hazard evaluation and reduction program and meet the scope of section 405(f)?

2. Which lead hazard reduction products are going to be required to be tested before they can be registered under this program?

There are several factors affecting the first question, such as the cost and size of the program; timeliness of implementation; cost to the government and industry; and acceptance by industry, state regulators, and consumers. The acceptance of a program will depend in part on the expertise of the staff administering the program and the recognition of the organization as an accredited certification/registration body.

Several issues associated with the second question will need to be discussed, such as testing methods, and protocols, development of testing criteria, voluntary consensus standards, cost of testing products, and reciprocity between states.

V. Public Docket

The official record for this notice has been established under docket control number ''OPPTS–00224.'' The record is available for inspection from 12 noon to 4:00 p.m., Monday through Friday, excluding legal holidays. The record is located at: TSCA Docket (7407), Office of Pollution Prevention and Toxics, Environmental Protection Agency, Room E–G99, 401 M St., SW., Washington, DC. 20460.

List of Subjects

Environmental protection.

Dated: September 18, 1997

William H. Saunders III,

Director, Office of Pollution Prevention and Toxics.

[FR Doc.97–25500 Filed 9–22–97; 3:06 p.m.]

Billing Code 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–767; FRL–5748–2]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–767, must be received on or before October 27, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

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

FOR FURTHER INFORMATION CONTACT: By mail: George LaRocca, Registration Division [PM-13], Office of Pesticide Programs, 401 M St., SW., Washington, DC 20460. Office location, telephone number and e-mail address: Rm. 204, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–6100, e-mail: larocca.george@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–767] (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 controlnumber [PF–767] 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: September 19, 1997.

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. AgrEvo USA Company

PP 2F4055, 6F3436, 4F2993, 6F3309

EPA has received a request from AgrEvo USA Company (acting as registered US agent for Hoechst Schering AgrEvo, S. A., Little Falls Centre, 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 removing the time limitation for tolerances established for residues of the insecticides and pyrethroid Deltamethrin and Tralomethrin in or on the following raw agricultural commodities: Deltamethrin - cottonseed at 0.04 parts per million (ppm) and cottonseed oil at 0.2 ppm; and Tralomethrin - broccoli at 0.50 ppm, cottonseed at 0.02 ppm, lettuce, head at 1.00 ppm, lettuce, leaf at 3.00 ppm, soybeans at 0.05 ppm, sunflower seed at 0.05 ppm and cottonseed oil at 0.20 ppm. The IUPAC name for deltamethrin is [(1*R*, 3*R*)-3(2,2-dibromovinyl)-2,2-dimethylcyclopropanecarboxylic acid (*S*)-alpha-cyano-3-phenoxybenzyl ester] and for tralomethrin is [(1*R*, 3*S*)3[(1′ RS)(1′,2′,2′,2′,-tetrabromo-ethyl)]-2,2-dimethylcyclopropane-carboxylic acid (*S*)-alpha-cyano-3-phenoxybenzyl ester]. The tolerances were originally requested in Pesticide Petition Numbers 2F4055, 6F3436, 4F2993, 6F3309. Based on the fact that tralomethrin is rapidly metabolized in plants and animals to deltamethrin, and the toxicological profile of the two compounds is similar, it is appropriate to consider combined exposure assessments for tralomethrin and deltamethrin. EPA has determined that the request 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 permanent tolerance. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Deltamethrin metabolism studies in tomatoes, corn, apples, and cotton demonstrate the same metabolic pathway. Furthermore, plant metabolism studies have been conducted following application of tralomethrin in cotton, corn, cabbage, and tomatoes. These studies have demonstrated that the metabolism of tralomethrin involves debromination to deltamethrin and its isomers. Thus, a similar metabolic pathway has been shown to occur in a variety of crops following either direct application of deltamethrin (cotton, corn, apples, and tomatoes) or in-plant formation of deltamethrin via debromination of applied tralomethrin (tomatoes, cotton, corn, and cabbage). As a result of this substantial information base, it is concluded that the residues of toxicological concern in/on growing crops following application of tralomethrin or deltamethrin are tralomethrin, cis-deltamethrin, and its isomers, trans-deltamethrin and alpha-R-deltamethrin.

2. *Analytical method*. Analytical methods for determining residues of tralomethrin and deltamethrin in the commodities for which registrations have been approved, have been previously submitted to, and reviewed by, the Agency. These methods, based on gas nyhromatography (GLC) equipped with an electron capture detector (ECD) and a DB-1 (or equivalent) capillary column, are used for the determination of tralomethrin, cis-deltamethrin, trans-deltamethrin, and alpha-R-deltamethrin in various raw agricultural, animal derived, and processed commodities. These methods were independently validated and are appropriate for the determination of residues of tralomethrin and deltamethrin in various food and feed commodities after application of these ingredients to target growing crops, and after use in food/feed handling establishments.

3. *Magnitude of residues*. Residues of tralomethrin, deltamethrin, and its metabolites are not expected to exceed the established tolerance levels as a result of the use of these active ingredients on target crops.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral LD_{50} values for deltamethrin in the rat are 66.7 mg/kg for males, 86 mg/kg for females and for tralomethrin 99 mg/kg for males, 157 mg/kg for females when administered in sesame oil. The oral LD_{50} for deltamethrin when administered in aqueous methyl cellulose was greater than 5,000 mg/kg for both sexes. The dermal LD_{50} in rabbits was greater than 2,000 mg/kg for both materials. Inhalation 4–hour LC_{50} values in the rat are 2.2 mg/L for deltamethrin and greater than 0.286 mg/L for tralomethrin.

2. *Genotoxicity*. No indication of genotoxicity was noted in a battery of *in vivo* and *in vitro* studies conducted with either deltamethrin or tralomethrin.

3. *Reproductive and developmental toxicity*— a. *Deltamethrin* A rat developmental toxicity study conducted with deltamethrin indicated a maternal no-observed-effect levels (NOEL) of 3.3 mg/kg/day based on clinical observations, decreased weight gain and mortality. The developmental NOEL was 11 mg/kg/day [highest dose tested] (HDT). In a rabbit developmental toxicity study with deltamethrin, the maternal NOEL was considered to be 10 mg/kg/day based on decreased defecation at 25 and 100 mg/kg/day, and mortality at 100 mg/kg/day. The developmental NOEL was considered to be 25 mg/kg/day based on retarded ossification of the pubic and tail bones at 100 mg/kg (HDT). A three-generation rat reproduction study and a more recent, two-generation rat reproduction study with deltamethrin indicated the NOEL for both parents and offspring was 80 ppm (4–12 mg/kg/day for adults and 18–44 mg/kg/day for offspring) based on clinical signs of toxicity, reduced weight gain and mortality at 320 ppm (HDT).

b. *Tralomethrin*. In a rat developmental toxicity study with tralomethrin the NOEL for maternal and developmental toxicity was judged to be greater than or equal to 18 mg/kg/day (HDT). No evidence of developmental toxicity was observed in either of two rabbit developmental toxicity studies conducted with tralomethrin. In one study, the maternal NOEL was 12.5 mg/kg/day based on mortality while the developmental NOEL was judged to be greater than or equal to 25 mg/kg/day (HDT). In the second study, the maternal NOEL was 8 mg/kg/day based on body weight effects while the developmental NOEL was 32 mg/kg/day (HDT). In a two-generation reproduction study with tralomethrin in rats, the parental NOEL was 0.75 mg/kg/day based on body weight deficits while the NOEL for offspring was 3.0 mg/kg/day, also based on body weight deficits.

4. *Subchronic toxicity*— a. *Deltamethrin*. A 90–day rat oral toxicity study was conducted with deltamethrin which was administered by gavage. The NOEL was judged to be 1.0 mg/kg/day based on reduced body weight gain and slight hypersensitivity. In a more recent 90–day rat dietary study with deltamethrin, the NOEL was judged to be 300 ppm (~23.9 mg/kg/day for males, 30.5 mg/kg/day for females) based on uncoordinated movement, unsteady gait, tremors, increased sensitivity to sound, shakes and spasmodic convulsions. The difference in the NOEL between the two studies is attributed to the different routes of exposure (gavage in oil vs. administered in diet). A 12-week study was conducted with deltamethrin in mice. The NOEL was 300 ppm (~61.5 mg/kg/day in males and 77.0 mg/kg/day in females) based on chronic contractions, convulsions, poor condition, decreased weight gain and mortality. Two 13–week dog studies were conducted with deltamethrin. In the first study, beagle dogs were administered deltamethrin by capsule using PEG 200 as a vehicle. The NOEL for this study was 1 mg/kg/day based on tremors, unsteadiness, jerking movements, salivation, vomiting, liquid

feces and/or dilatation of the pupils. In the second study, deltamethrin was administered by capsule without a vehicle to beagle dogs. The NOEL for this study was 10 mg/kg/day based on unsteady gait, tremors, head shaking, vomiting and salivation. The difference in toxicity between the two studies is attributed to the enhanced absorption resulting from the use of PEG 200 as a vehicle in the first study. A 21–day dermal toxicity study was conducted with deltamethrin in rats. The NOEL for systemic toxicity was determined to be 1,000 mg/kg/day. In a subchronic inhalation study, rats were exposed to aerosolized deltamethrin for 6 hours per day, 5 days per week, for a total of 14 days over 3 weeks. Based on slightly decreased body weights and neurological effects at higher dose levels, it was concluded that 3 µg/l was the NOEL for systemic effects in this study.

b. *Tralomethrin*. Tralomethrin was administrated by gavage in corn oil to rats for 13 weeks. Based on mortality, decreased activity and motor control, soft stools, labored breathing and significantly lower absolute and relative mean liver weights, the NOEL was considered to be 1 mg/kg/day. Tralomethrin was administered by capsule to beagle dogs for 13 weeks. The NOEL for this study was 1.0 mg/kg/day based on refusal of milk supplement, tremors, exaggerated patellar response, unsteadiness and uncoordinated movement. A 21-day dermal toxicity study was conducted with tralomethrin on rats. No systemic effects were observed, therefore, the systemic NOEL for this study was 1,000 mg/kg/day.

5. *Chronic toxicity*— a. *Deltamethrin*. Deltamethrin was administered in the diet to beagle dogs for 2 years. No treatment-related effects were observed and the NOEL was judged to be 40 ppm (~1.1 mg/kg/day). In a more recent study, deltamethrin was administered by capsule (without a vehicle) to beagle dogs for 1–year. The NOEL in this study was considered to be 1 mg/kg/day based on clinical signs, decreased food consumption and changes in several hematology and blood chemistry parameters. Two rat chronic toxicity/ oncogenicity studies were conducted with deltamethrin. In the first study, the test substance was administered via the diet to rats for 2 years. The NOEL for this study was 20 ppm (~1 mg/kg/day) based on slightly decreased weight gain. In a more recent study, deltamethrin was administered to rats in the diet for 2 years. The NOEL for this study was considered to be 25 ppm (~1.1 and 1.5 mg/kg/day for males and females, respectively), based on neurological signs, weight gain effects and increased incidence and severity of eosinophilic hepatocytes and/or balloon cells. No evidence of carcinogenicity was noted in either study. Two mouse oncogenicity studies were conducted with deltamethrin. In the first study, deltamethrin was administered in the diet for 2 years. No adverse effects were observed and the NOEL was judged to be 100 ppm (~12 and 15 mg/kg/day, respectively, for males and females). In a more recent study, deltamethrin was administered in the diet to mice for 97 weeks. The NOEL was considered to be 1,000 ppm (~15.7 and 19.6 mg/kg/day) based on a higher incidence of poor physical condition and a slight transient weight reduction. There was no evidence of oncogenicity in either study.

b. *Tralomethrin*. Tralomethrin was administered to beagle dogs by capsule for 1–year at initial dosages of 0, 0.75, 3.0 and 10.0 mg/kg/day. Due to trembling, ataxia, prostration and convulsions, the high dosage was lowered to 8 mg/kg/day at study week 4 and lowered again to 6 mg/kg/day on study week 14. On the fourteenth week of study, the 0.75 mg/kg/day dosage was raised to 1.0 mg/kg/day. Based on body weight changes, convulsions, tremors, ataxia and salivation, the NOEL for this study was considered to be 1 mg/kg/ day. Tralomethrin was administered by gavage to rats for 24 months. The NOEL for this study was 0.75 mg/kg/day based on salivation, uncoordinated movement, inability to support weight on limbs and decreased body weight parameters. No evidence of carcino-genicity was observed. A 2–year mouse oncogenicity study was conducted with tralomethrin administered by gavage. The NOEL was judged to be 0.75 mg/kg/day based on higher incidences of dermatitis and mortality, salivation, uncoordinated involuntary movements and aggressiveness. No evidence of oncogenicity was observed.

