Pretreatment Pilot Program would meet the eight Project XL criteria discussed earlier in this notice.

EPA believes stakeholder involvement in developing Local Pilot Pretreatment Programs is crucial to the success of the programs. Therefore, as part of the application, the POTW must clearly explain its process for involving stakeholders in the design of the pilot program. This process should be based upon the guidance set out in the April 23, 1997, **Federal Register** document. The support of parties that have a stake in the program is very important.

Once EPA has accepted a candidate based on its detailed proposal, the POTW, EPA, the State and local stakeholders should finalize a Final Project Agreement (FPA). The FPA is a non-binding agreement that enumerates the conditions of the project. (In order to expedite this process, EPA will develop a FPA template for these projects that will contain the elements that are anticipated to be common among these projects and shall make this available to the candidates.) The actual regulatory flexibility will be granted by modifying 40 CFR part 403 to allow these specific POTWs to operate Local Pilot Pretreatment Programs.

After an opportunity for public participation at the local level and the development of the Final Project Agreement, a selected POTW's Approval Authority would approve or disapprove the pilot program using the procedures in 40 CFR 403.18. The POTW may implement its Local Pilot Pretreatment Program once its NPDES permit has been modified to incorporate the program as an enforceable permit element.

As with any XL Project, EPA intends to work cooperatively with the POTWs that submit applications for Local Pilot Pretreatment Programs to develop and fine tune the applications. Applicants must recognize that EPA retains the ultimate authority to select projects based on a qualitative consideration of the criteria described earlier. Since these are pilot projects and there are a limited number of pilots that can be approved, projects that satisfy many or all of the criteria may not be chosen for Local Pilot Pretreatment Programs status. The decision of which projects will be selected will be based on an Agency decision about which projects are expected to best serve the objectives of this program. No person is required to submit a proposal or obtain approval as a condition of commencing or continuing a regulated activity. Accordingly, there will be no formal administrative review available for proposals that are not selected, nor does EPA believe there will be a right to judicial review.

Dated: June 20, 1998.

Michael B. Cook,

Director, Office of Wastewater Management.

[FR Doc. 98–16399 Filed 6–22–98; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–813; FRL–5795–1]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–813, must be received on or before July 23, 1998.

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

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

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

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

Product Manager	Office location/telephone number	Address
Mary Waller	Rm. 247, CM #2, 703–308–9354, e-mail:waller.mary@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
James Tompkins	Rm. 239, CM #2, 703–305–5687, e-mail: tompkins.james@epamail.epa.gov.	
Stephanie Willett	Rm. 202, CM #2, 703-305-5419, e-mail:willett.stephanie@epamail.epa.gov.	Do.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–813] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the

use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number (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: June 12, 1998.

James Jones,

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. AgrEvo USA Company

PP 4F4380

EPA has received a pesticide petition (PP [4F4380]) from AgrEvo USA Company, 2711 Centerville Road, Wilmington, DE 19808 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 flutolanil in or on the raw agricultural commodity of rice grain at 2.0 parts per million (ppm), rice straw at 12.0 ppm and in or on the processed commodities of rice hulls at 7.00 ppm and rice bran at 3.0 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. *Plant and animal metabolism*. The metabolism of flutolanil in plants and animals is adequately understood for the purposes of this petition. Animal studies in rats, ruminants, and poultry indicate that flutolanil is metabolized primarily to desisopropylflutolanil and its conjugates. Plant metabolism studies have been conducted in rice, cucumber, and peanuts. The metabolic profile for flutolanil was similar in all three crops. The major route of degradation was 4′-0-dealkylation to desisopropylflutolanil, followed by conjugation. Other metabolites may occur at very low levels due to hydroxylation and oxidation of the side chain, hydroxylation of the aniline ring, and methylation of the hydroxyl groups. These minor metabolites were also subject to conjugation. The residues of concern are the parent flutolanil and desisopropylflutolanil.

2. *Analytical method*. The analytical method designated as AU-95R-04 has been independently validated and is adequate for enforcement purposes. A multi-residue method for flutolanil has been previously submitted. It has the following disclaimer: The method is for use only by experienced chemists who have demonstrated knowledge of the principles of trace organic analysis and have proven skills and abilities to run a complex residue analytical method obtaining accurate results at the part per million level (PPML). Users of this method are expected to perform additional method validation prior to using the method for either monitoring or enforcement. The method can detect gross misuse.

3. *Magnitude of residues*. 24 field trials consisting of foliar applications to rice were conducted in California, Louisiana, Texas, Arkansas, Arizona, Missouri, and Mississippi. Applications of flutolanil formulated as 50WP or 70WP were made at a total seasonal rate of 1.0 lb active ingredient (a.i) per acre resulted in flutolanil-derived residues ranging from below the limit of detection (<0.05 ppm) to 1.66 ppm in whole rice grain and hulled rice and from 0.95 ppm to 11.28 ppm in rice straw.

A processing study was also conducted in Louisiana in which the 50WP formulation of flutolanil was applied to rice following label directions at a total rate of 1.0 lb active ingredient per acre. Residues of flutolanil were observed in all processed commodities and ranged from <0.05 ppm in polished rice to 1.37 ppm in grain dust below 420 microns.

B. Toxicological Profile

1. *Acute toxicity*. A battery of acute studies was conducted: the acute oral LD_{50} in rat and mice were >10,000 milligram/kilograms (mg/kg), Toxicity category IV; acute dermal LD_{50} in rat was >2,000 mg/kg, Toxicity category III; and acute inhalation LC_{50} in rat was >5.98 milligram/liter (mg/l), Toxicity category III. There was slight eye irritation; no dermal irritation; and no dermal sensitization.

2. *Genotoxicty*. Flutolanil has been tested in a battery of *in-vitro* and *in-vivo* assays. No evidence of genotoxicity was noted in gene mutation assays with *Salmonella, E. coli*, or mouse lymphoma cells; a mouse micronucleus assay or in an *in-vitro* unscheduled DNA synthesis assay. A weak positive response was noted in an *in-vitro* cytogenetics assay in Chinese hamster lung cells but no evidence of clastogenicity was noted in an *in-vitro* cytogenetics assay in human lymphocytes. The overall weight of evidence indicates that flutolanil is not genotoxic.

