control broadleaf weeds on cereal grain groups and soybeans.

2. File Symbol: 279–GRIR. Applicant: FMC Corporation. Product Name: Carfentrazone-ethyl (F8426) Technical. Herbicide. Active ingredient: Carfentrazone: ethyl α,2-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzenepropanoate at 90 percent. Proposed classification/Use: None. For formulation use only.

3. File Symbol: 279–GROU. Applicant: FMC Corporation. Product Name: Carfentrazone-ethyl (F8426) 40DF. Herbicide. Active ingredient: Carfentrazone: ethyl α,2-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzenepropanoate at 40 percent. Proposed classification/Use: None. For agricultural or commercial use only to control broadleaf weeds on cereal grain groups and soybeans.

Notice of approval or denial of an application to register a pesticide product will be announced in the **Federal Register**. The procedure for requesting data will be given in the **Federal Register** if an application is approved.

Comments received within the specified time period will be considered before a final decision is made; comments received after the time specified will be considered only to the extent possible without delaying processing of the application.

The official record for this notice, as well as the public version, has been established for this notice under docket number [OPP–30447] (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–30447]. 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: February 12, 1998.

**Donald R. Stubbs,**

*Acting Director, Registration Division, Office of Pesticide Programs.*

[FR Doc. 98–4814 Filed 2–24–98; 8:45 am]

BILLING CODE 6560–50–F

## ENVIRONMENTAL PROTECTION AGENCY

**[PF–790; FRL–5768–4]**

### 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–790, must be received on or before March 27, 1998.

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

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. 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/e-mail address | Address |
|---|---|---|
| George LaRocca (PM 21). | Rm. 204, CM #2, 703–305–6100, e-mail: larocca.george@epamail.epa.gov. | 1921 Jefferson Davis Hwy, Arlington, VA |
| James A. Tompkins (PM 25). | Rm. 239, CM #2, 703–305–5697, e-mail: tompkins.james@epamail.epa.gov. | Do. |
| Hoyt Jamerson (PM 05) | Rm. 268, CM #2, 703–308–9368, e-mail: jamerson.hoyt@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 raw 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 grantinig of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice, as well as the public version, has been established for this notice of filing under docket control number PF–790 (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

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

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

**List of Subjects**

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

Dated: February 11, 1998.

**Peter Caulkins,**

*Acting Director, Registration Division, Office of Pesticide Programs.*

**Summaries of Petitions**

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

**1. DowElanco**

*PP 1F3935*

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

*A. Residue Chemistry*

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

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

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

*B. Toxicological Profile*

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

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

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

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

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

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

7. *Endocrine Effects.* An evaluation of the potential effects on the endocrine systems of mammals has not been

determined; However, no evidence of such effects were reported in the chronic or reproductive toxicology studies described above. There was no observed pathology of the endocrine organs in these studies. There is no evidence at this time that triclopyr causes endocrine effects.

*C. Aggregate Exposure*

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Homeowner exposure will not be significant for the following reasons: the

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

There is a potential for post-application exposure to swimmers following applications to aquatic sites that may be used for recreational purposes. There are no triclopyr-specific exposure data to assess swimmer exposure. However, an assessment was conducted using information provided in EPA's Dermal Exposure Assessment: Principles and Applications. The dermal permeability constant (Kp) was calculated to be $6.5 \times 10^{-8}$ mg/cm$^2$/hr. The assessment of swimmer exposure was based on a 6–year old boy having a body weight of 21.9 kg and a surface area of 0.88 m$^2$. The swimming period was assumed to be 3 hours on the day of treatment in water containing 2.5 ppm triclopyr.

Total dermal exposure (mg) = 3 hr/day $\times$ 0.88 m$^2$ $\times$ $10^4$ cm$^2$/m$^2$ $\times$ $6.5 \times 10^{-8}$ mg/cm$^2$/hr = $1.716 \times 10^{-3}$ mg/day

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

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

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

*D. Cumulative Effects*

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

*E. Safety Determination*

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

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

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

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

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

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

Non-nursing Infants (Dietary + Drinking Water)

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

Determination of Safety for U.S. Population

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

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

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

*F. International Tolerances*

There are no established or proposed Codex MRLs for triclopyr residues. Therefore, there are no issues of compatibility with respect to U.S. tolerances and Codex MRLs. (PM 25)