6. *Animal metabolism*— a. *Deltamethrin*. The absorption of deltamethrin appears to be highly dependent upon the route and vehicle of administration. Once absorbed, deltamethrin is rapidly and extensively metabolized and excreted, primarily within the first 48 hours.

b. *Tralomethrin*. Tralomethrin is rapidly metabolized to deltamethrin after debromination. The metabolic pattern of the debrominated tralomethrin is exactly the same as that of the metabolic pattern of deltamethrin.

7. *Neurotoxicity*. Acute delayed neurotoxicity studies in hens were conducted for both deltamethrin and tralomethrin. In both cases, the study results were negative indicating that neither material causes delayed neurotoxicity.

8. *Endocrine effects*. No special studies have been conducted to investigate the potential of deltamethrin or tralomethrin to induce estrogenic or other endocrine effects. However, the standard battery of required toxicity studies has been completed. These studies include an evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. These studies are generally considered to be sufficient to detect any endocrine effects, yet no such effects were detected. Thus, the potential for deltamethrin or tralomethrin to produce any significant endocrine effects is considered to be minimal.

C. Aggregate Exposure

Based on the fact that tralomethrin is rapidly metabolized in plants and animals to deltamethrin, and the toxicological profile of the two compounds is similar, it is appropriate to consider combined exposure assessments for tralomethrin and deltamethrin. Deltamethrin and tralomethrin are broad spectrum insecticides used to control pests of crops, ornamental plants and turf, and domestic indoor and outdoor (including dog collars), commercial, and industrial food use areas. Thus, aggregate non-occupational exposure would include exposures resulting from non-food uses in addition to consumption of potential residues in food and water. Exposure via drinking water is expected to be negligible since deltamethrin binds tightly to soil and rapidly degrades in water.

1. *Dietary exposure*— a. *Food*. Food tolerances have been established (with expiration dates of November 15, 1997), for residues of tralomethrin and/or deltamethrin and its metabolites in or on a variety of raw agricultural commodities. These tolerances, in support of registrations, currently exist for residues of tralomethrin on broccoli, cottonseed, head lettuce, leaf lettuce, soybeans, sunflower seed, and cottonseed oil. Also, such tolerances, in support of registrations, currently exist for deltamethrin on cottonseed and cottonseed oil. Additionally, tolerances which are not time-limited have been established for tralomethrin to support its use in food/feed handling establishments, and for deltamethrin on tomatoes and concentrated tomato products to support the importation of tomato commodities treated with deltamethrin. Further, a food/feed

handling establishment use, and associated tolerances, is pending for deltamethrin. Potential acute exposures from food commodities were estimated using a Tier 3 acute dietary risk assessment (Monte Carlo Analysis) following EPA guidance. Potential chronic exposures from food commodities under the established food and feed additive tolerances for deltamethrin and tralomethrin, plus the pending tolerances for deltamethrin associated with use in food/feed handling areas, were estimated using NOVIGEN's DEEM (Dietary Exposure Evaluation Model). This chronic risk assessment was conducted using anticipated residues based on field trial or monitoring data, percent crop treated, and percent food handling establishments treated.

b. *Drinking water.* Tralomethrin and deltamethrin are immobile in soil and, therefore, will not leach into groundwater. Additionally, due to the insolubility and lipophilic nature of deltamethrin and tralomethrin, 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 (PRZM3). 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 (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.

2. *Non-dietary exposure.* As noted above, deltamethrin and tralomethrin are broad spectrum insecticides registered for use on a variety of food and non-food agricultural commodities. Additionally, registrations are held for non-agricultural applications including turf and lawn care treatments, broadcast carpet treatments (professional use only), indoor fogger, spot, crack and crevice treatments, insect baits, lawn and garden sprays and indoor and outdoor residential, industrial and institutional sites including those for Food/Feed Handling Establishments.

To evaluate non-dietary exposure, the ''flea infestation control'' senario was chosen to represent a plausible but worst case non-dietary (indoor and outdoor) non-occupational exposure. This scenario provides a situation where deltamethrin and/or tralomethrin is commonly used and they can be used concurrently for a multitude of uses, e.g., spot and/or broadcast treatment of infested indoor surfaces such as carpets and rugs, treatment of pets and treatment of the lawn. This hypothetical situation provides a very conservative, upper bound estimate of potential non-dietary exposures. Consequently, if health risks are acceptable under these conditions, the potential risks associated with other more likely scenarios would also be acceptable.

Because tralomethrin is rapidly metabolized to deltamethrin, and the toxicology profiles of deltamethrin and tralomethrin are virtually identical, a non-dietary and aggregate (non-dietary + chronic dietary) exposure/risk assessment has been conducted for the combination of both active ingredients. The total exposure to both materials was expressed as ''deltamethrin equivalents'' and these were compared to the toxicology endpoints identified for deltamethrin.

C. Cumulative Effects

When considering a tolerance, the Agency must consider ''available information'' concerning the cumulative effects of a particular pesticides residues and ''other substances that have a common mechanism of toxicity''. AgrEvo USA Company, acting as registered US agent for Hoechst Schering AgrEvo SA, believes that ''available information'' in this context includes not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments.

Further, AgrEvo does not have, at this time, available data to determine whether tralomethrin and/or deltamethrin have a common mechanism of toxicity with other substances. For the purposes of this tolerance action, therefore, no assumption has been made that tralomethrin and/or deltamethrin have a common mechanism of toxicity with other substances.

AgrEvo USA Company, acting as registered US agent for Hoechst Schering AgrEvo SA, will submit information for EPA to consider concerning potential cumulative effects of deltamethrin and/or tralomethrin consistent with the schedule established by EPA at 62 FR 42020 (August 4, 1997,) and other EPA publications pursuant to the Food Quality Protection Act (FQPA).

D. Safety Determination

1. *U.S. population.* The toxicity and residue data base for deltamethrin and tralomethrin are considered to be valid, reliable and essentially complete according to existing regulatory requirements. No evidence of oncogenicity has been observed for either compound. For acute exposures, the toxicology endpoint from the deltamethrin rat development toxicity study, 3.3 mg/kg/day, is used. For chronic exposures to deltamethrin and tralomethrin, the Reference Dose (RfD) of 0.01 mg/kg bodyweight/day established for deltamethrin based on the NOEL from the 2–year rat feeding study and a 100-fold safety factor to account for interspecies extrapolation and intraspecies variation is used.

For the overall U.S. population, acute dietary exposure at the 99.9th percentile results in a Margin of Exposure (MOE) of 5,382; the MOE for the 99th percentile is 16,661; and at the 95th percentile the MOE is 57,470. For the overall US population, chronic dietary exposure results in a utilization of 0.2 percent of the reference dose. Using an upper bound estimate of potential non-dietary exposures for a worst case scenario (flea treatment) results in an MOE of 160,000 for adults. Utilizing the scenario of chronic dietary exposure plus an upper bound estimate of potential non-dietary exposure from a worst case scenario (flea treatment), it is shown that for aggregate exposure to deltamethrin and tralomethrin there is an MOE of 83,000 for adults. There is generally no concern for MOE greater than 100. For chronic exposure, there is generally no concern for exposure below 100 percent 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.

In conclusion, there is reasonable certainty that no harm will result to the U.S. population, in general, from dietary or aggregate exposure to either deltamethrin and/or tralomethrin.

2. *Infants and children.* Data from developmental toxicity studies in rats and rabbits, and multigeneration reproduction studies in rats are generally used to assess the potential for increased sensitivity of infants and children. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism

resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to reproductive and other effects on adults and offspring from pre-natal and post-natal exposure to the pesticide. None of these studies conducted with deltamethrin or tralomethrin indicated developmental or reproductive effects as a result of exposure to these materials.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects in children is complete. Although no indication of increased susceptibility to younger animals was noted in any of the above studies, or in the majority of studies with other pyrethroids, several recent publications have reported that deltamethrin is more toxic to neonate and weanling animals than to adults. However, a joint industry group currently investigating this issue was unable to reproduce these findings. Furthermore, the RfD (0.01 mg/kg/day) that has been established for deltamethrin is already more than 1,000-fold lower than the lowest NOEL from the developmental and reproduction studies. Therefore, the RfD of 0.01 mg/kg/day is appropriate for assessing chronic aggregate risk to infants and children and an additional uncertainty factor is not warranted. Also, the NOEL of 3.3 mg/kg/day from the rat developmental toxicity study is appropriate to use in acute dietary, short term non-dietary, and aggregate exposure assessments.

For the population subgroup described as non-nursing infants, less than 1 year old, the MOE for acute dietary exposure at the 99.9th percentile is 13,853; at the 99th percentile the MOE is 74,022; and at the 95th percentile the MOE is 663,629. For the population subgroup described as children 1–6 years old, the MOE for acute dietary exposure is 2,300 for the 99.9th percentile; at the 99th percentile the MOE is 10,409; and at the 95th percentile the MOE is 42,070. For non-nursing infants, chronic dietary exposure results in a utilization of 0.3 percent of the reference dose, and for children 1–6 years old 0.4 percent of the reference dose is utilized. Using an upper bound estimate of potential non-dietary exposures for a worst case scenario (flea treatment) results in an MOE of 6,100 for infants less than 1 year old, and an MOE of 6,600 for children 1–6 years old. Utilizing the scenario of chronic dietary exposure plus an upper bound estimate of potential non-dietary exposure from a worst case scenario (flea treatment) it is shown that for aggregate exposure to deltamethrin and tralomethrin, there is an MOE of 5,800 for infants less than 1–year old, and an MOE of 6,100 for children 1-6 years old. There is generally no concern for MOE s greater than 100. For chronic exposure, there is generally no concern for exposure below 100 percent 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.

In summary, there is reasonable certainty that no harm will result to infants and children from aggregate exposure to either deltamethrin or tralomethrin.

E. International Tolerances

The proposed/established CODEX maximum residue levels (MRL) and for deltamethrin are as follows: cotton at 0.05 ppm and food/feed handling uses at 0.05 ppm. As far as can be determined at this time, no CODEX MRL's are established or proposed for tralomethrin.

