II. Public Record and Electronic Submissions

The official record for this notice, as well as the public version, has been established for this notice under docket number [OPP–30471] (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 notice 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 number [OPP–30471]. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pest, Product registration.

Dated: March 30, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 99–8774 Filed 4–7–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–869; FRL–6071–2]

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–869, must be received on or before May 10, 1999.

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 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. 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
Sidney Jackson	Rm. 272, CM #2, 703–305–7610, e-mail:jackson.sidney@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Lisa D. Jones	Rm. 259, CM #2, 703–308–9424, e-mail:jones.lisa@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 support 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–869] (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. Comments 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 (insert docket number) 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: April 2, 1999.

onald 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. Interregional Research Project No. 4 (IR-4)

PP 6E4766, 7E4898, 7E4899

EPA has received pesticide petitions [6E4766, 7E4898, 7E4899] from the Interregional Research Project Number 4 (IR-4) New Jersey Agricultural Experiment Station, P.O. Box 231, Rutgers University, New Brunswick, NJ 08903 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of the insecticide imidacloprid [1-[(6-chloro-3-pyridinyl)methyl]-N-nitro-2-imidazolidinimine and its metabolites containing the 6-chloropyridinyl moiety, all expressed as 1-[(6-chloro-3-pyridinyl)methyl]-N-nitro-2-imidazolidinimine, in or on the raw agricultural commodities (RAC):

1. *PP 6E4766* proposes the establishment of a tolerance for cucurbits vegetables (Crop Group 9) at 0.5 parts per million (ppm).

2. *PP 7E4898* proposes the establishment of a tolerance for tuberous and corm vegetables at 0.3 ppm and dasheen (taro) at 3.5 ppm.

3. *PP 7E4899* proposes the establishment of a tolerance for watercress, upland at 3.5 ppm.

EPA has determined that the petitions contain 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 these petitions. Additional data may be needed before EPA rules on these petitions. Imidacloprid is produced by the Bayer Corporation (Bayer), the registrant.

A. Residue Chemistry

1. *Plant and animal metabolism*. The nature of the imidacloprid residue in plants and livestock is adequately understood. The residues of concern are combined residues of imidacloprid and it metabolites containing the 6-chloropyridinyl moiety, all calculated as imidacloprid.

2. *Analytical method*. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloropyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary gas chromatography mass spectrometry (GC/MS) selective monitoring. This method has successfully passed a petition method validation in EPA labs. There is a confirmatory method specifically for imidacloprid and several metabolites utilizing GC/MS and high performance liquid chromotography using ultra-violet detection (HPLC-UV) which has been validated by the EPA as well. Imidacloprid and its metabolites are stable for at least 24 months in the commodities when frozen.

3. *Magnitude of residues*. For cucurbits, IR-4 performed 6 trials on cucumber, 6 trials on summer squash, and 6 trials on cantaloupe spread over two growing seasons (1992 and 1993). Trials conducted during the 1992 growing season used the following use pattern: i) a plant drench plus foliar applications, ii) a plant drench, iii) an in-furrow, and iv) a sidedress application. In 1993, IR-4 performed work on only the plant drench plus foliar treatment use pattern with a zero day pre-harvest interval (PHI).

The use pattern with the highest residue levels was the plant drench plus foliar application with a zero day. The maximum residues observed were 0.39 ppm for melon, 0.34 ppm for cucumber, and 0.28 ppm for summer squash. These maximum levels are all very similar and support the crop group concept and proposed 0.5 ppm proposed tolerance for imidacloprid on cucurbit vegetables.

Bayer believes that the data used to support the establishment of the imidacloprid 3.5 ppm leafy greens tolerance can be used to extend the tolerance to cover upland watercress. This is based on the similarities of upland watercress to upland cress and garden cress (members of crop subgroup 4A). The use patterns and restrictions for use on upland watercress would be the same as currently registered for garden cress and upland cress.

Even at exaggerated rates, imidacloprid residues in the potato tubers were only 0.25 ppm. Therefore, IR-4 contends that a crop subgroup tolerance for tuberous and corm vegetables to include dasheen (taro) is justified and appropriate, and no additional crop-specific data are required.

Although Dasheen (taro) leaves are seldom consumed, they are occasionally harvested from dasheen (taro) plantings grown primarily for the corms. In support of the proposed tolerance on dasheen (taro) leaves, IR-4 has noted that a tolerance of 3.5 ppm has been established on lettuce under pesticide petition (PP) 3F4231. IR-4 is requesting that the EPA use the data presented in PP 3F4231 to establish a tolerance for dasheen (taro) leaves. The proposed use pattern on taro does not include any foliar applications of imidacloprid. Therefore, it is unlikely that imidacloprid residues in or on taro leaves would exceed the proposed 3.5 ppm tolerance.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral lethal dose $(LD)_{50}$ values for imidacloprid technical ranged from 424-475 milligram/kilogram body weight (mg/kg bwt) in the rat. The acute dermal LD_{50} was greater than 5,000 mg/kg in rats. The 4-hour rat inhalation lethal concentration $(LC)_{50}$ was > 69 mg/cubic meters (m 3) air (aerosol). Imidacloprid was not irritating to rabbit skin or eyes. Imidacloprid did not cause skin sensitization in guinea pigs.