**2. DuPont Agricultural Products**

*PP 4F3003, 4F3120, 0F3852*

EPA has received a pesticide petition (PP 4F3003, 4F3120, 0F3852) from DuPont Agricultural Products, PO Box 80038, Wilmington, DE 19880–0038. proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of esfenvalerate (Asana XL Insecticide), ((*S*)-cyano-(3-phenoxyphenyl) methyl (*S*)-4-chloro-alpha-(1-methylethyl) benzeneacetate) in or on the raw agricultural commodities sorghum, sugarbeets and head lettuce (see section A3 for specific tolerance levels). The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using ultra violet (UV) detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

*A. Residue Chemistry*

1. *Plant and Animal metabolism*. The metabolism and chemical nature of residues of fenvalerate in plants and animals are adequately understood. The fate of fenvalerate has been extensively studied using radioactive tracers in plant and animal metabolism/nature of the residue studies previously submitted to the Agency. These studies have demonstrated that the parent compound is the only residue of toxicological significance. EPA has concluded that the qualitative nature of

the residue is the same for both fenvalerate and esfenvalerate.

2. *Analytical method*. There is a practical analytical method utilizing electron-capture gas chromatography with nitrogen phosphorous detection available for enforcement with a limit of detection that allows monitoring food with residues at or above tolerance levels. The limit of detection for updated method is the same as that of the current PAM II, which is 0.01 ppm.

3. *Magnitude of residues*. Fenvalerate is a racemic mixture of four isomers (*S,S*; *R,S*; *S,R*; and *R,R*). Technical Asana® (esfenvalerate) is enriched in the insecticidally active *S,S*-isomer (84%). Tolerance expressions are proposed for esfenvalerate based on the sum of all isomers. Tolerance of 5 parts per million (ppm) for head lettuce, 5.0 ppm for sorghum grain, 10.0 ppm for sorghum forage, 10.0 ppm for sorghum fodder, 0.03 ppm for whole eggs, 0.03 ppm for poultry meat, 0.3 ppm for poultry fat, 0.3 ppm for poultry meat by-products (except liver), and 0.03 ppm for poultry liver, 5 ppm for sugarbeet tops, 0.5 ppm for sugarbeet roots and 2.5 ppm sugarbeet pulp are proposed. Magnitude of residue studies support the proposed tolerance.

*B. Toxicological Profile*

1. *Acute toxicity*. A battery of acute toxicity studies places technical esfenvalerate in Toxicity Category II for acute oral toxicity (rat $LD_{50}$ 87.2 mg/kg), Category III for acute dermal (rabbit $LD_{50}$ >2,000 mg/kg) and primary eye irritation (mild irritation in rabbits), and Category IV for primary skin irritation (minimal skin irritation in rabbits that reversed within 72 hours after treatment). Acute inhalation on technical grade a.i. waived due to negligible vapor pressure. A dermal sensitization test on esfenvalerate in guinea pigs showed no sensitization.

2. *Genotoxicty*. Esfenvalerate was not mutagenic in reverse mutation assays in *S. typhimurium* and *E. Coli* and did not induce mutations Chinese hamster V79 cells or chromosome aberrations in Chinese hamster ovary cells. Esfenvalerate did not induce micronuclei in bone marrow of mice given up to 150 mg/kg intraperitoneally. Esfenvalerate did not induce unscheduled DNA synthesis in HeLa cells. Other genetic toxicology studies submitted on racemic fenvalerate indicate that the mixture containing equal parts of the four stereoisomers is not mutagenic in bacteria. The racemic mixture was also negative in a mouse host mediated assay and in a mouse dominant lethal assay.

3. *Reproductive and developmental toxicity*. Esfenvalerate was administered to pregnant female rats by gavage in a pilot developmental study at doses of 0, 1, 2, 3, 4, 5, and 20 mg/kg/day and a main study at 0, 2.5, 5, 10, and 20 mg/kg/day. Maternal clinical signs (abnormal gait and mobility) were observed at 2.5 mg/kg/day and above. A maternal NOEL of 2 mg/kg/day was established on the pilot study. The developmental NOEL was >20 mg/kg/day.

Esfenvalerate was administered by gavage to pregnant female rabbits in a pilot developmental study at doses of 0, 2, 3, 4, 4.5, 5, and 20 mg/kg/day and a main study at does of 0, 3, 10, and 20 mg/kg/day. Maternal clinical signs (excessive grooming) were observed at 3 mg/kg/day and above. A maternal NOEL of 2 mg/kg/day was established on the pilot study. The developmental NOEL was > 20 mg/kg/day.