F. Conclusions

The existing tolerances for deltamethrin and tralomethrin do not pose a significant risk to human health, including that of children, and are in compliance with the requirements of the FQPA of 1996. Therefore, the time limitations associated with these tolerances can be removed. (John Hebert)

2. Bayer Corporation

PP 4F3046, 9F3731, 3F4204, 4F4309, 4F4313, 2F4137, 4H5427, 9H5574, 3H5670, 4H5686, 4H5687

EPA has received a request regarding pesticide petitions (PP 4F3046, 9F3731, 3F4204, 4F4309, 4F4313, 2F4137, 4H5427, 9H5574, 3H5670, 4H5686, 4H5687) from Bayer Corporation, 8400 Hawthorn Road, P.O. Box 4913, Kansas City, MO 64210 to remove the time limitations on the established tolerances at 40 CFR § 180.436, § 185.1250 and § 186.1250 for the insecticide cyfluthrin, [cyano[4-fluoro-3-phenoxyphenyl]-methyl-3-[2,2-dicloroethenyl]-2,2-dimethyl- cyclopropanecarboxylate] in or on the raw agricultural commodities alfalfa, forage, at 5.0 ppm; alfalfa, hay, at 10.0 ppm; aspirated grain fractions at 300 ppm; carrots at 0.2 ppm; cattle, fat, at 1.0 ppm; cattle, meat, at 0.4 ppm; cattle, meat by-products (mbyp) at 0.4 ppm; corn, forage (sweet), at 15.0 ppm; corn, fodder (sweet), at 30 ppm; corn (sweet, K+CWHR), at 0.05 ppm; cottonseed at 1.0 ppm; cottonseed, oil, at 2.0 ppm; cottonseed, hulls, at 2.0 ppm; citrus, whole fruit, at 0.2 ppm; citrus oil, at 0.3 ppm; citrus dried pulp, at 0.3 ppm; eggs at 0.01 ppm; goats, fat, at 1.0 ppm; goats, meat, at 0.4 ppm; goats, meat by-products (mbyp) at 0.4 ppm; hogs, fat, at 1.0 ppm; hogs, meat, at 0.4 ppm; hogs, meat by-products (mbyp) at 0.4 ppm; horses, fat, at 1.0 ppm; horses, meat, at 0.4 ppm; horses, meat by-products (mbyp) at 0.4 ppm; milkfat, at 15.0 ppm (representing 0.5 ppm in whole milk); peppers, at 0.5 ppm; poultry, fat, at 0.01 ppm; poultry, meat, at 0.01 ppm; poultry, meat by-products (mbyp) at 0.01 ppm; radishes at 1.0 ppm; sheep, fat, at 1.0 ppm; sheep, meat, at 0.4 ppm; sheep, meat by-products (mbyp) at 0.4 ppm; sorghum, fodder, at 5.0 ppm; sorghum, forage, at 2.0 ppm; sorghum, grain at 4.0 ppm, sunflower, forage, at 1.0 ppm; sunflower, seed, at 0.02 ppm; sugarcane, at 0.05 ppm; sugarcane, molasses, at 0.2 ppm; tomatoes, at 0.2 ppm; tomato, concentrated products, at 0.5 ppm; and tomato, pomace (wet and dry) at 5.0 ppm. All data requested by EPA have been submitted. Therefore, a request for unconditional registration and removal of the time limitations on established tolerances is being made.

Consistent with section 408(d) of FFDCA, as recently amended by the Food Quality Protection Act, Bayer submitted a summary and authorization for the summary to be published in the **Federal Register** in a notice of receipt of the request. The summary represents the views of Bayer; EPA is in the process of evaluating the request. Consistent with section 408(d)(3), EPA is including the summary as a part of this notice of filing. EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting the request.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of cyfluthrin in plants is adequately understood. Studies have been conducted to delineate the metabolism of radio labeled cyfluthrin in various crops all showing similar results. The residue of concern is cyfluthrin.

2. *Analytical method*. Adequate analytical methodology (gas/liquid chromatography with an electron capture detector) is available for enforcement purposes.

3. *Magnitude of residues*. Cyfluthrin is the active ingredient in the registered end-use product Baythroid 2 Emulsifiable Pyrethroid Insecticide, EPA Reg. No. 3125–351. Baythroid 2 is registered for use on alfalfa, carrots,

citrus, cotton, peppers, radishes, sorghum, sugarcane, sweet corn, sunflowers and tomatoes.

Tolerances to support these uses were proposed in pesticide petitions 4F3046, 9F3731, 3F4204, 4F4309, 4F4313, 2F4137, and 4F4313 and food/feed additive petitions 4H5427, 9H5574, 3H5670, 4H5686, and 4H5687. Residue data covering all the uses associated with these petitions have been previously submitted to EPA for review and have been found by EPA to support the establishment of the tolerances. Consequently, regulations establishing these tolerances were promulgated in response to these petitions. See [53 FR 30676] (cottonseed), [60 FR 28353] (carrots, radishes, peppers and tomatoes), [60 FR 28353] (sugarcane), [61 FR 10678] (alfalfa, sunflowers, and sweet corn), [61 FR 39883] (sorghum), and [62 FR 25518] (citrus).

B. Toxicological Profile

The database for cyfluthrin is current and complete. Toxicology data cited in support of these tolerances include:

1. *Acute toxicity*. There is a battery of acute toxicity studies for cyfluthrin supporting an overall toxicity Category II.

2. *Genotoxicty*. Mutagenicity tests were conducted, including several gene mutation assays (reverse mutation and recombination assays in bacteria and a Chinese hamster ovary (CHO)/HGPRT assay); a structural chromosome aberration assay (CHO/sister chromatid exchange assay); and an unscheduled DNA synthesis assay in rat hepatocytes. All tests were negative for genotoxicity.

3. *Reproductive and developmental toxicity*. An oral developmental toxicity study in rats with a maternal and fetal NOEL of 10 milligrams/kilogram of body weight/day (mg/kg bw/day) (highest dose tested).

An oral developmental toxicity study in rabbits with a maternal NOEL of 20 mg/kg bw/day and a maternal Lowest Effect Level (LEL) of 60 mg/kg bw/day, based on decreased body weight gain and decreased food consumption during the dosing period. A fetal NOEL of 20 mg/kg bw/day and a fetal LEL of 60 mg/kg bw/day were also observed in this study. The LEL was based on increased resorptions and increased postimplantation loss.

A three-generation reproduction study in rats with systemic toxicity NOELs of 7.5 and 2.5 mg/kg bw/day for parental animals and their offspring, respectively. At higher dose levels, the body weights of parental animals and their offspring were reduced.

4. *Subchronic toxicity*. A subchronic toxicity feeding study using rats demonstrated a NOEL of 22.5 mg/kg bw/day, the highest dose tested.

A 6–month toxicity feeding study in dogs established a NOEL of 5 mg/kg bw/day. The LEL was 15 mg/kg bw/day based on clinical signs and reduced thymus weights.

5. *Chronic toxicity*. A 12–month chronic feeding study in dogs established a NOEL of 4 mg/kg bw/day. The lowest effect level (LEL) for this study is established at 16 mg/kg bw/day, based on slight ataxia, increased vomiting, diarrhea and decreased body weight.

A 24–month chronic feeding/carcinogenicity study in rats demonstrated a NOEL of 2.5 mg/kg bw/day and LEL of 6.2 mg/kg bw/day, based on decreased body weights in males, decreased food consumption in males, and inflammatory foci in the kidneys in females.

A 24–month carcinogenicity study in mice was conducted. Under the conditions of the study there were no carcinogenic effects observed. A 24–month chronic feeding/carcinogenicity study in rats was conducted. There were no carcinogenic effects observed under the conditions of the study.

6. *Animal metabolism*. A metabolism study in rats showed that cyfluthrin is rapidly absorbed and excreted, mostly as conjugated metabolites in the urine, within 48 hours. An enterohepatic circulation was observed.

7. *Metabolite toxicology*. No toxicology data have been required for cyfluthrin metabolites. The residue of concern is cyfluthrin.

8. *Endocrine effects*. There is no evidence of endocrine effects in any of the studies conducted with cyfluthrin, thus, there is no indication at this time that cyfluthrin causes endocrine effects.

C. Aggregate Exposure

1. *Dietary exposure— Food*. Dietary exposure was estimated using Novigen's Dietary Exposure Evaluation Model (DEEMa) software; results from field trial and processing studies; consumption data from the USDA Continuing Surveys of Food Intake by Individuals (CSFIIs), conducted from 1989 through 1992; and information on the percentages of the crop treated with Cyfluthrin.

Cyfluthrin is registered for use in alfalfa, citrus, sweet corn, cotton, sorghum, sunflower, sugarcane, carrots, peppers, radishes and tomatoes. In addition, it has an import tolerance for hops. Various formulations are registered for use in food handling establishments and in combination with another active ingredient, for use in field corn, pop corn and sweet corn.

Chronic dietary exposure estimates for the overall U.S. population were 0.5% of the Reference dose (RfD) (0.008 mg/kg bw/day). For the most highly exposed population subgroup, children 1 to 6 years of age, the exposure was estimated to be 0.000062 mg/kg bw/day, or 0.8% of the RfD. Acute dietary exposures were estimated for the overall US population, females 13 years and older, children, ages 1–6 and 7–12 years, infants, non-nursing and nursing. The exposure was compared to the NOEL of 20 mg/kg bw/day to estimate the Margins of Exposures (MOEs).

For the overall U.S. population the 95th, 99th and 99.9th percentile of exposure the MOEs were calculated as 29,981; 9,519; and 3,658 respectively.

For women aged 13 years and older the 95th, 99th and 99.9th percentile of exposure the MOEs were calculcated as 45,996; 20,103 and 10,011 respectively.

Lastly, for the potentially highest exposed population subgroup, non-nursing infants, the 95th, 99th and 99.9th percentile of exposure to the MOEs were calculated at 16,107; 3,072; and 1,343, respectively.

2. *Drinking water*. Cyfluthrin is immobile in soil, therefore, will not leach into groundwater. Additionally, due the insolubility and lipophilic nature of cyfluthrin, 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 (PRZM3). Based on this screening assessment, the potential concentrations of a pyrethroid in ground water at 2 meters are essentially zero (much less than 0.001 parts 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 52 parts per trillion. Concentration in actual drinking water would be much lower. Based on these analyses, the contribution of water to the dietary risk estimate is negligible.

3. *Non-dietary exposure*. Non-occupational exposure to cyfluthrin may occur as a result of inhalation or contact from indoor residential, indoor commercial, and outdoor residential uses. Pursuant to the requirements of FIFRA as amended by the Food Quality Protection Act of 1996, non-dietary and aggregate risk analyses for cyfluthrin

were conducted. The analyses include evaluation of potential non-dietary acute application and post-application exposures. Non-occupational, non-dietary exposure was assessed based on the assumption that a flea infestation control scenario represents a ''worst case'' scenario. For the flea control infestation scenario indoor fogger, and professional residential turf same day treatments were included for cyfluthrin. Deterministic (point values) were used to present a worse case upper-bound estimate of non-dietary exposure. The non-dietary exposure estimates were expressed as systemic absorbed doses for a summation of inhalation, dermal, and incidental ingestion exposures. These worst-case non-dietary exposures were aggregated with chronic dietary exposures to evaluate potential health risks that might be associated with cyfluthrin products. The chronic dietary exposures were expressed as an oral absorbed dose to combine with the non-dietary systemic absorbed doses for comparison to a systemic absorbed dose (NOEL). Results for each potential exposed subpopulation (of adults, children 1–6 years, and infants <1 year) were compared to the systemic absorbed dose NOEL for cyfluthrin to provide estimates of MOE.

