quizalofop ethyl residues at tolerance levels of 1.0 ppm, we calculate that the TMRC in the infants' and children's diets would be 0.000847 mg/kg/day or 9.4% of the RfD.

As indicated above, infants and children have a low potential for quizalofop ethyl exposure because of both the low levels of canola oil in the diet, and the absence of detectable residues in field-treated canola. The toxicology profile of quizalofop ethyl demonstrates low mammalian toxicity. Because there was no evidence that offspring were uniquely susceptible to the toxic effects of quizalofop ethyl, an additional 10-fold uncertainty factor should not be required to protect infants and children. Therefore, the RfD of 0.009 mg/kg/day, which utilizes a 100-fold safety factor, is appropriate to assure a reasonable certainty of no harm to infants and children from aggregate exposure to quizalofop ethyl.

F. International Tolerances

Harmonization of Tolerances: Since there are no Mexican or Codex MRLs/tolerances, compatibility is not a problem at this time. Compatibility cannot be achieved with the Canadian negligible residue type limit at 0.1 ppm at the USA use pattern, which had findings of real residues above 0.1 ppm. (James Tompkins)

[FR Doc. 97–32935 Filed 12–16–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–782; FRL–5759–1]

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 food commodities.

DATES: Comments, identified by the docket control number PF–782, must be received on or before January 16, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow 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. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail: miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
James Tompkins (PM 25).	Rm. 239, CM #2, 703–305–5697, e-mail: tompkins.james@epamail.epa.gov.	Do.

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 food 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 granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–782] (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 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–782] and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: December 3, 1997.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. 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 6F4772

EPA has received a pesticide petition (PP 6F4772) from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268, 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 fluroxypyr methylheptyl ester (MHE) and its only significant metabolite fluroxypyr, free and conjugated, all expressed as fluroxypyr in or on the raw agricultural commodities wheat, barley, and oats as follows: 0.5 parts per million (ppm) (grain), 10 ppm (straw and forage), 20 ppm (hay), and 0.5 ppm (aspirated grain fractions, wheat). Because residues of fluroxypyr MHE or fluroxypyr, free or conjugated, may occur in animal feeds derived from wheat, barley, and oats, the following meat and milk tolerances are also being proposed: 0.1 ppm (meat, fat, milk, and meat byproducts except for kidney) and 0.5 ppm (kidney). The proposed analytical method is based on gas chomatography (GC) with mass spectral (MS) detection. 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 metabolism of fluroxypyr MHE in plants (wheat) and animals (goats and poultry) is adequately understood for the purposes of this tolerance. A rotational crop study showed no carryover of significant fluroxypyr MHE related residues in representative test crops except for cereal grains for which tolerances are being proposed.

2. *Analytical method*. There is a practical method (GC with MS detection) for measuring levels of fluroxypyr MHE in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set for the proposed tolerances. Fluroxypyr has been tested through the FDAs Multiresidue Methodology, Protocols C, D. and E. The results have been published in the FDA Pesticide Analytical Manual, Volume I.

3. *Magnitude of residues*. Magnitude of residue studies were conducted for wheat, barley, and oats. Residues of fluroxypyr MHE did not concentrate in process fractions in samples treated at a 7.5 X application rate.

B. Toxicological Profile

1. *Acute toxicity*. Fluroxypyr MHE has low acute toxicity. The rat oral LD_{50} is >5000 mg/kg, the rabbit dermal LD_{50} is >2000 mg/kg, and the rat inhalation LC_{50} is >1.0 mg/l (1,000 mg/cubic meter), the maximum attainable concentration. In addition, fluroxypyr MHE is not a skin sensitizer in guinea pigs, has no dermal irritation in rabbits, and shows mild ocular irritation in rabbits. The end use formulation of fluroxypyr MHE has a similar low acute toxicity profile.

2. *Genotoxicity*. Short term assays for genotoxicity consisting of a bacterial reverse mutation assay (Ames test), an *in vitro* assay for cytogenetic damage using the Chinese hamster ovary cells, an in vitrochromosomal aberration assay using rat lymphocytes, and an *in vivo* cytogenetic assay in the mouse bone marrow (micronucleus test) have been conducted with fluroxypyr MHE. DowElanco believes that these studies show a lack of genotoxicity. In addition, short term assays for genotoxicity consisting of an Ames metabolic activation test, point mutations at the HGPRT-Locus of Chinese hamster ovary cells, *in vivo* and *in vitro* chromosomal aberrations in the Chinese hamster ovary cells, unscheduled DNA synthesis in human embryonic cells, and an assay in mouse lymphoma cells have been conducted with fluroxypyr. DowElanco believes that the weight of evidence also indicates a lack of genotoxicity.

3. *Reproductive and developmental toxicity*. Developmental studies in rats and rabbits were conducted with both fluroxypyr MHE and fluroxypyr. Studies with fluroxypyr MHE showed maternal and fetal no observed effect levels (NOELs) of 300 milligram/kilogram (mg/kg/day) (rat) and 500 mg/kg/day (rabbit). Studies with fluroxypyr showed no observed adverse effect levels (NOAELs) in the rat of 250 mg/kg/day for maternal effects and 500 mg/kg/day for fetal effects and a NOEL in the rabbit of 250 mg/kg/day for both maternal and fetal effects. DowElanco believes that these studies show that fluroxypyr and fluroxypyr MHE are not teratogenic nor will they interfere with in utero development. Two multi-generation reproduction studies were conducted with fluroxypyr in rats. The first in Wistar rats showed no effect on fertility or reproductive performance and had a NOAEL of 500 mg/kg/day (highest dose tested). The second study in Sprague-Dawley rats showed a parental NOEL for systemic effects of 100 mg/kg/day in male rats and 500 mg/kg/day in female rats. The NOEL for reproductive effects was 750 mg/kg/day for males and 1,000 mg/kg/day for females (highest dose tested). The NOEL for neonatal effects was 500 mg/kg/day.