A two-generation feeding study with esfenvalerate was conducted in the rat at dietary levels of 0, 75, 100, and 300 ppm. Skin lesions and minimal (non biologically significant) parental body weight effects occurred at 75 ppm. The NOEL for reproductive toxicity was 75 ppm (4.2–7.5 mg/kg/day) based on decreased pup weights at 100 ppm.

4. *Subchronic toxicity*. Two 90-day feeding studies with esfenvalerate were conducted in rats - one at 50, 150, 300, and 500 ppm esfenvalerate, and a second at 0, 75, 100, 125, and 300 ppm to provide additional dose levels. The NOEL was 125 ppm (6.3 mg/kg/day) based on clinical signs (jerky leg movements) observed at 150 ppm (7.5 mg/kg/day) and above. A 90-day feeding study in mice was conducted at 0, 50, 150, and 500 ppm esfenvalerate with a NOEL of 150 ppm (30.5 mg/kg) based on clinical signs of toxicity at 500 ppm (106 mg/kg). Three-month subchronic study in dogs was satisfied by one-year oral study in dogs, in which the NOEL was 200 ppm (5 mg/kg/day). A 21-day dermal study in rabbits with fenvalerate conducted at 100, 300, and 1,000 mg/kg/day with an NOEL of 1,000 mg/kg/day.

5. *Chronic toxicity*. In a one-year study, dogs were fed 0, 25, 50, or 200 ppm esfenvalerate with no treatment related effects at any dietary level. The NOEL was 200 ppm (5 mg/kg/day). An effect level for dietary administration of esfenvalerate for dogs of 300 ppm had been established earlier in a three week pilot study used to select dose levels for the chronic dog study.

One chronic study with esfenvalerate and three chronic studies with fenvalerate have been conducted in mice.

In an 18-month study, mice were fed 0, 35, 150, or 350 ppm esfenvalerate. Mice fed 350 ppm were sacrificed within the first two months of the study after excessive self-trauma related to skin stimulation and data collected were not used in the evaluation of the oncogenic potential of esfenvalerate. The NOEL was 35 ppm (4.29 and 5.75 mg/kg/day for males and females, respectively) based on lower body weight and body weight gain at 150 ppm. Esfenvalerate did not produce carcinogenicity. In a 2-year feeding study, mice were administered 0, 10, 50, 250 or 1,250 ppm fenvalerate in the diet. The NOEL was 10 ppm (1.5 mg/kg/day) based on granulomatous changes (related to fenvalerate only, not esfenvalerate) at 50 ppm (7.5 mg/kg/day). Fenvalerate did not produce carcinogenicity. In an 18-month feeding study, mice were fed 0, 100, 300, 1,000, or 3,000 ppm fenvalerate in the diet. The NOEL is 100 ppm (15.0 mg/kg/day) based on fenvalerate-related microgranulomatous changes at 300 ppm (45 mg/kg/day). No compound related oncogenicity occurred. Mice were fed 0, 10, 30, 100, or 300 ppm fenvalerate for 20 months. The NOEL was 30 ppm (3.5 mg/kg/day) based on red blood cell effects and granulomatous changes at 100 ppm (15 mg/kg/day). Fenvalerate was not carcinogenic at any concentration.

In a two-year study, rats were fed 1, 5, 25, or 250 ppm fenvalerate. A 1,000 ppm group was added in a supplemental study to establish an effect level. The NOEL was 250 ppm (12.5 mg/kg/day). At 1,000 ppm (50 mg/kg/day), hind limb weakness, lower body weight, and higher organ-to-body weight ratios were observed. Fenvalerate was not carcinogenic at any concentration. (A conclusion that fenvalerate is associated with the production of spindle cell sarcomas at 1,000 ppm was retracted by EPA).

EPA has classified esfenvalerate in Group E - evidence of noncarcinogenicity for humans.

6. *Animal metabolism*. After oral dosing with fenvalerate, the majority of the administered radioactivity was eliminated in the initial 24 hours. The metabolic pathway involved cleavage of the ester linkage followed by hydroxylation, oxidation, and conjugation of the acid and alcohol moieties.