2. *Genotoxicty*. Extensive mutagenicity studies conducted to investigate point and gene mutations, DNA damage and chromosomal aberration, both using *in vitro* and *in vivo* test systems show imidacloprid to be non-genotoxic.

3. *Reproductive and developmental toxicity*. A 2-generation rat reproduction study gave a no-observed adverse effect level (NOAEL) of 100 ppm (8 mg/kg bwt). Rat and rabbit developmental toxicity studies were negative at doses up to 30 mg/kg bwt and 24 mg/kg bwt, respectively.

4. *Subchronic toxicity*. 90-day feeding studies were conducted in rats and dogs. The NOAEL's for these tests were 14 mg/kg bwt/day (150 parts per million (ppm)) and 5 mg/kg bwt/day (200 ppm) for the rat and dog studies, respectively.

5. *Chronic toxicity/carcinogenicity*. A 2-year rat feeding/carcinogenicity study was negative for carcinogenic effects under the conditions of the study and had a NOAEL of 100 ppm (5.7 mg/kg/bwt in male and 7.6 mg/kg bwt female) for noncarcinogenic effects that included decreased body weight gain in females at 300 ppm and increased thyroid lesions in males at 300 ppm and females at 900 ppm. A 1-year dog feeding study indicated a NOAEL of 1,250 ppm (41 mg/kg bwt). A 2-year mouse carcinogenicity study that was negative for carcinogenic effects under conditions of the study and had a NOAEL of 1,000 ppm (208 mg/kg/day).

Imidacloprid has been classified under ''Group E'' (no evidence of carcinogenicity) by EPA's reference dose (RfD) committee. There is no cancer risk associated with exposure to this chemical. The RfD based on the 2-year rat feeding/carcinogenic study with a NOAEL of 5.7 mg/kg bwt and 100-fold uncertainty factor, is calculated to be 0.057 mg/kg bwt.

6. *Endocrine disruption*. The toxicology database for imidacloprid is

current and complete. Studies in this database include evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following short- or long-term exposure. These studies revealed no primary endocrine effects due to imidacloprid.

C. Aggregate Exposure

Imidacloprid is a broad-spectrum insecticide with excellent systemic and contact toxicity characteristics with both food and non-food uses. Imidacloprid is currently registered for use on various food crops, tobacco, turf, ornamentals, buildings for termite control, and cats and dogs for flea control. Those potential exposures are addressed below:

1. *Dietary exposure*. For purposes of assessing the potential acute and chronic dietary exposure, the registrant, Bayer, has estimated exposure based on the Theoretical Maximum Residue Contribution (TMRC). The TMRC is obtained by using a model which multiplies the tolerance level residue for each commodity by consumption data. The consumption data, based on the National Food Consumption Survey (NFCS) 1989-92 data base, estimates the amount of each commodity and products derived from the commodities that are eaten by the U.S. population and various population subgroups.

i. *Acute*. For acute dietary exposure the model calculates a margin of exposure (MOE) by dividing the estimated human exposure into the NOAEL from the appropriate animal study. Commonly, EPA finds MOEs lower than 100 to be unacceptable. The EPA has determined that a NOAEL of 24 mg/kg/day from a developmental toxicity study in rabbits should be used to assess acute toxicity and the risk assessment should evaluate acute exposure to females 13 years.

The MOE for imidacloprid derived from previously established tolerances, including time limited tolerances, plus the use on dasheen (taro) proposed by IR-4 would be 628 for the U.S. population (48 States), 258 for nursing infants, and 929 for females 13+ years at the 99 percentile. These MOEs do not exceed the EPA's level of concern for acute dietary exposure.

ii. *Chronic*. The EPA has determined that the RfD based on the 2-year rat feeding/carcinogenic study with a NOAEL of 5.7 mg/kg bwt and 100-fold uncertainty factor, is calculated to be 0.057 mg/kg bwt. As published in the **Federal Registers** of December 13, 1995 (60 FR 64006), and June 12, 1996 (61 FR 2674) (petition to establish tolerances on leafy green vegetables (PP 5F4522/R2237)), the TMRC from published uses is 0.008358 mg/kg bwt/day which utilizes 14.7% of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (< 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day, which utilizes 27.1% of the RfD. Using these conservative assumptions, Bayer has determined that the TMRC from published and proposed uses is 0.008498 mg/kg bwt/day (15% of the RfD) for the general population and 0.015684 mg/kg/day (27.5% of the RfD) for the most highly exposed subgroup in the population, non-nursing infants (< 1 year old). Therefore, Bayer concludes that dietary exposure from the existing uses and proposed uses on cucurbits will not exceed the reference dose for any subpopulation (including infants and children).

iii. *Drinking water*. The EPA has determined that imidacloprid is persistent and could potentially leach into groundwater. However, there is no established Maximum Contamination Level (MCL) or health advisory levels established for imidacloprid in drinking water. EPA's ''Pesticides in Groundwater Database'' has no entry for imidacloprid. In addition, Bayer is not aware of imidacloprid being detected in any wells, ponds, lakes, streams, etc. from its use in the U.S. In studies conducted in 1995, imidacloprid was not detected in 17 wells on potato farms in Quebec, Canada. Therefore, Bayer concludes that contributions to the dietary burden from residues of imidacloprid in water would be inconsequential.