4. *Subchronic toxicity*. Fluroxypyr MHE showed a NOEL of 1,000 mg/kg/day in a 90-day rat dietary study and a 21-day rabbit dermal study. Ninety day feeding studies with fluroxypyr showed NOELs of 80 mg/kg/day (Wistar rats), 700 mg/kg/day (Fischer 344 rats), 1342 mg/kg/day (male mice), and 1,748 mg/kg/day (female mice). In a 4-week dietary, range finding study with fluroxypyr in dogs the NOEL was >50 mg/kg/day.

5. *Chronic toxicity*. Based on chronic testing with fluroxypyr in the mouse, dog, and rat (two studies), a reference dose (RfD) of 0.8 mg/kg/day is proposed for fluroxypyr and fluroxypyr MHE. The RfD has incorporated a 100-fold safety factor to the NOEL found in the rat chronic test. NOELs found in the chronic dietary studies are as follows: 150 mg/kg/day (dog), 300 mg/kg/day (mouse), 80 mg/kg/day (Wistar rats), 100 mg/kg/day (male Fischer 344 rats), and 500 mg/kg/day (female Fischer 344 rats).

6. *Animal metabolism*. Both fluroxypyr and fluroxypyr MHE have been evaluated in rat metabolism studies. In summary, these studies show that fluroxypyr MHE is rapidly hydrolyzed and the fate of the hydrolysis products, fluroxypyr and 1-methylheptanol, are independent of whether they were given as the ester or the acid. Fluroxypyr, *per se*, was extensively absorbed and rapidly excreted principally unchanged in the urine. 1-Methylheptanol also was rapidly absorbed and rapidly eliminated. Repeated administration of fluroxypyr MHE was not associated with accumulation in tissues. Also, the metabolism and pharmacokinetics of methylheptanol are comparable to that of the methylheptyl portion of fluroxypyr MHE.

7. *Metabolite toxicology*. Administration of fluroxypyr, as the acid or methylheptyl ester, in a variety of toxicological studies has produced similar effects. The principal response to sufficiently high dosages, whether administered over the short-term or, in some cases, over a lifetime, was nephrosis. Fluroxypyr is an organic acid that is actively excreted into the urine by the kidney. Thus, the target organ and dose response relationship for fluroxypyr toxicity are entirely consistent with the data on the toxicokinetics of fluroxypyr. Metabolism studies have shown that fluroxypyr MHE is rapidly and completely hydrolyzed to fluroxypyr acid and methylheptanol.

8. *Carcinogenicity*. Using the Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), it is proposed that fluroxypyr and fluroxypyr MHE be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. DowElanco believes that there was no evidence of carcinogenicity in an 18-month mouse feeding study and a 24-month rat feeding study at all dosages tested. The NOELs shown in the mouse and rat oncogenicity studies were 1,000 and 320 mg/kg/day, respectively. A maximum tolerated dose was achieved at the top dosage level tested in both of these studies based on excessive renal toxicity. Thus, the doses tested are adequate for identifying a cancer risk. Accordingly, DowElanco believes that a cancer risk assessment is not needed.

9. *Endocrine effects*. There is no evidence to suggest that fluroxypyr and fluroxypyr HME have an effect on any endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. An over estimation of dietary exposure from use of fluroxypyr MHE on wheat, barley, oats is determined by basing the TMRC on the conservative assumptions that all cereal grain commodities will have tolerance level residues of fluroxypyr and that 100% of the wheat, barley, and oat crops grown in the U.S. are treated with fluroxypyr MHE. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related foodstuffs consumed by various population subgroups. There are no other established U.S. tolerances or exemption from tolerances for fluroxypyr MHE and no other registered uses for fluroxypyr MHE on food or feed crops in the United States. The use of a tolerance level and 100% of crop treated clearly results in an overestimate of human exposure and a safety determination for the use of fluroxypyr MHE on wheat, barley, and oats that is based on a conservative exposure assessment.

ii. *Drinking water*. Another potential source of dietary exposure are residues in drinking water. Based on the available environmental studies conducted with fluroxypyr MHE and fluroxypyr wherein the properties of these materials show little persistence in the soil environment, there is no anticipated exposure to residues of fluroxypyr MHE and fluroxypyr in drinking water. In addition, there is no established Maximum Concentration Level for residues of fluroxypyr MHE and fluroxypyr in drinking water.

2. *Non-dietary exposure*. There are no other uses currently registered for fluroxypyr MHE and fluroxypyr. The proposed use on wheat, barley, and oats involves application of fluroxypyr MHE to crops grown in an agriculture environment. Thus, the potential for non-occupational exposure to the general population is not expected to be significant.

D. Cumulative Effects

The potential for cumulative effects of fluroxypyr MHE and fluroxypyr and other substances that have a common mechanism of toxicity is also considered. There is no reliable information to indicate that toxic effects produced by fluroxypyr MHE and fluroxypyr would be cumulative with those of any other pesticide chemical. Thus, it is appropriate to consider only the potential risks of fluroxypyr MHE and fluroxypyr in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions and the proposed RfD, the dietary exposure to fluroxypyr MHE use on wheat, barley, and oats will utilize 0.2% of the RfD for the U.S. population. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Since there are no anticipated residues in drinking water or from other non-occupational sources and no reliable information exists on cumulative effects due to common mechanism of toxicity, the aggregate exposure to fluroxypyr MHE is adequately represented by the dietary route. Thus, DowElanco believes that there is reasonable certainty that no harm will result from aggregate exposure to fluroxypyr MHE residues on wheat, barley, and oats.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of fluroxypyr MHE, data from developmental toxicity studies in rats and rabbits and a 2-generation reproduction study in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability and potential systemic toxicity of mating animals and on various parameters associated with the well-being of pups.