7. *Metabolite toxicology*. The parent molecule is the only moiety of toxicological significance appropriate for regulation in plant and animal commodities.

8. *Other potential toxicology considerations - endocrine effects.*

Estrogenic effects have not been observed in any studies conducted on fenvalerate or esfenvalerate. In subchronic or chronic studies there were no lesions in reproductive systems of males or females. In the recent reproduction study with esfenvalerate, full histopathological examination of the pituitary and the reproductive systems of males and females was conducted. There were no compound-related gross or histopathological effects. There were also no compound-related changes in any measures of reproductive performance including mating, fertility, or gestation indices or gestation length in either generation. There have been no effects on offspring in developmental toxicity studies. EPA is required to develop an endocrine disrupter screening program by August 3, 1999. EPA will decide whether further testing of esfenvalerate is required at that time.

*C. Aggregate Exposure*

1. *Dietary exposure*. Tolerances have been established for the residues of fenvalerate/esfenvalerate, in or on a variety of agricultural commodities. In addition, pending tolerance petitions exist for use of esfenvalerate on sugarbeets, sorghum, head lettuce, celery, pistachios, and a number of other minor use commodities. For purposes of assessing dietary exposure, chronic and acute dietary assessments have been conducted using all existing and pending tolerances for esfenvalerate. EPA recently reviewed the existing toxicology data base for esfenvalerate and selected the following toxicological endpoints. For acute toxicity, EPA established a NOEL of 2.0 mg/kg/day from rat and rabbit developmental studies based on maternal clinical signs at higher concentrations. An MOE of 100 was required. For chronic toxicity EPA established the RfD for esfenvalerate at 0.02 mg/kg/day. This RfD was also based on a NOEL of 2.0 mg/kg/day in the rat developmental study with an uncertainty factor of 100. Esfenvalerate is classified as a Group E. There is no evidence of carcinogenicity in either rats or mice.

2. *Food*. A chronic dietary exposure assessment was conducted using Novigen's DEEm (Dietary Exposure Estimate Model). Anticipated residues and adjustment for percent crop treated were used in the chronic dietary risk assessment. The percentages of the Reference Dose (RfD) utilized by the most sensitive sub-population, children 1–6 yrs., was 4.6% based on a daily dietary exposure of 0.000911 mg/kg/day. Chronic exposure for the overall Us population was 1.9% of the RfD based on a dietary exposure of 0.000376 mg/kg/day. This assessment has been approved by EPA and included pending tolerances and all food tolerances for incidental residues from use in food handling establishments. EPA 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. Esfenvalerate is classified as a Group E carcinogen - no evidence of carcinogenicity in rats or mice. Therefore, a carcinogenicity risk analysis is not required.

Potential acute exposures from food commodities were estimated using a Tier 3 (Monte Carlo) Analysis and appropriate processing factors for processed food and distribution analysis. This analysis used field trial data to estimate exposure and federal and market survey information to derive the percent of crop treated. EPA considered these data reliable and used the upper end estimate of percent crop treated in order to not underestimate any significant subpopulation. Regional consumption information was taken into account. The Margins of Exposure (MOEs) for the most sensitive sub-population (children 1–6 yr.) were 202 and 103 at the 99$^{th}$, and 99.9$^{th}$ percentile of exposure, respectively, based on daily exposures of 0.009908 and 0.019445 mg/kg/day. The MOEs for the general population are 355 and 171 at the 99$^{th}$ and 99.9$^{th}$ percentile of exposure, respectively, based on daily exposure estimates of 0.005635 and 0.011717 mg/kg/day. The EPA has stated there is no cause for concern if total acute exposure calculated for the 99.9$^{th}$ percentile yields an MOE of 100 or larger. This acute dietary exposure estimate is considered conservative and EPA considered the MOEs adequate in a recent final rule (62 FR 63019).

3. *Drinking water*. Esfenvalerate is immobile in soil and will not leach into groundwater. Due to the insolubility and lipophilic nature of esfenvalerate, any residues in surface water will rapidly and tightly bind to soil particles and remain with sediment, therefore not contributing to potential dietary exposure from drinking water. A screening evaluation of leaching potential of a typical pyrethroid was conducted using EPA's Pesticide Root Zone Model (PRZM). Based on this screening assessment, the potential concentrations of a pyrethroid in ground water at depths of 1 and 2 meters are essentially zero (much less than 0.001 parts per billion).

