it is believed that metolachlor will be infrequently found in groundwater (less than 5% of the samples analyzed), and when found, it will be in the low ppb range.

3. *Non-dietary exposure*. Although metolachlor may be used on turf and ornamentals in a residential setting, that use represents less than 0.1 percent of the total herbicide market for residential turf and landscape uses. Currently, there are no acceptable, reliable exposure data available to assess any potential risks from non-dietary exposure. However, given the small amount of material that is used, Novartis believes that the potential for non-occupational exposure to the general population is unlikely.

D. Cumulative Effects

The potential for cumulative effects of metolachlor and other substances that have a common mechanism of toxicity has also been considered. Novartis believes that consideration of a common mechanism of toxicity with other registered pesticides in this chemical class (chloroacetamides) is not appropriate. EPA concluded that the carcinogenic potential of metolachlor is not the same as other registered chloroacetamide herbicides, based on differences in rodent metabolism (EPA Peer Review of metolachlor, 1994). Novartis maintains that only metolachlor should be considered in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions described above, based on the completeness and reliability of the toxicity data, Novartis has concluded that aggregate exposure to metolachlor including the proposed new uses on peppers and grasses grown for seed will utilize approximately 3.0% 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. Therefore, Novartis believes that there is a reasonable certainty that no harm will result from aggregate exposure to metolachlor or metolachlor residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of metolachlor, data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat have been considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from chemical exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to a chemical on the reproductive capability of mating animals and data on systemic toxicity.

Developmental toxicity (reduced mean fetal body weight, reduced number of implantations/dam with resulting decreased litter size, and a slight increase in resorptions/dam with a resulting increase in post-implantation loss) were observed in studies on metolachlor in rats and rabbits. The NOEL's for developmental effects in both rats and rabbits were established at 360 mg/kg/day. The developmental effect observed in the metolachlor rat study is believed to be a secondary effect resulting from maternal stress (lacrimation, salivation, decreased body weight gain and food consumption and death) observed at the limit dose of 1,000 mg/kg/day.

A 2-generation reproduction study was conducted with metolachlor at feeding levels of 0, 30, 300 and 1,000 ppm. The reproductive NOEL of 300 ppm (equivalent to 23.5 to 26 mg/kg/day) was based upon reduced pup weights in the F1a and F2a litters at the 1,000 ppm dose level (equivalent to 75.8 to 85.7 mg/kg/day). The NOEL for parental toxicity was equal to or greater than the 1,000 ppm dose level.

Section 408 of the FFDCA 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 relative to pre- and post-natal effects for children is complete. Further, for the chemical metolachlor, the NOEL of 9.7 mg/kg/day from the metolachlor chronic dog study, which was used to calculate the RfD (discussed above), is already lower than the developmental NOEL's of 360 mg/kg/day from the metolachlor developmental toxicity studies in rats and rabbits. In the metolachlor reproduction study, the lack of severity of the pup effects observed (decreased body weight) at the systemic lowest-observed-effect level (LOEL) (equivalent to 75.8 to 85.7 mg/kg/day) and the fact that the effects were observed at a dose that is nearly 10 times greater than the NOEL in the chronic dog study (9.7 mg/kg/day) suggest there is no additional sensitivity for infants and children. Therefore, Novartis concludes that an additional uncertainty factor is not warranted to protect the health of infants and children and that the RfD at 0.1 mg/kg/day based on the chronic dog study is appropriate for assessing aggregate risk to infants and children from use of metolachlor.

Using the exposure assumptions described above, Novartis concludes that the approximate percentages of the RfD that will be utilized by aggregate exposure to residues of metolachlor including published and pending tolerances is 1% for U. S. population, for nursing infants less than 1%, 3% for non-nursing infants, 3% for children 1 to 6 years old and 2% for children 7 to 12 years old.

Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Novartis concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to metolachlor residues.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRL's) established for residues of metolachlor in or on raw agricultural commodities. (Sidney Jackson)

[FR Doc. 98–5563 Filed 3–3–98:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–792; FRL–5772–6]

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–792, must be received on or before April 3, 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. 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: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Jim Tompkins (PM 25) ..	Rm. 239, CM #2, 703–305–5697, e-mail:tompkins.jim@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA

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–792] (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 [PF–792] 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: February 12, 1998.