The large MOEs for cyfluthrin clearly demonstrate a substantial degree of safety. The total non-dietary MOEs are 3,800, 2,700, and 2,500 for adults, children (1–6 years), and infants (<1 year), respectively. The aggregate MOE for adults is approximately 3,800 and the MOEs for infants and children exceed 2,500.

The non-dietary methods used in the analyses can be characterized as highly conservative. This is due to the conservatism inherent in the calculation procedures and input assumptions. An example of this is the conservatism inherent in the jazzercise over representation of residential post-application exposures. It is important to acknowledge that these MOEs are likely to significantly underestimate actual MOEs due to a variety of conservative assumptions and biases inherent in the derivatization of exposure by this method. Therefore, it can be concluded that large MOEs associated with potential non-dietary and aggregate exposures to cyfluthrin will result in little or no health risks to exposed persons. The aggregate risk analysis demonstrates compliance with the health-based requirements of the Food Quality Protection Act of 1996 and supports the continued registration and use of residential, commercial, and agricultural products containing cyfluthrin.

D. Cumulative Effects

Further, Bayer does not have, at this time, available data to determine whether cyfluthrin has a common mechanism of toxicity with other substances. For the purposes of this tolerance action, therefore, no assumption has been made that cyfluthrin has a common mechanism of toxicity with other substances.

Bayer will submit information for EPA to consider concerning potential cumulative effects of cyfluthrin consistent with the schedule established by EPA in the **Federal Register** of August 4, 1997, (62 FR 42020) and other EPA publications pursuant to the Food Quality Protection Act.

E. Safety Determination

1. *U.S. population*. Based on the exposure assessments described above and on the completeness and reliability of the toxicity data, it can be concluded that total aggregate exposure to cyfluthrin from all uses will utilize less than 1% percent of the RfD for chronic dietary exposures and that MOEs in excess of 1,000 exist for aggregate exposure to cyfluthrin for non-occupational exposure. EPA generally has no concerns for exposures below 100 percent 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. Margins of exposure of 100 or more (300 for infants and children) also indicate an adequate degree of safety. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to cyfluthrin residues.

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

The toxicology data which support these tolerances include:

An oral developmental toxicity study in rats with a maternal and fetal NOEL of 10 mg/kg bw/day (HDT).

An oral developmental toxicity study in rabbits with a maternal NOEL of 20 mg/kg bw/day and a maternal LEL of 60 mg/kg bw/day, based on decreased body weight gain and decreased food consumption during the dosing period. A fetal NOEL of 20 mg/kg bw/day and a fetal LEL of 60 mg/kg bw/day were also observed in this study. The LEL was based on increased resorptions and increased postimplantation loss.

An oral developmental toxicity study performed with beta-cyfluthrin, the resolved isomer mixture of cyfluthrin, has been submitted to the Agency and is currently under review.

A developmental toxicity study in rats exposed via inhalation to liquid aerosols of cyfluthrin revealed developmental toxicity, but only in the presence of maternal toxicity. The developmental NOEL was 0.46 mg/m^3 on the basis of reduced placental and fetal weights, and delayed ossification. The NOEL for overt maternal toxicity was < 0.46 mg/m^3, the lowest dose tested (LDT).

A three-generation reproduction study in rats with systemic toxicity NOELs of 7.5 and 2.5 mg/kg bw/day for parental animals and their offspring, respectively. At higher dose levels, the body weights of parental animals and their offspring were reduced. Another multiple-generation reproduction study in rats has been submitted to the Agency and is currently under review.

The Agency used the rabbit developmental toxicity study with a maternal NOEL of 20 mg/kg bw/day to assess acute dietary exposure and determine a MOE for the overall U.S. population and certain subgroups. Since this toxicological endpoint pertains to developmental toxicity the population group of concern for this analysis was women aged 13 and above, the subgroup which most closely approximates women of child-bearing age. The MOE is calculated as the ratio of the NOEL to the exposure. The Agency calculated the MOE to be over 600. Generally, MOE's greater than 100 for data derived from animal studies are regarded as showing no appreciable risk.

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.

The results of the three-generation study in rats provided evidence suggesting that, with respect to effects of cyfluthrin on body weight, pups were more sensitive than adult rats. Thus, the Agency determined that an additional 3–fold uncertainty factor (UF) should be used in risk assessments to ensure adequate protection of infants and children.

Generally, EPA considers MOEs of at least 100 to indicate an adequate degree of safety. With an additional 3× uncertainty factor, this would be 300 for infants and children. Using the exposure assessments described above and based on the described toxicity data aggregate exposure to infants and children indicate a MOE in excess of 2,500. Thus, it can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to cyfluthrin residues.

3. *Conclusions.* The available data indicate that there is reasonable certainty of no harm from the aggregate exposure from all currently registered uses of cyfluthrin. Thus, consistent with the provisions of the FFDCA as amended August 3, 1996, the time limitations on established cyfluthrin tolerance should be removed.

F. International Tolerances

Codex maximum residue levels (MRLs) are established for residues of cyfluthrin on milk (0.01 mg/kg); cottonseed (0.05 mg/kg); peppers, sweet (0.2 mg/kg); and tomatoes (0.5 mg/kg). (Stephanie Willett)

3. DuPont Agricultural Products

PP–7F2013

EPA has received a request from DuPont Agricultural Products, P. O. Box 80038, Wilmington, DE 19880–0038 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by removing the time limitation for a tolerance established for residues of the insecticide and pyrethroid fenvalerate, including the *s,s*-enriched isomer esfenvalerate (Asana® XL Insecticide), ((*S*)-cyano-(3-phenoxyphenyl)methyl (*S*)-4-chloro-alpha-(1-methylethyl)benzeneacetate in or on the raw agricultural commodity cottonseed at 0.2 parts per million (ppm). The tolerance was originally requested in PP–7F2013. EPA has determined that the request 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 and chemical nature of residues of fenvalerate in plants is 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.

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.

3. *Magnitude of residues*. Tolerances are based on the sum of all isomers of fenvalerate. Fenvalerate is a racemic mixture of four isomers (about 25% each). This product was registered as Pydrin® . However since 1992, an *S,S*-isomer enriched formulation, Asana® (esfenvalerate), has been the only fenvalerate formulation sold in the U.S. for agricultural use. Since the *S,S*-isomer is the insecticidally active isomer, the use rate for Asana® is four times lower than that for Pydrin® . A petition is pending (PP–4F4329), to convert tolerances (still to be expressed as the sum of all isomers) based on the use rates for Asana®. Bridging residue studies have shown Asana® residues to be 3–4 times lower than Pydrin® residues.

EPA has established a tolerance of 0.2 ppm for fenvalerate on cottonseed. Magnitude of residue and processing studies support this tolerance. This request is for the removal of the time limitation currently imposed on the tolerance of 0.2 ppm for fenvalerate on cottonseed.

B. Toxicological Profile

The following studies have been submitted to EPA:

1. *Acute toxicity*. A rat acute oral study on esfenvalerate technical has an LD_{50} of 87.2 milligram (mg)/kilogram (kg). A rabbit acute dermal study on esfenvalerate has an LD_{50} of >2,000 mg/kg. Acute inhalation on technical grade active ingredient (a.i.) waived due to negligible vapor pressure. A primary eye irritation test using esfenvalerate in the rabbit showed mild irritation (conjunctivitis) that cleared by day 7. A primary dermal irritation test using esfenvalerate in the rabbit which showed minimal irritation that reversed within 72 hours after treatment. A dermal sensitization test on esfenvalerate in guinea pigs which showed no sensitization.

2. *Genotoxicty*. Esfenvalerate was not mutagenic in reverse mutation assays in Salmonella and *E. Coli in vitro* assay in Chinese hamster lung cells. Esfenvalerate did not induce chromosome aberrations in an *in vitro* assay 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.

3. *Reproductive and developmental toxicity*. In a pilot developmental study in the rat with doses of 0, 1, 2, 3, 4, 5, and 20 mg/kg/day esfenvalerate maternal clinical signs of abnormal gait or mobility occurred at 4 mg/kg/day and above. In a developmental study in the rat with doses of 0, 2.5, 5, 10, and 20 mg/kg/day esfenvalerate by gavage maternal signs observed at 2.5 mg/kg/day were erratic jerking and extension of forelimbs, rapid side-to-side head movement, and excessive grooming. There was no maternal No-Observed-Effect-Level (NOEL) in the main study but a NOEL of 2 mg/kg/day was established on the pilot study. There were no fetal or developmental effects in either study at 20 mg/kg/day, the highest dose tested. Therefore, the fetal/developmental NOEL was ≧20 mg/kg/day.

In a pilot developmental study in the rabbit with doses of 0, 2, 3, 4, 4.5, 5, and 20 mg/kg/day esfenvalerate by gavage. The maternal NOEL was 2 mg/kg/day based on excessive grooming at 3 mg/kg/day and above. In a developmental study in the rabbit with doses of 0, 3, 10, and 20 mg/kg/day esfenvalerate by gavage there was no maternal NOEL in the main study, but a maternal NOEL of 2 mg/kg/day was established in the pilot study. There were no fetal or developmental effects in either study at the highest dose tested. Therefore, the fetal/developmental NOEL was ≧20 mg/kg/day.

A two-generation feeding study with esfenvalerate in the rat at dietary levels of 0, 75, 100, or 300 ppm. The high dietary concentration was lowered to 150 ppm for the second generation. Very mild body weight effects and sores at 75 ppm in both generations were considered secondary effects caused by scratching related to skin stimulation from dermal exposure. Therefore 75 ppm (4.2 mg/kg/day for first generation parental males, 5.6 mg/kg/day for first generation parental females, 6.0 mg/kg/day for second generation parental males, and 7.3 mg/kg/day for second generation parental females) was considered an No-Observed-Adverse-Effect-Level (NOAEL) for both adult rats and their offspring. Effects were observed in adults and pups of both generations at 100 ppm and above. Pups

were no more sensitive than adult animals.

4. *Subchronic toxicity*. A 90–day feeding study in rats was conducted at 0, 75, 100, 125, and 300 ppm esfenvalerate with a NOEL of 125 ppm (6.3 mg/kg/day). This study provided intermediate dose levels to supplement a 90–day feeding study in rats conducted at 0, 50, 150, 300 and 500 ppm esfenvalerate with a NOEL of 50 ppm (2.5 mg/kg/day) based on jerky leg movements 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 and 2,000 ppm fenvalerate with a NOEL of 50 ppm esfenvalerate (10.5 mg/kg/day) based on lower glucose and triglycerides at 150 ppm. Neurologic symptoms were observed with 500 ppm esfenvalerate and 2,000 ppm fenvalerate.

A 3–month subchronic study in dogs is satisfied by a 1–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 was conducted at 100, 300, and 1,000 mg/kg/day with an NOAEL of 1,000 mg/kg/day.

5. *Chronic toxicity*. In a 1–year study in which 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 the 2–week pilot study used to select dose levels for the chronic-dog study.

In a 20–month study with fenvalerate in mice fed 0, 10, 30, 100, and 300 ppm the NOEL was 30 ppm (~ 6 mg/kg/day) based on red blood cell effects and granulomatous changes at 100 ppm. Fenvalerate was not carcinogenic at any concentration.

In a 18–month study in mice fed 0, 35, 150, and 350 ppm esfenvalerate. Mice fed the 350 ppm dose were sacrificed within the first two months of the study, after excessive morbidity and mortality due to self-trauma induced by pharmacological effects related to skin stimulation. Therefore, data collected from the 350 ppm group 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 study with fenvalerate in rats fed 1, 5, 25, and 250 ppm a 1,000 ppm group was added to establish an effect level. The NOEL was 250 ppm (12.5 mg/kg/day). At 1,000 ppm, hind limb weakness, lower body weight, and higher organ-to-body weight ratios were observed. Fenvalerate was not carcinogenic at any concentration.