2. *Non-dietary exposure*—i. *Residential Turf*. Bayer has conducted an exposure study to address the potential exposures of adults and children from contact with imidacloprid treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied are young children. Margins of safety (MOS) of 7,587 - 41,546 for 10-year old children and 6,859 - 45,249 for 5-year old children were estimated by comparing dermal exposure doses to the imidacloprid NOAEL of 1,000 mg/kg/day established in a 15-day dermal toxicity study in rabbits. The estimated safe residue levels of imidacloprid on treated turf for 10-year old children ranged from 5.6 - 38.2 $\mu g/cm^2$ and for 5-year old children from 5.1 - 33.5 $\mu g/cm^2$. This compares with the average imidacloprid transferable residue level of 0.080 $\mu g/cm^2$ present immediately after the sprays have dried. These data indicate that children can safely contact imidacloprid-treated turf as soon after application as the spray has dried.

ii. *Termiticide— Imidacloprid is registered as a termiticide*. Due to the nature of the treatment for termites, exposure would be limited to that from inhalation and was evaluated by Bayer. Data indicate that the MOS for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are 8.0×10^7 and 2.4×10^8, respectively - and exposure can thus be considered negligible.

iii. *Tobacco smoke*. Studies have been conducted to determine residues in tobacco and the resulting smoke following treatment. Residues of imidacloprid in cured tobacco following treatment were a maximum of 31 ppm (7 ppm in fresh leaves). When this tobacco was burned in a pyrolysis study only 2% of the initial residue was recovered in the resulting smoke (main stream plus side stream). This would result in an inhalation exposure to imidacloprid from smoking of approximately 0.0005 mg per cigarette. Using the measured subacute rat inhalation NOAEL of 5.5 mg/m^3, Bayer believes that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

iv. *Pet treatment*. Bayer concludes that human exposure from the use of imidacloprid to treat dogs and cats for fleas does not pose unacceptable risks to human health since imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

D. Cumulative Effects

No other chemicals having the same mechanism of toxicity are currently registered, therefore, Bayer concludes that there is no risk from cumulative effects from other substances with a common mechanism of toxicity.

E. Safety Determination

1. *U.S. population—U.S. population in general*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, Bayer concludes that total aggregate exposure to imidacloprid from all current uses including those currently proposed will utilize little more than 15% of the RfD for the U.S. population. EPA generally has no concerns for exposures below 100% of the RfD, because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. Thus, it can be

concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of imidacloprid, the data from developmental studies in both rat and rabbit and a 2-generation reproduction study in the rat have been considered. The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development. The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through 2-generations, as well as any observed systemic toxicity.

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 effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to pre- and post-natal effects is complete. Further for imidacloprid, the NOAEL of 5.7 mg/kg bwt from the 2-year rat feeding/carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOAELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a 100-fold uncertainty factor is already used to calculate the RfD, Bayer surmises that an additional uncertainty factor is not warranted and that the RfD at 0.057 mg/kg bwt/day is appropriate for assessing aggregate risk to infants and children.

Using the conservative exposure assumptions described above under aggregate exposure, Bayer has determined from a chronic dietary analysis that the percent of the RfD utilized by aggregate exposure to residues of imidacloprid ranges from 9.3% for nursing infants up to 32.2% for children (1-6 years). EPA generally has no concern for exposure below 100% of the RfD. In addition, the MOEs for all infant and children population groups do not exceed EPA's level of concern for acute dietary exposure. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Bayer concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the residues of imidacloprid, including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

No CODEX Maximum Residue Levels (MRLs) have been established for residues of imidacloprid on any crops at this time.

2. IR-4 Project

PP 8E5034

EPA has received a pesticide petition (8E5034) from the Interregional Research Project Number 4 (IR-4), 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 the insecticide, spinosad in or on the raw agricultural commodities (RAC) tuberous and corm vegetables (crop subgroup 1C) at 0.03 parts per million (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. Spinosad is produced by Dow AgroSciences, Inc. (Dow), the registrant,

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of spinosad in plants (apples, cabbage, cotton, tomato, and turnip), and animals (goats and poultry) is adequately understood for the purposes of this tolerance. A rotational crop study showed no carryover of measurable spinosad related residues in representative test crops.

2. *Analytical method*. There is a practical method (immunoassay) for detecting (0.005 ppm) and measuring (0.01 ppm) levels of spinosad in or on food with a limit of detection that allows monitoring of food with residues at or above the level set for this tolerance. The method has had a successful method tryout in the EPA's laboratories.