Donald R. Stubbs,

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 Zeneca Ag Products

PP OF3860

EPA has received a pesticide petition (PP 0F3860) from Zeneca Ag Products, 1800 Concord Pike, P. O. Box 15458, Wilmington, DE 19850–5458, requesting pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.489 by removing the expiration date of April 10, 1998 for residues of sulfosate (glyphosate-trimesium; sulfonium, trimethyl salt with *N*-(phosphonomethyl)glycine (1:1)) in or on the raw agricultural commodities (RACs) for soybean forage (2.00 ppm, of which no more than 1 ppm is trimethylsulfonium (TMS)), soybean aspirated grain fractions (210.00 ppm, of which no more than 60 ppm is TMS), soybean hay (5.00 ppm, of which no more than 2 ppm is TMS), and soybean seed (3.00 ppm, of which no more than 1 ppm is TMS). 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 support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of sulfosate has been studied in corn, grapes, and soybeans. EPA has concluded that the nature of the residue is adequately understood and that the residues of concern are the parent ions only *N*-(phosphonomethyl)-glycine anion (PMG) and trimethylsulfonium cation (TMS).

2. *Analytical method*. Gas chromatography/mass selective detector methods have been developed for PMG analysis in crops, animal tissues, milk, and eggs. Gas chromatography detection methods have been developed for TMS in crops, animal tissues, milk, and eggs.

3. *Magnitude of residues— magnitude of residues in crops*—i. *Soybeans*. A total of 20 field residue trials were conducted in Regions 2 (3 trials), 4 (4 trials), and 5 (13 trials). The first application was a preplant or preemergence broadcast application at a rate of 8.0 lbs ai/A. A spot treatment was made to a 10% area of each plot 43 – 99 days after the initial treatment. The spot application rate was 2–20 lbs ai/A on a treated basis. Forage samples were harvested at the R3 (early pod) stage of soybean development from each treated plot 7–14 days after the spot application in 6 trials and prior to the spot application in 12 trials. A wiper application was made in all trials approximately 1 week prior to harvest of mature seed. Hay was collected at normal harvest, 7–8 weeks following the spot application in most trials. Seed were collected at normal harvest approximately 1 week after the wiper application. Analysis of the treated samples showed maximum residues were < 0.78 ppm in forage, 1.19 ppm in hay and 0.73 ppm in seed for TMS; and 0.60 ppm in forage, 2.7 ppm in hay, and 1.7 ppm in seed for PMG. These data

support the following tolerances for residue of sulfosate: soybean forage - 2.0 ppm (of which no more than 1.0 ppm is TMS); soybean hay 5.0 ppm (of which no more than 2.0 ppm is TMS); and soybean seed 3.0 ppm (of which no more than 1.0 ppm is TMS).

Concentration of residues is seen in aspirated grain fractions. The appropriate concentration factors for aspirated grain fractions are 73.8 (PMG) and 57.5 (TMS). The appropriate tolerance for aspirated grain fractions is 210 ppm (of which no more than 60 ppm is TMS).

ii. *Magnitude of residue in animals*—a. *Ruminants.* The maximum practical dietary burden in dairy cows for sulfosate results from a diet of soybean RAC's for a total dietary burden of 54.4 ppm. In a cow feeding study one of the dosing levels was 50 ppm, very close to the estimated ruminant dietary burden. Based on these results, the appropriate tolerance levels are: 0.1 ppm for cattle, goat, hog, horse, and sheep fat; 1 ppm for cattle, goat, hog, horse, and sheep meat by-products; 0.2 ppm for cattle, goat, hog, horse, and sheep meat; and 0.2 ppm in milk.

b. *Poultry.* The maximum poultry dietary burden for sulfosate results from a diet comprised of soybean and corn RACs for a total dietary burden of 2.7 ppm. Comparison to a poultry feeding study at a dosing level of 5 ppm indicates that the appropriate tolerance levels would be 0.05 ppm for poultry liver, fat, and meat; 0.10 ppm for poultry meat by-products; and 0.02 ppm for eggs.

B. Toxicological Profile

1. *Acute toxicity.* Several acute toxicology studies have been conducted placing technical grade sulfosate in Toxicity Category III and Toxicity Category IV. The acute oral LD_{50} in rat for sulfosate technical is 750 mg/kg.

