(NS), Control and Operating Leases and Agreements, To serve portion of eastern United States.

Summary: EPA expressed environmental concerns for the project's potential impact to air quality and requested additional information regarding the noise analysis and mitigation for communities identified by-the STB's environmental justice review.

ERP No. DS–APH–A82124–00 Rating EO2, Logs, Lumber and Other Unmanufactured Wood Articles Importation, Additional Updated Information, Improvements to the existing system to Prohibit Introduction of Plant Pests into the United States.

Summary: EPA had environmental objections to the proposed project and requested additional information. The objections focused on the adequacy of analysis for assessing risk and consequences of pest introduction, compliance of regulations by exporters, human health effects of the program, and the comparison of alternatives. EPA also had concerns about the use of methyl bromide in the program.

ERP No. DS–NOA–L91001–AK Rating EC2, Juneau Consolidated Facility, Additional Information, Space for the University of Alaska Fairbanks School of Fisheries and Ocean Science (UAF), Possible Site Lena Point, Fisheries Management Operation, ''Vision for 2005'', Juneau, AK.

Summary: EPA still has environmental concerns regarding water quality/groundwater, noise, wastewater treatment, and mitigation measures. Clarification of these issues was requested.

Final EISs

ERP No. F–AFS–J65261–MT, Beaverhead Forest Plan Riparian Amendment, Implementation, Beaverhead—Deerlodge National Forest, Beaverhead, Madison, Silver Bow, Deer Lodge and Gallatin Counties, MT.

Summary: EPA expressed environmental concerns regarding the anticipated slow rate of improvement or restoration of existing degraded riparian areas with the preferred alternative. EPA recommended more timely attainment of proper functioning condition and desired future condition, and recommended inclusion of water quality protection language in the objectives.

ERP No. F–IBR–J64006–ND, Arrowwood National Wildlife Refuge, Implementation, Water Management Capability to Mitigate for Past, Present and Future Impacts of Jamestown Reservoir, Stutsman and Foster Counties, ND.

Summary: EPA expressed environmental concerns over potential water quality impacts, and recommended that downstream water quality should be monitored and an adaptive operations plan be developed to ensure that water quality standards are met.

Dated: February 17, 1998.

B. Katherine Biggs,

Associate Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 98–4369 Filed 2–19–98; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[PF–791; FRL–5768–9]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various agricultural commodities.

DATES: Comments, identified by the docket control number PF–791, must be received on or before March 23, 1998.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: James A. Tompkins, Product Manager (PM) 25, Registration Division, (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 239, 1921 Jefferson Davis Hwy., Arlington, VA., (703) 305–5697; e-mail: Tompkins.jim@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various raw agricultural commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports grantinig of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice, as well as the public version, has been established for this notice of filing under docket control number PF–791 (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number (insert docket number) and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

Authority: 21 U.S.C. 346a.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and

pests, Reporting and recordkeeping requirements.

Dated: February 12, 1998.

Donald R. Stubbs,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Below summaries of the pesticide petitions are printed. The summaries of the petitions were prepared by the petitioners. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. DowElanco

PP 1F3935

EPA has received a pesticide petition (PP 1F3935) from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268-1054 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of triclopyr, (3,5,6-trichloro-2-pyridinyl)oxyacetic acid and its metabolites 3,5,6-trichloro-2-pyridinol (TCP) and 2-methoxy-3,5,6-trichloropyridine (TMP) in or on the raw agricultural commodity fish at 3.0 parts per million (ppm), and shellfish at 5.0 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Analytical method.* Adequate methodology is available for the enforcement of tolerances for triclopyr residues of concern. Gas chromatography methods are available for the determination of triclopyr residues of concern. Residues of triclopyr, 3,5,6-trichloro-2-pyridinol, and 2-methoxy-3,5,6-trichloropyridine can be separately determined. The limits of quantitation are 0.01 – 0.05 ppm in fish and shellfish, depending on the compound being analyzed. The water method has a limit of quantitation of 0.1 parts per billion (ppb).

2. *Magnitude of residues.* In field studies, triclopyr and its metabolites in water have half-lives of 0.5 – 15 days. Triclopyr residues in lake water treated at the maximum label rate were below 0.5 ppm within 3 – 14 days. In pond water where whole ponds were treated at the maximum label rate, residues were below 0.5 ppm by 28 days after treatment. After 42 days in both lakes and ponds, residues were non-detectable (<0.010 ppm) to 0.013 ppm.

Residues of triclopyr and its metabolites 3,5,6-trichloro-2-pyridinol and 2-methoxy-3,5,6-trichloropyridine reach a maximum concentration in fish at 3–14 days after treatment of water, and total residues of triclopyr and its metabolites were detectable in the edible flesh at a maximum level of 3.0 ppm in fish and 5.0 ppm in shellfish. Residues in fish and shellfish decline as residues in water dissipate.

B. Toxicological Profile

1. *Acute toxicity.* The developmental no-effect level (NOEL) of 30 milligrams/kilograms/day (mg/kg/day) from a rabbit developmental study was recommended for the acute dietary risk assessment. At the lowest effect level (LEL) of 100 mg/kg/day, there were embryotoxic and fetotoxic effects associated with significant maternal toxicity, including death. Acute exposure assessment will evaluate risk to pregnant females age 13 and older.

2. *Short- and Intermediate-Term Toxicity.* Based on the available data, short- and intermediate-term dermal and inhalation risk assessments are not required. A systemic NOEL of 1,000 mg/kg/day, the highest dose tested (HDT), was determined in a 21–day dermal toxicity study in rabbits. The LC_{50} from the acute inhalation study in rats was determined to be > 2.6 mg/L (Toxicity Category III).

3. *Chronic toxicity.* The Reference Dose (RfD) for triclopyr is 0.05 mg/kg/day. This RfD is based on a 2-generation reproductive toxicity study in rats with a NOEL of 5.0 mg/kg/day using an uncertainty factor of 100. At the next higher dose level of 25 mg/kg/day, an increased incidence of slight degeneration of the proximal tubules of the kidneys was observed in some P1 and P2 parents of both sexes. Chronic exposure assessment will evaluate risk using this RfD.

4. *Carcinogenicity.* Environmental Protection Agency's Cancer Peer Review Committee (CPRC) concluded that triclopyr should be classified as a ''Group D chemical'' - not classifiable as to human carcinogenicity. A cancer risk assessment is not required.