FFDCA section 408 provides that EPA may apply an additional safety 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 the current toxicological data requirements, the database for fluroxypyr MHE relative to pre- and post-natal effects for children is complete. Further, for fluroxypyr MHE, the NOEL in the chronic feeding studies which was used to calculate the RfD (0.8 mg/kg/day) is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of more than three.

Concerning the reproduction studies in rats, the pup effects shown at the highest dose tested (1,000 mg/kg/day) were attributed to maternal toxicity. Therefore, DowElanco concludes that an additional uncertainty factor is not needed and that the RfD at 0.8 mg/kg/day is appropriate for assessing risk to infants and children.

As noted above for the general U.S. population, aggregate exposure for infants and children will result from the dietary (i.e. not drinking water or non-occupational) route of exposure. In addition, there is no reliable information that shows cumulative effects based on a common mechanism of toxicity for infants and children. Using the conservative exposure assumptions previously described, the percent RfD utilized by the aggregate dietary exposure to residues of fluroxypyr MHE on wheat, barley, and oats is 0.6% for children 1 to 6 years old, the most sensitive population subgroup. Thus, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, DowElanco believes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to fluroxypyr MHE residues on wheat, barley, and oats.

F. International Tolerances

There are no Codex maximum residue levels established for residues of fluroxypyr MHE and fluroxypyr on any food or feed crop. (J. Miller)

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

PP 1F4032

EPA has received a pesticide petition (PP 1F4032) from E.I. du Pont de Nemours and Company, Barley Mill Plaza, Walker's Mill Bldg. 37, Wilmington, 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 ethametsulfuron in or on the raw agricultural commodity canola at 0.1 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. *Plant metabolism*. The qualitative nature of the residues of ethametsulfuron methyl is adequately understood. The unmetabolized parent compound was the major residue found in a canola metabolism study up to 30 days after application. The principal route of metabolic breakdown of ethametsulfuron methyl in canola is dealkylation from the triazine ring. The initial step in the metabolic breakdown is deethylation to form *O*-deethyl ethametsulfuron methyl. Further metabolism forms *N*-demethyl-*O*-deethyl ethametsulfuron methyl and more minor polar metabolites. For purposes of establishing the proposed tolerance, the parent compound ethametsulfuron methyl is the only residue of concern.

The available metabolism studies indicate that total radioactive residues found in mature seeds, when rapeseed was treated at a rate equivalent to the proposed application rate, ranged from 0.008 to 0.012 ppm. These terminal residues may consist of the parent compound, *O*-deethyl ethametsulfuron methyl, O-deethyl-N-demethyl ethametsulfuron methyl and other minor metabolites.

2. *Analytical method*. Analytical methods are available to measure the parent compound in oil seeds, and in oil seed processing fractions. The quantification of ethametsulfuron methyl is by normal phase high performance liquid chromatography (HPLC) using a photoconductivity detector. The Limit of Quantitation (LOQ) for the analytical method is 0.02 ppm.

3. *Magnitude of residues*—i. *Magnitude of the residue in plants.* The results of the seed analyses from canola/seed show that no detectable residues of ethametsulfuron methyl were found in canola/seed harvested 60 to 137 days after treatment at exaggerated rates of 3X of the normal application rate.

ii. *Magnitude of the residue in processed commodities*. Analyses of canola processed fractions (whole seed, pressed cake, desolventized meal, crude oil, pressed oil, solvent extracted oil, degummed oil, refined washed oil, refined bleached oil, and deodorized oil) show that levels of ethametsulfuron methyl were found to be less than 0.02 ppm, the limit of quantitation of the method in all of the fractions evaluated. All of the processed fractions were obtained from seed harvested 92 days after application at proposed use rates and exaggerated rates.

B. Toxicological Profile

1. *Acute toxicity*. Based on EPA criteria, ethametsulfuron methyl is relatively non-toxic, and be categorized as Toxicity Category IV (oral and inhalation routes) and Category III (dermal exposure). LD_{50}s are >5,000 mg/kg for acute oral toxicity in rats, >2,000 mg/kg for acute dermal toxicity in rabbits, and >5.7 mg/L for acute inhalation toxicity in rats. For technical grade active ingredient, primary eye irritation in rabbits is classified as Tox Cat II. For formulated product, primary eye irritation in rabbits is classified as Tox Cat IV. Primary dermal irritation in rabbits is classified as Tox Cat IV. Dermal sensitization in guinea pigs is classified as ''Not a skin sensitizer.''

2. *Genotoxicity*. This compound was negative in the following tests that have been conducted to determine the genotoxic and mutagenic potential of ethametsulfuron methyl: Mutagenicity assays conducted in bacteria (Ames test) and in cultured Chinese Hamster Ovary cells; a test that measures the induction of chromosomal aberrations in bone marrow cells isolated from rats treated with ethametsulfuron methyl; micronuclei induction in bone marrow cells from mice; and negative in a text that measures DNA damage in cultured rat liver cells. Based on the weight of these data, E.I du Pont concludes that ethametsulfuron methyl is neither genotoxic or mutagenic.

3. *Reproductive and developmental toxicity.* A 2-generation, four litter reproduction study with CD rats treated with dietary levels of 0, 250, 5,000, 20,000 ppm of ethametsulfuron methyl failed to reveal any evidence suggestive of an adverse effect on reproductive potential. A NOEL was indicated at the mid dose level of 5,000 ppm (equivalent to approx. 433 mg/kg b.w./day, actual intake) based on significantly ($p<0.5$) decreased body weights in the high dose treated F0 and F1 generation males.