2. *Genotoxicty.* Mutagenicity data includes two Ames tests with *Salmonella typhimurium*; a sex linked recessive lethal test with *Drosophila melanoga*; a forward mutation (mouse lymphoma) test; an *in vivo* bone marrow cytogenetics test in rats; a micronucleus assay in mice; an *in vitro* chromosomal aberration test in Chinese hamster ovary cells (CHO) (no aberrations were observed either with or without S9 activation and there were no increases in sister chromatid exchanges); and a morphological transformation test in mice (all negative). A chronic feeding/carcinogenicity study was conducted in male and female rats fed dose levels of 0, 100, 500 and 1,000 ppm (0, 4.2., 21.2 or 41.8 mg/kg/day in males and 0, 5.4, 27.0 or 55.7 mg/kg day in females). No carcinogenic effects were observed under the conditions of the study. The systemic NOEL of 1,000 ppm (41.1/55.7 mg/kg/day for males and females, respectively) was based on decreased body weight gains (considered secondary to reduced food consumption) and increased incidences of chronic laryngeal and nasopharyngeal inflammation (males). A chronic feeding/carcinogenicity study was conducted in male and female mice fed dosage levels of 0, 100, 1,000 and 8,000 ppm (0, 11.7, 118 or 991 mg/kg/day in males and 0, 16, 159 or 1,341 mg/kg/day in females). No carcinogenic effects were observed under the conditions of the study at dose levels up to and including the 8,000 ppm HDT (highest dose may have been excessive). The systemic NOEL was 1,000 ppm based on decreases in body weight and feed consumption (both sexes), increases in the incidences of white matter degeneration in the lumbar spinal cord (males only), and increased incidences of duodenal epithelial hyperplasia (females only). Sulfosate is classified as a Group E carcinogen based on no evidence of carcinogenicity in rat and mouse studies.

3. *Reproductive and developmental toxicity.* A developmental toxicity study in rats was conducted at doses of 0, 30, 100 and 333 mg/kg/day. The maternal (systemic) NOEL was 100 mg/kg/day, based on decreased body weight gain and food consumption, and clinical signs (salivation, chromorhinorrhea, and lethargy) seen at 333 mg/kg/day. The reproductive NOEL was 100 mg/kg/day, based on decreased mean pup weight. The decreased pup weight is a direct result of the maternal toxicity. A developmental toxicity study was conducted in rabbits at doses of 0, 10, 40 and 100 mg/kg/day with developmental and maternal toxicity NOELs of 40 mg/kg/day based on the following:

i. *Maternal effects.* Six of 17 dams died (2 of the 4 non-gravid dams); 4 of 11 dams aborted; clinical signs - higher incidence and earlier onset of diarrhea, anorexia, decreased body weight gain and food consumption.

ii. *Fetal effects.* decreased litter sizes due to increased post-implantation loss, seen at 100 mg/kg/day (HDT). The fetal effects were clearly a result of significant maternal toxicity. A two generation reproduction study in rats fed dosage rates of 0, 150, 800 and 2,000 ppm (equivalent to calculated doses of 0, 7.5, 40, and 100 mg/kg/day for males and females, based on a factor of 20). The maternal (systemic) NOEL was 150 ppm (7.5 mg/kg/day), based on decreases in body weight and body weight gains accompanied by decreased food consumption, and reduced absolute and sometimes relative organ (thymus, heart, kidney & liver) weights seen at 800 and 2,000 ppm (40 and 100 mg/kg/day). The reproductive NOEL was 150 ppm (7.5 mg/kg/day), based on decreased mean pup weights during lactation (after day 7) in the second litters at 800 ppm (40 mg/kg/day) and in all litters at 2,000 ppm (100 mg/kg/day), and decreased litter size in the F0a and F1b litters at 2,000 ppm (100 mg/kg/day). The statistically significant decreases in pup weights at the 800 ppm level were borderline biologically significant because at no time were either the body weights or body weight gains less than 90% of the control values and because the effect was not apparent in all litters. Both the slight reductions in litter size at 2,000 ppm and the reductions in pup weights at 800 and 2,000 ppm appear to be secondary to the health of the dams. There was no evidence of altered intrauterine development, increased stillborns, or pup anomalies. The effects are primarily a result of feed palatability leading to reduced food consumption and decreases in body weight gains in the dams.