3. *Reproductive and developmental toxicity*. A 3-generation rat reproduction study was conducted at dietary concentrations of 0, 1,000 and 10,000 ppm. The NOEL for this study is considered to be 1,000 ppm (63 milligram/kilograms/day (mg/kg/day), based on reduced pup weights late in lactation at 10,000 ppm. Because the Agency considered this study supplementary, a 2-generation rat reproduction study subsequently was conducted at dietary concentrations of 200, 2,000, and 20,000 ppm. No adverse findings were noted at any dose level and the NOEL was considered to be 20,000 ppm 1,936 mg/kg/day. The Agency, however, has concluded that the NOEL of the original study 63 mg/kg/day should continue to be used for risk assessment.

Developmental toxicity (teratology) studies were conducted in both rats and rabbits at dose levels of 0, 40, 200, and 1,000 mg/kg/day. No significant maternal or developmental toxicity was noted in either study. Thus, both the maternal and developmental NOEL's for both rats and rabbits were considered to be 1,000 mg/kg/day highest dose tested (HDT).

4. *Subchronic toxicity*. A 90-day rat feeding study was conducted at dose levels of 500, 4,000 and 20,000 ppm. The NOEL in this study was considered to be 500 ppm (37 mg/kg/day for males and 44 mg/kg/day for females) based on increased liver weights at 4,000 ppm and slightly decreased body weights at 20,000 ppm.

In a 90-day oral toxicity study in dogs, flutolanil was administered via capsule at dose levels of 0, 80, 400 and 2,000 mg/kg/day. The NOEL was determined to be 80 mg/kg/day based on enlarged livers and increased glycogen deposition at 400 and 2,000 mg/kg/day, and increased alkaline phosphatase and cholesterol levels and thyroid/parathyroid organ weights at 2,000 mg/kg/day.

In a 21-day dermal toxicity study, flutolanil was applied dermally to rats for 15-days over a 21-day interval at dose levels of 0 and 1,000 mg/kg/day. No evidence of dermal irritation or systemic toxicity was observed. Thus, the NOEL was considered to be 1,000 mg/kg/day.

5. *Chronic toxicity*. In a 2-year chronic toxicity/oncogenicity study, flutolanil was administered to rats at dietary levels of 0, 40, 200, 2,000 and 10,000 ppm. The NOEL was considered to be 2,000 ppm (86.9 mg/kg/day for males and 103.1 mg/kg/day for females) based on reduced body weight gain in males and increased liver weights in females at 10,000 ppm. No evidence of carcinogenicity was observed.

In a 78-week carcinogenicity study, flutolanil was administered to mice at dietary concentrations of 0, 300, 1,500, 7,000 and 30,000 ppm. The NOEL was considered to be 7,000 ppm (735 mg/kg/day for males) and 1,500 ppm (162 mg/kg/day for females) based on decreased body weight gains at the higher level(s). No evidence of carcinogenicity was observed.

A 2-year chronic toxicity study was conducted in beagle dogs at dose levels of 0, 50, 250, and 1,250 mg/kg/day. The NOEL was considered to be 250 mg/kg/day based on decreased weight gain at 1,250 mg/kg/day.

6. *Animal metabolism*. Studies in rats, ruminants, and poultry suggest that flutolanil is not well-absorbed following oral administration. Once absorbed, however, it is rapidly metabolized, primarily to desisopropylflutolanil and its conjugates, and rapidly excreted via urine and feces.

7. *Endocrine disruption*. No special studies have been conducted to investigate the potential of flutolanil to induce estrogenic or other endocrine effects. However, no evidence of such effects has been observed in the subchronic, chronic, or reproductive studies previously discussed. Thus, the potential for flutolanil to cause endocrine effects is considered to be minimal.

C. Aggregate Exposure

1. *Dietary exposure*. Includes food and drinking water—i. *Food*. Time-limited tolerances have been previously established for flutolanil in or on rice commodities, and tolerances with no time limitations are established for peanut commodities, meat, milk, and eggs. Potential dietary exposures to flutolanil from these food commodities were assessed using the exposure one software system (TAS, Inc.) and food consumption data from the 1977-1978 USDA Continuing Surveys of Food Consumption by Individuals (CSFCI). For the purposes of this assessment, it was assumed that 100% of all of the above commodities were at the existing tolerance levels for flutolanil.

ii. *Drinking water*. The potential for flutolanil to leach into groundwater has been assessed in two terrestrial field dissipation studies, a long-term terrestrial field dissipation study, and an aquatic field dissipation study. Under field conditions, the half-life of flutolanil varied from 101 to 123 days in the long-term field soil dissipation study, which was consistent with the other field studies, and was approximately 180 days in the aquatic environment. Flutolanil strongly adsorbs to soil following application and did not exhibit mobility under either terrestrial or aquatic conditions. The water solubility of flutolanil is quite low (5.0 ppm). Based on these environmental fate data and the conditions of use, the potential for movement of flutolanil into groundwater is very low, and as such the potential contribution of any such residues to the total dietary intake of flutolanil will be negligible. No maximum contaminant level (MCL) or Health Advisory Level for residues of flutolanil in drinking water has been established.

2. *Non-dietary exposure*. As prostar 50WP (EPA Reg No. 45639-153) is a professional turf and ornamental fungicide, flutolanil is used primarily (>95%) on golf courses for control of brown patch disease (Rhizoctonia solani). Very limited use of prostar 50WP may occur on commercial ornamental turf by professional lawn care applicators or on sod farms. The product is rarely, if ever, used on homeowner turf due to the fact that the diseases it controls (Brown patch, Fry ring, snow molds) occur in high-fertility, high-maintenance turf (e.g. golf courses), not in homeowner lawns. Thus, non-dietary exposure to flutolanil would be minimal. Furthermore, no dermal toxicity endpoints of concern have been identified for flutolanil. Thus, an assessment of non-dietary exposure and risk is not considered to be necessary.

D. Cumulative Effects

Flutolanil has demonstrated only minimal toxicity in animal studies. The mechanism of this toxicity is unknown. Furthermore, there are no available data to indicate that flutolanil has a common mechanism of toxicity with other substances. Thus, only the potential risks from flutolanil are being considered in this document.

E. Safety Determination

1. *U.S. population*. Based on the existing and proposed tolerances in rice, peanuts, and secondary commodities, the Theoretical Maximum Residue Contribution (TMRC) of the current action is estimated to be 0.001124 mg/kg/day for the U.S. population in general. This exposure would utilize less than 1% of the RfD. There is generally no concern for exposures below 100% of the RfD since the RfD represents the exposure level at or below which daily exposure over a lifetime will not pose any appreciable risks to human health. Therefore, there is a reasonable certainty that no harm will result to the U.S. population in general from aggregate exposure to flutolanil.