5. *Animal metabolism.* Disposition and metabolism of ^{14}C-triclopyr in rats demonstrated that triclopyr was well absorbed after oral administration. Excretion was relatively rapid with a majority of radioactivity eliminated in the urine by 24 hours. At the high dose of 60 mg/kg, urinary elimination of ^{14}C-triclopyr was decreased due to apparent saturation of renal elimination mechanisms. Fecal elimination of ^{14}C-triclopyr was a minor route of excretion, as was elimination via exhaled air. Unmetabolized parent chemical represented >90% of urinary radioactivity, with the remainder accounted for by the metabolite 3,5,6-trichloro-2-pyridinol (3,5,6-TCP), and possible glucuranide and/or sulfate conjugates of 3,5,6-TCP. Plasma elimination following intravenous administration of ^{14}C-triclopyr was consistent with a one-compartment model with an elimination half-life of 3.6 hr and zero-order kinetics from 0–12 hours at the 60 mg/kg dose.

6. *Bioequivalency.* Toxicology studies conducted with triclopyr have been performed using both the free acid or the triethylamine salt from of triclopyr. Bioequivalency of the two chemical forms of triclopyr has been addressed through the conduct of special studies with the triethylamine from of triclopyr. These studies, which included data on comparative disposition, plasma half-life, tissue distribution, hydrolytic cleavage under physiological and environmental conditions for triclopyr triethylamine salt were found to adequately address the issue of Bioequivalency. In addition, subchronic toxicity studies supported the pharmacokinetics data in demonstrating bioequivalence. Therefore, studies conducted with any one from of triclopyr can be used to support the toxicology database as a whole.

7. *Endocrine effects.* An evaluation of the potential effects on the endocrine systems of mammals has not been determined; However, no evidence of such effects were reported in the chronic or reproductive toxicology studies described above. There was no observed pathology of the endocrine organs in these studies. There is no evidence at this time that triclopyr causes endocrine effects.

C. Aggregate Exposure

1. *Dietary exposure.* The RfD for triclopyr is based upon the 2-generation reproduction toxicity study in rats with a NOEL of 5.0 mg/kg/day, the lowest dose tested. An uncertainty factor of 10 for interspecies differences in response and an uncertainty factor of 10 for intraspecies differences in response was applied. Thus, the RfD for triclopyr was established at 0.05 mg/kg/day by the RfD Peer Review Committee on September 4, 1996.

A chronic dietary exposure analysis was performed using tolerance level

residues and 100 percent crop treated information to estimate the Theoretical Maximum Residue Contribution (TMRC) for the general population and 22 subgroups. Existing tolerances, including the proposed tolerances for fish and shellfish, result in a TMRC that represents 1.25% of the RfD for the U.S. general population. The highest subgroup, Non-Nursing Infants (<1 year old) occupies 2.65% of the RfD. The chronic analysis for triclopyr is a worse case estimate of dietary exposure with all residues at tolerance level and 100 percent of the commodities assumed to be treated with triclopyr. Based on the risk estimates calculated in this analysis, the chronic dietary risk from the uses currently registered is not of concern.

Since the toxicological endpoint to which exposure is being compared in the acute dietary risk analysis is a developmental NOEL (30 mg/kg/day), females (13+ years) are the sub population of particular interest. The Margin of Exposure (MOE) is a measure of how close the high end exposure comes to the NOEL (the highest dose at which no effects were observed in the laboratory test), and is calculated as the ratio of the NOEL to the exposure (NOEL/exposure = MOE.) Generally, acute dietary margins of exposure greater than 100 tend to cause no dietary concern. The high end MOE value of 1,639 is above the acceptable level and demonstrates no acute dietary concern.

An acute dietary exposure analysis was performed using tolerance level residues and 100 percent crop treated to estimate the high end exposure for the general population and females (13+, pregnant, non-nursing). The high end exposure was assumed to be the upper 0.5% of consumers, that is, the 99.5 percentile. The resulting exposure estimates and margins of exposure are as follows:

Population Subgroup	Exposure (mg/kg BW/day)	MOE
U.S. Population	0.01359	2208
Females	0.01831	1639

These high end MOE values are above the acceptable level and demonstrate no acute dietary concerns.

2. *Drinking water.* The use of triclopyr as described on the label allows only slight additional exposure of triclopyr to humans. The proposed labeling requires that the product not be applied within one-quarter mile of a potable water intake and that treated water not be used for domestic purposes until the residue level is demonstrated to be at or below 0.5 ppm as determined by laboratory analysis or immunoassay. The basis for these restrictions is a series of aquatic dissipation studies conducted in lakes and ponds. In these studies, triclopyr was applied to lakes and ponds at the maximum concentration of 2.5 ppm triclopyr in water. Triclopyr residues in the lakes at one-quarter mile from the treatment areas were well below 0.1 ppm throughout the study, with a maximum reported value of 0.058 ppm. Within the treatment area, triclopyr residues of less than 0.5 ppm were reported at 3 – 14 days after treatment in the Lake Minnetonka and Lake Seminole studies. In seven test ponds treated with triclopyr at a water concentration of 2.5 ppm, total residues of triclopyr were less than 0.5 ppm by 28 days after application, with the highest residue value being 0.193 ppm. At 42 days after treatment, total residues in both treated lakes and ponds ranged from non-detectable to 0.013 ppm.

If the proposed labeling is followed precisely, that is, potable water is not collected within one-quarter mile of a treated area, there will be little contribution from water to the ''risk cup'' for triclopyr. If drinking water is collected from the treatment area when water analysis indicates triclopyr residues are 0.5 ppm or less, the risk is still acceptable on an acute basis. On a chronic basis, the value of 0.013 ppm, found to be the highest triclopyr residue at 42 days after treatment in all studies, uses only 0.9% of the RfD for females (13+, pregnant, not nursing) and 2.6% of the RfD for children (1–6 years).

For a worst case estimate of potential drinking water exposure, the water residue at the proposed allowable water level at 0.5 ppm was utilized. When this residue level is considered, the following analysis indicates no level of concern for acute exposure:

For a 60 kg pregnant female consuming 2 liters a day (Acute)

(0.5 mg/L × 2 L/day) / 60 kg = 0.0167 mg/kg/day

MOE = NOEL / Exposure = (30 mg/kg/day) / (0.0167 mg/kg/day) = 1796

For a 60 kg pregnant female consuming 2 liters a day (Chronic)

(0.013 mg/kg/day × 2 L/day) / 60 kg = 0.00043 mg/kg/day

% RfD = (0.00043 mg/kg/day × 100) / (0.05 mg/kg/day) = 0.9 %

For a 10 kg child consuming 1 liter a day (Acute)

(0.5 mg/L × 1 L/day) / 10 kg = 0.05 mg/kg/day

MOE = (30 mg/kg/day) / (0.05 mg/kg/day) = 600

For a 10 kg child consuming 1 liter a day (Chronic)

(0.013 mg/L × 1 L/day) / 10 kg = 0.0013 mg/kg/day

% RfD = (0.0013 mg/kg/day × 100) / (0.05 mg/kg/day) = 2.6 %

3. *Non-dietary exposure.* There are potential exposures to homeowners during usual use-patterns associated with triclopyr. These involve application of triclopyr-containing products by means of aerosol cans, pump spray bottles, squeeze bottles, ''weed sticks,'' hose-end sprayers, power sprayers, paint brush, rotary and drop spreaders. It is unlikely that power sprayers will be used by homeowners; this is an application method requiring special applicator equipment more apt to be used by agricultural or commercial applicator.