4. *Subchronic toxicity.* Two subchronic 90–day feeding studies with dogs and a 1–year feeding study in dogs have been conducted. In the 1–year study dogs were fed 0, 2, 10 or 50 mg/kg/day. The No Observable Effect Level (NOEL) was determined to be 10 mg/kg/day based on decreases in lactate dehydrogenase (LDH) at 50 mg/kg/day. In the first 90–day study, dogs were fed dosage levels of 0, 2, 10 and 50 mg/kg/day. The NOEL in this study was 10 mg/kg/day based on transient salivation, and increased frequency and earlier onset of emesis in both sexes at 50 mg/kg/day. A second 90–day feeding study with dogs dosed at 0, 10, 25 and 50 mg/kg/day was conducted to refine the threshold of effects. There was evidence of toxicity at the top dose of 50 mg/kg/day with a no observed effect level of 25 mg/kg/day. Adverse effects from oral exposure to sulfosate occur at or above 50 mg/kg/day. These effects consist primarily of transient salivation, which is regarded as a pharmacological rather than toxicological effect, emesis and non-biologically significant hematological changes. Exposures at or below 25 mg/kg/day have not resulted in significant biological adverse effects. In addition, a comparison of data from the 90–day and 1–year studies indicates that there is no evidence for increased toxicity with time. The overall NOEL in the dog is 25 mg/kg/day.

5. *Chronic toxicity.* A chronic feeding/carcinogenicity study was conducted in male and female rats fed dose levels of 0, 100, 500 and 1,000 ppm (0, 4.2, 21.2 or 41.8 mg/kg/day in males and 0, 5.4, 27.0 or 55.7 mg/kg day in females). No carcinogenic effects were observed under the conditions of the study. The systemic NOEL of 1,000 ppm (41.1/55.7 mg/kg/day for males and females, respectively) was based on decreased body weight gains (considered secondary to reduced food consumption) and increased incidences of chronic laryngeal and nasopharyngeal inflammation (males). A chronic feeding/carcinogenicity study was conducted in male and female mice fed dosage levels of 0, 100, 1,000 and 8,000 ppm (0, 11.7, 118 or 991 mg/kg/day in males and 0, 16, 159 or 1,341 mg/kg/day in females). No carcinogenic effects were observed under the conditions of the study at dose levels up to and including the 8,000 ppm HDT (highest dose may have been excessive). The systemic NOEL was 1,000 ppm based on decreases in body weight and feed consumption (both sexes), increases in the incidences of white matter degeneration in the lumbar spinal cord (males only), and increased incidences of duodenal epithelial hyperplasia (females only). Sulfosate is classified as a Group E carcinogen based on no evidence of carcinogenicity in rat and mouse studies.

6. *Animal metabolism*. The metabolism of sulfosate has been studied in animals. The residues of concern for sulfosate in meat, milk, and eggs are the parent ions PMG and TMS only.

7. *Metabolite toxicology.* There are no metabolites of toxicological concern. Only the parent ions, PMG and TMS are of toxicological concern.

C. Aggregate Exposure

1. *Dietary (food) exposure.* For the purposes of assessing the potential dietary exposure, Zeneca has utilized the tolerance level for all existing tolerances and proposed tolerances; and 100% crop treated acreage for all commodities. Assuming that 100% of foods, meat, eggs, and milk products will contain sulfosate residues and those residues will be at the level of the tolerance results in an overestimate of human exposure. This is a very conservative approach to exposure assessment. For all existing tolerances, all proposed tolerances, and the proposed maximum permissible levels proposed in this notice of filing, the potential exposure for the U.S. population is 0.0184 milligrams per kilogram of bodyweight per day (mg/kg bwt/day). Potential exposure for children's population subgroups range from 0.0151 mg/kg bwt/day for nursing infants (< 1 year old) to 0.0763 mg/kg bwt/day for non-nursing infants (< 1 year old).

2. *Drinking water.* Sulfosate adsorbs fairly strongly to soil and would not be expected to move vertically below the 6 inch soil layer. The *N*-phosphonomethyl moiety is readily degraded by soil microbes to AMPA with a half-life of 48 to 72 hours. AMPA is further degraded to CO_2. In addition, the trimethylsulfonium moiety degrades rapidly to CO_2 with a half-life of 72 hours. Therefore, sulfosate would not be a contaminant of groundwater. Additionally, since sulfosate has no aquatic uses, residues are not expected in drinking water.