2 *Infants and children*. Data from reproductive and developmental toxicity studies are generally used to assess the potential for increased sensitivity of infants and children. No evidence of developmental toxicity was noted in rats or rabbits, even at the limit dose of 1,000 mg/kg/day. Reduced pup weights in the absence of parental toxicity were noted at the HDL (10,000 ppm) in a 3-generation rat reproduction study. However, no such effects were noted in a subsequent reproduction study, even at a HDT (20,000 ppm). Furthermore, the reduced weight gain in the first study began late in the lactation period, at a time when the pups were likely ingesting significant quantities of diet. Feed intake is much higher in young animals than in adults and the apparent increase in sensitivity may simply reflect the higher test material intake in these pups on a mg/kg basis compared to the adults. Thus, AgrEvo believes that the overall weight of evidence does not indicate any special concern for infants and children, and that no additional safety factor is necessary.

Based on the existing and proposed tolerances in rice, peanuts, and secondary commodities, the Theoretical Maximum Residue Contribution (TMRC) from the current petition is estimated to be 0.006218 mg/kg/day for the most highly exposed sub-population, non-nursing infants (less

than 1-year old).. This exposure would utilize less than 1 % of the RfD. Therefore, there is a reasonable certainty that no harm will result to infants or children from aggregate exposure to flutolanil.

F. International Tolerances

No CODEX tolerances have been established or proposed for residues of flutolanil. (Mary Waller).

2. Bayer Corporation

PP 6F4631

EPA has received a pesticide petition (PP 6F4631) from Bayer Corporation, 8400 Hawthorn Road, P.O. Box 4913, Kansas City, MO 64120–0013 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.527 by establishing tolerances for inadvertent residues of *N*-(4-fluorophenyl)-*N*-(1-methylethyl)-2- [[5-(trifluoromethyl)-1,3,4-thiadiazol-2-yl]oxy]acetamide [hereafter referred to as flufenacet, the proposed common chemical name] and metabolites containing the 4-fluoro-*N*-methylethyl benzenamine moiety in or on the raw agricultural commodities of Crop Group 15 (cereal grains), Crop Group 16 (forage, stover and hay of cereal grains), Crop Group 17 (grass forage, and grass hay), alfalfa forage, alfalfa hay, alfalfa seed, clover forage, and clover hay at 0.1 parts per million (ppm) when present therein as a result of the application of flufenacet to field corn and soybeans as a herbicide. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in field corn, soybeans, livestock and rotational crops is adequately understood. The residues of concern for the tolerance expression are *N*-(4-fluorophenyl)-*N*-(1-methylethyl)-2-[[5-(trifluoromethyl)-1,3,4-thiadiazol-2-yl]oxy]acetamide parent and its metabolites containing the 4-fluoro-*N*-methylethyl benzenamine moiety. Based on the results of animal metabolism studies it is unlikely that secondary residues would occur in animal commodities from the use of flufenacet on field corn and soybeans.

2. *Analytical method*. An adequate analytical method, gas chromatography/mass spectrometry with selected ion monitoring, is available for enforcement purposes. Because of the long lead time from establishing these tolerances to publication of the enforcement methodology in the Pesticide Analytical Manual, Vol. II, the analytical methodology is being made available in the interim to anyone interested in pesticide enforcement when requested from: Calvin Furlow, Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Room 119E, CM #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703–305–5937).

3. *Magnitude of residues*. Time limited tolerances exist for the combined residues of flufenacet, *N*-(4-fluorophenyl)-*N*-(1-methylethyl)-2-[[5-(trifluoromethyl)-1,3,4-thiadiazol-2-yl]oxy]acetamide and its metabolites containing the 4-fluoro-*N*-methylethyl benzenamine moiety in or on field corn grain at 0.05 ppm, field corn forage at 0.4 ppm, field corn stover at 0.4 ppm, and soybean seed at 0.1 ppm. The petitioner, Bayer Corporation has amended its petition (PP 6F4631) to include tolerances for residues of *N*-(4-fluorophenyl)-*N*-(1-methylethyl)-2-[[5-(trifluoromethyl)-1,3,4-thiadiazol-2-yl]oxy]acetamide and its metabolites containing the 4-fluoro-*N*-methylethyl benzenamine moiety at 0.1 ppm for residues in or on the raw agricultural commodities of Crop Group 15 (cereal grains), Crop Group 16 (forage, stover and hay of cereal grains), Crop Group 17 (grass forage and grass hay), alfalfa forage, alfalfa hay, alfalfa seed, clover forage, and clover hay. The proposed tolerance levels are adequate to cover residues likely to be present in rotational crops planted after corn or soybeans which were treated with flufenacet.

B. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study with a LD_{50} of 1,617 milligrams/kilograms for males and 589 mg/kg for females.

2. *Genotoxicty*. Flufenacet was negative for mutagenic/genotoxic effects in a Gene mutation/*In vitro* assay in bacteria, a Gene mutation/*In vitro* assay in chinese hamster lung fibroblasts cells, a Cytogenetics/*In vitro* assay in chinese hamster ovary cells, a Cytogenetics/*In vivo* mouse micronucleus assay, and an *In vitro* unscheduled DNA synthesis assay in primary rat hepatocytes.

3. *Reproductive and developmental toxicity*. A two-generation rat reproduction study with a parental systemic no observed effect level (NOEL) of 20 ppm [1.4 mg/kg/day in males and 1.5 mg/kg/day in females] and a reproductive NOEL of 20 ppm [1.3 mg/kg/day] and a parental systemic lowest observed effect level (LOEL) of 100 ppm [7.4 mg/kg/day in males and 8.2 mg/kg/day in females] based on increased liver weight in F1 females and hepatocytomegaly in F1 males and a reproductive LOEL of 100 ppm [6.9 mg/kg/day] based on increased pup death in early lactation (including cannibalism) for F1 litters and the same effects in both F1 and F2 pups at the high dose level of 500 ppm [37.2 mg/kg/day in F1 males and 41.5 mg/kg/day in F1 females, respectively]. A rat developmental study with a maternal NOEL of 25 mg/kg/day and with a maternal LOEL of 125 mg/kg/day based on decreased body weight gain initially and a developmental NOEL of 25 mg/kg/day and a developmental LOEL of 125 mg/kg/day based on decreased fetal body weight, delayed development [mainly delays in ossification in the skull, vertebrae, sternebrae, and appendages], and an increase in the incidence of extra ribs. A rabbit developmental study with a maternal NOEL of 5 mg/kg/day and a maternal LOEL of 25 mg/kg/day based on histopathological finds in the liver and a developmental NOEL of 25 mg/kg/day and a developmental LOEL of 125 mg/kg/day based on increased skeletal variations.