Surface water concentrations for pyrethroids were estimated using PRZM3 and Exposure Analysis Modeling System (EXAMS) using Standard EPA cotton runoff and Mississippi pond scenarios. The maximum concentration predicted in the simulated pond was 0.052 parts per billion. Concentrations in actual drinking water would be much lower than the levels predicted in the hypothetical, small, stagnant farm pond model since drinking water derived from surface water would be treated before consumption. Chronic drinking water exposure was estimated to be 0.000001 mg/kg/day for both the U.S. general population and for non-nursing infants. Less than 0.1% of the RfD was occupied by both population groups.

Using these values, the contribution of water to the acute dietary risk estimate was estimated for the U.S. population to be 0.000019 mg/kg/day at the 99$^{th}$ percentile and 0.000039 mg/kg/day at the 99.9$^{th}$ percentile resulting in MOEs of 105,874 and 51,757, respectively. For the most sensitive subpopulation, non-nursing infants less than 1 year old, the exposure is 0.000050 mg/kg/day and 0.000074 mg/kg/day at the 99$^{th}$ and 99.9$^{th}$ percentile, respectively, resulting in MOEs of 39,652, and 27,042, respectively. Therefore there is reasonable certainty of no harm from drinking water.

4. *Non-dietary exposure*. Esfenvalerate is registered for non-crop uses including spray treatments in and around commercial and residential areas, treatments for control of ectoparasites on pets, home care products including foggers, pressurized sprays, crack and crevice treatments, lawn and garden sprays, and pet and pet bedding sprays. For the non-agricultural products, the very low amounts of active ingredient they contain, combined with the low vapor pressure ($1.5 \times 10^{-9}$ mm Mercury at 25° C.) and low dermal penetration, would result in minimal inhalation and dermal exposure.

To assess risk from (nonfood) short and intermediate term exposure, EPA has recently selected a toxicological endpoint of 2.0 mg/kg/day, the NOEL from the rat and rabbit developmental studies. For dermal penetration/absorption, EPA selected 25% dermal absorption based on the weight-of-evidence available for structurally related pyrethroids. For inhalation exposure, EPA used the oral NOEL of 2.0 mg/kg/day and assumed 100% absorption by inhalation. Individual non-dietary risk exposure analyses were conducted using a flea infestation scenario that included pet spray, carpet

and room treatment, and lawn care, respectively. The total potential short- and intermediate-tern aggregate non-dietary exposure including lawn, carpet, and pet uses are: 0.000023 mg/kg/day for adults, 0.00129 mg/kg/day for children 1–6 years and 0.00138 mg/kg/day for infants less than one year old. EPa concluded (62 FR 63019) that the potential non-dietary exposure for esfenvalerate are associated with substantial margins of safety.

5. *Aggregate exposure - dietary and non-dietary exposure.* EPA has concluded that aggregate chronic exposure to esfenvalerate from food and drinking water will utilize 1.9% of the RfD for the U.S. population based on a dietary exposure of 0.000377 mg/kg/day. The major identifiable subgroup with the highest aggregate exposure are children 1–6 years old. 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.

The acute aggregate risk assessment takes into account exposure from food and drinking water. The potential acute exposure from food and drinking water to the overall U.S. population provides an acute dietary exposure of 0.011756 mg/kg/day with an MOE of 170. This acute dietary exposure estimate is considered conservative, using anticipated residue values and percent crop-treated data in conjunction with Monte Carlo analysis.

Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. The potential short- and intermediate-term aggregate risk for the U.S. population is an exposure of 0.0082 mg/kg/day with an MOE of 244.

It is important to acknowledge that these MOEs are likely to significantly underestimate the actual MOEs due to a variety of conservative assumptions and biases inherent in the exposure assessment methods used for their derivation. Therefore, it can be concluded that the potential non-dietary and dietary aggregate exposures for esfenvalerate are associated with a substantial degree of safety. EPA has previously determined (62 FR 63019) that there was reasonable certainty that no harm will result from aggregate exposure to esfenvalerate residues. Head lettuce was included in that risk assessment.

*D. Cumulative Effects*

Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity''. In a recent Final Rule on esfenvalerate (62 FR 63019) EPA concluded, ''Available information'' in this context might include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides as to which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed). Although esfenvalerate is similar to other members of the synthetic pyrethroid class of insecticides, EPA does not have, at this time, available data to determine whether esfenvalerate has a common method of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, esfenvalerate does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that esfenvalerate has a common mechanism of toxicity with other substances.