3. *Non-dietary exposure.* Since sulfosate is not registered for residential or turf uses, and does not represent groundwater contamination concern, exposures from other than dietary or occupational sources are not expected to occur.

D. Cumulative Effects

There is no information to indicate that toxic effects produced by sulfosate are cumulative with those of any other chemical compound.

E. Safety Determination

The appropriate toxicity endpoint for use in determining a Reference Dose (RfD) is the NOEL of 25 mg/kg/day, based on the 90–day dog study. Adverse effects resulting from exposure to sulfosate occur at or above approximately 40 mg/kg/day across all species tested (rat, mouse, rabbit and dog). The RfD based on a 90–day dog feeding study (NOEL of 25 mg/kg/day) using a hundredfold safety factor is calculated to be 0.25 mg/kg/day.

1. *U.S. population.* Using the conservative assumptions of 100% of all crops treated and assuming all residues are at the tolerance level for all established and proposed tolerances, the aggregate exposure to sulfosate will utilize 7.4% of the RfD for the U.S. population. Generally there are no concerns for exposures below 100% of the RfD.

2. *Infants and children*. The database on sulfosate relative to pre- and post-natal toxicity is complete. Because the developmental and reproductive effects occurred in the presence of parental (systemic) toxicity, these data do not suggest an increased pre- or post-natal sensitivity of children and infants to sulfosate exposure. Therefore, Zeneca concludes, upon the basis of reliable data, that a hundredfold uncertainty factor is adequate to protect the safety of infants and children and an additional safety factor is unwarranted.

Using the conservative assumptions of 100% of all crops treated and assuming all residues are at the tolerance level for all established and proposed tolerances described above, we conclude that the percent of the RfD that will be utilized by aggregate exposure to residues of sulfosate ranges from 6.1% for nursing infants up to 30.5% for non-nursing infants (< 1 year old).

F. International Tolerances

There are no Codex Maximum Residue Levels established for sulfosate.

2. Zeneca Ag Products

PP 9F3796

EPA has received a pesticide petition (PP 9F3796) from Zeneca Ag Products, 1800 Concord Pike, P. O. Box 15458, Wilmington, DE 19850–5458, requesting pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.489 by removing the expiration date of March 9, 1998 for residues of sulfosate (glyphosate-trimesium; sulfonium, trimethyl salt with *N*-(phosphonomethyl)glycine (1:1)) in or on the raw agricultural commodities (RACs) for cattle, goat, hog, horse, sheep and poultry fat (0.10 ppm), meat by products (1.00 ppm), and meat (0.20 ppm); poultry liver (0.05 ppm), poultry meat by-products (0.10 ppm), and poultry meat (0.05 ppm); corn fodder (0.30, of which no more than 0.20 is trimethylsulfonium TMS)), corn forage (0.10 ppm), and corn grain (0.20 ppm, of which no more than 0.10 ppm is TMS); milk (0.20 ppm); and eggs (0.02 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 support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of sulfosate has been studied in corn, grapes, and soybeans. EPA has concluded that the nature of the residue is adequately understood and that the residues of concern are the parent ions only *N*-(phosphonomethyl)-glycine anion (PMG) and trimethylsulfonium cation (TMS).

2. *Analytical method.* Gas chromatography/mass selective detector methods have been developed for PMG

analysis in crops, animal tissues, milk, and eggs. Gas chromatography detection methods have been developed for TMS in crops, animal tissues, milk, and eggs.

3. *Magnitude of residues—crops*— i. *Corn*. A total of 25 field residue trials were conducted in Regions 1 (2 trials), 2 (2 trials), 5 (18 trials), 7 (1 trial), 8 (1 trial), and 10 (1 trial). The first application was a preemergence broadcast application at a rate of 8.0 lbs ai/A. A spot treatment was made to a 10% area of each plot 30–57 days after the initial treatment. The application rate was 2–20 lbs ai/A on a treated basis. Forage samples were harvested from each treated plot 2–8 weeks after the second application. Fodder and grain samples were obtained at maturity. Analysis of the treated samples showed maximum residues were < 6.1 ppm in forage, 0.13 ppm in fodder and 0.06 ppm in grain for TMS; and < 0.1 ppm in forage, < 0.1 ppm in fodder and 0.07 ppm in grain for PMG. These data support the following tolerances for residue of sulfosate: corn forage - 0.10 ppm; corn fodder - 0.30 ppm (of which no more than 0.2 ppm is TMS); and corn grain - 0.20 ppm (of which no more than 0.10 ppm is TMS). There is no concentration of residues in corn processed fractions.