3. *Magnitude of residues*. Magnitude of residue studies were conducted for potatoes at 14 sites. No quantifiable residues were observed in treated field samples at an application rate of 0.11 pounds active ingredient (lb a.i.) per acre or at an exaggerated application rate of 0.55 lb a.i. per acre. A potato processing study is not required because there were no quantifiable residues in the RAC even at the 5x application rate (5x is the maximum theoretical concentration factor for potato). Potato is the representative crop for the tuberous and corm vegetables crop subgroup 1C.

B. Toxicological Profile

1. *Acute toxicity—Spinosad has low acute toxicity*. The rat oral lethal dose $(LD)_{50}$ is 3,738 milligram kilogram (mg/kg) for males and > 5,000 mg/kg for females, whereas the mouse oral LD_{50} is > 5,000 mg/kg. The rabbit dermal LD_{50} is > 5,000 mg/kg and the rat inhalation lethal concentration $(LC)_{50}$ is > 5.18 mg/liter(l) air. In addition, spinosad is not a skin sensitizer in guinea pigs and does not produce significant dermal or ocular irritation in rabbits. End use formulations of spinosad that are water based suspension concentrates have similar low acute toxicity profiles.

2. *Genotoxicty*. 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 vitro* mammalian gene mutation assay using mouse lymphoma cells, an *in vitro* assay for DNA damage and repair in rat hepatocytes, and an *in vivo* cytogenetic assay in the mouse bone marrow (micronucleus test) have been conducted with spinosad. These studies show a lack of genotoxicity.

3. *Reproductive and developmental toxicity*. Spinosad caused decreased body weights in maternal rats given 200 mg/kg/day by gavage, highest dose tested (HTD). This was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The no-observed adverse effect levels (NOAELs) for maternal and fetal toxicity in rats were 50 and 200 mg/kg/day, respectively. A teratology study in rabbits showed that spinosad caused decreased body weight gain and a few abortions in maternal rabbits given 50 mg/kg/day, HTD. Maternal toxicity was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOAELs for maternal and fetal toxicity in rabbits were 10 and 50 mg/kg/day, respectively. In a 2-generation reproduction study in rats, parental toxicity was observed in both males and females given 100 mg/kg/day HTD. Perinatal effects (decreased litter size and pup weight) at 100 mg/kg/day were attributed to maternal toxicity. The NOAEL for maternal and pup effects was 10 mg/kg/day.

4. *Subchronic toxicity*. Spinosad was evaluated in 13-week dietary studies and showed NOAELs of 4.89 and 5.38 mg/kg/day, respectively in male and female dogs; 6 and 8 mg/kg/day, respectively in male and female mice; and 33.9 and 38.8 mg/kg/day, respectively in male and female rats. No dermal irritation or systemic toxicity occurred in a 21-day repeated dose dermal toxicity study in rabbits given 1,000 mg/kg/day.

5. *Chronic toxicity*. Based on chronic testing with spinosad in the dog and the rat, the EPA has set a reference dose (RfD) of 0.027 mg/kg/day for spinosad. The RfD has incorporated a 100-fold safety factor to the NOAELs found in the chronic dog study to account for inter- and intra-species variation. The NOAELs shown in the dog chronic study were 2.68 and 2.72 mg/kg/day, respectively for male and female dogs. The NOAELs (systemic) shown in the rat chronic/carcinogenicity/ neurotoxicity study were 9.5 and 12.0 mg/kg/day, respectively for male and female rats. Using the Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), it is proposed that spinosad be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. 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 NOAELs shown in the mouse carcinogenicity study were 11.4 and 13.8 mg/kg/day, respectively for male and female mice. A maximum tolerated dose was achieved at the top dosage level tested in both of these studies based on excessive mortality. Thus, the doses tested are adequate for identifying a cancer risk. Accordingly, a cancer risk assessment is not needed.

6. *Animal metabolism*. There were no major differences in the bioavailability, routes or rates of excretion, or metabolism of spinosyn A and spinosyn D following oral administration in rats. Urine and fecal excretions were almost completed in 48-hours post-dosing. In addition, the routes and rates of excretion were not affected by repeated administration.

7. *Metabolite toxicology*. The residue of concern for tolerance setting purposes is the parent material (spinosyn A and spinosyn D). Thus, there is no need to address metabolite toxicity.

8. *Neurotoxicity*. Spinosad did not cause neurotoxicity in rats in acute, subchronic or chronic toxicity studies.

9. *Endocrine disruption*. There is no evidence to suggest that spinosad has an effect on any endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure from use of spinosad on tuberous and corm vegetables as well as from other existing and pending spinosad crop uses, a conservative estimate of aggregate exposure is determined by basing the theoretical maximum residue concentration (TMRC) on the proposed tolerance level for spinosad and assuming that 100% of these proposed new crops and other pending and existing (registered for use) crops grown in the United State were treated with spinosad. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related food stuffs consumed by various population subgroups. 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 spinosad on crops cited in this summary 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 spinosad wherein it's properties show little or no mobility in soil, Dow concludes that there is no anticipated exposure to residues of spinosad in drinking water. In addition, there is no established maximum concentration level (MCL) for residues of spinosad in drinking water.