A developmental toxicity study of ethametsulfuron methyl in rabbits indicated that dams administered 4,000 mg/kg (highest dose tested) had a higher mortality rate, lower food consumption and body weight gains, increased incidences of gross clinical signs of toxicity and of abortions, and increased absolute and relative liver weights. Absolute and relative liver weights were also slightly greater for dams administered 1,000 mg/kg. There were no compound-related effects observed for dams administered 250 mg/kg.

Dams administered 4,000 mg/kg also had a decrease in the number of live fetuses. This was related to an increase in the number of early resorptions. There were no other compound-related effects on the dams, nor were there any effects on fetal weights, malformations or variations incidences. The NOELs for this study were 250 mg/kg for the dams and 1,000 mg/kg for the fetus. Ethametsulfuron methyl was neither teratogenic in rabbits nor uniquely toxic to the conceptus.

A developmental toxicity study was also conducted in rats treated at doses of 0, 60, 250, 1,000, or 4,000 mg/kg. Among the dams of the groups given ethametsulfuron methyl, no compound-related mortality or clinical abnormalities were observed. For the treatment period, the high dose group had a lower weight gain and significantly decreased food consumption compared to the control group. No other significant differences in body weight changes or food consumption were observed. A significant trend was indicated for mean fetal weight and the mean fetal weight of the high dose group was lower than that of the control group. No significant differences were observed in the rates of malformations or developmental variations. Under the conditions of the study, the apparent no effect level for the dam and fetus was 1,000 mg/kg/day. Thus ethametsulfuron methyl was not uniquely toxic to the conceptus nor was it teratogenic in rats.

4. *Subchronic toxicity—i. Rat.* A 90-day feeding study followed by a 1-generation reproduction phase in rats at dietary levels of 0, 100, 1,000, and 5,000 ppm of ethametsulfuron methyl failed to elicit any signs of overt toxicity or any adverse effect on reproductive performance at levels as high as 5,000 ppm (equivalent to 0.5% of the diet or approximately 409 mg/kg b.w./day, actual intake). The No Observed Adverse Effect Level (NOAEL) for this study was, therefore, the high dose level of 5,000 ppm.

ii. *Mouse.* A 90-day dietary feeding study in CD-1 mice at levels of 0, 50, 500, 2,500 and 5,000 ppm indicated a No Observed Effect Level (NOEL) for females and a NOAEL for males set at

the high dose level of 5,000 ppm (equivalent to approximate 687 mg/kg b.w./day, actual intake for males).

iii. *Dog.* Dietary administration of technical ethametsulfuron methyl to dogs for 90 days at levels of 0, 100, 3,500 or 10,000 ppm failed to reveal any evidence of treatment-related toxicity at levels as high as 10,000 ppm (equivalent to 1% of the diet or approximately 386 mg/kg b.w./day, based on actual intake).

5. *Chronic toxicity*—i. *Rat.* Administration of ethametsulfuron methyl to Sprague-Dawley rats for up to 24 months at dietary levels of 0, 50, 500 and 5,000 ppm revealed a NOAEL for in-life parameters of 5,000 ppm (equivalent to 238.5 mg/kg b.w./day, actual intake), based on questionable toxicological significance of decreased ($p<0.05$) serum sodium levels in both the 5,000 ppm treated males and females during the first 12 months of treatment. The effects on serum sodium levels in the high dose groups were described as mild (representing a decrease in 2-6% of the control values) and occurring in the absence of any associated pathological changes in the kidney. Treatment with the test material at dietary levels as high as 5,000 ppm (equivalent to 0.5% of the diet) failed to elicit any evidence of treatment-related neoplastic potential.

ii. *Dogs.* Chronic dietary administration of the test material to dogs at levels of 0, 250, 3,000 and 15,000 ppm for 1-year indicated a NOEL of 3,000 ppm, equivalent to approximately 87 mg/kg b.w./day actual intake, based on compound-related effects expressed in the 15,000 ppm treated group as decreased body weight gain and food efficiency values in the males. Significantly decreased serum sodium levels in both sexes at the high dose treated level were not associated with any evidence of renal pathology. In the absence of any collaborative clinical or pathological findings differences in organ weights relative to body or brain weight were considered to be of doubtful biological significance.

iii. *Mouse.* Administration of the test material to CD-1 mice at dietary levels of 0, 25, 500, and 5,000 ppm for the period of up to 78 weeks failed to reveal any overt signs of treatment-related toxicity of dietary levels of up to 5,000 ppm (equivalent to 818 mg/kg bwt/day, actual intake). Although a direct effect of treatment on body weights or weight gains could not be established, overall body weight gain in the 5,000 ppm treated male mice was depressed (non-significant, $p>0.05$) by 10% when compared to the controls. There was no evidence of any treatment-related oncogenic potential.

6. *Animal metabolism*. When administered via oral gavage to rats, ethametsulfuron methyl was rapidly metabolized and excreted in the urine and feces. Within 3 days, greater than 90% of the administered dose was excreted by male rats and greater than 80% was excreted by females. Approximately 50% of the administered dose was excreted as unchanged ethametsulfuron methyl. The remainder was converted predominately to *N*-demethyl ethametsulfuron methyl and *O*-deethyl ethametsulfuron methyl, which are considered by by-products of cytochrome P450-mediated reactions. Less than 0.02% of the administered dose remained in the carcass or tissues. There was no significant or preferential accumulation of ethametsulfuron methyl or its metabolites in any tissue. Because of the short excretion half-life, repeated daily exposures are not expected to result in significant body burdens of ethametsulfuron methyl.

7. *Metabolite toxicology*. There is no evidence that the metabolites of ethametsulfuron methyl as identified as either the plant or animal metabolism studies are of any toxicological significance.