ii. *Animals— ruminants*. The maximum practical dietary burden in dairy cows for sulfosate results from a diet of soybean RAC's for a total dietary burden of 54.4 ppm. In a cow feeding study one of the dosing levels was 50 ppm, very close to the estimated ruminant dietary burden. Based on these results, the appropriate tolerance levels are: 0.1 ppm for cattle, goat, hog, horse, and sheep fat; 1 ppm for cattle, goat, hog, horse, and sheep meat by-products; 0.2 ppm for cattle, goat, hog, horse, and sheep meat; and 0.2 ppm in milk.

iii. *Poultry*. The maximum poultry dietary burden for sulfosate results from a diet comprised of soybean and corn RACs for a total dietary burden of 2.7 ppm. Comparison to a poultry feeding study at a dosing level of 5 ppm indicates that the appropriate tolerance levels would be 0.05 ppm for poultry liver, fat, and meat; 0.10 ppm for poultry meat by-products; and 0.02 ppm for eggs.

B. Toxicological Profile

1. *Acute toxicity*. Several acute toxicology studies have been conducted placing technical grade sulfosate in Toxicity Category III and Toxicity Category IV. The acute oral LD_{50} in rat for sulfosate technical is 750 mg/kg.

2. *Genotoxicty*. Mutagenicity data include two Ames tests with *Salmonella typhimurium*; a sex linked recessive lethal test with *Drosophila melanoga*; a forward mutation (mouse lymphoma) test; an *in vivo* bone marrow cytogenetics test in rats; a micronucleus assay in mice; an *in vitro* chromosomal aberration test in Chinese hamster ovary cells (CHO) (no aberrations were observed either with or without S9 activation and there were no increases in sister chromatid exchanges); and a morphological transformation test in mice (all negative). A chronic feeding/carcinogenicity study was conducted in male and female rats fed dose levels of 0, 100, 500 and 1,000 ppm (0, 4.2., 21.2 or 41.8 mg/kg/day in males and 0, 5.4, 27.0 or 55.7 mg/kg day in females). No carcinogenic effects were observed under the conditions of the study. The systemic NOEL of 1,000 ppm (41.1/55.7 mg/kg/day for males and females, respectively) was based on decreased body weight gains (considered secondary to reduced food consumption) and increased incidences of chronic laryngeal and nasopharyngeal inflammation (males). A chronic feeding/carcinogenicity study was conducted in male and female mice fed dosage levels of 0, 100, 1,000 and 8,000 ppm (0, 11.7, 118 or 991 mg/kg/day in males and 0, 16, 159 or 1,341 mg/kg/day in females). No carcinogenic effects were observed under the conditions of the study at dose levels up to and including the 8,000 ppm HDT (highest dose may have been excessive). The systemic NOEL was 1,000 ppm based on decreases in body weight and feed consumption (both sexes), increases in the incidences of white matter degeneration in the lumbar spinal cord (males only), and increased incidences of duodenal epithelial hyperplasia (females only). Sulfosate is classified as a Group E carcinogen based on no evidence of carcinogenicity in rat and mouse studies.

3. *Reproductive and developmental toxicity*. A developmental toxicity study in rats was conducted at doses of 0, 30, 100 and 333 mg/kg/day. The maternal (systemic) NOEL was 100 mg/kg/day, based on decreased body weight gain and food consumption, and clinical signs (salivation, chromorhinorrhea, and lethargy) seen at 333 mg/kg/day. The reproductive NOEL was 100 mg/kg/day, based on decreased mean pup weight. The decreased pup weight is a direct result of the maternal toxicity. A developmental toxicity study was conducted in rabbits at doses of 0, 10, 40 and 100 mg/kg/day with developmental and maternal toxicity NOELs of 40 mg/kg/day based on the following:

i. *Maternal effects*. Six of 17 dams died (2 of the 4 non-gravid dams); 4 of 11 dams aborted; clinical signs - higher incidence and earlier onset of diarrhea, anorexia, decreased body weight gain and food consumption.