4. *Subchronic toxicity*. A 84–day rat feeding study with a No Observed Effect Level (NOEL) less than 100 ppm [6.0 mg/kg/day] for males and a NOEL of 100 ppm [7.2 mg/kg/day] for females and with a Lowest Observed Effect Level (LOEL) of 100 ppm [6.8 mg/kg/day] for males based on suppression of thyroxine (T4) level and a LOEL of 400 ppm [28.8 mg/kg/day] for females based on hematology and clinical chemistry findings. A 13–week mouse feeding study with a NOEL of 100 ppm [18.2 mg/kg/day for males and 24.5 mg/kg/day for females] and a LOEL of 400 ppm [64.2 mg/kg/day for males and 91.3 mg/kg/day for females] based on histopathology of the liver, spleen and thyroid. A 13–week dog dietary study with a NOEL of 50 ppm [1.70 mg/kg/day for males and 1.67 mg/kg/day for females] and a LOEL of 200 ppm [6.90 mg/kg/day for males and 7.20 mg/kg/day for females] based on evidence that the bio-transformation capacity of the liver has been exceeded, (as indicated by increase in LDH, liver weight, ALK and hepatomegaly), globulin and spleen pigment in females, decreased T4 and

ALT values in both sexes, decreased albumin in males, and decreased serum glucose in females. A 21–day rabbit dermal study with the dermal irritation NOEL of 1,000 mg/kg/day for males and females and a systemic NOEL of 20 mg/kg/day for males and 150 mg/kg/day for females and a systemic LOEL of 150 mg/kg/day for males and 1,000 mg/kg/day for females based on clinical chemistry data (decreased T4 and FT4 levels in both sexes) and centrilobular hepatocytomegaly in females.

5. *Chronic toxicity*. A 1–year dog chronic feeding study with a NOEL was 40 ppm [1.29 mg/kg/day in males and 1.14 mg/kg/day in females] and a LOEL of 800 ppm [27.75 mg/kg/day in males and 26.82 mg/kg/day in females] based on increased alkaline phosphatase, kidney, and liver weight in both sexes, increased cholesterol in males, decreased T2, T4 and ALT values in both sexes, and increased incidences of microscopic lesions in the brain, eye, kidney, spinal cord, sciatic nerve and liver. A rat chronic feeding/carcinogenicity study with a NOEL less than 25 ppm [1.2 mg/kg/day in males and 1.5 mg/kg/day in females] and a LOEL of 25 ppm [1.2 mg/kg/day in males and 1.5 mg/kg/day in females] based on methemoglobinemia and multi-organ effects in blood, kidney, spleen, heart, and uterus. Under experimental conditions the treatment did not alter the spontaneous tumor profile. In a mouse carcinogenicity study the NOEL was less than 50 ppm [7.4 mg/kg/day] for males and the NOEL was 50 ppm [9.4 mg/kg/day] for females and the LOEL was 50 ppm [7.4 mg/kg/day] for males and the LOEL was 200 ppm [38.4 mg/kg/day] for females based on cataract incidence and severity. There was no evidence of carcinogenicity for flufenacet in this study.

6. *Animal metabolism*. A rat metabolism study showed that radio-labeled flufenacet was rapidly absorbed and metabolized by both sexes. Urine was the major route of excretion at all dose levels and smaller amounts were excreted via the feces. A 55–day dog study with subcutaneous administration of Thiadone [flufenacet metabolite] supports the hypothesis that limitationsin glutathione interdependent pathways and antioxidant stress result in metabolic lesions in the brain and heart following flufenacet exposure.

7. *Endocrine disruption*. EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other effect. The Agency is currently working with interested stakeholders, including other government agencies, public interest groups, industry and research scientists in developing a screening and testing program and a priority setting scheme to implement this program. Congress has allowed 3 years from the passage of FQPA (August 3, 1999) to implement this program. At that time, EPA may require further testing of this active ingredient and end use products for endocrine disrupter effects. Based on the toxicological findings for flufenacet relating to endocrine disruption effects, flufenacet should be considered as a candidate for evaluation as an endocrine disrupter when the criteria are established.

C. Aggregate Exposure

In examining aggregate exposure, FFDCA section 408 requires that EPA take into account available and reliable information concerning exposure from the pesticide residue in the food in question, residues in other foods for which there are tolerances, residues in groundwater or surface water that is consumed as drinking water, and other non-occupational exposures through pesticide use in gardens, lawns, or buildings (residential and other indoor uses).

1. *Dietary exposure*—i. *Food*. Dietary exposure to residues of a pesticide in a food commodity are estimated by multiplying the average daily consumption of the food forms of that commodity by the tolerance level or the anticipated pesticide residue level. The Theoretical Maximum Residue Contribution (TMRC) is an estimate of the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance. In evaluating food exposures, varying consumption patterns of major identifiable subgroups of consumers, including infants and children is taken into account. The TMRC is a ''worst case'' estimate since it is based on the assumptions that food contains pesticide residues at the tolerance level and that 100% of the crop is treated by pesticides that have established tolerances. Using tolerance levels and percent crop treated, the residues in the diet (food only) are calculated to be 0.0001 milligrams/kilogram of body weight per day (mg/kg bwt/day) or 2.6% of the RfD for the general U.S. population and 0.00023 mg/kg bwt/day or 5.8% of the RfD for children aged 1–6 years.

ii. *Drinking water*. Residues of flufenacet in drinking water may comprise up to 0.0039 mg/kg bwt/day (0.0040–0.0001 mg/kg bwt/day) for the U.S. population and 0.0038 mg/kg bwt/day (0.00400–0.00023 mg/kg bwt/day) for children 1–6 years old (the group exposed to the highest level of flufenacet residues in both food and water). The drinking water levels of concern (DWLOCs) for chronic exposure to flufenacet in drinking water calculated for the U.S. population was 136 parts per billion (ppb) assuming that an adult weighs 70 kg and consumes a maximum of 2 liters of water per day. For children (1–6 years old), the DWLOC was 37.7 ppb assuming that a child weighs 10 kg and consumes a maximum of 1 liter of water per day. The drinking water estimated concentration (DWECs) for groundwater (parent flufenacet and degradate thiadone) calculated from the monitoring data is 0.03 ppb for chronic concentrations which does not exceed DWLOC of 37.7 ppb for children (1–6 years old). The DWEC for surface water based on the computer models PRZM 2.3 and EXAMS 2.97.5 was calculated to be 14.2 ppb for chronic concentration (parent flufenacet and degradate thiadone) which does not exceed the DWLOC of 37.7 ppb for children (1–6 years old).