8. *Endocrine effects*. No special studies investigating potential estrogenic or endocrine effects of ethametsulfuron methyl have been conducted. However, the standard battery of required toxicology studies have been completed. These include an evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. Based on these studies there is no evidence to suggest that ethametsulfuron methyl has an effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure—i. Food*. Based on the residue data and the proposed single-crop use, potential for dietary exposure of ethametsulfuron methyl from food sources is extremely low. Residue studies have shown no residues above the LOQ (residues <0.02 ppm) in any canola seed samples evaluated, including the canola oil processed fractions. No dietary exposure is anticipated from secondary residues in meat or milk. Although canola meal is considered a minor feedstuff for cattle and poultry (representing a maximum of 15% of an animal's diet), field residue studies showed ethametsulfuron methyl residues were all below the LOQ (<0.02 ppm) in all of the canola RACs and processed fractions, including meal, even when the crop was treated at 2-3X the proposed maximum use rate.

Direct human consumption of canola as a food commodity in the United States is extremely low. Canola is a minor crop in the U.S., and the only canola fraction used as a food product is the refined canola oil. A dietary risk evaluation (DRES) was conducted to determine the theoretical maximum residue contribution of ethametsulfuron methyl in the diet as a result of agricultural use on canola. Unfortunately, consumption data for canola oil does not exist in the 1977-1979 food consumption database used in EPA's DRES system, since at that time, canola oil was not a significant part of the U.S. diet. Since 1977 more canola oil is used in U.S. homes, although total production and usage are still minor when compared to other edible oils such as soybean oil.

Conservative assumptions were used to estimate canola consumption in the United States. The USDA's Oilseed Analysis Division has indicated that an average of 1.1 billion pounds of canola oil was used in the United States annually over the past 5 years. The dietary exposures that might occur by way of canola oil consumption can be estimated by taking the average annual use of canola oil in the United States (includes both domestically produced and imported canola oils) and dividing it by the approximate US population of 266.3 million people. This provides a per-capita consumption estimate for the general population. Using this approach, total canola oil consumption on a grams per kg body weight per day was calculated by dividing by the average days in a year and average body weight of a person (60 kg). The 60 kg value is used by the US EPA as part of their ''Food Factor'' system, and is also supported by taking the average weight of children between the ages of 6 months to 19 years (36 kg) and the average weight of adults of 70 kg and assuming a 69 year life span (as proposed in the review draft of the US EPA's Exposure Factors Handbook). Using these assumptions, canola oil consumption was calculated to be 0.088 g/kg bw/day.

While this method provides a useful approximation of canola consumption, this is clearly a conservative estimate for risk assessment purposes, since this estimate assumes that all of the canola oil used in the US is indeed ingested. In reality, not all the oil that is used in cooking or deep-fat frying is consumed but instead, is discarded or recycled. Another indication that the consumption value of 0.088 g/kg bw/day is an over-estimate is from the USDA's 1989-1992 food survey (not yet included in the EPA's DRES system),

which indicates canola oil consumption is 0.00023 g/kg/day for the general U.S. population.

Using the consumption estimate of 0.088 g canola oil/ kg bw/day for the general US population, and assuming that 100% of the canola crop is treated with ethametsulfuron methyl and all canola consumed contains residues at the proposed tolerance level of 0.1 ppm, the theoretical maximum residue contribution of ethametsulfuron methyl in the diet is calculated to be 0.00001 mg/kg/day or <0.01% of the RfD of 0.87 mg/kg/day.

ii. *Drinking water.* Another potential source of dietary exposure to pesticides are residues in drinking water. There is no established Maximum Concentration Level (MCL) for ethametsulfuron methyl in water. Based on the low use rate of ethametsulfuron methyl, and a use pattern that is not widespread (since the only proposed use is on a minor crop), DuPont does not anticipate residues of ethametsulfuron in drinking water and exposure from this route is unlikely.

2. *Non-dietary exposure.* Ethametsulfuron methyl is not registered for any use which could result in non-occupational, non-dietary exposure to the general population. Ethametsulfuron methyl is a herbicide with proposed use only on canola. There are no other food uses, nor are there any residential or non-crop uses of this active ingredient. Therefore, the only potential for non-occupational aggregate exposure would come from dietary intake.

D. Cumulative Effects

Ethametsulfuron methyl belongs to the sulfonylurea class of compounds. Other compounds in this class are registered herbicides. However, the herbicidal activity of the sulfonylureas is due to the inhibition of acetolactase synthase (ALS), an enzyme only found in plants. ALS is part of the biosynthetic pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the low toxicity of the sulfonylurea compounds in animals. There is no evidence to indicate or suggest that ethametsulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemicals.

E. Safety Determination

1. *U.S. population.* Using the conservative exposure assumptions described above and based on the most sensitive species chronic NOEL of 87 mg/kg and a Reference Dose (RfD) of 0.87 mg/kg/day, the proposed use of ethametsulfuron methyl on canola is expected to utilize 0.001% of the RfD for the general U.S. population. Generally, exposures below 100 percent of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose risk to human health. Thus, DuPont concludes that there is a reasonable certainty that no harm will result from aggregate exposure to ethametsulfuron methyl resulting from proposed agricultural use on canola.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of ethametsulfuron methyl, data were considered from developmental toxicity studies in the rat and rabbit, and a multi-generation reproduction study in rats. The developmental toxicity studies demonstrated that even at the high oral doses used in these studies (up to 4,000 mg/kg in rabbits and rats), no teratogenic effects were found in either species nor was the compound found to be uniquely toxic to the conceptus.

The 2-generation reproduction study in rats treated at dietary levels as high as 20,000 ppm on a daily basis throughout 2 generations (equivalent to 1,582 mg/kg/day for males and 1817 mg/kg/day for females), showed no evidence of effects on reproductive performance in the adults, or evidence of gross or histopathological effects in the adult or weanling rats in any test group. This study indicates that ethametsulfuron methyl is not a reproductive toxicant.