*E. Safety Determination*

Both the chronic and acute toxicological endpoints are derived from maternal NOEL's of 2.0 mg/kg/day in developmental studies in rats and rabbits. There were no fetal effects. In addition, no other studies conducted with fenvalerate or esfenvalerate indicate that immature animals are more sensitive than adults. Therefore, the safety factor used for protection of adults is fully appropriate for the protection of infants and children; no additional safety factor is necessary as described below.

1. *U.S. population.* A chronic dietary exposure assessment using anticipated residues, monitoring information, and percent crop treated indicated the percentage of the Reference Dose (RfD) utilized by the General Population to be 1.9%. There is generally 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.

For acute exposure, a Margin of Exposure (MOE) of greater than 100 is considered an adequate MOE. A Tier 3 acute dietary exposure assessment found the General Population to have MOE's of 355 and 171 at the 99$^{th}$ and 99.9$^{th}$ percentile of exposure, respectively. These values were generated using actual field trial residues and market share data for percentage of crop treated. These results depict an accurate exposure pattern at an exaggerated daily dietary exposure rate.

Short- and intermediate-term aggregate exposure risk from chronic dietary food and water plus indoor and outdoor residential exposure for the U.S. population is an exposure of 0.0082 mg/kg/day with an MOE of 244. Therefore, there is a reasonable certainty that no harm will result from chronic dietary, acute dietary, non-dietary, or aggregate exposure to esfenvalerate residues.

2. *Infants and children.* FFDCA section 408 provides that EPA shall

apply an additional tenfold margin of safety for infants and children unless EPA determines that a different margin of safety will be safe for infants and children. EPA has stated that reliable data support using the standard MOE and uncertainty factor (100 for combined inter- and intra-species variability) and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor. In a recent Final Rule (62 FR 63019), EPA concluded that reliable data support use of the standard 100-fold uncertainty factor for esfenvalerate, and that an additional uncertainty factor is not needed to protect the safety of infants and children. This decision was based on: no evidence of developmental toxicity at a doses up to 20 mg/kg/day (ten times the maternal NOEL) in prenatal developmental toxicity studies in both rats and rabbits; offspring toxicity only at dietary levels which were also found to be toxic to parental animals in the two generation reproduction study; and no evidence of additional sensitivity to young rats or rabbits following pre- or postnatal exposure to esfenvalerate.

A chronic dietary exposure assessment found the percentages of the RfD utilized by the most sensitive sub-population to be 4.6% for children 1–6 yr based on a dietary exposure of 0.000912 mg/kg/day. The % RfD for nursing and non-nursing infants was 1.1% and 2.7%, respectively. The Agency has no cause for concern if RfD are below 100%.

The most sensitive sub-population, children 1–6 year, had acute dietary MOEs of 202 and 103 at the 99th and 99.9th percentile of exposure, respectively. Nursing infants had MOEs of 195 and 146 at the 99th, and 99.9th percentile of exposure, respectively. Non-nursing infants had MOEs of 304 and 158 at the 99th and 99.9th percentile of exposure, respectively. The Agency has no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger.

EPA has recently concluded that the potential short- or intermediate-term aggregate exposure of esfenvalerate from chronic dietary food and water plus indoor and outdoor residential exposure to children (1–6 years old) is 0.0113 mg/kg/day with an MOE of 177. For infants (less than 1 year old) the exposure is 0.0098 mg/kg/day with an MOE of 204. There is reasonable certainty that no harm will result to infants and children from aggregate exposure to esfenvalerate residues (62 FR 63019).

*F. International Tolerances*

Codex maximum residue levels (MRL's) have been established for residues of fenvalerate on a number of crops that also have U.S. tolerances. There is a Codex MRL of 2 ppm fenvalerate on head lettuce. Thus any imported head lettuce is expected to have lower residue values than the proposed section 408 tolerance of 5 ppm esfenvalerate on head lettuce. There are also some minimal differences between the section 408 tolerances and certain Codex MRl values for other commodities. These differences could be caused by differences in methods to establish tolerances, calculate animal feed, dietary exposure, and as a result of different agricultural practices. Therefore, some harmonization of these maximum residue levels will be required. (PM 13)

**3. Interregional Research Project No. 4 (IR-4)**

*PP 5E4598*

EPA has received a pesticide petition (PP) from the Interregional Research Project No. 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, 21 U.S.C. 346a(d), to amend 40 CFR 180.472 by extending the effective date for the time-limited tolerance established for indirect or inadvertant combined residues of the insecticide imidacloprid in or on the raw agricultural commodity cucurbit vegetable crop group at 0.2 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 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 GC-MS selective ion 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 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.