2. *Non-dietary exposure*. There are no non-food uses of flufenacet currently registered under the Federal Insecticide, Fungicide and Rodenticide Act, as amended. No non-dietary exposures are expected for the general population.

D. Cumulative Effects

Flufenacet is structurally a thiadiazole. EPA is not aware of any other pesticides with this structure. For flufenacet, EPA has not yet conducted a detailed review of common mechanisms to determine whether it is appropriate, or how to include this chemical in a cumulative risk assessment. After EPA develops a methodology to address common mechanism of toxicity issues to risk assessments, the Agency will develop a process (either as part of the periodic review of pesticides or otherwise) to reexamine these tolerance decisions. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, flufenacet does not appear to produce a toxic metabolite produced by other substances. For the purposes of these tolerance actions; therefore, EPA has not assumed that flufenacet has a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*—i. *Acute risk*. The acute endpoint for flufenacet and its metabolites is 75 mg/kg/day. The acute

exposure for flufenacet and its metabolites is 0.0015 mg/kg/day for the general U.S. population and 0.002 mg/kg/day for children 1–6 years of age. The DWLOC for acute exposure to flufenacet in drinking water calculated for the U.S. population was 2.87 ppm and for children (1–6 years old) was 813 ppb. These figures were calculated as follows. First, the acceptable acute exposure to flufenacet in drinking water was obtained by subtracting the acute dietary food exposures from the ratio of the acute LOEL to the acceptable margin of exposure (MOE) for aggregate exposure. Then, the DWLOCs were calculated by multiplying the acceptable exposure to flufenacet in drinking water by estimated body weight (70 kg for adults, 10 kg for children) and then dividing by the estimated daily drinking water consumption (2 L/day for adults, 1 L/day for children). The Agency's SCI-Grow model estimates peak levels of flufenacet and its metabolite thiadone in groundwater to be 15.3 ppb. PRZM/EXAMS estimates peak levels of flufenacet and its metabolite thiadone in surface water to be 17 ppb. EPA's acute drinking water level of concern is well above the estimated exposures for flufenacet in water for the U.S. population and subgroup with highest estimated exposure.

ii. *Chronic risk*. The chronic endpoint for flufenacet is 0.004 mg/kg bwt/day. Using tolerance levels and percent crop treated, the residues in the diet (food only) are calculated to be 0.0001 mg/kg bwt/day or 2.6% of the Reference dose (RfD) for the general U.S. population and 0.00023 mg/kg bwt/day or 5.8% of the RfD for children aged 1–6 years. Therefore, residues of flufenacet in drinking water may comprise up to 0.0039 mg/kg bwt/day (0.0040–0.0001 mg/kg bwt/day) for the U.S. population and 0.0038 mg/kg bwt/day (0.00400–0.00023 mg/kg bwt/day) for children 1–6 years old (the group exposed to the highest level of flufenacet residues in both food and water). The DWLOCs for chronic exposure to flufenacet in drinking water calculated for the U.S. population was 136 ppb assuming that an adult weighs 70 kg and consumes a maximum of 2 liters of water per day. For children (1–6 years old), the DWLOC was 37.7 ppb assuming that a child weighs 10 kg and consumes a maximum of 1 liter of water per day. The drinking water estimated concentration (DWECs) for groundwater (parent flufenacet and degradate thiadone) calculated from the monitoring data is 0.03 ppb for chronic concentrations which does not exceed the DWLOC of 37.7 ppb for children (1–6 years old). The DWEC for surface water based on the computer models PRZM 2.3 and EXAMS 2.97.5 was calculated to be 14.2 ppb for chronic concentration (parent flufenacet and degradate thiadone) which does not exceed the DWLOC of 37.7 ppb for children (1–6 years old). EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to flufenacet residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of flufenacet, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Although there is no indication of increased sensitivity to young rats or rabbits following pre- and/or post-natal exposure to flufenacet in the standard developmental and reproductive toxicity studies, an additional developmental neurotoxicity study, which is not normally required, is needed to access the susceptibility of the offspring in function/neurological development. Therefore, EPA has required that a developmental neurotoxicity study be conducted with flufenacet and a threefold safety factor for children and infants will be used in the aggregate dietary acute and chronic risk assessment. Although there is no indication of additional sensitivity to young rats or rabbits following pre- and/or post-natal exposure to flufenacet in the developmental and reproductive toxicity studies; the Agency concluded that the FQPA safety factor should not be removed but instead reduced because:

(i) There was no assessment of susceptibility of the offspring in functional/neurological developmental and reproductive studies.

(ii) There is evidence of neurotoxicity in mice, rats, and dogs.

(iii) There is concern for thyroid hormone disruption.

F. International Tolerances

There are no Codex Alimentarius Commission (Codex) Maximum Residue Levels (MRLs) for flufenacet. (James A. Tompkins).

3. FMC Corporation

PP 8F4970

EPA has received pesticide petitions (PP 8F4970) from FMC Corporation, 1735 Market Street,Philadelphia, PA 19103, proposing pursuant to section 408 (d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.418 by establishing a tolerance for residues of the insecticide zeta-cypermethrin (±α-Cyano(3-phenoxyphenyl)methyl (±) cis, trans 3-(2,2-dichloroethenyl)-2,2-dimethylcyclopropanecarboxylate)in or on the raw agricultural commodity Brassica vegetables, head and stem at 2.0 ppm and Brassica vegetables, leafy at 14.0 ppm; and the leafy vegetables (except Brassica vegetables) group at 10.0 ppmn. 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 cypermethrin in plants is adequately understood. Studies have been conducted to delineate the metabolism of radio labelled cypermethrin in various crops all showing similar results. The residue of concern is the parent compound only.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of cypermethrin in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances (Gas Chromatography with Electron Capture Detection (GC/ECD).