Homeowner exposure will not be significant for the following reasons: the percent ai in products for homeowner use is less than that for agricultural or industrial use; the areas treated are usually limited in size; all products are intended for outdoor use which is likely to reduce the concentration in the environment by allowing dissipation in the outdoor air; the application methods recommended or commonly used by homeowners are not expected to provide significant exposure. Additionally, no toxicological endpoints of concern have been identified by EPA for dermal exposure to triclopyr, therefore, no exposure assessment is required for this exposure; an inhalation exposure assessment is also not required and no chronic use pattern is expected for homeowner use of triclopyr products.

There is a potential for post-application exposure to swimmers following applications to aquatic sites

that may be used for recreational purposes. There are no triclopyr-specific exposure data to assess swimmer exposure. However, an assessment was conducted using information provided in EPA's Dermal Exposure Assessment: Principles and Applications. The dermal permeability constant (Kp) was calculated to be 6.5×10^{-8} mg/cm^2/hr. The assessment of swimmer exposure was based on a six-year old boy having a body weight of 21.9 kg and a surface area of 0.88 m^2. The swimming period was assumed to be 3 hours on the day of treatment in water containing 2.5 ppm triclopyr.

Total dermal exposure (mg) = 3 hr/day $\times$ 0.88 m^2 $\times$ 104 cm^2/m^2 $\times$ 6.5×10^{-8} mg/cm^2/hr = 1.716×10^{-3} mg/day

Oral absorption could also account for a portion of the exposure. It was assumed that 1% of the water in residence in the mouth while breathing will be swallowed.

Oral exposure = 3 hr/day $\times$ 0.05 L/hr $\times$ 2.5 mg/L = 0.375 mg/day

Combining the dermal exposure and oral exposure for a 21.9 kg child, the swimming exposure for one day was estimated to be 0.377 mg/day ÷ 21.9 kg = 0.017 mg/kg/day. Compared to the acute NOEL of 30 mg/kg/day, an MOE of 1,765 was obtained. No dermal or inhalation endpoint has been established for triclopyr, so this represents a very conservative estimate of the risk due to swimming in triclopyr-treated waters.

D. Cumulative Effects

The potential for cumulative effects of triclopyr and other substances that have a common mechanism of toxicity was considered. The mammalian toxicity of triclopyr is well defined. However, the biochemical mechanism of toxicity of this compound is not known. No reliable information exists to indicate that toxic effects produced by triclopyr would be cumulative with those of other similar compounds. Therefore, consideration of a common mechanism of toxicity with other compounds is not appropriate. Thus, only the potential risks of triclopyr are considered in the aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Because of the toxicological characteristics of triclopyr (no dermal endpoint of concern), post-application exposure assessment was not necessary. Residential exposure is considered to be negligible. Swimming in treated water was shown to be a minimal risk. Therefore, residential and swimming exposure were not considered in the aggregate risk calculation.

For the population subgroup of concern, pregnant females age 13 and older, an MOE of 857 was estimated for the acute aggregate dietary risk (food + water) from exposures to triclopyr residues.

MOE = (30 mg/kg/day) / (0.0183 + 0.0167) mg/kg/day = 857

Using the TMRC exposure assumptions described above, the percentage of the RfD that will be utilized by aggregate exposures (food + water) to residues of triclopyr ranges from 2.1% to 5.3% for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is non-nursing infants <1 year old. The water exposure value used the highest water residue concentration at 42 days after treatment of lakes and ponds (the longest sampling time interval common to all studies), 0.013 ppm, in the calculations below:

Total U.S. Population (Dietary + Drinking Water)

(0.00062 + 0.00043) mg/kg/day $\times$ 100 / (0.05 mg/kg/day) = 2.1% Rfd

Non-nursing Infants (Dietary + Drinking Water)

(0.00133 + 0.0013) mg/kg/day $\times$ 100 / (0.05 mg/kg/day) = 5.3% Rfd

Determination of Safety for U.S. Population

Based on the current state of knowledge for this chemical, the RfD approach accurately reflects the exposure of the U.S. population, infants and children to triclopyr.

2. *Infants and children*. Studies cited earlier in this document indicate that triclopyr is not a selective developmental toxicant, and an additional uncertainty factor for infants and children is unnecessary. This decision is based on the following data.

Since the developmental and reproductive NOELs were either the same or greater than the maternal or parental, it is unlikely that there is additional risk concern for immature or developing organisms which is not reflected by the risk assessment utilizing the established reference dose. The effects noted for the RfD NOEL are parental effects, not developmental.

F. International Tolerances

There are no established or proposed Codex MRLs for triclopyr residues. Therefore, there are no issues of compatibility with respect to U.S. tolerances and Codex MRLs.

2. E.I. du Pont de Nemours & Company

PP 6F4706

EPA has received a pesticide petition (PP 6F4706)from E.I. du Pont de Nemours & Company, Barley Mill Plaza, P.O. Box 80038, Willimington, DE 19880–0038. proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of rimsulfuron: *N*-((4,6-dimethoxypyrimidin-2-yl) aminocarbonyl) -3- (ethylsulfonyl) -2-pyridinesulfonamide in or on the raw agricultural commodity tomato fruit at 0.10 parts per million. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. With the initial establishment of rimsulfuron tolerances in field corn and potatoes, the EPA determined that the nature of plant residues was adequately understood for the purposes establishing those tolerances. A metabolism study on tomatoes was conducted at the following use rates:

Tomatoes grown in field	72 g active ingredient per hectare (approx. 1 oz. ai per acre, maximum proposed use rate).
Tomatoes grown in greenhouse	172, 350, and ca. 700 g ai per hectare (2.5, 5, and 10 oz. ai per acre or up to 10 times the proposed maximum use rate).

No residues of rimsulfuron or any metabolite were detected in any tomato fruit, immature or mature. Detection limits for the study were 0.004 ppm for the field-grown samples and 0.013 ppm for the greenhouse-grown samples. This study conclusively shows that application of rimsulfuron to tomatoes, when used in accordance with the proposed label directions, will not result in detectable residue of rimsulfuron or its metabolites in tomato fruit. Therefore, the nature of rimsulfuron residues (i.e., their absence) has been established for tomato fruit, the only raw agricultural commodity established for tomatoes.