*B. Toxicological Profile*

1. *Acute toxicity*. The acute oral $LD_{50}$ values for imidacloprid technical ranged from 424 – 475 milligram (mg)/kilogram (kg) body weight (bwt) in the rat. The acute dermal $LD_{50}$ was greater than 5,000 mg/kg in rats. The 4–hour rat inhalation $LC_{50}$ was > 69 mg/meter3 (m3) 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-effect level (NOEL) 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 NOEL's for these tests were 14 mg/kg bwt/day (150 ppm) and 5 mg/kg bwt/day (200 ppm) for the rat and dog studies, respectively.

5. *Chronic toxicity*. A 2-year rat feeding/carcinogenicity study was negative for carcinogenic effects under the conditions of the study and had a NOEL 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 NOEL 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 NOEL of 1,000 ppm (208 mg/kg/day).

6. *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.

7. *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. Bayer has concluded that these studies revealed no primary endocrine effects due to imidacloprid.

*C. Aggregate Exposure*

Imidacloprid is a broad-spectrum insecticide with 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. These potential exposures are addressed below:

1. *Dietary exposure*. For purposes of assessing the potential acute and chronic dietary exposure, 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 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.

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

The MOE for imidacloprid derived from previously established tolerances and pending tolerances, including IR-4's cucurbit petition, would be 366 for the U.S. population (48 states), 323 for non-nursing infants, 101 for children (ages 1–6 years), 420 for children (ages 7–12 years), 622 for males 13+ years, and 554 for females 13+ years at the 99.9 percentile. These MOEs do not exceed the EPA's level of concern for acute dietary exposure.

ii. *Chronic*. For purposes of assessing the potential chronic dietary exposure, the model uses the reference dose (RfD) which the EPA has determined to be 0.057 mg/kg/day. This is based on the 2–year rat feeding/carcinogenic study with a NOEL of 5.7mg/kg/bwt and 100–fold uncertainty factor. In conducting this exposure assessment, very conservative assumptions (100% of all commodities contain imidacloprid residues and those residues are at the level of the tolerance) result in a large overestimate of human exposure.

3. *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.

4. *Non-dietary exposure* —i. *Residential Tur*. 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 no-observable effect level 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 µg/cm$^2$ and for 5–year-old children from 5.1 – 33.5 µg/cm$^2$. This compares with the average imidacloprid transferable residue level of 0.080 µg/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 EPA's Occupational and Residential Exposure Branch's (OREB) and Bayer. Data indicate that the Margins of Safety for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are $8.0 \times 10^7$ and $2.4 \times 10^8$, 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 percent 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 NOEL of 5.5 mg/m3, it is apparent that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

iv. *Pet Treatment*. Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA's Occupational and Exposure Branch (OREB) who have concluded that due to the fact that 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*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, it can be concluded that total aggregate exposure to imidacloprid from all current uses including those currently proposed will utilize little more than 14.3% of the RfD for the U.S. population from food, water and non-occupational sources. 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. In addition, the MOEs for all population groups does not exceed the EPA's level of concern for acute dietary exposure. Thus, Bayer concludes 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 two 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 NOEL 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 NOELs 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, it is surmised 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 percent 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. (PM 05)

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## ENVIRONMENTAL PROTECTION AGENCY

**[PF–795; FRL–5775–3]**

### 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–795, must be received on or before March 27, 1998.
**ADDRESSES:** By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 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 Virginia address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

**FOR FURTHER INFORMATION CONTACT:** By mail: Paul Schroeder, Registration Division, (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M. St., SW., Washington, DC 20460. Office location and telephone number: Rm. 255, CM #2, 1921 Jefferson Davis Highway, Arlington, VA, 703–305–6602, e-mail: schroeder.paul@epamail.epa.gov.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–795] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number [PF–795] 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 18, 1998.

**James Jones,**

*Director, Registration Division, Office of Pesticide Programs.*

### Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required