3. *Magnitude of residues*. Crop field trial residue data from studies conducted at the maximum label rates for head and stem Brassica vegetables, leafy Brassica greens, and leafy vegetables (except Brassica vegetables) group, show that the proposed zeta-cypermethrin tolerances on Brassica vegetables, head and stemat 2.0 ppm and Brassica vegetables, leafy at 14.0 ppm; and the leafy vegetables (except Brassica vegetables) group at 10.0 ppm will not be exceeded when the zeta-cypermethrin products labeled for these uses are used as directed.

B. Toxicological Profile

1. *Acute toxicity*. For the purposes of assessing acute dietary risk, FMC has used the no-observed-effected label (NOEL) of 3.8 mg/kg/day based on the NOEL of 7.5 mg/kg/day from the cypermethrin chronic feeding/ oncogenicity study in rats and a correction factor of two to account for the differences in the percentage of the biologically active isomer. The LOEL of 50.0 mg/kg/day was based on neurological signs which were displayed during week one of the study. This acute dietary end point is used to determine acute dietary risks to all population subgroups.

2. *Genotoxicity*. The following genotoxicity tests were all negative: *in vivo* chromosomal aberration in rat bone marrow cells; *in vitro* cytogenic chromosome aberration; unscheduled DNA synthesis;CHO/HGPTT mutagen assay; weakly mutagenic: gene mutation (Ames).

3. *Reproductive and developmental toxicity*. No evidence of additional sensitivity to young rats was observed following pre- or postnatal exposure to zeta-cypermethrin.

i. A 2-generation reproductive toxicity study with zeta-cypermethrin in rats demonstrated a NOEL of 7.0 mg/kg/day and a LOEL of 27.0 mg/kg/day for parental/systemic toxicity based on body weight, organ weight, and clinical signs. There were no adverse effects in reproductive performance. The NOEL for reproductive toxicity was considered to be > 45.0 mg/kg/day the highest dose tested (HDT).

ii. A developmental study with zeta-cypermethrin in rats demonstrated a maternal NOEL of 12.5 mg/kg/day and a LOEL of 25 mg/kg/day based on decreased maternal body weight gain, food consumption and clinical signs. There were no signs of developmental toxicity at 35.0 mg/kg/day, the higest dose level tested (HDLT).

iii. A developmental study with cypermethrin in rabbits demonstrated a maternal NOEL of 100 mg/kg/day and a LOEL of 450 mg/kg/day based on decreased body weight gain. There were no signs of developmental toxicity at 700 mg/kg/day, the HDLT.

4. *Subchronic toxicity— Short- and intermediate-term toxicity*. The NOEL of 3.8 mg/kg/day based on the NOEL 7.5 mg/kg/day from the cypermethrin chronic feeding/oncogenicity study in rats and a correction factor of two to account for the biologically active isomer would also be used for short-and intermediate-term MOE calculations (as well as acute, discussed in (1) above). The LOEL of 50.0 mg/kg/day was based on neurological signs which were displayed during week one of the study.

5. *Chronic toxicity*. The reference dose (RfD) of 0.0125 mg/kg/day for zeta-cypermethrin is based on a NOEL of 2.5 mg/kg/day from a cypermethrin rat reproduction study and an uncertainty factor of 200 (used to account for the differences in the percentage of the biologically active isomer). The endpoint effect of concern was based on consistent decreased body weight gain in both sexes at the LOEL of 7.5 mg/kg/ day.

Cypermethrin is classified as a Group C chemical (possible human carcinogen with limited evidence of carcinogenicity in animals) based upon limited evidence for carcinogenicity in femalemice; assignment of a Q* has not been recommended.

6. *Animal metabolism*. The metabolism of cypermethrin in animals is adequately understood. Cypermethrin has been shown to be rapidly absorbed, distributed, and excreted in rats when administered orally. Cypermethrin is metabolized by hydrolysis and oxidation.

7. *Metabolite toxicology*. The Agency has previously determined that the metabolites of cypermethrin are not of toxicological concern and need not be included in the tolerance expression.

8. *Endocrine disruption*. No special studies investigating potential estrogenic or other endocrine effects of cypermethrin have been conducted. However, no evidence of such effects were reported in the standard battery of required toxicology studies which have been completed and found acceptable. Based on these studies, there is no evidence to suggest that cypermethrin has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Permanent tolerances, in support of registrations, currently exist for residues of zeta-cypermethrin on cottonseed; pecans; lettuce, head; onions, bulb; and cabbage and livestock commodities of cattle, goats, hogs, horses, and sheep. For the purposes of assessing the potentialdietary exposure for these existing and the subject proposed tolerances, FMC has utilized available information on anticipated residues, monitoring data and percent crop treated as follows:

ii. *Acute exposure and risk*. Acute dietary exposure risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1-day or single exposure. For the purposes of assessing acute dietary risk for zeta-cypermethrin, FMC has used the NOEL of 3.8 mg/kg/ day based on the NOEL of 7.5 mg/kg/ day from the cypermethrin chronic feeding/oncogenicity study in rats and a correction factor of two to account for the differences in the percentage of the biologically active isomer. The LOEL of 50.0 mg/kg/day was based on neurological signs which were displayed during week one of this study. This acute dietary endpoint is used to determine acute dietary risks to all population subgroups. Available information on anticipated residues, monitoring data and percent crop treated was incorporated into a Tier 3 analysis, using Monte Carlo modeling for commodities that may be consumed in a single serving. These assessments show that the margins of exposure (MOE) are significantly greater than the EPA standard of 100 for all subpopulations. The 95th percentile of exposure for the overall U. S. population was estimated to be 0.000708 mg/kg/day (MOE of 5364); 99th percentile 0.002677 mg/kg/day (MOE of 1420); and 99.9th percentile 0.012098 mg/kg/day (MOE of 314). The 95th percentile of exposure for all infants <1- year old was estimated to be 0.000264 mg/kg/day (MOE of 14394); 99th percentile 0.00189 mg/kg/day (MOE of 2011); and 99.9th percentile 0.018164 mg/kg/day (MOE of 209). The 95th percentile of exposure for nursing infants <1-year old was estimated to be 0.000026 mg/kg/day (MOE of 147540); 99th percentile 0.000484 mg/kg/day (MOE of 7843); and 99.9th percentile 0.002004 mg/kg/day (MOE of 1896).The 95th percentile of exposure for non-nursing infants < 1- year old was estimated to be 0.000367mg/kg/day (MOE of 10342); 99th percentile 0.005649 mg/kg/day (MOE of 673); and 99.9th percentile 0.019823 mg/kg/day (MOE of 192). The 95th percentile of exposure for children 1 to 6-years old (the most highly exposed population subgroup) and children 7 to 12-years old was estimated to be, respectively, 0.000742 mg/kg/day (MOE of 5120) and 0.00748 mg/kg/day (MOE of 5077); 99th percentile 0.003061 mg/kg/day (MOE of 1241) and 0.002638 (MOE of 1440); and 99.9th percentile 0.031769 mg/kg/day (MOE of 120) and 0.013432 (MOE of 283). Therefore, FMC concludes that the acute dietary risk of zeta-cypermethrin, as estimated by the dietary risk assessment, does not appear to be of concern.