2. *Analytical method*. Adequate analytical methodology, high-pressure liquid chromatography with Uv detection, is available for enforcement purposes. The method is ''Analytical Method for the Quantitiation of DPX-E9636 (rimsulfuron) in Various Crop Matrices and Their Processed Fractions'', DuPont Report No. AMR 3424–95, EPA MRID No. 43979002. The method involves liquid chromatography utilizing eluent and column switching with UV/VIS detection at 254 nm. The limit of quantitation for rimsulfuron in tomatoes is 0.05 ppm. EPA offers enforcement methodology to anyone interested in pesticide enforcement when requested by mail from: Calvin Furlow, Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 1130A, CM#2, 1921 Jefferson Davis Hwy., Arlington, VA 22202.

3. *Magnitude of residues*. —i. *Plant residues.* Magnitude of residues of rimsulfuron in tomato fruit were determined following application of rimsulfuron at the proposed maximum annual use rate of 1.0 oz ai/acre (1×), and at twice that rate (2×). An additional test was conducted at an exaggerated rate of 5.0 oz ai/acre (5×) in an attempt to generate quantifiable residues in tomato fruit (RAC) for a processing study.

Seventeen tests, each containing one control and two treatment plots, were established in California, Florida, Indiana, Maryland, and Pennsylvania. Row-crop tomato samples were collected approximately 45 days following the final application; staked tomatoes were collected immediately following the final application. Tomato samples were analyzed using the procedures described in DuPont Method No. AMR 3424–95, Analytical Method for the Quantitation of DPX-E9636 in Various Crop Matrices and Their Processed Fractions. The overall mean percent recovery of 52 control tomato samples fortified at either 0.05 or 0.10 ppm was 86%, with a relative standard deviation of 4%. Results of freezer storage stability study indicate that rimsulfuron is stable up to 6 months in tomatoes stored at −20C +/− 5C.

No quantifiable residues (<0.05 ppm) of rimsulfuron were found in any of the tomato samples treated at 1.0, 2.0, and 5.0 oz ai/acre. A processing study was not necessary since all 1× and 5× samples did not have rimsulfuron present with a limit of quantitation of 0.05 ppm.

Data generated from this study support the use of rimsulfuron on tomatoes at a maximum seasonal use rate of 1.0 oz ai/acre with a minimum preharvest interval of 45 days. Study results also support the petition for a 0.10 ppm tolerance of rimsulfuron on tomatoes.

ii. *Animal residues.* EPA determined, upon granting field corn and potato tolerances, that there is no reasonable expectation of residues occurring in meat, milk, poultry, or eggs from these tolerances. Tomato fruit and its processed commodities (i.e., tomato paste and puree) are not considered by the EPA to be animal feed items. Further, no residues would be available to enter animal feed based on results from the tomato metabolism study and magnitude or residue study discussed above. Therefore, there remains a reasonable expectation that no residue of rimsulfuron will occur in meat, milk, poultry, or eggs from all rimsulfuron tolerances, current (field corn and potatoes) and proposed (tomatoes).

B. Toxicological Profile

1. *Acute toxicity*. Technical rimsulfuron has been placed in acute toxicology category III based on overall results from several studies. This compound was placed in toxicology category III for acute dermal toxicity (LD_{50} > 2,000 mg/kg; rabbits) and eye irritation (effects reversible within 72 hours; rabbits). Acute oral toxicity (LD_{50} > 5,000 mg/kg; rats), acute inhalation toxicity (LC_{50} > 5.4 mg/L, rats) and skin irritation (no observed irritation; rabbits) results were assigned toxicology category IV. Technical rimsulfuron is not a dermal sensitizer.

2. *Genotoxicty*. Technical rimsulfuron was negative for genotoxicity in a battery of in vitro and in vivo tests. These tests included the following: mutagenicity in bacterial (Ames test) and mammalian (CHO/HGPRT assay) cells; in vitro cytogenetics (chromosomal aberration in human lymphocytes); in vivo cytogenetics (bone marrow micronucleus assay in mice); and unscheduled DNA synthesis in rat primary hepatocytes.

3. *Reproductive and developmental toxicity*. A two-generation reproduction study was conducted in rats with dietary technical rimsulfuron concentrations of 0, 50, 3,000 or 15,000 ppm. The study was negative for reproductive toxicity and there was no indication that offspring were more susceptible to rimsulfuron administration than parents. The NOEL was 3,000 ppm (or 165 to 264 mg/kg/day for P1 and F1 males and females and their offspring). This was based on the following effects at 15,000 ppm (830 to 1,316 mg/kg/day): lower food consumption and/or food efficiency in P1 males and females and F1 females; decreased mean body weights and/or body weight gain by P1 and F1 males and females; lower mean body weights and increased incidence of small body size for F2 pups during lactation.

A developmental study was conducted in rats administered technical rimsulfuron by gavage at 0, 200, 700, 2,000 or 6,000 mg/kg/day. There were no systemic or developmental effects observed up to and including the highest dose tested. The NOEL was therefore > 6,000 mg/kg/day.

A developmental study was conducted in rabbits administered technical rimsulfuron by gavage at 0, 25, 170, 500 or 1,500 mg/kg/day. The NOELs for maternal and offspring toxicity were 170 and 500 mg/kg/day, respectively. The maternal NOEL was based on reduced body weight and mortality at higher doses. These maternal effects precluded any evaluation of adverse effects in fetuses at 1,500 mg/kg/day; however, there were no systemic or developmental effects observed among fetuses at 500 mg/kg/day and below.

4. *Subchronic toxicity.* A 90-day study in mice was conducted at dietary concentrations of 0, 50, 375, 1,500 or 7,500 ppm. The NOELs were 375 ppm (56.0 mg/kg/day) for male mice and 7,500 ppm (1,575 mg/kg/day) for female mice. The NOEL for males was based on slight reductions in mean body weight gain and food efficiency at 1,500 ppm (228 mg/kg/day).

Technical rimsulfuron was administered in the diets of rats at 0, 50, 1,500, 7,500 or 20,000 ppm for 90 days. The NOEL was 1,500 ppm (102 and 120 mg/kg/day for males and females, respectively) based on reduced mean body weights and body weight gains and increased relative liver weights at

7,500 ppm (495 and 615 mg/kg/day for males and females, respectively).

Dogs were administered technical rimsulfuron in their diets at 0, 250, 5,000 or 20,000 ppm for 90 days. The NOEL was 250 ppm (9.63 and 10.6 mg/kg/day for males and females, respectively). This was based on slight increases in liver and/or kidney weights, increased urine volume and decreased urine osmolarity at 5,000 ppm (193 and 189 mg/kg/day for males and females, respectively).