2. *Non-dietary exposure*. Spinosad is currently registered for use on a number of crops including cotton, fruits, and vegetables in the agriculture environment. Spinosad is also currently registered for outdoor use on turf and ornamentals at low rates of application (0.04 to 0.54 lb a.i. per acre) and indoor use for drywood termite control (extremely low application rates used with no occupant exposure expected). Thus, Dow believes that the potential for non-dietary exposure to the general population is considered negligible.

D. Cumulative Effects

The potential for cumulative effects of spinosad and other substances that have a common mechanism of toxicity is also considered. In terms of insect control, spinosad causes excitation of the insect nervous system, leading to involuntary muscle contractions, prostration with tremors, and finally paralysis. These effects are consistent with the activation of nicotinic acetylcholine receptors by a mechanism that is clearly novel and unique among known insecticidal compounds. Spinosad also has effects on the gamma aminobatopic acid (GABA) receptor function that may contribute further to its insecticidal activity. Based on results found in tests with various mammalian species, spinosad appears to have a mechanism of toxicity like that of many amphiphilic cationic compounds. There is no reliable information to indicate that toxic effects produced by spinosad would be cumulative with those of any other pesticide chemical. Thus Dow contends that it is appropriate to consider only the potential risks of spinosad in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions and the proposed RfD described above, the aggregate exposure to spinosad use on tuberous and corm vegetables and other pending and existing crop uses will utilize 25.5% of the RfD for the U.S. population. A more realistic estimate of dietary exposure and risk relative to a chronic toxicity endpoint is obtained if average (anticipated) residue values from field trials are used. Inserting the average residue values in place of tolerance residue levels produces a more realistic, but still conservative risk assessment. Based on average or anticipated residues in a dietary risk analysis, the use of spinosad on tuberous and corm vegetables and other pending and existing crop uses will utilize 4.1% 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. Thus, Dow believes that there is reasonable certainty that no harm will result from aggregate exposure to spinosad residues on existing and pending crop uses.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of spinosad, 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 spinosad relative to pre- and post-natal effects for children is complete. Further, for spinosad, the NOAELs in the dog chronic feeding study which was used to calculate the

RfD (0.027 mg/kg/day) are already lower than the NOAELs from the developmental studies in rats and rabbits by a factor of more than 10-fold.

Concerning the reproduction study in rats, the pup effects shown at the HDT were attributed to maternal toxicity. Therefore, the registrant concludes that an additional uncertainty factor is not needed and that the RfD at 0.027 mg/kg/day is appropriate for assessing risk to infants and children.

In addition, the EPA has determined that the 10x factor to account for enhanced sensitivity of infants and children is not needed because:

i. The data provided no indication of increased susceptibility of rats or rabbits to in utero and/or post-natal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and two-generation reproduction in rats, effects in the offspring were observed only at or below treatment levels which resulted in evidence of parental toxicity.

ii. No neurotoxic signs have been observed in any of the standard required studies conducted.

iii. The toxicology data base is complete and there are no data gaps.

Using the conservative exposure assumptions previously described (tolerance level residues), the percent RfD utilized by the aggregate exposure to residues of spinosad on tuberous and corm vegetables and other pending and existing crop uses is 51.2% for children 1 to 6 years old, the most sensitive population subgroup. If average or anticipated residues are used in the dietary risk analysis, the use of spinosad on these crops will utilize 9.4% of the RfD for children 1 to 6 years old. Thus, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, the registrant concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to spinosad residues on the above proposed use including other pending and existing crop uses.

F. International Tolerances

There are no Codex maximum residue levels established for residues of spinosad on tuberous and corm vegetables or any other food or feed crop.

3. Zeneca Ag. Products

PP 7F4854, 7F4876, and 7F4853

EPA has received pesticide petitions [7F4854, 7F876, and 7F4853] from Zeneca Ag.Products, 1800 Concord Pike, P. O. Box 15458, Wilmington, DE 19850-5458 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of sulfosate (the trimethylsulfonium salt of glyphosate, also known as glyphosate-trimesium in or on the raw agricultural commodity (RAC) the fruiting vegetables (except cucurbits) group at 0.05 ppm; the edible-podded legume vegetables subgroup at 0.5 ppm (of which no more than 0.3 ppm is trimethylsulfonium (TMS)), the succulent shelled pea and bean subgroup at 0.2 ppm (of which no more than 0.1 ppm is TMS); the dried shelled pea and bean (except soybean) subgroup at 6 ppm (of which no more than 1.5 ppm is TMS); in cattle, goat, hog, sheep, and horse kidney at 3.5 ppm; in cattle, goat, hog, sheep, and horse meat by-products, except liver and kidney, at 2.5 ppm; and to increase the tolerance in cattle, goat, hog, sheep, and horse fat to 0.2 ppm; in cattle, goat, hog, sheep, and horse meat to 0.6 ppm; in cattle, goat, hog, sheep, and horse liver to 0.75 ppm; in milk to 1.1 ppm; in poultry liver to 0.1 ppm; in poultry meat by-products to 0.25 ppm; in or on soybean seed to 21 ppm (of which no more than 13 ppm is TMS); in soybean hulls to 45 ppm (of which no more than 25 ppm is TMS); and in aspirated grain fractions to 1,300 ppm (of which no more than 720 ppm is TMS) at parts per million (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 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 only residues of concern are the parent ionsN-(phosphonomethyl)-glycine anion (PMG) and trimethylsulfonium cation (TMS).