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. *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.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing dietary exposure, chronic and acute dietary assessments have been conducted using all existing and pending tolerances for esfenvalerate. The toxicological endpoints used in both dietary assessments are derived from maternal NOEL's of 2.0 mg/kg/day from rat and rabbit teratology studies. There were no fetal effects in these studies.

2. *Food*. A chronic dietary exposure assessment using anticipated residues and monitoring data and adjusting for percent crop treated, found the percentages of the Reference Dose (RfD) utilized by the most sensitive sub-population (children 1–6 years) to be 5.2%. Chronic exposure for the overall U.S. population was 2.1% of the RfD. This assessment included pending tolerances and all food tolerances for incidental residues from use in food handling establishments.

A Tier 3 acute dietary assessment indicated the most sensitive sub-population was children 1–6 years with Margin of Exposures (MOEs) of 352, 200, and 103 at the 95th, 99th, and 99.9th percentile of exposure, respectively. The MOEs for nursing infants are 410, 199, and 151 at the 95th, 99th, and 99.9th percentile of exposure, respectively. The MOEs for non-nursing infants are 661, 270, and 134 at the 95th, 99th, and 99.9th percentile of exposure, respectively. The MOEs for the general population are 742, 352, and 170 at the 95th, 99th, and 99.9th percentile of exposure, respectively. This analysis used field trial data to estimate exposure and market share information for the percent of crop treated. It used Monte Carlo modeling and appropriate processing factors for processed food and distribution analysis. Food handling establishment commodities are not relevant to this type of analysis and EPA methodology does not include them in Tier 3 exposure modeling.

3. *Drinking water*. Esfenvalerate is immobile in soil and, therefore, will not leach into groundwater. Additionally, 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 (PRZM3). 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 (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 pound model since drinking water derived from surface water would be treated before consumption. Based on these analyses, the contribution of water to the dietary risk estimate is negligible.

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 (1.5×10^{-9} millimeters (mm) Mercury at 25°C) and low dermal penetration, would result in minimal inhalation and dermal exposure.

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 pet spray product assessment indicated MOEs of 740,000, 2,600, and 2,500 for adults, children 1–6 years, and children < 1 year, respectively. The carpet and room treatment assessment indicated MOEs of 110,000, 4,500, and 4,200 for adults, children 1–6 years, and children < 1 year, respectively. The lawn care assessment indicated MOEs of 700,000, 26,000, and 24,000 for adults, children 1–6 years, and children < 1 year, respectively.

5. *Aggregate exposure— Dietary and non-dietary*. Based on the toxicity endpoints selected for esfenvalerate, absorbed doses were combined and compared to the relevant systemic NOEL for estimating MOEs.

The non-dietary risk analysis MOEs combined with the chronic dietary risk analysis MOEs indicated aggregate MOEs of 4,400, 860, and 1,000 for adults, children 1–6 years, and children < 1 year, respectively.

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. The aggregate risk analyses demonstrate compliance with the health-based requirements of the Food Quality Protection Act of 1996 (FQPA) (7 U.S.C. 136 note) and supports the continued registration and use of residential, agricultural, and commercial products containing this a.i.

D. Cumulative Effects

Section 408(b)(2)(D)(v) of the FFDCA 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''. At this time, available methodologies do not exist to resolve the complex scientific issues concerning common mechanism of toxicity of pyrethroids in a meaningful way. DuPont intends to submit information for EPA to consider concerning potential cumulative effects of esfenvalerate consistent with the schedule established by EPA at 62 FR 42020 (August 4, 1997)(FRL–5734–6) and other EPA publications pursuant to the FQPA.

In consideration of potential cumulative effects of esfenvalerate 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 esfenvalerate would be cumulative with those of other chemical compounds. In addition, since esfenvalerate does not appear to produce a toxic metabolite produced by other substances; only the potential risks of esfenvalerate have been considered in this assessment of its aggregate exposure.

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.

1. *U.S. population*. A chronic dietary exposure assessment using anticipated residues, monitoring information, and percent crop treated indicated the percentage of the RfD utilized by the general population to be 2.1%. 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 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 742, 352, 170 at the 95th, 99th, and 99.9th 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.

The aggregate exposure to use of esfenvalerate as pet spray, carpet treatment, lawn care, and in the diet indicated an MOE of 4,400 for adults.

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*. A chronic dietary exposure assessment found the percentages of the RfD utilized by the most sensitive sub-population to be 5.2% for children 1–6 years. The acute dietary exposure assessment found the most sensitive sub-population to be children 1–6 years with MOEs of 352, 200, and 103 at the 95th, 99th, and 99.9th percentile of exposure, respectively. Nursing infants had MOEs of 410, 199, and 151 at the 95th, 99th, and 99.9th percentile of exposure, respectively. Non-nursing infants had MOEs of 661, 270, and 134 at the 95th, 99th, and 99.9th percentile of exposure, respectively. The aggregate exposure to use of esfenvalerate as pet spray, carpet treatment, lawn care, and in the diet indicated an MOE of 860 for children 1–6 years and an MOE of 1,000 for children < 1 year.

Thus, there is reasonable certainty that no harm to infants and children will result from chronic dietary, acute dietary, non-dietary, or aggregate exposure to esfenvalerate residues.

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. Several of these MRL's are different than the proposed U.S. tolerances for esfenvalerate. Therefore, some harmonization of these maximum residue levels is desirable. (John Hebert)

4. FMC Corporation

PP 2F2623, 4F2986, 3F2824, 7F3498, and 4F3011

EPA has received a request regarding pesticide petitions (PP 2F2623, 4F2986, 3F2824, 7F3498, and 4F3011) from FMC Corporation, 1735 Market Street, Philadelphia, PA 19103. The request proposes to remove any time limitations on established tolerances for residues of the insecticide zeta-cypermethrin (*s*-Cyano(3-phenoxyphenyl)methyl (±) *cis, trans* 3-(2,2-dichloroethenyl)-2,2-dimethylcyclopropanecarboxylate) in or on the raw agricultural commodities cottonseed at 0.5 ppm, pecans 0.05 ppm, lettuce, head at 10.0 ppm, onions, bulb at 0.10 ppm and cabbage at 2.0 ppm (established at 40 CFR 180.418). These tolerances were established under (PP) 2F2623, 4F2986, 3F2824, 7F3498, and 4F3011. EPA has determined that the request 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 requests. Additional data may be needed before EPA rules on the requests.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of cypermethrin in plants is adequately understood. Studies have been conducted to delineate the metabolism of radiolabelled 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 cotton, pecans, head lettuce, bulb onions, and cabbage show that the established cypermethrin tolerances on cottonseed of 0.5 ppm, pecans 0.05 ppm, lettuce, head at 10.0 ppm, onions, bulb at 0.10 ppm and cabbage at 2.0 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 NOEL of 0.5 mg/kg/day based on the NOEL of 1.0 mg/kg/day from the cypermethrin chronic toxicity study in dogs and a correction factor of two to account for the differences in the percentage of the biologically active isomer. The LOEL of this study of 5.0 mg/kg/day was based on gastrointestinal disturbances observed in the first week of the study. This acute dietary endpoint 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.

a. A two-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).

b. A developmental study 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 highest dose level tested.

c. 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 highest dose level tested.

4. *Subchronic toxicity—Short- and intermediate-term toxicity*. The systemic NOEL of 2.5 mg/kg/day based on the systemic NOEL of 5.0 mg/kg/day from the cypermethrin chronic toxicity study in dogs 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). This NOEL was based on neurotoxic clinical signs observed in the first week of treatment of the study.

5. *Chronic toxicity*—a. The RfD has been established at 0.0050 mg/kg/day. This RfD is based on a cypermethrin chronic toxicity study in dogs with a NOEL of 1.0 mg/kg/day, based on gastrointestinal disturbances observed at the LOEL of 5.0 mg/kg/day during the first week of the study; an uncertainty factor of 200 is used to account for the differences in the percentage of the biologically active isomer.

b. 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 female mice; 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*—a. *Food*. Tolerances have been established for the residues of the insecticide zeta-cypermethrin, in or on a variety of raw agricultural commodities. 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 potential dietary exposure for these existing tolerances, FMC has utilized available information on anticipated residues, monitoring data and percent crop treated as follows:

b. *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 one day or single exposure. For the purposes of assessing acute dietary risk for zeta-cypermethrin, FMC has used the NOEL of 0.5 mg/kg/day based on the NOEL of 1.0 mg/kg/day from the cypermethrin chronic toxicity study in dogs and a correction factor of two to account for the differences in the percentage of the biologically active isomer. The LOEL of this study of 5.0 mg/kg/day was based on gastrointestinal disturbances observed in the first week of the 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 0.000528 mg/kg/day (MOE of 947); 99th percentile 0.001746 mg/kg/day (MOE of 286); and 99.9th percentile 0.004069 mg/kg/day (MOE of 123).

The 95th percentile of exposure for all infants < 1 year old was estimated to be 0.000560 mg/kg/day (MOE of 892); 99th percentile 0.000885 mg/kg/day (MOE of 565); and 99.9th percentile 0.001260 mg/kg/day (MOE of 397).

The 95th percentile of exposure for nursing infants < 1 year old was estimated to be 0.000207 mg/kg/day (MOE of 2,417); 99th percentile 0.000569 mg/kg/day (MOE of 879); and 99.9th percentile 0.001442 mg/kg/day (MOE of 347).

The 95th percentile of exposure for non-nursing infants < 1 year old was estimated to be 0.000607 mg/kg/day (MOE of 824); 99th percentile 0.000925 mg/kg/day (MOE of 540); and 99.9th percentile 0.001190 mg/kg/day (MOE of 420).

The 95th percentile of exposure for children 1 to 6 years old and 7 to 12 years old (the most highly exposed population subgroup) was estimated to be, respectively, 0.000740 mg/kg/day (MOE of 676) and 0.000596 mg/kg/day (MOE of 839); 99th percentile 0.001856 mg/kg/day (MOE of 269) and 0.002047 mg/kg/day (MOE 244); and 99.9th percentile 0.005021 mg/kg/day (MOE of 100) and 0.004843 (MOE of 103). 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.

c. *Chronic exposure and risk*. The acceptable reference dose (RfD) of 0.0050 mg/kg/day for zeta-cypermethrin is based on a NOEL of 1.0 mg/kg/day from the cypermethrin chronic dog 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 were based on gastrointestinal disturbances observed in the first week of the study at the LOEL of 5.0 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 0.000017 mg/kg body weight (bwt)/day and utilize 0.3 percent 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 0.000011 mg/kg/day and 0.000002 mg/kg/day and utilizes 0.2 percent and 0 percent of the RfD, respectively. The ARC for children 1–6 years old and children 7–12 years old (subgroups most highly exposed) are estimated to be 0.000027 mg/kg bwt/day and 0.000022 mg/kg bwt/day and utilizes 0.5 percent and 0.4 percent 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 percent of the RfD. Therefore, FMC concludes that the chronic dietary risk of 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 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 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) 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 acceptable reference dose (RfD) for zeta-cypermethrin is 0.0005 mg/kg/day, based on a NOEL of 1.0 mg/kg/day and a LOEL of 5.0 mg/kg/day from the cypermethrin chronic dog 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 Anticipated Residue Contribution (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 0.000017 mg/kg body weight (bwt)/day and utilize 0.3 percent 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 0.000011 mg/kg/day and 0.000002 mg/kg/day and utilizes 0.2 percent and 0 percent of the RfD, respectively. The ARC for children 1–6 years old and children 7–12 years old (subgroups most highly exposed) are estimated to be 0.000027 mg/kg bwt/day and 0.000022 mg/kg bwt/day and utilizes 0.5 percent and 0.4 percent 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 percent 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 947; 286 at the 99th percentile; and 123 at the 99.9th percentile.