As mentioned previously, canola oil is a very minor component of the diet, and thus had not been included as part of the 1977-79 food survey used in EPA's DRES system. DuPont is not aware of specific food survey data concerning consumption of canola oil by infants and children. However, the 1977-79 food survey database does provide consumption data for other edible oils for each of the population subgroups, including infants and children. This data indicate that non-nursing infants consume more soybean and coconut oil than any of the other 22 population subgroups, specifically consuming 4.2 times more soybean oil and 49.1 times more coconut oil than the consumption by the general US population. The data also show that children 1-6 consume more corn, cottonseed, peanut and sunflower oil than any other subgroup listed, to a maximum of 2 times more than the general U.S. population. Using these data and making the most conservative assumption to extrapolate to canola oil, we can estimate that infants and children consume 49 times more canola oil than does the U.S. population, and calculate an approximate daily consumption of 4.3 g canola oil/kg body weight. If we use the additional conservative assumptions that all the canola oil consumed contains ethametsulfuron methyl residues at tolerance levels of 0.1 ppm, we calculate that the maximum theoretical residue concentration of ethametsulfuron methyl in the infants' and children's diets would be 0.00049 mg/kg/day or <0.05% of the RfD.

As indicated above, DuPont concludes that infants and children have a low potential for ethametsulfuron methyl exposure because of both the low level of canola oil in the diet, and the absence of detectable residues in field-treated canola. The toxicology profile of ethametsulfuron methyl demonstrates low mammalian toxicity, and results from the developmental and reproduction studies indicate that there is no additional sensitivity for infants and children. Therefore, DuPont concludes that an additional safety (uncertainty) factor is not warranted and the RfD of 0.87 mg/kg body weight/day, which utilizes a 100-fold safety factor, is appropriate to assure a reasonable certainty of no harm to infants and children from aggregate exposure to ethametsulfuron methyl.

F. International Tolerances

Ethametsulfuron methyl and its end-use product Muster are registered only in Canada on canola/rape and mustard with a MRL value of 0.1 ppm. A CODEX tolerance for ethametsulfuron methyl has not been established. (Jim Tompkins)

3. Monsanto

PP 7F4840

EPA has received a pesticide petition (PP 7F4840) from Monsanto, Suite 1100, 700 14th St., NW., Washington, DC 2005, 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 sulfosulfuron 1-(4,6-dimethoxypyrimidin-2-yl)-3-[(2-ethanesulfonyl-imdazo[1,2-a]pyridine-3-yl)sulfony]urea in or on the raw agricultural commodities. The proposed analytical method involves hydrolyzing sulfosulfuron and its imadazopyridine-containing metabolites under acidic conditions to the common chemophore, ethyl sulfone. Ethyl sulfone is then separated and quantitated by High Performance Liquid Chromatography (HPLC) with fluorescence detection. 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*. Metabolism of sulfosulfuron in plants is negligible. The nature of the major sulfosulfuron residues in wheat matrices depends primarily on the mode of application with reliance upon metabolism in the soil.

Postemergence applications result in residues that are mostly made up of parent compound, with small amounts of five to six metabolites that together make up less than 15% of the total radioactive residue.

Preemergence application result in soil degradation of the parent compound followed by uptake primarily of the imidazopyridine ring-containing metabolites and small amounts of the parent compound. The pyrimidine ring-containing metabolites under these conditions are tightly bound to the soil, resulting in negligible uptake of these residues. Little further metabolism of the imidazopyridine metabolites takes place in the plant. The predominant residues resulting from preemergence applications were sulfonamide (22% TRR) and guanidine (18.3% TRR).

In both cases, translocation of residue to the grain is negligible. The highest residues are observed following postemergence applications and the residues are primarily parent compound.

In rotational crops, residues were low, with the TRR's not exceeding 0.01 ppm in most crops. The most abundant metabolite was sulfonamide, with low levels of a sulfonamide-sugar conjugate and parent compound also observed.

2. *Analytical method*. The primary crop (wheat) residue and the secondary (animal products) residues are analyzed as total residue by hydrolyzing sulfosulfuron and its imadazopyridine-containing metabolites under acidic conditions to the common chemophore, ethyl sulfone. Ethyl sulfone is then separated and quantitated by High Performance Liquid Chromatography (HPLC) with fluorescence detection.

3. *Magnitude of residues*. EPA has received a pesticide petition from Monsanto Company proposing to amend 40 CFR part 180 by establishing a tolerance for residues of the herbicide sulfosulfuron in or on the following:

Commodity	Part per million (ppm)
Wheat	
grain	0.01 ppm (limit of quantitation 0.008 ppm)
straw	0.1 ppm
hay	0.3 ppm
forage	3.0 ppm
Animal Products	
milk	0.004 ppm
fat	0.004 ppm
meat	0.004 ppm
muscle	0.004 ppm
meat by-products	0.1 ppm
kidney	0.1 ppm
liver	0.1 ppm

B. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study with an LD_{50} of >5,000 mg/kg, EPA Category IV. A rabbit acute dermal study with an LD_{50} of >5,000 mg/kg, EPA Category IV. A rat inhalation study with and LC_{50} of >3.0 mg/l, the highest concentration generated, EPA Category IV. A primary eye irritation study in the rabbit showing moderate eye irritation, EPA Category III. A primary dermal irritation study in the rabbit showing essentially no irritation, EPA Category IV. A dermal sensitization study in the guinea pig showing no potential for sensitization. Acute and subchronic neurotoxicity studies in rats demonstrating no neurotoxicity potential. Sulfosulfuron has a low order of acute toxicity.

2. *Genotoxicity*. An *in vitro* Ames/*Salmonella* mutagenicity assay in five commonly used strains was negative for mutagenic potential.