2. *Analytical method*. Gas chromatography/mass selective (GC/MS) 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*—i. *Magnitude of residues in crops—Soybeans*. Residue data are available for sulfosate in a total of 20 trials conducted in 3 different EPA regions and 15 different States representing 99% of the soybean production in the U.S. The proposed tolerance of 21 ppm (of which no more than 13 ppm is TMS) for soybean seed will accommodate any residue resulting from the proposed use pattern.

Soybean seed for processing were obtained and samples were processed into hulls, meal, crude oil, refined oil, and soapstock. Aspirated grain fractions were also collected. Analysis of the treated samples showed that residue of both TMS and PMG accumulated in hulls but did not accumulate in any other processed fractions. The proposed tolerance of 45 ppm (of which no more than 25 ppm is TMS) for soybean hulls and 1,300 ppm (of which no more than 720 ppm is TMS) for aspirated grain fractions will accommodate any residue resulting from the proposed use pattern.

ii. *Fruiting vegetables (except curcurbits) group*. Residue data are available for sulfosate in a total of 12 trials in tomatoes conducted in 5 EPA regions and 5 different states; a total of 6 trials in bell peppers conducted in 5 EPA regions and 6 different States; and a total of 3 trials in chili peppers conducted in 3 EPA regions and 3 different States. The residue levels were below the limit of quantitation (LOQ) of 0.05 ppm in all samples. The proposed tolerance of 0.05 ppm will accommodate any residue resulting from the proposed use pattern.

Tomato fruits for processing were obtained and samples were processed into puree and paste. After adjusting the results for the exaggerated rate, no concentration occurred in the puree and paste. No tolerances are required for puree and paste at the proposed use rates.

iii. *Edible podded legume vegetables subgroup*. Residue data are available for sulfosate in a total of 9 trials conducted in 5 different EPA regions and 8 different States representing 94% of the edible podded beans and peas in the U.S. The proposed tolerance of 0.5 ppm (of which no more than 0.3 ppm is TMS) for the Edible podded legume vegetables subgroup will accommodate any residue resulting from the proposed use pattern.

iv. *Succulent shelled pea and bean subgroup*. Residue data are available for sulfosate in a total of 12 trials in 6 different EPA regions and 10 different States representing 97% of the green peas and lima beans in the United States. The proposed tolerance of 0.2 ppm (of which no more than 0.1 ppm is TMS) for the Succulent shelled pea and bean subgroup will accommodate any residue resulting from the proposed use pattern.

v. *Dried shelled pea and bean (except soybean) subgroup*. Residue data are available for sulfosate in a total of 14 trials conducted in 5 different EPA Regions and in 8 States representing 97% of dried pea and 96% of dried bean production in the United States. The proposed tolerance of 6 ppm (of which no more than 1.5 ppm is TMS) for the Dried shelled pea and bean (except soybean) subgroup will accommodate any residue resulting from the proposed use pattern.

vi. *Magnitude of residue in animals—Ruminants*. The maximum dietary burden in dairy cows results from a diet comprised of 20% aspirated grain fractions, 60% wheat forage, and 20% wheat hay for a total dietary burden of 409 ppm. The maximum dietary burden in beef cows results from a diet comprised of 20% aspirated grain fractions, 25% wheat forage, 25% wheat hay, 20% soybean hulls, and 10% soybean seed for a total dietary burden of 378 ppm. Comparison to a ruminant feeding study at a dosing level of 300 ppm indicates that the appropriate tolerance levels are 0.75 ppm in cattle, goat, hog, sheep, and horse liver; 3.5 ppm in cattle, goat, hog, sheep, and horse kidney; 2.5 ppm in cattle, goat, hog, sheep, and horse meat by-products, except kidney and liver; 0.6 ppm in cattle, goat, hog, sheep, and horse meat; 1.1 ppm in milk; and 0.2 ppm in cattle, goat, hog, sheep, and horse fat. All of these tolerances exceed existing tolerances in 40 CFR 180.489.

vii. *Poultry*. The maximum dietary burden in poultry results from a diet comprised of 40% soybean meal, 20% soybean hulls, 20% soybean seed, and 20% wheat milled by-products for a total dietary burden of 24 ppm. Comparison to a poultry feeding study at a dosing level of 50 ppm indicates that the appropriate tolerance levels are below established tolerances for poultry meat, fat, and eggs. The appropriate tolerance for poultry liver is 0.1 ppm and for poultry meat by-products is 0.25 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Several acute toxicology studies have been conducted placing technical grade sulfosate in Toxicity Category III and IV.