ii. *Fetal effects*. Decreased litter sizes due to increased post-implantation loss, seen at 100 mg/kg/day (HDT). The fetal effects were clearly a result of significant maternal toxicity. A two generation reproduction study in rats fed dosage rates of 0, 150, 800 and 2,000 ppm (equivalent to calculated doses of 0, 7.5, 40, and 100 mg/kg/day for males and females, based on a factor of 20). The maternal (systemic) NOEL was 150 ppm (7.5 mg/kg/day), based on decreases in body weight and body weight gains accompanied by decreased food consumption, and reduced absolute and sometimes relative organ (thymus, heart, kidney & liver) weights seen at 800 and 2,000 ppm (40 and 100 mg/kg/day). The reproductive NOEL was 150 ppm (7.5 mg/kg/day), based on decreased mean pup weights during lactation (after day 7) in the second litters at 800 ppm (40 mg/kg/day) and in all litters at 2,000 ppm (100 mg/kg/day), and decreased litter size in the F0a and F1b litters at 2,000 ppm (100 mg/kg/day). The statistically significant decreases in pup weights at the 800 ppm level were borderline biologically significant because at no time were either the body weights or body weight gains less than 90% of the control values and because the effect was not apparent in all litters. Both the slight reductions in litter size at 2,000 ppm and the reductions in pup weights at 800 and 2,000 ppm appear to be secondary to the health of the dams. There was no evidence of altered intrauterine development, increased stillborns, or pup anomalies. The effects are primarily a result of feed palatability leading to reduced food consumption and decreases in body weight gains in the dams.

4. *Subchronic toxicity*. Two subchronic 90–day feeding studies with dogs and a 1–year feeding study in dogs have been conducted. In the 1–year study dogs were fed 0, 2, 10 or 50 mg/kg/day. The No Observable Effect Level (NOEL) was determined to be 10 mg/kg/day based on decreases in lactate dehydrogenase (LDH) at 50 mg/kg/day. In the first 90–day study, dogs were fed dosage levels of 0, 2, 10 and 50 mg/kg/day. The NOEL in this study was 10 mg/kg/day based on transient salivation, and increased frequency and earlier onset of emesis in both sexes at 50 mg/kg/day. A second 90–day feeding study with dogs dosed at 0, 10, 25 and 50 mg/kg/day was conducted to refine the

threshold of effects. There was evidence of toxicity at the top dose of 50 mg/kg/day with a no observed effect level of 25 mg/kg/day. Adverse effects from oral exposure to sulfosate occur at or above 50 mg/kg/day. These effects consist primarily of transient salivation, which is regarded as a pharmacological rather than toxicological effect, emesis and non-biologically significant hematological changes. Exposures at or below 25 mg/kg/day have not resulted in significant biological adverse effects. In addition, a comparison of data from the 90 day and 1 year studies indicates that there is no evidence for increased toxicity with time. The overall NOEL in the dog is 25 mg/kg/day.

5. *Chronic toxicity.* A chronic feeding/carcinogenicity study was conducted in male and female rats fed dose levels of 0, 100, 500 and 1,000 ppm (0, 4.2., 21.2 or 41.8 mg/kg/day in males and 0, 5.4, 27.0 or 55.7 mg/kg day in females). No carcinogenic effects were observed under the conditions of the study. The systemic NOEL of 1,000 ppm (41.1/55.7 mg/kg/day for males and females, respectively) was based on decreased body weight gains (considered secondary to reduced food consumption) and increased incidences of chronic laryngeal and nasopharyngeal inflammation (males). A chronic feeding/carcinogenicity study was conducted in male and female mice fed dosage levels of 0, 100, 1,000 and 8,000 ppm (0, 11.7, 118 or 991 mg/kg/day in males and 0, 16, 159 or 1,341 mg/kg/day in females). No carcinogenic effects were observed under the conditions of the study at dose levels up to and including the 8,000 ppm HDT (highest dose may have been excessive). The systemic NOEL was 1,000 ppm based on decreases in body weight and feed consumption (both sexes), increases in the incidences of white matter degeneration in the lumbar spinal cord (males only), and increased incidences of duodenal epithelial hyperplasia (females only). Sulfosate is classified as a Group E carcinogen based on no evidence of carcinogenicity in rat and mouse studies.