An *in vitro* CHO/HGPRT Gene Mutation assay was negative for mutagenicity up to the limit of solubility.

An *in vitro* chromosomal aberration test in cultured mammalian cells demonstrated the induction of chromosomal aberrations only under conditions of prolonged incubation at high dose levels that exceeded the solubility of the test material. The mechanism responsible for this induction and the biological relevance of the effect is not clear. Other, more relevant, chromosomal aberration tests (see below) were negative.

An *in vitro* chromosome aberration study in human lymphocytes was negative for chromosomal aberrations.

An *in vivo* bone marrow micronucleus assay in the mouse was negative for chromosomal effects.

The weight of evidence demonstrates that sulfosulfuron does not produce significant genotoxic or mutagenic effects.

3. *Reproductive and developmental toxicity*. A developmental study in the rat demonstrated no signs of maternal or developmental toxicity up to the maximum dose level of 1,000 mg/kg/day. The NOEL was considered to be 1,000 mg/kg/day.

A developmental study in the rabbit demonstrated no signs of maternal or developmental toxicity up to the maximum dose level of 1,000 mg/kg/day. The NOEL was considered to be 1,000 mg/kg/day.

A 2-generation reproduction study in the rat demonstrated a subchronic toxicity NOEL of 5,000 ppm based on body weight and food consumption decreases, urinary bladder calculi formation and minor bladder and kidney pathology. There were no effects on reproduction or fertility up to 20,000 ppm, the highest dose tested. Sulfosulfuron demonstrates no reproductive effects in rats and no teratogenic or developmental effects in rats and rabbits.

4. *Subchronic toxicity*. A 28-day dermal study in the rat with a NOEL of at least 1,000 mg/kg/day, the highest dose tested. A 90-day feeding study in the rat resulted in only mild body weight/weight gain effects at 20,000 ppm, the highest dose tested. The NOEL for both males and females was considered to be 6,000 ppm. A 90-day feeding study in the dog demonstrated subchronic toxicity, primarily in the urinary bladder, secondary to urinary crystal formation and urolithiasis at dose levels of 300 and 1,000 mg/kg/day in females and at 1,000 mg/kg/day in males. The NOEL was considered to be 100 mg/kg/day in females and 300 mg/kg/day in males. Sulfosulfuron has a low order of subchronic toxicity, related only to the precipitation of test material in the urinary bladder of dogs at high doses.

5. *Chronic toxicity*. A 1-year study in the dog demonstrated toxicity in the urinary bladder secondary to urinary crystal and calculus formation at 500 mg/kg/day in a single male animal. Urinary crystal formation was observed in females at 500 mg/kg/day with no subsequent pathology. The NOEL was considered to be 100 mg/kg/day for male and female dogs.

A combined chronic toxicity/oncogenicity study in the rat demonstrated chronic toxicity, primarily in the urinary bladder, in males and females at 5,000 and females at 20,000 mg/kg/day. The NOEL for chronic toxicity was considered to be 500 ppm or 24.4 mg/kg/day. This is the

lowest NOEL and is used in the calculation of the Reference Dose (RfD).

An 18-month oncogenicity study in the mouse demonstrated chronic toxicity, primarily in the urinary bladder, of male mice at 3,000 and 7,000 ppm. No chronic toxicity was observed in females. The NOEL for chronic toxicity was considered to be 700 ppm for male mice and 7,000 ppm for female mice. Sulfosulfuron demonstrates chronic toxicity related only to the formation of crystals and calculi of the compound in the urinary bladders of mice, rats and dogs.

6. *Carcinogenicity*. An 18-month oncogenicity study in the mouse demonstrated a small increase in the incidence of benign mesenchymal tumors of the urinary bladder submucosa in male mice with urinary bladder calculi at 7,000 ppm. However, these tumors are reportedly unique to Swiss-derived mice and were considered to be of biological relevance only to the mouse by a Independent Working Group on Mouse Mesenchymal Tumors convened by the International Life Sciences Institute (ILSI).

A combined chronic toxicity/ oncogenicity study in the rat (same as above) demonstrated a urinary bladder transitional cell carcinoma and a urinary bladder transitional cell papilloma in two females at 5,000 mg/ kg/day, probably secondary to urinary system calculi formation and (chronic) irritation.

The low incidences of oncogenicity observed in the oncogenicity studies conducted with sulfosulfuron are either considered to be relevant to the mouse only or a secondary threshold effect related to chronic irritation resulting from bladder stone formation at high doses. Sulfosulfuron is not considered to be a primary oncogen.

Using the Guidelines for Carcinogenic Risk Assessment published September 24, 1986, Monsanto believes that the EPA would classify sulfosulfuron as a Group ''C'' carcinogen, without quantitative risk assessment, i.e., using the margin of exposure (MOE) approach for risk assessment. Under the proposed guidelines published April 10, 1996, however, Monsanto believes that sufosulfuron should be included in the ''Not Likely Human Carcinogen'' category based upon mechanistic considerations. To quote the 1996 EPA guideline document discussing a similar effect in a rat study:

''A major uncertainty is whether the profound effects of (substance 5) may be unique to the rat. Even if (substance 5) produced stones in humans, there is only limited evidence that humans with bladder stones develop cancer. Most often human bladder stones are either passed in the urine or lead to symptoms resulting in their removal.''

In either case, a Margin of Exposure assessment or reference dose (RfD) approach would be utilized. Since the chronic NOEL for male rats is lower than the oncogenic NOEL for female rats (24 mg/kg/day vs 30 mg/kg/day), the male rat chronic NOEL was used with a 100 fold safety factor for a reference dose of 0.24 mg/kg/day, for the quantitation of human risk.

