antifoam adjuvant. Following three broadcast applications at a target rate of 1.50 lb ai/A/application, with a 14-day RTI and a 14-day PHI, residues of GA and NAG in/on pear were less than the LOQ (0.05 ppm). However, one pear trial found MPP residues above the LOQ. Specifically, the pear HAFT for MPP was 0.081 ppm, and the maximum residue was 0.089 ppm. For total glufosinate ammonium, the pear HAFT was 0.181 ppm with a maximum residue of 0.189 ppm. Results from the residue decline study with PHIs of 7, 9, 14, 16, and 21 days showed all residues of concern were below the LOQ at all sampling intervals.

The established apple tolerance of 0.05 ppm is based on the same use pattern used in the pear trials. Accordingly, it is appropriate to recommend a pome fruit tolerance based on the pear trials. The maximum total glufosinate ammonium residues of 0.189 ppm indicates a pome fruit tolerance level of 0.25 ppm is appropriate.

Olive

For olive, three crop field trials were conducted. In all trials, ammonium sulfate fertilizer was added to each tank mixture. In two of the trials, a non-ionic antifoam adjuvant was also added to the tank mixture. Following three broadcast applications at a target rate of 1.50 lb ai/A/application, with a 14-day RTI and a 14-day PHI, there were no residues of GA, MPP, or NAG detected above the LOQ (0.05 ppm) in/on olive samples. There were no residue decline studies performed for olive. The appropriate tolerance level for olive is 0.15 ppm [computed as the sum of the LOQs for each of the three residues of concern].

Sweet Corn

For sweet corn, twelve crop field trials were conducted (see Table B.2). Two of the trials in Arlington, Wisconsin were conducted in the same field in the same year, and therefore cannot be considered independent. In all trials, ammonium sulfate fertilizer was added to each tank mixture, but no adjuvant was used. Following two foliar applications at a rate of 0.375 lb ai/A, with a RTI of 14 days and a PHI of 30-50 days, maximum total glufosinate ammonium residues in/on sweet corn ears, forage and stover were 0.19, 0.79, and 3.70 ppm, respectively. The tolerance levels for sweet corn commodities were determined using the Organization for Economic Cooperation and Development (OECD) tolerance calculation procedures. Thus, the recommended tolerance levels for sweet corn ears, forage, and stover are 0.30, 1.5, and 6.0 ppm, respectively.

TABLE B.1. Summary of Total Glufosinate Ammonium Residues (sum of GA + MPP + NAG) in Fruits and Olive.

Commodity	Total Applic. Rate (lb ai/A)	PHI (days)	Residue Levels (ppm)					
			n ¹	Min.	Max.	HAFT	Median	Mean
Orange	4.38 to 4.61	14	24	<0.15	<0.15	<0.15	NA	NA
Lemon	4.50 to 4.53	14	10	<0.15	<0.15	<0.15	NA	NA
Grapefruit	4.47 to 4.58	14	12	<0.15	<0.15	<0.15	NA	NA
Pear	4.45 to 4.57	14	12	<0.15	0.189	0.181	0.15	0.155
Cherry	3.00 to 3.11	14	12	<0.15	0.198	0.183	0.15	0.156
Plum	2.92 to 3.03	14	12	<0.15	0.166	0.1655	0.15	0.153
Peach	2.86 to 3.07	14	18	<0.15	<0.15	<0.15	NA	NA
Olive	4.50 to 4.52	14	6	<0.15	<0.15	<0.15	NA	NA

¹ Two values per field trial.

TABLE B.2. Summary of Total Glufosinate Ammonium Residues (sum of GA + MPP + NAG) in Sweet Corn.								
Commodity	Total Applic. Rate (lb ai/A)	PHI (days)	Residue Levels (ppm)					
			n ¹	Min.	Max.	HAFT	Median	Mean
Sweet Corn, Ears	0.75	30-50	11	0.10	0.19	0.19	0.10	0.11
Sweet Corn, Forage	0.75	30-50	11	0.18	0.79	0.79	0.31	0.39
Sweet Corn, Stover	0.75	30-50	11	0.16	3.7	3.7	1.53	1.42

¹ One value per field trial: replicate averages.