iii. *Chronic exposure and risk*. RfD of 0.0125 mg/kg/day for zeta-cypermethrin is based on a NOEL of 2.5 mg/kg/day from a cypermethrin rat reproduction

study and an uncertainty factor of 200 (used to account for the differences in the percentage of the biologically active isomer). The endpoint effect of concern was based on consistent decreased body weight gain in both sexes at the LOEL of 7.5 mg/kg/day. A chronic dietary exposure/risk assessment has been performed for zeta-cypermethrin using the above RfD. Available information on anticipated residues, monitoring data and percent crop treated was incorporated into the analysis to estimate the anticipated residue contribution (ARC). The ARC is generally considered a more realistic estimate than an estimate based on tolerance level residues. The ARC are estimated to be 0.000098 mg/kg body weight/day (mg/kg/bwt/day) and utilize 0.8 % of the RfD for the overall U. S. population. The ARC for non-nursing infants (<1-year) and nursing infants (<1-year) are estimated to be 0.00016 mg/kg/day and 0.00001 mg/kg/day and utilizes 1.3 % and 0.1 % of the RfD, respectively. The ARC for children 1-6 years old (subgroup most highly exposed) and children 7-12 years old are estimated to be 0.000172 mg/kg bwt/day and 0.000092 mg/kg bwt/day and utilizes 1.4 % and 0.7 % of the RfD, respectively. Generally speaking, the EPA has no cause for concern if the total dietary exposure from residues for uses for which there are published and proposed tolerances is less than 100 % of the RfD. Therefore, FMC concludes that the chronic dietary risk of zeta-cypermethrin, as estimated by the dietary risk assessment, does not appear to be of concern.

2. *Drinking water*. Laboratory and field data have demonstrated that cypermethrin is immobile in soil and will not leach into groundwater. Other data show that cypermethrin is virtually insoluble in water and extremely lipophilic. As a result, FMC concludes that residues reaching surface waters from field runoff will quickly adsorb to sediment particles and be partitioned from the water column. Further, a screening evaluation of leaching potential of a typical pyrethroid was conducted using EPA's Pesticide Root Zone Model (PRZM3). Based on this screening assessment, the potential concentrations of a pyrethroid in groundwater at depths of 1 and 2 meters are essentially zero (<0.001 part per billion (PPB). 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 PPB. 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 normally be treated before consumption. Based on these analyses, the contribution of water to the dietary risk estimate is negligible. Therefore, FMC concludes that together these data indicate that residues are not expected to occur in drinking water.

3. *Non-dietary exposure*. Zeta-cypermethrin is registered for agricultural crop applications only, therefore non-dietary exposure assessments are not warranted.

D. Cumulative Effects

In consideration of potential cumulative effects of cypermethrin and other substances that may have a common mechanism of toxicity, to our knowledge there are currently no available data or other reliable information indicating that any toxic effects produced by cypermethrin would be cumulative with those of other chemical compounds; thus only the potential risks of cypermethrin have been considered in this assessment of its aggregate exposure. FMC intends to submit information for the EPA to consider concerning potential cumulative effects of cypermethrin consistent with the schedule established by EPA at 62 FR 42020 (August 4, 1997) (FRL 5734-6) and other EPA publications pursuant to the Food Quality Protection Act.

E. Safety Determination

1. *U.S. population*. Based on a complete and reliable toxicology database, the RfD for zeta-cypermethrin is 0.0125 mg/kg/day, based on a NOEL of 2.5 mg/kg/day and a LOEL of 7.5 mg/kg/day from the cypermethrin rat reproduction study and an uncertainty factor of 200. Available information on anticipated residues, monitoring data and percent crop treated was incorporated into an analysis to estimate the ARC for 26 population subgroups. The ARC is generally considered a more realistic estimate than an estimate based on tolerance level residues. The ARC are estimated to be 0.000098 mg/kg/bwt/day and utilize 0.8 of the RfD or the overall U. S. population. The ARC for non-nursing infants (<1-year) and nursing infants (<1- year) are estimated to be 0.00016 mg/kg/day and 0.00001 mg/kg/day and utilizes 1.3 % and 0.1 % of the RfD, respectively. The ARC for children 1-6 years old (subgroup most highly exposed) and children 7-12 years old are estimated to be 0.000172 mg/kg bwt/day and 0.000092 mg/kg bwt/day and utilizes 1.4 % and 0.7 % of the RfD, respectively. Generally speaking, the EPA has no cause for concern if the total dietary exposure from residues for uses for which there are published and proposed tolerances is less than 100 % of the RfD. Therefore, FMC concludes that the chronic dietary risk of zeta-cypermethrin, as estimated by the aggregate risk assessment, does not appear to be of concern.

For the overall U.S. population, the calculated margins of exposure (MOE) at the 95th percentile was estimated to be 5364; 1420 at the 99th percentile; and 314 at the 99.9th percentile. For all infants < 1-year old, the calculated MOE at the 95th percentile was estimated to be 14394; 2011 at the 99th percentile; and 209 at the 99.9th percentile. For nursing infants < 1-year old, the calculated MOE at the 95th percentile was estimated to be 147540; 7843 at the 99th percentile; and 1896 at the 99.9th percentile. For non-nursing infants < 1-year old, the calculated MOE at the 95th percentile was estimated to be 10342; 673 at the 99th percentile; and 192 at the 99.9th percentile. For the most highly exposed population subgroup, children 1- 6 years old, and for children 7-12 years old, the calculated MOEs at the 95th percentile were estimated to be, respectively, 5120 and 5077; 1241 and 1440 at the 99th percentile; and 120 and 283 at the 99.9th percentile. Therefore, FMC concludes that there is reasonable certainty that no harm will result from acute exposure to zeta-cypermethrin.