For all infants < 1 year old, the calculated margins of exposure (MOE) at the 95th percentile was estimated to be 892; 565 at the 99th percentile; and 397 at the 99.9th percentile.

For nursing infants < 1 year old, the calculated margins of exposure (MOE) at

the 95th percentile was estimated to be 2,417; 879 at the 99th percentile; and 347 at the 99.9th percentile.

For non-nursing infants < 1 year old, the calculated margins of exposure (MOE) at the 95th percentile was estimated to be 824; 540 at the 99th percentile; and 420 at the 99.9th percentile. For the most highly exposed population subgroups, children 1–6 years old and children 7–12 years old, the calculated MOEs at the 95th percentile were estimated to be, respectively, 676 and 839; 269 and 244 at the 99th percentile; and 100 and 103 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*— a. *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 two-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.

b. *Developmental toxicity studies.* In the prenatal developmental toxicity studies in rats and rabbits, there was no evidence of developmental toxicity at the highest doses tested (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.

c. *Reproductive toxicity study.* In the two-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, highest dose tested.

d. *Pre- and post-natal sensitivity*— i. *Pre-natal.* There was no evidence of developmental toxicity in the studies at the highest doses tested 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.

ii. *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.

F. 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 percent 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.

Subchronic toxicity— Short- and intermediate-term toxicity. The systemic NOEL of 2.5 mg/kg/day based on the systemic NOEL of 5.0 mg/kg/day from the cypermethrin chronic toxicity study in dogs 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). This NOEL was based on neurotoxic clinical signs observed in the first week of treatment of the study.

G. International Tolerances

There are no Codex, Canadian, or Mexican residue limits for residues of zeta-cypermethrin in or on cotton, pecans, lettuce, head, onions, bulb, or cabbage. (Stephanie Willett)

5. FMC Corporation

PP 2F2623, 4F2986, 3F2824, 7F3498, 4F3011, 4F4291

EPA has received a request regarding (PP 2F2623, 4F2986, 3F2824, 7F3498, 4F3011, 4F4291) from FMC Corporation, 1735 Market Street, Philadelphia, PA 19103. The request proposes to remove any time limitations on established tolerances for residues of the insecticide cypermethrin (±-alpha -Cyano(3-phenoxyphenyl)methyl (±) *cis, trans* 3-(2,2-dichloroethenyl)-2,2-dimethylcyclopropanecarboxylate) in or on the raw agricultural commodities cottonseed at 0.5 ppm, pecans 0.05 ppm, lettuce, head at 10.0 ppm, onions, bulb at 0.10 ppm, cabbage at 2.0 ppm, *Brassica*, head and stem at 2.0 ppm and *Brassica*, leafy at 14.0 ppm (established at 40 CFR 180.418). These tolerances were established under [PP] 2F2623, 4F2986, 3F2824, 7F3498, 4F3011, and 4F4291. EPA has determined that the request 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 requests. Additional data may be needed before EPA rules on the requests.

A. Residue Chemistry

1. *Plant metabolism.* The metabolism of cypermethrin in plants is adequately understood. Studies have been conducted to delineate the metabolism of radiolabelled 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 cotton, pecans, head lettuce, bulb onions, cabbage, *Brassica*, head and stem, and *Brassica*, leafy show that the established cypermethrin tolerances on cottonseed of 0.5 ppm, pecans 0.05 ppm, lettuce, head at 10.0 ppm, onions, bulb at 0.10 ppm, cabbage at 2.0 ppm, *Brassica*, head and stem at 2.0 ppm and *Brassica*, leafy at 14.0 ppm will not be exceeded when the 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 NOEL of 1.0 mg/kg/day from the chronic toxicity study in dogs. The LOEL of this study of 5.0 mg/kg/day was based on gastrointestinal disturbances observed in the first week of the study. This acute dietary endpoint is used to determine acute dietary risks to all population subgroups.

2. *Genotoxicty.* The following genotoxicity tests were all negative:

gene mutation (Ames); chromosome aberration in Chinese hamster bone marrow cells; host mediated assay in mice; dominant lethal assay in mice.

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

a. A three-reproductive toxicity study in rats demonstrated a NOEL of 2.5 mg/kg/day and a LOEL of 7.5 mg/kg/day for parental/systemic toxicity based on decreased body weight gain in both sexes. There were no adverse effects in reproductive performance. The NOEL for reproductive toxicity was considered to be 37.5 mg/kg/day, the highest dose level tested.

b. A developmental study in rats demonstrated a maternal NOEL of 17.5 mg/kg/day and a LOEL of 35 mg/kg/day based on decreased body weight gain. There were no signs of developmental toxicity at 70 mg/kg/day, the highest dose level tested.

c. A developmental study 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 highest dose level tested.

4. *Subchronic toxicity*. Short- and intermediate-term toxicity. The systemic NOEL of 5.0 mg/kg/day from the chronic toxicity study in dogs is also used for short- and intermediate-term MOE calculations (as well as acute, discussed in (1) above). This NOEL was based on neurotoxic clinical signs observed in the first week of treatment of the study.

5. *Chronic toxicity*— a. The RfD has been established at 0.010 mg/kg/day. This RfD is based on a chronic toxicity study in dogs with a NOEL of 1.0 mg/kg/day, based on gastrointestinal disturbances observed at the LOEL of 5.0 mg/kg/day during the first week of the study; an uncertainty factor of 100 is used.

b. 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 female mice; 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— Food* . Tolerances have been established for the residues of cypermethrin, in or on a variety of raw agricultural commodities. Tolerances, in support of registrations, currently exist for residues of cypermethrin on cottonseed; pecans; lettuce, head; onions, bulb; cabbage; *Brassica*, head and stem; *Brassica*, leafy and livestock commodities of cattle, goats, hogs, horses, and sheep. A pending tolerance for onions, green also exists. For the purposes of assessing the potential dietary exposure for these existing and pending tolerances, FMC has utilized available information on anticipated residues, monitoring data and percent crop treated as follows:

i. *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 one day or single exposure. For the purposes of assessing acute dietary risk for cypermethrin, the maternal NOEL of 1.0 mg/kg/day from the chronic toxicity study in dogs was used. The LOEL of this study of 5.0 mg/kg/day was based on gastrointestinal disturbances observed in the first week of the study. This acute dietary endpoint was 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 MOEs 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 0.00067 mg/kg/day (MOE of 1,493); 99th percentile 0.002109 mg/kg/day (MOE of 474); and 99.9th percentile 0.004543 mg/kg/day (MOE of 220). The 95th percentile of exposure for all infants < 1 year old was estimated to be 0.000562 mg/kg/day (MOE of 1,780); 99th percentile 0.000896 mg/kg/day (MOE of 1,116); and 99.9th percentile 0.001362 mg/kg/day (MOE of 734). The 95th percentile of exposure for nursing infants < 1 year old was estimated to be 0.000213 mg/kg/day (MOE of 4,706); 99th percentile 0.000587 mg/kg/day (MOE of 1,704); and 99.9th percentile 0.001660 mg/kg/day (MOE of 602). The 95th percentile of exposure for non-nursing infants < 1 year old was estimated to be 0.000613 mg/kg/day (MOE of 1,631); 99th percentile 0.000939 mg/kg/day (MOE of 1,065); and 99.9th percentile 0.001224 mg/kg/day (MOE of 817). The 95th percentile of exposure for children 1 to 6 years old (the most highly exposed population subgroup) was estimated to be 0.000819 mg/kg/day (MOE of 1,221); 99th percentile 0.002400 mg/kg/day (MOE of 417); and 99.9th percentile 0.005694 mg/kg/day (MOE of 176). Therefore, FMC concludes that the acute dietary risk of cypermethrin, as estimated by the dietary risk assessment, does not appear to be of concern.

ii. *Chronic exposure and risk*. The acceptable RfD is based on a NOEL of 1.0 mg/kg/day from the chronic dog study and an uncertainty factor of 100 is 0.010 mg/kg/day. The endpoint effect of concern were based on gastrointestinal disturbances observed in the first week of the study at the LOEL of 5.0 mg/kg/day. A chronic dietary exposure/risk assessment has been performed for 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 0.000024 mg/kg bwt/day and utilize 0.2% of the RfD for the overall U. S. population. The ARC for non-nursing infants (< 1 year) and children 1–6 years old (subgroups most highly exposed) are estimated to be 0.000018 mg/kg bwt/day and 0.000042 mg/kg bwt/day and utilizes 0.2% and 0.4% 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 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 (much less than 0.001 parts 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*. Analyses were conducted which included an evaluation of potential non-dietary (residential) applicator, post-application and chronic dietary aggregate exposures associated with cypermethrin products used for residential flea infestation control and agricultural/commercial applications. The aggregate analysis conservatively assumes that a person is concurrently exposed to the same active ingredient via the use of consumer or professional flea infestation control products and to chronic level residues in the diet.

In the case of potential non-dietary health risks, conservative point estimates of non-dietary exposures, expressed as total systemic absorbed dose for each product use category (indoor total release fogger and lawn care) and exposed population group (adults, children 1–6 years, and infants < 1 year) are compared to the systemic absorbed dose No-Observed-Effects-Level (NOEL) for cypermethrin to provide estimates of the MOEs. Based on the toxicity endpoints selected by EPA for cypermethrin, inhalation and incidental oral ingestion absorbed doses were combined and compared to the relevant systemic NOEL for estimating MOEs.

In the case of potential aggregate health risks, the above mentioned conservative point estimates of non-dietary exposure (expressed as systemic absorbed dose) are combined with estimates (arithmetic mean values) of chronic average dietary (oral) absorbed doses. These aggregate absorbed dose estimates are also provided for adults, children 1–6 years and infants < 1 year. The combined or aggregated absorbed dose estimates (summed across non-dietary and chronic dietary) are then compared with the systemic absorbed dose NOEL to provide estimates of aggregate MOEs.

The total non-dietary MOEs (combined across all product use categories) for the inhalation + incidental oral routes are 97,000 for adults, 2,100 for children 1–6 years old, and 1,900 for infants (< 1 year). The aggregate MOE (inhalation + incidental oral + chronic dietary, summed across all product use categories) was estimated to be 66,000 for adults, 2,000 for children 1–6 years old and 1,900 for infants (<1 year). It can be concluded that the potential non-dietary and aggregate (non-dietary + chronic dietary) exposures for cypermethrin are associated with substantial margins of safety.

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) 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 acceptable RfD is 0.010 mg/kg/day, based on a LOEL of 5.0 mg/kg/day from the chronic dog study and an uncertainty factor of 100. Available information on anticipated residues, monitoring data and percent crop treated was incorporated into an analysis to estimate the Anticipated Residue Contribution (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 0.000024 mg/kg body weight (bwt)/day and utilize 0.2% of the RfD for the overall U. S. population. The ARC for non-nursing infants (<1 year) and children 1–6 years old (subgroups most highly exposed) are estimated to be 0.000018 mg/kg bwt/day and 0.000042 mg/kg bwt/day and utilizes 0.2% and 0.4% 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 cypermethrin, as estimated by the aggregate risk assessment, does not appear to be of concern.