6. *Animal metabolism.* The metabolism of sulfosate has been studied in animals. The residues of concern for sulfosate in meat, milk, and eggs are the parent ions PMG and TMS only.

7. *Metabolite toxicology.* There are no metabolites of toxicological concern. Only the parent ions, PMG and TMS are of toxicological concern.

C. Aggregate Exposure

1. *Dietary (food) exposure.* For the purposes of assessing the potential dietary exposure, Zeneca has utilized the tolerance level for all existing tolerances, and proposed Tolerances; and 100% crop treated acreage for all commodities. Assuming that 100% of foods, meat, eggs, and milk products will contain sulfosate residues and those residues will be at the level of the tolerance results in an overestimate of human exposure. This is a very conservative approach to exposure assessment. For all existing tolerances and the proposed maximum permissible levels proposed in this notice of filing, the potential exposure for the U.S. population is 0.0184 mg/kg bwt/day. Potential exposure for children's population subgroups range from 0.0151 mg/kg bwt/day for nursing infants (< 1 year old) to 0.0763 mg/kg bwt/day for non-nursing infants (> 1 year old).

2. *Drinking water.* Sulfosate adsorbs fairly strongly to soil and would not be expected to move vertically below the 6 inch soil layer. The *N*-phosphonomethyl moiety is readily degraded by soil microbes to AMPA with a half-life of 48 to 72 hours. AMPA is further degraded to CO_2. In addition, the trimethylsulfonium moiety degrades rapidly to CO_2 with a half-life of 72 hours. Therefore, sulfosate would not be a contaminant of groundwater. Additionally, since sulfosate has no aquatic uses, residues are not expected in drinking water.

3. *Non-dietary exposure.* Since sulfosate is not registered for residential or turf uses, and does not represent groundwater contamination concern, exposures from other than dietary or occupational sources are not expected to occur.

D. Cumulative Effects

There is no information to indicate that toxic effects produced by sulfosate are cumulative with those of any other chemical compound.

E. Safety Determination

The appropriate toxicity endpoint for use in determining a Reference Dose (RfD) is the NOEL of 25 mg/kg/day, based on the 90–day dog study. Adverse effects resulting from exposure to sulfosate occur at or above approximately 40 mg/kg/day across all species tested (rat, mouse, rabbit and dog). The RfD based on a 90–day dog feeding study (NOEL of 25 mg/kg/day) using a hundredfold safety factor is calculated to be 0.25 mg/kg/day.

1. *U.S. population.* Using the conservative assumptions of 100% of all crops treated and assuming all residues are at the tolerance level for all established and proposed tolerances, the aggregate exposure to sulfosate will utilize 7.4% of the RfD for the US population. Generally there are no concerns for exposures below 100 percent of the RfD.

2. *Infants and children.* The database on sulfosate relative to pre- and post-natal toxicity is complete. Because the developmental and reproductive effects occurred in the presence of parental (systemic) toxicity, these data do not suggest an increased pre- or post-natal sensitivity of children and infants to sulfosate exposure. Therefore, Zeneca concludes, upon the basis of reliable data, that a hundredfold uncertainty factor is adequate to protect the safety of infants and children and an additional safety factor is unwarranted. Using the conservative assumptions of 100% of all crops treated and assuming all residues are at the tolerance level for all established and proposed tolerances described above, we conclude that the percent of the RfD that will be utilized by aggregate exposure to residues of sulfosate ranges from 6.1% for nursing infants up to 30.5% for non-nursing infants (< 1 year old).

F. International Tolerances.

There are no Codex Maximum Residue Levels established for sulfosate.

[FR Doc. 98–5257 Filed 3–3–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPPT–59362A; FRL–5775–1]

Certain Chemicals; Extension of Test Marketing Period for Test Marketing Exemption

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces EPA's approval of an extension of the test marketing period for a test marketing exemption (TME) under section 5(h)(1) of the Toxic Substances Control Act (TSCA) and 40 CFR 720.38. EPA designated the original test marketing application as TME–97–9. Therefore, this extension is a modification of the previously granted TME. The test marketing conditions are described below.

DATES: This notice becomes effective on February 25, 1998.

FOR FURTHER INFORMATION CONTACT: Shirley D. Howard, New Chemicals Notice Management Branch, Chemical Control Division (7405), Office of Pollution Prevention and Toxics, Environmental Protection Agency, Rm.