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 no-observable effect level (NOAEL) 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 highest dose tested (HDT) which may have been excessive. The systemic NOAEL was 1,000 ppm based on decreases in body weight and feed consumption (both sexes) 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) NOAEL 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 NOAEL 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 NOAELs of 40 mg/kg/day based on the following: (1) Maternal effects: 6 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; and, (2) 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 2-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 conversion factor of 1 mg/kg-day = 20 ppm). The maternal (systemic) NOAEL 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 and liver) weights seen at 800 and 2,000 ppm (40 and 100 mg/kg/day). The reproductive NOAEL 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 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 NOAEL 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 NOAEL 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 NOAEL 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 NOAEL 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 NOAEL 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 NOAEL was 1,000 ppm based on decreases in body weight and feed consumption (both sexes) 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.

8. *Endocrine disruption*. Current data suggest that sulfosate is not an endocrine disruptor.

C. Aggregate Exposure

1. *Dietary exposure*.—i. *Food*. For the purposes of assessing the potential dietary exposure, Zeneca has utilized the tolerance level for all existing and pending tolerances; and the proposed maximum permissible levels of 0.05 ppm for the fruiting vegetables (except cucurbits) group; 0.5 ppm for the edible-podded legume vegetables subgroup; 0.2 ppm for the succulent shelled pea and bean subgroup; 6 ppm for the dried shelled pea and bean (except soybean) subgroup; 3.5 ppm for cattle, goat, hog, sheep, and horse kidney; 2.5 ppm for cattle, goat, hog, sheep, and horse meat by-products, except liver and kidney; 0.6 ppm for cattle, goat, hog, sheep, and horse meat; 0.75 ppm for cattle, goat, hog, sheep, and horse liver; 1.1 ppm for milk; 0.1 ppm for poultry liver; 0.25 ppm for poultry meat by-products; 21 ppm for soybean seed; 45 ppm for soybean hulls; 1300 ppm for aspirated grain fractions; 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.

ii. *Chronic exposure*. For all existing tolerances and pending tolerances; and the proposed maximum permissible levels proposed in this notice of filing, the potential exposure for the U.S. population is 0.018 mg/kg bwt/day (7.4% of RfD). Potential exposure for children's population subgroups range from 0.015 mg/kg bwt/day (6.1% of RfD) for nursing infants (<1 year old) to 0.076 mg/kg bwt/day (30.5%) for non-nursing infants. The chronic dietary risk due to food does not exceed the level of concern (100%) Acute exposure. The exposure to the most sensitive population subgroup, in this instance non-nursing infants, was 23.2% of the acute RfD. The acute dietary risk due to food does not exceed the level of concern (100%).

iii. *Drinking water*. Results from computer modeling indicate that sulfosate in groundwater will not contribute significant residues in drinking water as a result of sulfosate use at the recommended maximum annual application rate (4.00 lbs. a.i. acre -1). The computer model uses conservative numbers, therefore it is unlikely that groundwater concentrations would exceed the estimated concentration of 0.00224 parts per billion (ppb), and sulfosate should not pose a threat to ground water.

The surface water estimates are based on an exposure modeling procedure called Generic Expected Environmental Concentration (GENEEC). The assumptions of 1 application of 4.00 lbs. a.i. acre -1 resulted in calculated estimated maximum concentrations of 64 ppb (acute, based on the highest 56-day value) and 43 ppb (chronic, average). GENEEC modeling procedures assumed that sulfosate was applied to a 10-hectare field that drained into a 1-hectare pond, 2-meters deep with no outlet.

As a conservative assumption, because sulfosate residues in ground water are expected to be insignificant compared to surface water, it has been assumed that 100% of drinking water consumed was derived from surface water in all drinking water exposure and risk calculations.

To calculate the maximum acceptable acute and chronic exposures to sulfosate in drinking water, the dietary food exposure (acute or chronic) was subtracted from the appropriate (acute or chronic) RfD. Drinking water levels of concern (DWLOCs) were then calculated using the maximum acceptable acute or chronic exposure, default body weights (70 kg - adult, 10 kg - child), and drinking water consumption figures (2 liters - adult, 1 liter - child).

The maximum concentration of sulfosate in surface water is 64 ppb. The acute DWLOCs for sulfosate in surface water were all greater than 7,700 ppb. The estimated average concentration of sulfosate in surface water is 43 ppb which is much less than the calculated levels of concern (> 1,700 ppb) in drinking water as a contribution to chronic aggregate exposure. Therefore, for current and proposed uses of sulfosate, Zeneca concludes with reasonable certainty that residues of sulfosate in drinking water would not result in unacceptable levels of aggregate human health risk.

2. *Non-dietary exposure*. Sulfosate is currently not registered for use on any residential non-food sites. Therefore, residential exposure to sulfosate residues will be through dietary exposure only.

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

1. *U.S. population*—i. *Acute risk*. Since there are no residential uses for sulfosate, the acute aggregate exposure only includes food and water. 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 17.3% of the acute RfD for the U.S. population. The estimated peak concentrations of sulfosate in surface and ground water are less than DWLOCs for sulfosate in drinking water as a contribution to acute aggregate exposure. Residues of sulfosate in drinking water do not contribute significantly to the aggregate acute human health risk considering the

present uses and uses proposed in this action.

ii. *Chronic risk*. Using the conservative exposure assumptions described above, the aggregate exposure to sulfosate from food will utilize 7.4% of the chronic RfD for the U.S. population. The estimated average concentrations of sulfosate in surface and ground water are less than DWLOCs for sulfosate in drinking water as a contribution to chronic aggregate exposure. Residues of sulfosate in drinking water do not contribute significantly to the aggregate chronic human health risk considering the present uses and uses proposed in this action.