5. *Chronic toxicity*. An 18-month mouse study was conducted with dietary concentrations of 0, 25, 250, 2,500 or 7,500 ppm technical rimsulfuron. This product was not oncogenic in mice. The systemic NOEL was 2,500 ppm (351 and 488 mg/kg/day for males and females, respectively) based on decreased mean body weights in both sexes and increased incidence of spontaneous, age-related artery and tunica degeneration in the testes for this mouse strain at the highest dose tested, 7,500 ppm (1,127 and 1,505 mg/kg/day for males and females, respectively). The latter was observed in the absence of any effect on spermatogenesis. An increased incidence of dilation and cysts in the glandular stomach of males was also observed at 7,500 ppm.

A 2-year chronic toxicity/oncogenicity study was conducted in rats fed diets that contained 0, 25, 300, 3,000 or 10,000 ppm technical rimsulfuron. This product was not oncogenic in rats. The systemic NOELs were 300 ppm (11.8 mg/kg/day) for males and 3,000 ppm (163 mg/kg/day) for females. The NOELs were defined by decreased body weight gain and increased relative liver weights at 3,000 ppm (121 mg/kg/day) and 10,000 ppm (569 mg/kg/day) for males and females, respectively.

Technical rimsulfuron was administered for one year to dogs at dietary concentrations of 0, 50, 2,500 or 10,000 ppm. The NOELs were 50 ppm (1.6 mg/kg/day) for males and 2,500 ppm (86.5 mg/kg/day) for females. The NOEL for males was based on the following effects observed at 2,500 ppm (81.8 mg/kg/day): increased absolute liver and kidney weights; and increased incidence of seminiferous tubule degeneration and increased numbers of spermatid giant cells present in the epididymides. The NOEL for females was based on the following effects observed at 10,000 ppm (358.5 mg/kg/day): decreased body weight and body weight gain; increased serum cholesterol levels and alkaline phosphatase activity, increased absolute liver weight and increased relative liver and kidney weights.

6. *Animal metabolism*. The metabolism of rimsulfuron in animals (rat, goat and hen) is adequately understood and is similar among the species evaluated. Rimsulfuron was rapidly eliminated via urinary and fecal excretion in the rat. Approximately 60 to 70% of the administered dose to rats was excreted within 24 hours. There were no volatile metabolites detected and unmetabolised rimsulfuron was the major component in the urine (42 – 55%) and feces (5 – 16%). The major metabolic pathway in rats involved a contraction of the sulfonylurea bridge followed by dealkylation, hydroxylation and/or conjugation reactions. Cleavage of the sulfonylurea bridge was observed; however, it was considered to be a minor pathway. Elimination of administered rimsulfuron was similarly rapid for the goat and hen. Tissue residue levels were generally less than 0.3% of the administered dose for the rat, goat and hen. There was no evidence of accumulation of rimsulfuron or its metabolites in tissues of any of the species or in milk and eggs.

7. *Metabolite toxicology*. Common metabolic pathways for rimsulfuron were demonstrated in the rat, goat and hen as well as plants (corn, tomatoes and potatoes). When evaluated for acute toxicity and mutagenicity, two of the major metabolites, i.e., one resulting from contraction of the sulfonylurea bridge and one from the cleavage of this bridge, were found to be of low toxicity and were negative in the Ames test. The existing metabolism studies indicate that the metabolites formed are unlikely to accumulate in humans or in animals that may be exposed to these residues in the diet. The fact that no quantifiable residues were found in treated crops further indicates that exposures to and accumulation of metabolites are unlikely. Because of the above, toxicology studies on metabolites were not required.

C. Aggregate Exposure

1. *Dietary exposure — Residue of concern*. When tolerances were established on field corn and potatoes, EPA determined that the residue of concern was rimsulfuron. The metabolism study conducted on tomatoes (see Plant Metabolism Section) showed no residues of rimsulfuron are present in the tomato fruit. Therefore, the residue of concern continues to be rimsulfuron.

2. *Food*. For the general U.S. population, acute dietary exposure assessments were not considered relevant for rimsulfuron for the following reasons: rimsulfuron presents very low acute toxicity based on animal testing; and no detectable residues have been demonstrated in edible portions of treated crops.

The Agency has conducted chronic dietary exposure assessments for rimsulfuron and the results are summarized below. The Reference Dose (RfD) is based on a NOEL of 1.6 mg/kg/day established in the 1-year feeding study with dogs and combines an uncertainty factor of 100. EPA calculated the RfD to be 0.016 mg/kg/day. The theoretical maximum residue contribution (TMRC) for these tolerances for the overall U.S. population is 1.47×10^{-4} mg/kg/day or 0.92% of the RfD based on current (field corn and potatoes) tolerances and would be 2.21×10^{-4}, or 1.4% of the RfD when the proposed tolerance on tomatoes is included.

For infants and children, the TMRC for the most exposed subgroup, children (1 to 6 years old), is 2.37×10^{-4} mg/kg/day, respectively, or 1.95% of the RfD based on current (field corn and potatoes) tolerances and would be 4.37×10^{-4} mg/kg/day, or 2.73% of the RfD, when the proposed tomato tolerance is included. As with calculations for the general US population, these values assume the residues are at the established tolerance level and that 100 percent of the crop is treated.

3. *Drinking water*. Another potential dietary source of exposure of the general population to residues of pesticides is residues in drinking water. There have been no field studies or monitoring programs conducted to assess rimsulfuron residues in groundwater or drinking water. Several factors indicate very low potential that rimsulfuron will be present in raw or finished drinking water: low use rate (1 oz a.i./acre), rapid hydrolysis (half-life < 7 days), short half-lives under field conditions (7–18 days), absence of leaching in field soil dissipation studies. Water solubility for rimsulfuron is as follows:

Unbuffered Water:.		< 10 ppm
Buffers:	pH 5	135 ppm
.....	pH 7	7,300 ppm
.....	pH 9	5,560 ppm (rapidly decomposes at pH 9); K_{oc} is less than 100.

Computer modeling, taking into account use rate, physical properties, and degradation rates, predicts low probability of rimsulfuron being present in ground- or drinking water. Given that only 2.73% of the RfD is attained by the TMRC for the population sub-group with the highest theoretical dietary exposure (children 1–6 years old; see above), there is ample allowance for safe exposure to rimsulfuron via drinking water.

4. *Non-dietary exposure*. Rimsulfuron is not registered for any use which could result in non-occupational, or non-dietary exposure to the general population.