For the overall U.S. population, the calculated MOE at the 95th percentile was estimated to be 1,493; 474 at the 99th percentile; and 220 at the 99.9th percentile. For all infants < 1 year old, the calculated MOE at the 95th percentile was estimated to be 1,780; 1,116 at the 99th percentile; and 734 at the 99.9th percentile. For nursing infants < 1 year old, the calculated MOE at the 95th percentile was estimated to be 4,706; 1,704 at the 99th percentile; and 602 at the 99.9th percentile. For non-nursing infants < 1 year old, the calculated MOE at the 95th percentile was estimated to be 1,631; 1,065 at the 99th percentile; and 817 at the 99.9th percentile. For the most highly exposed population subgroup, children 1 – 6 years old, the calculated MOE at the 95th percentile was estimated to be 1,221 ; 417 at the 99th percentile; and 176 at the 99.9th percentile. Therefore, FMC concludes that there is reasonable certainty that no harm will result from acute exposure to cypermethrin.

2. *Infants and children*— a. *General*. In assessing the potential for additional sensitivity of infants and children to residues of cypermethrin, FMC considered data from developmental toxicity studies in the rat and rabbit, and a three-reproductive study in the rat. The data demonstrated no indication of increased sensitivity of rats or rabbits to in utero and/or postnatal exposure to 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.

b. *Developmental toxicity studies*. In the prenatal developmental toxicity studies in rats and rabbits, there was no evidence of developmental toxicity at the highest doses tested (70 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 17.5 mg/kg/day in rats and 100 mg/kg/day in rabbits.

c. *Reproductive toxicity study*. In the three-reproduction study in rats, offspring toxicity (reduced mean litter weight gain) was observed only at the highest dietary level tested (37.5 mg/kg/day), while toxicity in the parental animals was observed at the lower treatment levels. The parental systemic NOEL was 2.5 mg/kg/day and the parental systemic LOEL was 7.5 mg/kg/day. There were no developmental (pup) or reproductive effects up to 37.5 mg/kg/day (highest dose tested).

d. *Pre- and post-natal sensitivity*—i. *Pre-natal*. There was no evidence of developmental toxicity in the studies at the highest doses tested in the rat (70 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.

ii. *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.

e. *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.

F. International Tolerances

There are no Codex, Canadian, or Mexican residue limits for residues of cypermethrin in or on cotton; pecans; lettuce, head; onions, bulb; cabbage; *Brassica*, head and stem, or *Brassica*, leafy. (Stephanie Willett)

6. FMC Corporation, Agricultural Products Group

PP 6F3453, 7F3546, 5F4484, and 0E3921

EPA has received a request to remove the time limitations on established tolerances from FMC Corporation, Agricultural Products Group, 1735 Market Street, Philadelphia, Pennsylvania 19103 and from the Interregional Research Project No. 4 (IR-4), New Jersey Agricultural Experiment Station, P.O. Box 231, Rutgers University, New Brunswick, NJ 08903. The request proposes to remove the time limitations on established tolerances for residues of the insecticide bifenthrin ((2-methyl [1,1′-biphenyl]-3-yl) methyl-3-(2-chloro-3,3,3,-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylate), in or on the raw agricultural commodities cottonseed at 0.5 parts per million (ppm); corn, grain (field, seed, and pop) at 0.05 ppm; hops, dried at 10.0 ppm; and strawberries at 3.0 ppm (established at 40 CFR 180.442). These tolerances were established under [PP] 6F3453, 7F3546, 5F4484, and 0E3921. EPA has determined that the request 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 requests. Additional data may be needed before EPA rules on the requests.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of bifenthrin in plants is adequately understood. Studies have been conducted to delineate the metabolism of radiolabeled bifenthrin 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 bifenthrin 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) analytical method P-2132M.

3. *Magnitude of residues*. Crop field trial residue data from studies conducted at the maximum label rates for cotton; corn (field, seed, pop); strawberries, and hops show that the established bifenthrin tolerances on cottonseed of 0.5 ppm; corn, grain (field, seed, and pop) of 0.05 ppm; corn, fodder of 5.0 ppm; corn, forage of 2.0 ppm; strawberries of 3.0 ppm, and hops, dried of 10.0 ppm will not be exceeded when the bifenthrin 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 maternal NOEL of 1.0 mg/kg/day from the oral developmental toxicity study in rats. The maternal LEL of this study of 2.0 mg/kg/day was based on tremors from day 7-17 of dosing. This acute dietary endpoint is used to determine acute dietary risks to all population subgroups.

2. *Genotoxicty*. The following genotoxicity tests were all negative: gene mutation in Salmonella (Ames); chromosomal aberrations in Chinese hamster ovary and rat bone marrow cells; HGPRT locus mutation in mouse lymphoma cells; and unscheduled DNA synthesis in rat hepatocytes.

3. *Reproductive and developmental toxicity*— a. *Parental toxicity*. In the rat reproduction study, parental toxicity occurred as decreased body weight at 5.0 mg/kg/day with a NOEL of 3.0 mg/kg/day. There were no developmental (pup) or reproductive effects up to 5.0 mg/kg/day (highest dose tested).

b. *Post-natal sensitivity*. 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.

4. *Subchronic toxicity*. Short- and intermediate-term toxicity. The maternal NOEL of 1.0 mg/ kg/day from the oral developmental toxicity study in rats is also used for short- and intermediate-term margins of exposure (MOE) calculations (as well as acute, discussed in (1) above). The maternal lowest effect level (LEL) of this study of 2.0 mg/kg/day was based on tremors from day 7–17 of dosing.

5. *Chronic toxicity*—a. The reference dose (RfD) has been established at 0.015 mg/kg/day. This RfD is based on a 1 year oral feeding study in dogs with a NOEL of 1.5 mg/kg/day, based on intermittent tremors observed at the Lowest Observed Effect Level (LOEL) of 3.0 mg/kg/day; an uncertainty factor of 100 is used.

b. Bifenthrin is classified as a Group C chemical (possible human carcinogen) based upon urinary bladder tumors in mice; assignment of a Q^* has not been recommended.

6. *Animal metabolism*. The metabolism of bifenthrin in animals is adequately understood. Metabolism studies in rats with single doses demonstrated that about 90% of the

parent compound and its hydroxylated metabolites are excreted.

7. *Metabolite toxicology.* The Agency has previously determined that the metabolites of bifenthrin 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 bifenthrin 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 bifenthrin has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure— Food.* Tolerances have been established for the residues of bifenthrin, in or on a variety of raw agricultural commodities. Tolerances, in support of registrations, currently exist for residues of bifenthrin on hops; strawberries; corn grain, forage, and fodder; cottonseed; and livestock commodities of cattle, goats, hogs, horses, sheep, and poultry. Additionally, time-limited tolerances associated with emergency exemptions were recently established for broccoli, cauliflower, raspberries, cucurbits, and canola. A pending tolerance for artichokes also exists. For the purposes of assessing the potential dietary exposure for these existing and pending tolerances as well as the existing time-limited tolerances under FIFRA section 18 emergency exemptions, FMC has utilized available information on anticipated residues, monitoring data and percent crop treated as follows:

i. *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 bifenthrin, the maternal NOEL of 1.0 mg/kg/day from the oral developmental toxicity study in rats was used. The maternal LEL of this study of 2.0 mg/kg/day was based on tremors from day 7–17 of dosing. This acute dietary endpoint was 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 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 0.000362 mg/kg/day (MOE of 2,762); 99th percentile 0.000732 mg/kg/day (MOE of 1,367); and 99.9th percentile 0.002282 mg/kg/day (MOE of 438). The 95th percentile of exposure for all infants < 1 year old was estimated to be 0.000652 mg/kg/day (MOE of 1,534); 99th percentile 0.001138 mg/kg/day (MOE of 879); and 99.9th percentile 0.001852 mg/kg/day (MOE of 540). The 95th percentile of exposure for nursing infants < 1 year old was estimated to be 0.000193 mg/kg/day (MOE of 5,180); 99th percentile 0.000456 mg/kg/day (MOE of 2,192); and 99.9th percentile 0.000475 mg/kg/day (MOE of 2,107). The 95th percentile of exposure for non-nursing infants < 1 year old was estimated to be 0.000766 mg/kg/day (MOE of 1,306); 99th percentile 0.001203 mg/kg/day (MOE of 832); and 99.9th percentile 0.001977 mg/kg/day (MOE of 506). The 95th percentile of exposure for children 1 to 6 years old (the most highly exposed population subgroup) was estimated to be 0.000632 mg/kg/day (MOE of 1,583); 99th percentile 0.001196 mg/kg/day (MOE of 836); and 99.9th percentile 0.005277 mg/kg/day (MOE of 190). Therefore, FMC concludes that the acute dietary risk of bifenthrin, as estimated by the dietary risk assessment, does not appear to be of concern.

ii. *Chronic exposure and risk.* The acceptable RfD is based on a NOEL of 1.5 mg/kg/day from the chronic dog study and an uncertainty factor of 100 is 0.015 mg/kg/day. The endpoint effect of concern were tremors in both sexes of dogs at the LEL of 3.0 mg/kg/day. A chronic dietary exposure/risk assessment has been performed for bifenthrin 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 0.00002 mg/kg body weight (bwt)/day and utilize 0.1% of the RfD for the overall U.S. population. The ARC for non-nursing infants (< 1 year) and children 1–6 years old (subgroups most highly exposed) are estimated to be 0.000042 mg/kg bwt/day and 0.000032 mg/kg bwt/day and utilizes 0.3% and 0.2% 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 bifenthrin, as estimated by the dietary risk assessment, does not appear to be of concern.

2. *Drinking water.* Laboratory and field data have demonstrated that bifenthrin is immobile in soil and will not leach into groundwater. Other data show that bifenthrin 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 (much less than 0.001 parts 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.* Analyses were conducted which included an evaluation of potential non-dietary (residential) applicator, post-application and chronic dietary aggregate exposures associated with bifenthrin products used for residential flea infestation control and agricultural/commercial applications. The aggregate analysis conservatively assumes that a person is concurrently exposed to the same active ingredient via the use of consumer or professional flea infestation control products and to chronic level residues in the diet. In the case of potential non-dietary health risks, conservative point estimates of non-dietary exposures, expressed as total systemic absorbed dose (summed across inhalation and incidental ingestion routes) for each relevant product use category (i.e., lawn care) and receptor subpopulation (i.e., adults, children 1–6 years and infants < 1 year) are compared to the systemic

absorbed dose NOEL for bifenthrin to provide estimates of the MOEs. Based on the toxicity endpoints selected by EPA for bifenthrin, inhalation and incidental oral ingestion absorbed doses were combined and compared to the relevant systemic NOEL for estimating MOEs. In the case of potential aggregate health risks, the above-mentioned conservative point estimates of inhalation and incidental ingestion non-dietary exposure (expressed as systemic absorbed dose) are combined with estimates (arithmetic mean values) of chronic average dietary (oral) absorbed doses. These aggregate absorbed dose estimates are also provided for adults, children 1–6 years and infants < 1 year. The combined or aggregated absorbed dose estimates (summed across non-dietary and chronic dietary) are then compared with the systemic absorbed dose NOEL to provide estimates of aggregate MOEs. The non-dietary and aggregate (non-dietary + chronic dietary) MOEs for bifenthrin indicate a substantial degree of safety. The total non-dietary (inhalation + incidental ingestion) MOEs for post-application exposure for the lawn care product evaluated was estimated to be > 51,000 for adults, 1,900 for children 1–6 years old and 1,800 for infants < 1 year. The aggregate MOE (inhalation + incidental oral + chronic dietary, summed across all product use categories) was estimated to be 25,000 for adults, 1,800 for children 1–6 years old and 1,600 for infants (< 1 year). It can be concluded that the potential non-dietary and aggregate (non-dietary + chronic dietary) exposures for bifenthrin are associated with substantial margins of safety.