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 100-fold uncertainty factor is adequate to protect the safety of infants and children and an additional safety factor is unwarranted.

i. *Acute risk*. Using the conservative exposure assumptions described above, the aggregate exposure to sulfosate from food will utilize 23.2% of the acute RfD for the most highly exposed group, non-nursing infants. The estimated peak concentrations of sulfosate in surface and ground water are less than DWLOCs for sulfosate in drinking water as a contribution to acute aggregate exposure. Residues of sulfosate in drinking water do not contribute significantly to the aggregate acute human health risk considering the present uses and uses proposed in this action.

ii. *Chronic risk*. Using the conservative exposure assumptions described above, we conclude that the percent of the RfD that will be utilized by aggregate exposure to residues of sulfosate is 30.5% for non-nursing infants, the most highly exposed group. The estimated average concentrations of sulfosate in surface and ground water are less than DWLOCs for sulfosate in drinking water as a contribution to chronic aggregate exposure. Residues of sulfosate in drinking water do not contribute significantly to the aggregate chronic human health risk considering the present uses and uses proposed in this action.

F. International Tolerances

There are no Codex Maximum Residue Levels established for sulfosate.

[FR Doc. 99–8775 Filed 4–7–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6321–3]

Notice of Proposed Administrative Settlement Pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act, as Amended by the Superfund Amendments and Reauthorization Act

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice; request for public comment.

SUMMARY: In accordance with section 122(i) of the Comprehensive Environmental Response, Compensation and Liability Act, as amended by the Superfund Amendments and Reauthorization Act (''CERCLA''), notice is hereby given that a proposed administrative cost recovery settlement concerning the Caelus Devices Removal Site in Hollister, California was executed by the Agency on March 19, 1999. The proposed settlement resolves an EPA claim under section 107 of CERCLA against the following Respondents: the United States Navy, Helen Sperber, and Victor Edmundson. The proposed settlement was entered into under the authority granted EPA in section 122(h) of CERCLA, and requires the Respondents to pay $124,195.84 to the Hazardous Substances Superfund in settlement of past costs. For thirty (30) days following the date of publication of this document, the Agency will receive written comments relating to the settlement. The Agency's response to any comments received will be available for public inspection at three locations: the Hollister Public Library; the Environmental Protection Agency, Region 9, Library & Resource Center, 75 Hawthorne Street, San Francisco, California, 94105; and the Environmental Protection Agency, Region 9, Ms. Danielle Carr, Regional Hearing Clerk, 75 Hawthorne Street, San Francisco, California, 94105.

DATES: Comments must be submitted on or before May 10, 1999.

ADDRESSES: The proposed settlement as set forth in the Administrative Consent Order may be obtained from Ms. Danielle Carr, Regional Hearing Clerk, Environmental Protection Agency, Region 9, 75 Hawthorne Street, San Francisco, California, 94105.

Comments regarding the proposed settlement should be addressed to Ms. Danielle Carr, Regional Hearing Clerk, Environmental Protection Agency, Region 9 at the address provided above, and should reference the Caelus Devices Removal Site located in Hollister, California (EPA Docket No. 99–05).

FOR FURTHER INFORMATION CONTACT: Julia A. Jackson, Assistant Regional Counsel, Environmental Protection Agency, Region 9, 75 Hawthorne Street, San Francisco, California 94105, (415) 744–1348.

Dated: March 30, 1999.

Keith Takata,

Director, Superfund Division.

[FR Doc. 99–8778 Filed 4–7–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6321–5]

Memphis Container Site; Notice of Proposed Settlement

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of Proposed Settlement.

SUMMARY: Pursuant to section 122(h) of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), the United States Environmental Protection Agency (EPA) proposes to enter into an Agreement for the recovery of past response costs with Buckman Laboratories, Inc., Perma-Fix of Memphis, Inc., Croda Inks Corporation, IBC Manufacturing Company and Memphis Light, Gas & Water Division, (Settling Parties). Pursuant to the Agreement, the Settling Parties will reimburse EPA for a portion of response costs at the Memphis Container Superfund Site (the ''Site'') located in Memphis, Shelby County, Tennessee. EPA will consider public comments on the proposed settlement for thirty days. EPA may withdraw from or modify the proposed settlement should such comments disclose facts or considerations which indicate the proposed settlement is inappropriate, improper, or inadequate. Copies of the proposed settlement are available from: Ms. Paula V. Batchelor, U.S. Environmental Protection Agency, Region 4, Program Services Branch, Waste Management Division, 61 Forsyth Street, S.W., Atlanta, Georgia 30303.

Written comments may be submitted to Ms. Batchelor at the above address