D. Cumulative Effects

Rimsulfuron belongs to the sulfonylurea class of crop protection chemicals. Other structurally similar compounds in this class are registered herbicides. However, the herbicidal activity of sulfonylureas is due to the inhibition of acetolactate synthase (ALS), an enzyme found only in plants. This enzyme is part of the biosynthesis pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the relatively low toxicity of sulfonylurea herbicides in animals. There is no reliable information that would indicate or suggest that rimsulfuron has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population*. Based on the completeness and reliability of the toxicology database and using the conservative assumptions presented earlier, EPA has established an RfD of 0.016 mg/kg/day. This was based on the NOEL for the 1-year dog study of 1.6 mg/kg/day and employed a 100–fold uncertainty factor. It has been concluded that the aggregate exposure for existing crops (corn and potatoes) would utilize 0.92% of the RfD and that the addition of tomatoes would increase utilization to 1.4% of the RfD. Generally, exposures below 100% of the RfD are of no concern because it represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is reasonable certainty that no harm will result from aggregate exposures to rimsulfuron residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of rimsulfuron, data from the previously discussed developmental and multigeneration reproductive toxicity studies were considered.

Developmental studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during pre-natal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from pre-natal and post-natal exposures to the pesticide. The studies with rimsulfuron demonstrated no evidence of developmental toxicity at exposures below those causing maternal toxicity. This indicates that developing animals are not more sensitive to the effects of rimsulfuron administration than adults.

FFDCA section 408 provides that EPA may apply an additional uncertainty factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database. Based on current toxicological data requirements, the database for rimsulfuron relative to pre- and post-natal effects for children is complete. In addition, the NOEL of 1.6 mg/kg/day in the 1-year dog study and upon which the RfD is based is much lower than the NOELs defined in the reproduction and developmental toxicology studies. Conservative assumptions utilized to estimate aggregate dietary exposures of infants and children to rimsulfuron demonstrated that only 1.95% of the RfD was utilized for current tolerances (corn and potatoes) and the addition of tomatoes would only increase utilization to 2.73% of the RfD for the highest exposed group. Based on these exposure estimates and the fact that the current database demonstrates that the developing offspring or young animals are not uniquely susceptible to rimsulfuron administration, the extra 10–fold uncertainty factor is not warranted for these groups. Therefore, it may be concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposures to rimsulfuron.

F. International Tolerances

The following international tolerances (or Maximum Residue Levels, MRL's) exist:

Country	Tolerance in ppm	Crop
Australia	0.05	Tomatoes
Austria	0.1	Corn, Potato
Belgium	0.05	Corn
Bulgaria	0.5	Corn-Fodder
Canada	0.1	Corn, Potato
Croatia	0.1	Fodder
Czech. Rep.	0.05	Corn, Grain, Potato
Germany	0.05	Corn, Potato
Hungary	0.2	Corn
Italy	0.10	Corn, Potato, Tomatoes
Romania	0.05	Corn, Potato

Country	Tolerance in ppm	Crop
Slovakia	0.05	Corn, Grain
Spain	0.05	Corn, Tomatoes
United States	0.1	Corn, Potato

3. Monsanto Company

PP 2E4118 and 7F4886

EPA has received a pesticide petitions (PP 2E4118 and 7F4886) from Monsanto Company, 700 14th St.,NW., Suite 1100, Washington, D.C. 20005, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for glyphosate [(*N*-phosphonomethyl) glycine] in or on the imported raw agricultural commodities barley grain at 20 parts per million (ppm); barley, bran and pearled barley at 60 ppm; cereal grains group (except wheat, corn, oats, grain sorghum, and barley at 0.1 ppm; canola, seed at 10 ppm; canola, meal at 25 ppm; legume vegetables (succulent or dried) group (except soybeans) at 5 ppm. (PP 2E4118) and in or on the raw agricultural commodities beets, sugar, tops (leaves) at 10 ppm; beets, sugar, root at 10 ppm; and beets, sugar, pulp, dried at 25 ppm (PP 7F4886). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in plants is adequately understood and consists of the parent, glyphosate and its metabolite AMPA (aminomethyl-phosphonic acid). Only glyphosate parent is to be regulated in plant and animal commodities since the metabolite AMPA is not of toxicological concern in food.

2. *Analytical method*. Adequate methodology High Pressure Liquid Chromatograpy (HPLC) with fluorometric detection is available for enforcement purposes, and the methodology has been published in the Pesticide Analytical Manual (PAM), Vol. II. This method has a limit of detection (0.05 ppm) that allows monitoring of food with residues at or above the levels set in these tolerances.

3. *Magnitude of residues*. The submitted residue data adequately support the proposed tolerances on Barley, grain (20 ppm); Barley, bran and pearled barley (60 ppm); Canola, seed (10 ppm); Canola, meal (25 ppm); and Legume vegetables (succulent or dried) group (except soybeans) (5 ppm), Sugar beet roots at 10 ppm, Sugar beet tops at 10 ppm and Sugar beet dried pulp at 25 ppm. Any secondary residues occurring in liver or kidney of cattle, goats, horses, and sheep and liver and kidney of poultry will be covered by existing tolerances.

B. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study with a combined LD_{50} of > 5000 mg/kg.

A rabbit acute dermal LD_{50} of > 5000 mg/kg.

A primary eye irritation study in the rabbit which showed severe irritation for glyphosate acid. However, glyphosate is normally formulated as one of several salts and eye irritation studies on the salts showed essentially no irritation.

A primary dermal irritation study which showed essentially no irritation.

A primary dermal sensitization study which showed no sensitization.

2. *Genotoxicty*. A number of mutagenicity studies were conducted and were all negative. These studies included: chromosomal aberration *in vitro* (no aberrations in Chinese hamster ovary cells were caused with or without S9 activation); DNA repair in rat hepatocyte; *in vivo* bone marrow cytogenic test in rats; rec-assay with *B. subtilis*; reverse mutation test with *S. typhimurium*; Ames test with *S. typhimurium*; and dominant-lethal mutagenicity test in mice.

3. *Reproductive and developmental toxicity*. An oral developmental toxicity study with rats given doses of 0, 300, 1,000 and 3,500 mg/kg/day with a maternal no observable effect level (NOEL) of 1,000 mg/kg/day based on clinical signs of toxicity, body weight effects and mortality, and a fetal NOEL of 1,000 mg/kg/day based on reduced body weights and delayed sternebrae maturation at the highest dose tested of 3,500 mg/kg/day.

An oral developmental toxicity study with rabbits given doses of 0, 75, 175 and 350 mg/kg/day with a maternal of NOEL of 175 mg/kg/day based on clinical signs of toxicity and mortality, and a fetal NOEL of 350 mg/kg/day based on no developmental toxicity at any dose tested.

A 3-generation reproduction study with rats fed dosage levels of 0, 3, 10 and 30 mg/kg/day with a NOEL for systemic and reproductive/ developmental parameters of 30 mg/kg/ day based on no adverse effects noted at any dose level.