7. *Animal metabolism*. An animal metabolism study was conducted in the rat using sulfosulfuron radiolabeled in both the pyrimidine and iminodazopyridine rings to detect possible cleavage of the sulfonylurea bond. Following oral dosing of sulfosulfuron, absorption was found to be greater at low doses (>90%) than at the higher doses (40%). Sulfosulfuron was readily excreted, mostly unchanged, with urinary excretion the major route of elimination at low doses and fecal excretion the major route at high doses. Greater than 90% of the dose was excreted 3 days after administration. Expiration as carbon dioxide or volatiles was not a significant route of elimination. Metabolism of sulfosulfuron in the rat occurred to only a limited extent with demethylation and pyrimidine ring hydroxylation as the major metabolic routes, yielding desmethyl-sulfosulfuron and 5-hydroxy-sulfosulfuron as the major metabolites. There was no evidence of bio-retention of sulfosulfuron or its metabolites; tissue and blood levels were negligible, with no individual tissue showing levels exceeding 0.2% of the dose.

8. *Metabolite toxicology*. Dietary residues are comprised almost entirely of parent sulfosulfuron and the imidazopyridine-containing metabolites sulfonamide and guanidine. Specific toxicology data is not available on these metabolites, but the structures do not suggest any specific toxicologic concern and the level of dietary exposure is low. These metabolites are not considered to present a significant toxicological risk.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Estimates of dietary exposure to residues of sulfosulfuron utilized the proposed tolerance-level residues for wheat grain (0.01 ppm) and for the following animal products: milk (0.004 ppm), fat (0.004 ppm), meat (0.004 ppm) and meat by-products (0.1 ppm, including kidney and liver). One-hundred percent market share was assumed as well as the assumption that no loss of residue would occur due to processing and cooking. A Reference Dose (RfD) of 0.24 mg/kg/day was assumed based on the low NOEL from the chronic/oncogenicity study in rats (≈ 24 mg/kg/day) with a safety factor of 100. Since the present label lists only wheat or fallow as approved rotations, no residues were entered for rotational crops. Using these conservative assumptions, dietary residues of sulfosulfuron contribute only 0.000149 mg/kg/day (0.006% of the RfD) for children 1-6 years, the most sensitive sub-population. For the U.S. Population as a whole, the exposure was only 0.000048 mg/kg/day (0.02% of the RfD).

ii. *Drinking water*. Given the low use rates, rapid soil degradation, strong soil binding characteristics and low soil mobility of sulfosulfuron, the risk of significant ground and surface water contamination and exposure via drinking water is considered to be negligible. Assuming that 10% of the RfD is allocated to drinking water exposure (0.024 mg/kg/day), and the average, 70 kg human consumes 2 liters of water per day, a Maximum Allowable Concentration value for drinking water of 0.84 mg/l is proposed for sulfosulfuron.

2. *Non-dietary exposure*. Sulfosulfuron is proposed for a variety of non-crop uses including roadsides, fencerows, industrial sites, parks, apartment complexes, schools and other public areas. Exposure assessments have been made for mixer/loaders and applicators in these situations (occupational exposure) and the cumulative (amortized) daily exposure from both these activities has been estimated to be less than 0.5 µg/kg/day, or approximately 0.2% of the RfD. The non-occupational exposure in these locations to the casual passer-by would be expected to be orders of magnitude less. The exposure in either instance does not present a significant exposure risk.

D. Cumulative Effects

Sulfosulfuron falls into the common category of sulfonylurea (SU) herbicides; however there is no information to suggest that any of the SU's have a common mechanism of mammalian toxicity or even produce similar effects. It is not appropriate to combine exposures in this case, and Monsanto is considering only the potential risk of sulfosulfuron in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. As presented above, the exposure of the U.S. General population to sulfosulfuron is low, and the risks, based on comparisons to the reference dose, are negligible. Margins

of safety are expected to be considerable. Monsanto concludes that there is a reasonable certainty that no harm will result to the U.S. Population from aggregate exposure to sulfosulfuron residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of sulfosulfuron, Monsanto considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in rats. No developmental or reproductive effects were observed up to the highest dose tested in each of the three studies. The Observed NOEL's were 1,000 mg/kg/day, 1,000 mg/kg/day and 20,000 ppm, respectively. Using the same conservative assumptions that were made previously for the dietary exposure analysis for the U.S. General population, the percent of the RfD utilized by pre-adult sub-populations are: all infants-0.03%; nursing infants-0.005%; non-nursing infants-0.04%; children, 1-6 years-0.06%; children, 7-12 years-0.04%. Monsanto concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to sulfosulfuron residues.

F. International Tolerances

There are currently no international (Codex) tolerances established for sulfosulfuron. Sulfosulfuron is currently registered on wheat in Switzerland, Poland, the Czech Republic, Slovakia and South Africa. Petitions for tolerances for sulfosulfuron in/on wheat have been submitted in Canada, Australia and the European Union. (Jim Tompkins)

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ENVIRONMENTAL PROTECTION AGENCY

[PF–785; FRL–5760–5]

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 food commodities.

DATES: Comments, identified by the docket control number [PF–785], must be received on or before January 16, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow 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. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: Amelia M. Acierto, Registrtion Division (7505W), Office of Pesticide Programs, U.S. Environmental Protection Agency, 401 M Street, S.W., Washington, D.C. 20460. Office location, telephone number and e-mail address: Rm. 4W60 4th floor, CS1, 2800 Crystal Drive, Arlington VA, (703)308-8377, e-mail: acierto.amelia@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 food 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 granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–785] (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 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–785] and appropriate petition number. Electronic comments on notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: December 4, 1997

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. 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. Ecolab Inc.

PP 7E4922

EPA has received a pesticide petition (PP 7E4922) from Ecolab Inc., 370 N. Wabasha Street, St. Paul, Minnesota 55102, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.1001(c) to establish an exemption from the requirement of a tolerance for the residues of