2. *Infants and children*—i. *General*. In assessing the potential for additional sensitivity of infants and children to residues of zeta-cypermethrin, FMC considered data from developmental toxicity studies in the rat and rabbit, and a 2-generation reproductive study in the rat. The data demonstrated no indication of increased sensitivity of rats to zeta-cypermethrin or rabbits to cypermethrin in utero and/or postnatal exposure to zeta-cypermethrin or cypermethrin. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. FFDCA section 408 provides that EPA may apply an additional margin of safety for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database.

ii. *Developmental toxicity studies*. In the prenatal developmental toxicity studies in rats and rabbits, there was no evidence of developmental toxicity at the HDT (35.0 mg/kg/day in rats and 700 mg/kg/day in rabbits). Decreased body weight gain was observed at the maternal LOEL in each study; the maternal NOEL was established at 12.5 mg/kg/day in rats and 100 mg/kg/day in rabbits.

iii. *Reproductive toxicity study*. In the 2-generation reproduction study in rats, offspring toxicity (body weight) and parental toxicity (body weight, organ weight, and clinical signs) was observed at 27.0 mg/kg/day and greater. The parental systemic NOEL was 7.0 mg/kg/day and the parental systemic LOEL was 27.0 mg/kg/day. There were no developmental (pup) or reproductive effects up to 45.0 mg/kg/day, HDT.

iv. *Pre- and post-natal sensitivity*—a. *Pre-natal*. There was no evidence of developmental toxicity in the studies at the HDT in the rat (35.0 mg/kg/day) or in the rabbit (700 mg/kg/day). Therefore, there is no evidence of a special dietary risk (either acute or chronic) for infants and children which would require an additional safety factor.

b. *Post-natal*. Based on the absence of pup toxicity up to dose levels which produced toxicity in the parental animals, there is no evidence of special post-natal sensitivity to infants and children in the rat reproduction study.

c. *Conclusion*. Based on the above, FMC concludes that reliable data support use of the standard 100-fold uncertainty factor, and that an additional uncertainty factor is not needed to protect the safety of infants and children. As stated above, aggregate exposure assessments utilized significantly less than 1 % of the RfD for either the entire U. S. population or any of the 26 population subgroups including infants and children. Therefore, it may be concluded that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to cypermethrin residues.

3. *Subchronic toxicity— Short- and intermediate-term toxicity*. The NOEL of 3.8 mg/kg/day based on the NOEL 7.5 mg/kg/day from the cypermethrin toxicity/oncogenicity study in rats and a correction factor of two to account for the biologically active isomer would also be used for short- and intermediate-term MOE calculations (as well as acute, discussed in (E.1.) above). The LOEL of this study of 50.0 mg/kg/day was based on neurological signs observed in the first week of the study.

F. International Tolerances

There are no Codex, Canadian, or Mexican residue limits for residues of zeta-cypermethrin in or on Brassica, head and stem vegetables; Brassica, leafy vegetables; and leafy vegetables (except Brassica vegetables) group. (Stephaine Willette).

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–6114–3]

Proposed CERCLA Prospective Purchaser Agreement and Proposed CERCLA Section 122(h)(1) Administrative Cost Recovery Settlement Agreement for the Ingram-Richardson Site

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposal of CERCLA Prospective Purchaser Agreement and Proposal of CERCLA section 122(h)(1) Administrative Cost Recovery Settlement Agreement for the Ingram-Richardson site.

SUMMARY: In accordance with the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), 42 U.S.C. 9601 *et seq*., as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA), Pub. L. 99–499, notification is hereby given that a proposed Agreement and Covenant Not to Sue (Agreement) for the Ingram-Richardson Site (the Site) located near Frankfort, in Clinton County, Indiana, has been executed by Clinton County, Indiana (the County), Frankfort Market Place, Inc. (Frankfort Market Place), and Kelly Strange (Mr. Strange). The proposed Agreement has been submitted to the Attorney General for approval. The proposed Agreement would resolve certain potential claims of the United States under sections 106 and 107 of CERCLA, 42 U.S.C. 9606 and 9607, against the County, as the prospective purchaser of the Site. The proposed Agreement also would resolve the potential liability of Frankfort Market Place and Mr. Strange (who are alleged to be past and current owners and operators of the Site) under CERCLA section 107 for certain past response costs incurred in connection with the Site, pursuant to the administrative cost recovery settlement authority conferred by CERCLA section 122(h)(1), 42 U.S.C. 9622(h)(1).

The components of the proposed Agreement relating to the County would require the County to pay $7,500 to the United States and to demolish unusable buildings on the Site before redeveloping the Site for use as a residential treatment center for juveniles. The United States would remove the CERCLA lien currently placed on the Site property.

The components of the proposed Agreement relating to Frankfort Market Place and Mr. Strange provide that: (1) Frankfort Market Place and Mr. Strange will pay $7,500 to the United States, to be applied toward more than $2.789 million in unreimbursed past response costs incurred in connection with removal action undertaken at the Site; (2) Frankfort Market Place and Mr. Strange will convey their ownership interest in the Site to the County, at no cost to the County; and (3) the United States will grant Frankfort Market Place and Mr. Strange a covenant not to sue for past response costs incurred in connection with the removal action (and will dismiss without prejudice a pending, unanswered civil judicial complaint filed by the United States against Frankfort Market Place under CERCLA section 107), and those parties will obtain contribution protection as provided by CERCLA sections 113(f)(2) and 122(h)(4) upon satisfactory completion of their obligations under the Agreement.

The Site is not on the NPL, and no further response activities at the Site are anticipated at this time.

DATES: Comments on the proposed Agreement must be received by July 23, 1998.

ADDRESSES: A copy of the proposed Agreement is available for review at U.S. EPA, Region 5, 77 West Jackson Boulevard, Chicago, Illinois 60604. Please contact Karen Peaceman at (312) 353–5751 prior to visiting the Region 5 office.

Comments on the proposed Agreement should be addressed to Karen Peaceman, Office of Regional Counsel, U.S. EPA, Region 5, 77 West Jackson Boulevard, (Mail Code C–14J), Chicago, Illinois 60604.

FOR FURTHER INFORMATION CONTACT: Karen Peaceman at (312) 353–3751 of the U.S. EPA Region 5 Office of Regional Counsel.

A 30-day period, commencing on the date of publication of this notice, is open for comments on the proposed Agreement. Comments should be sent to the addressee identified in this document.

Doug Ballotti,

Acting Director, Superfund Division, Region #5.

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