A 2-generation reproduction study with rats fed dosage levels of 0, 100, 500 and 1,500 mg/kg/day with a NOEL for systemic and developmental parameters of 500 mg/kg/day based on body weight effects, clinical signs of toxicity in adult animals and decreased pup bodyweights, and a reproductive NOEL of 1,500 mg/kg/day.

4. *Subchronic toxicity*. A 90-day feeding study in rats fed dosage levels of 0, 1,000, 5,000 and 20,000 ppm with a NOEL of 20,000 ppm based on no effects even at the highest dose tested.

A 90-day feeding study in mice fed dosage levels of 0, 5,000, 10,000 and 50,000 with a NOEL of 10,000 ppm based on body weight effects at the high dose.

A 90-day feeding study in dogs given glyphosate, via capsule, at doses of 0, 200, 600 and 2,000 mg/kg/day with a NOEL of 2,000 mg/kg/day based on no effects even at the highest dose tested.

5. *Chronic toxicity*. A 12-month oral study in dogs given glyphosate, via capsule, at doses of 0, 20, 100 and 500 mg/kg/day with a NOEL of 500 mg/kg/ day based on no adverse effects at any dose level.

A 26-month chronic/feeding oncogenicity study with rats fed dosage levels of 0, 3, 10 and 31 mg/kg/day (males) and 0, 3, 11 and 34 mg/kg/day (females) with a systemic NOEL of 31 mg/kg/day (males) and 34 mg/kg/day (females) based on no carcinogenic or other adverse effects at any dose level.

A 24-month chronic/feeding oncogenicity study with rats fed dosage levels of 0, 89, 362 and 940 mg/kg/day (males) and 0, 113, 457 and 1,183 mg/ kg/day (females) with a systemic NOEL of 362 mg/kg/day based on body weight

effects in the female and eye effects in males. There was no carcinogenic response at any dose level.

6. *Carcinogenicity*. A mouse oncogenicity study with mice fed dosage levels of 0, 150, 750 and 4,500 mg/kg/day with a NOEL of 750 mg/kg/day based on body weight effects and microscopic liver changes at the high dose. There was no carcinogenic effect at the highest dose tested of 4,500 mg/kg/day.

Glyphosate is classified as a Group E (evidence of non-carcinogenicity for humans), based upon lack of convincing carcinogenicity evidence in adequate studies in two animal species. This classification is based on the following findings: (1) There were no tumor findings in the chronic testing that were determined to be compound related; (2) glyphosate was tested up to the limit dose on the rat and up to levels higher than the limit dose in mice; and (3) there is no evidence of genotoxicity for glyphosate.

7. *Animal metabolism*. The nature of the residue in animals is adequately understood and consists of the parent, glyphosate and its metabolite AMPA (aminomethyl-phosphonic acid).

8. *Metabolite toxicology*. Only glyphosate parent is to be regulated in plant and animal commodities since the metabolite AMPA is not of toxicological concern in food.

9. *Endocrine disruption*. The toxicity studies required by EPA for the registration of pesticides measure numerous endpoints with sufficient sensitivity to detect potential endocrine-modulating activity. No effects have been identified in subchronic, chronic or developmental toxicity studies to indicate any endocrine-modulating activity by glyphosate. In addition, negative results were obtained when glyphosate was tested in a dominant-lethal mutation assay. While this assay was designed as a genetic toxicity test, agents that can affect male reproduction function will also cause effects in this assay. More importantly, the multi-generation reproduction study in rodents is a complex study design which measures a broad range of endpoints in the reproductive system and in developing offspring that are sensitive to alterations by chemical agents. Glyphosate has been tested in two separate multi-generation studies and each time the results demonstrated that glyphosate is not a reproductive toxin.

C. Aggregate Exposure

1. *Dietary exposure — Food*. For purposes of assessing the potential exposure under these tolerances, dietary exposure was estimated based on the Theoretical Maximum Residue Contribution (TMRC) from the all present tolerances for glyphosate and the additional exposure that could result if the proposed tolerances are established on barley grain at 20 ppm, barley bran and pearled barley at 60 ppm, canola seed at 10 ppm, canola meal at 25 ppm, legume vegetables (succulent or dried) group (except soybeans) at 5 ppm, sugar beet roots at 10 ppm, sugar beet tops at 10 ppm and sugar beet dried pulp at 25 ppm. The TMRC is obtained by multiplying the tolerance level residue for each food commodity by the consumption data which estimates the amount of those products eaten by various population subgroups. In conducting this exposure assessment, very conservative assumptions were made -- 100% of these crops will contain glyphosate residues and those residues would be at the level of the tolerance -- which result in an overestimate of human exposure. Thus, in making a safety determination for these tolerances, EPA is taking into account this conservative exposure assessment.

Secondary residues in animal commodities may occur from these uses through the feeding of barley grain and canola meal to livestock. Since these proposed tolerances do not arise from changes in U.S. registrations involving the use of glyphosate herbicides on barley, canola, or legume vegetables, it has been concluded that livestock feed items derived from these crops are not likely to enter channels of trade in the United States. Based on these considerations and the results of animal feeding studies and the amount of glyphosate residues expected in animal feeds, EPA has concluded that there is no reasonable expectation that such secondary residues of glyphosate will exceed existing tolerances in edible animal products.

2. *Drinking water*. In examining aggregate exposure, FQPA directs EPA to consider available information concerning exposures from the pesticide residue via drinking water. The lifetime health advisory and maximum contaminant level (MCL), for glyphosate are both 700 parts per billion in the EPA Office of Drinking Water's ''Drinking Water Health Advisory; Pesticides.'' The MCL represents the level at which no known or anticipated adverse health effects will occur, allowing for an adequate margin of safety, and is based on the reference dose (RfD). Environmental Fate data for glyphosate indicate little potential for the chemical to migrate to drinking. Glyphosate is not highly mobile and not persistent in a soil or water environment. Because the Agency lacks sufficient water-related exposure data to complete a comprehensive drinking water risk assessment for many pesticides, EPA has commenced and nearly completed a process to identify a reasonable yet conservative bounding figure for the potential contribution of water related exposures to the aggregate risk posed by a pesticide. In developing the bounding figure, EPA estimated residue levels in water for a number of specific pesticides using various data sources. The Agency then applied the estimated residue levels, in conjunction with appropriate toxicological endpoints (RfDs or acute dietary NOELs) and assumptions about body weight and consumption, to calculate, for each pesticide, the increment of aggregate risk contributed by consumption of contaminated water. While EPA has not yet pinpointed the appropriate bounding figure for consumption of contaminated water, the ranges the Agency is continuing to examine are all below the level that would cause glyphosate to exceed the RfD if the tolerances being considered in this document were granted. The Agency has therefore concluded that the potential exposures associated with glyphosate in water, even at higher levels the Agency may consider a conservative upper bound, would not prevent the Agency from determining that there is a reasonable certainty of no harm if the tolerance is granted.

3. *Non-dietary exposure*. Glyphosate is registered for use on non-food sites such as around ornamental, shade trees, shrubs, walks, driveways, flowerbeds, home lawns, farmsteads including building foundations, along and in fences, in dry ditches and canals, along ditchbanks, farm roads, shelterbelts, forestry, Christmas trees, and industrial sites and other non-crop or industrial areas such as airports, lumber yards, manufacturing sites, utility substations, parking areas, petroleum tank farms, and pumping station.

Margins of Exposure (MOEs) are determined for non-dietary exposure based on toxicological endpoints and measured or estimated exposures. Since glyphosate is a class E chemical (evidence of non-carcinogenicity for humans), the 21 day dermal study lacked any observable effects at the limit dose, and no adverse effects were observed in developmental toxicity studies in rats up to 1,000 mg/kg/day and rabbits up to 175 mg/kg/day, no toxicological endpoints are applicable. Because available data indicated no evidence of significant toxicity via the dermal or inhalations routes, MOEs were not calculated and risk

assessments are not required for non-occupational (residential uses).

D. Cumulative Effects

EPA does not have, at this time, available data to determine whether glyphosate has a common mechanism of toxicity with other substances or how to include it in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on common mechanism of toxicity, glyphosate does not produce a toxic metabolite which is common to other substances. For the purposes of this tolerance action, therefore EPA has assumed that glyphosate does not have a common mechanism of toxicity with other substances. A condition of the registrations associated with these tolerances will be that the registrant will provide common mechanism data in a timely manner when and if the Agency asks for it. After EPA develops methodologies for more fully applying common mechanism of toxicity issues to risk assessments, the Agency will develop a process to reexamine those tolerance decisions made earlier.

E. Safety Determination

1. *U.S. population* —i. *Acute dietary exposure*. Based on the available acute toxicity data, glyphosate does not pose any acute dietary risks, and an acute dietary risk assessment is not required.

ii. *Chronic dietary exposure*. Using the TAS Exposure 1 software and 1977–78 consumption data, a chronic dietary exposure estimate was based on 100% of the crops treated and all residues at tolerance levels to provide the TMRC. Based this assessment the combined new proposed tolerances contribute dietary exposure equal to 0.36% of the RfD for U.S. population and 0.69% of the RfD for non- nursing infants under 1 year old. Total estimated dietary exposure from glyphosate residues in food, taking into account both existing and these proposed uses will be 1.4% of the RfD for the overall U.S. population and 3.1% of the RfD for non- nursing infants, the most highly exposed population subgroup. An additional risk assessment for residential uses was not required because of no evidence of significant toxicology via dermal or inhalation routes. Even though an appropriate bounding figure for consumption of contaminated water has not been determined, the ranges being examine are all below the level that would cause glyphosate to exceed the RfD. Generally there is no concern for exposures below 100 percent of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, there is reasonable certainty that no harm will occur from aggregate exposure to glyphosate.

2. *Infants and children*. FFCDA section 408 provides that an additional tenfold margin of exposure (safety) for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database unless it is determined that a different margin of exposure (safety) will be safe for infants and children. Monsanto believes that reliable data support using the standard margin of exposure (usually 100× for combined inter- and intra-species variability) without the additional tenfold margin of exposure when a complete data base under existing guidelines exists and when the nature of the findings from these studies do not raise concerns regarding the adequacy of the standard margin of exposure.

The toxicological database for evaluating pre- and post-natal toxicity for glyphosate is considered to be complete at this time. Risk to infants and children for glyphosate was determined by the use of two developmental toxicity studies in rats and rabbits and a two-generation reproduction study in rats. The developmental toxicity studies evaluates the potential for adverse effects on the developing organism resulting from exposure during prenatal development. The reproduction study provides information relating to effects from exposure to the chemical on the reproductive capability of both (mating) parents and on systemic toxicity, in addition to information on prenatal development. The results of these studies indicate that glyphosate does not produce birth defects and is not a reproductive toxin.

In the rabbits, no developmental toxicity was observed at the highest dose tested (HDT) where significant maternal toxicity occurred (death and clinical signs at 350 mg/kg/day, highest dose tested HDT). Because no developmental toxicity was observed at any dose level, the developmental NOEL is considered to be 350 mg/kg/day. In the rat developmental toxicity study, severe maternal (systemic) and developmental toxicity was noted at 3,500 mg/kg/ day HDT. The HDT in this study was 3.5 times higher than the limit dose that is currently required by the guidelines. The maternal and developmental (pup) NOEL was 1,000 mg/kg/day. No effects on reproductive parameters were observed.

In the rat 2-generation reproduction study, parental toxicity was observed at 1,500 mg/kg/day as soft stools, decreased food consumption and body weight gain; therefore, the systemic NOEL is considered to be 500 mg/kg/day. Developmental (pup) toxicity was also only exhibited at 1,500 mg/kg/day as decreased body weight gain of the $F1_a$, $F2_a$, and $F2_b$ male and female pups during the second and third weeks of lactation. Glyphosate did not affect the ability of rats to mate, conceive, carry or deliver normal offspring at any dose level.

The RfD is based on the NOEL for maternal toxicity in the rabbit developmental study. No developmental effects were noted in the study. In the rat developmental study, effects were noted only at doses 20-fold higher than the NOEL used for the RfD. No pre- or post-natal effects were seen in any study in the absence of maternal toxicity. In the rat reproduction study, developmental effects were noted at doses 5 times higher than the NOEL used for the RfD. The Agency does not believe the effects seen in these studies are of such concern to require an additional safety factor. Accordingly, the Agency believes the RfD has an adequate margin of protection for infants and children. The dietary exposure from current and proposed uses of glyphosate ranges from 1% of the RfD for nursing infants (less than 1 year old) to 3% for non-nursing infants and children 1 to 6 years old. Monsanto has concluded that there is reasonable certainty that no harm will occur to infants and children from aggregate exposure to glyphosate.

F. International Tolerances

Codex MRLs have been established for residues of glyphosate in or on Barley Grain at 20 ppm, Dry Peas at 5 ppm, Dry Beans at 2 ppm, and Rape (Canola) Seed at 10 ppm. The proposed tolerances will achieve harmonization with these existing MRLs. The increase in U.S. tolerances on legume vegetables up to 5 ppm was recommended in 1993 in the glyphosate Reregistration Eligibility Decision.

The proposed U. S. tolerances are also consistent with the MRLs presently established for these commodities by other trade partner countries such as Canada, the European Union, and Japan.

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