D. Cumulative Effects

In consideration of potential cumulative effects of bifenthrin 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 bifenthrin would be cumulative with those of other chemical compounds; thus only the potential risks of bifenthrin 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 bifenthrin consistent with the schedule established by EPA in the **Federal Register** of August 4, 1997 (62 FR 42020) (FRL–5734–6), and other EPA publications pursuant to the FQPA.

E. Safety Determination

1. *U.S. population.* Based on a complete and reliable toxicology data base, the acceptable reference dose (RfD) is 0.015 mg/kg/day, based on a NOEL of 1.5 mg/kg/day from the chronic dog study and an uncertainty factor of 100. Available information on anticipated residues, monitoring data and percent crop treated was incorporated into an analysis to estimate the Anticipated Residue Contribution (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 0.00002 mg/kg body weight (bwt)/day and utilize 0.1% of the RfD for the overall U.S. population. The ARC for non-nursing infants (< 1 year) and children 1–6 years old (subgroups most highly exposed) are estimated to be 0.000042 mg/kg bwt/day and 0.000032 mg/kg bwt/day and utilizes 0.3% and 0.2% 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 bifenthrin, as estimated by the aggregate risk assessment, does not appear to be of concern. For the overall U.S. population, the calculated MOE at the 95th percentile was estimated to be 2,762; 1,367 at the 99th percentile; and 438 at the 99.9th percentile. For all infants < 1 year old, the calculated MOE at the 95th percentile was estimated to be 1,534; 879 at the 99th percentile; and 540 at the 99.9th percentile. For nursing infants < 1 year old, the calculated MOE at the 95th percentile was estimated to be 5,180; 2,192 at the 99th percentile; and 2,107 at the 99.9th percentile. For non-nursing infants < 1 year old, the calculated MOE at the 95th percentile was estimated to be 1,306; 832 at the 99th percentile; and 506 at the 99.9th percentile. For the most highly exposed population subgroup, children 1–6 years old, the calculated MOE at the 95th percentile was estimated to be 1,583; 836 at the 99th percentile; and 190 at the 99.9th percentile. Therefore, FMC concludes that there is reasonable certainty that no harm will result from acute exposure to bifenthrin.

2. *Infants and children*— a. *General.* In assessing the potential for additional sensitivity of infants and children to residues of bifenthrin, FMC considered data from developmental toxicity studies in the rat and rabbit, and a two-generation reproductive 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 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 data base.

b. *Developmental toxicity studies.* In the rabbit developmental study, there were no developmental effects observed in the fetuses exposed to bifenthrin. The maternal NOEL was 2.67 mg/kg/day based on head and forelimb twitching at the LOEL of 4 mg/kg/day. In the rat developmental study, the maternal NOEL was 1 mg/kg/day, based on tremors at the LOEL of 2 mg/kg/day. The developmental (pup) NOEL was also 1 mg/kg/day, based upon increased incidence of hydroureter at the LOEL 2 mg/kg/day. There were 5/23 (22%) litters affected (5/141 fetuses since each litter only had one affected fetus) in the 2 mg/kg/day group, compared with zero in the control, 1, and 0.5 mg/kg/day groups. According to recent historical data (1992-1994) for this strain of rat, incidence of distended ureter averaged 11% with a maximum incidence of 90%.

c. *Reproductive toxicity study.* In the rat reproduction study, parental toxicity occurred as decreased body weight at 5.0 mg/kg/day with a NOEL of 3.0 mg/kg/day. There were no developmental (pup) or reproductive effects up to 5.0 mg/kg/day (highest dose tested).

d. *Pre- and post-natal sensitivity*— i. *Pre-natal.* Since there was not a dose-related finding of hydroureter in the rat developmental study and in the presence of similar incidences in the recent historical control data, the marginal finding of hydroureter in rat fetuses at 2 mg/kg/day (in the presence of maternal toxicity) is not considered a significant developmental finding. Nor does it provide sufficient evidence of a special dietary risk (either acute or chronic) for infants and children which would require an additional safety factor.

ii. *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.

e. *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 bifenthrin residues.

F. International Tolerances

There are no Codex, Canadian, or Mexican residue limits for residues of bifenthrin in or on cotton; corn, field, seed, pop; strawberries; or hops. (Adam Heyward)

7. McLaughlin Gormley King Company

PP 7F4915

EPA has received a pesticide petition (PP 7F4915) from McLaughlin Gormley King Company, 8810 Tenth Avenue North, Minneapolis, MN 55427, 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 (*RS*)-2-Methyl-4-oxo-3-(2-propynyl) cyclopent-2-enyl (1*RS*)-cis, trans-chrysanthemate (common name, prallethrin; trade name ETOC®), a Type I synthetic pyrethroid in or on food commodities at 1 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Analytical method*. An adequate analytical method is available to detect residues of ETOC® in or on food commodities. Prallethrin can be extracted from samples and analyzed by gas chromatography, with final electron capture detection. The method has been confirmed through an independent laboratory validation.

2. *Magnitude of residues*. Studies were conducted to determine residues resulting from the application of ETOC® by ULV spray and contact spray in a simulated feed or food processing situation, and in a simulated warehouse situation. No residues were detected following contact sprays in either situation, with the exception of a trace amount in a peanut sample after the tenth treatment at 4X the normal application rate. No residues were detected in covered commodities after ULV spraying of ETOC®, but residues were detected in uncovered commodities and samples with permeable wrapping.

B. Toxicological Profile

1. *Acute toxicity*. An oral dosage rat study reported Category II toxicity with the LD_{50} being 640 mg/kg for males and 460 mg/kg for females. An acute dermal study with rats reported Category IV toxicity. An acute inhalation study with rats reported Category II toxicity with an LC_{50} of 0.288 mg/liter for males and 0.333 mg/liter for females. Rabbits were tested for eye and skin irritation. Eye irritation was minimal (Category III) and there was no skin irritation (Category IV). ETOC® is not a skin sensitizer, based on a Guinea Pig dermal sensitization study. Rats were dosed at 30, 100, and 300 mg/kg by oral gavage to test acute neurotoxicity. While there was some temporary motor activity reduction, there were no permanent treatment-related anomalies.

2. *Genotoxicity*. A bacterial reverse mutation test using *Salmonella typhimurium* and *Escherichia coli* indicated that ETOC® was not mutagenic. A gene mutation assay with Chinese hamster lung cells in both the presence and absence of S9 metabolic activation reported no mutagenicity. An *in vitro* chromosomal aberration test reported clastogenic potential against Chinese hamster ovary cells (CHO-K1) in the presence of S9 mix. An *in vivo* mouse bone marrow micronucleus test did not induce micronuclei formation in bone marrow cells of mice. An *in vivo/in vitro* unscheduled DNA synthesis test reported no induction of DNA damage in rat hepatocytes *in vivo*.

3. *Reproductive and developmental toxicity*. A range-finding study was conducted by administering 30, 60, 100, 300, 600, and 800/1,000 mg/kd/day by oral gavage to rabbits on days 7 through 19 of presumed gestation. Significantly decreased body weights occurred in those rabbits receiving 300 mg/kg/day and above, food consumption decreased at 100 mg/kg/day and above, and deaths occurred at 300 mg/kg/day and above. Doses as high as 100 mg/kg/day did not produce adverse effects in the offspring. ETOC® was then administered by oral gavage at doses of 10, 30, 100, and 200 mg/kg/day to rabbits on days 7 through 19 of presumed gestation. The maternal NOAEL was 100 mg/kg/day. The 200 mg/kg/day dosage caused reduced maternal body weight gains and reduced absolute and relative feed consumption values. The developmental NOAEL was reported as 200 mg/kg/day. ETOC® is not considered a developmental toxin. A teratology study was conducted by administering 10, 30, 100, and 300 mg/kg/day by oral gavage to rats on days 6–15 of presumed gestation. The developmental NOEL was >300 mg/kg/day and the developmental LOEL was not determined. Compound related maternal mortality was reported at 300 mg/kg/day. The maternal LOEL was 30 mg/kg/day, as determined by increased mortality at 300 mg/kg/day levels, clinical signs at the 30, 100, and 300 mg/kg/day dosages, and decreased body weight gain and food consumption. Rats were dosed with 12.5, 25.0, and 50 mg/kg/day by subcutaneous injection on days 7 through 17 of presumed gestation. No NOEL or LOEL was established, but the occurrence of lumbar rib variants was significantly higher in the offspring of the 50 mg/kg/day group than in the controls. Rabbits were dosed at 1, 3, and 10 mg/kg/day by subcutaneous injection on days 6 through 18 of presumed gestation. No effects were reported on either the dams or the offspring. ETOC® was incorporated into the feed at concentrations of 120, 600, 3,000, and 6,000 ppm to evaluate the reproductive effects on two generations of rats. The systemic toxicity and reproductive toxicity NOEL's were both established at 600 ppm, and the LOEL's were both 3,000 ppm, respectively. There were dosage-dependent effects on weight gains, body weights, feed consumption values, liver weights, and reduction of pup body weight at the 3,000 and 6,000 ppm dose levels. There were no adverse effects on viability or fertility in either generation up to the 6,000 ppm level.

4. *Subchronic toxicity*. A 21–day dermal toxicity rat study was conducted at 30, 150 and 750 mg/kg/day. The test article was considered a mild irritant. The dermal NOEL was 150 mg/kg/day and the systemic NOEL was 30 mg/kg/day. A 13–week oral mouse study was conducted at inclusion levels of 300, 3,000, 6,000, or 12,000 ppm. The NOEL was 3,000 ppm, and the LOEL was 6,000 ppm. A 3–month feeding study incorporating 100, 300, 1,000, and 3,000 ppm into the diet of rats reported a NOEL of 300 ppm, and a LOEL of 1,000 ppm. EPA later recommended raising the NOEL to 1,000 ppm and the LOEL to 3,000 ppm. A 3–month oral study on beagle dogs dosed at 3, 10, and 30 mg/kg/day, administered by capsule, reported a NOEL of 3 mg/kg/day and a LOEL of 10 mg/kg/day. A 4–week inhalation study exposed rats to 1.01, 4.39, and 19.6 mg/m3 of 92.0% ETOC®, with median aerodynamic particle diameter of 3.77 to 4.89 m. The NOEL was 1.01 mg/m^3 and the LOEL was 4.39 mg/m^3.

5. *Chronic toxicity*. A 52–week oral toxicity study was conducted on beagle dogs administered dosage levels of 2. 5, 5.0, 10.0 or 20.0 mg/kg/day. The NOEL was reported at 2.5 mg/kg/day; EPA's RfD/Peer Review Committee later recommended 5 mg/kg/day in a DER dated June 6, 1995. The LOEL was 5.0 mg/kg/day based upon reduced weight gain, clinical signs, elevated cholesterol levels and deposition of lipofuscin in renal and bladder epithelium. A 106–week combined oral toxicity and oncogenicity study was performed on rats using dietary concentrations of 80, 400, and 2,000 ppm. It was determined that there was no carcinogenic potential in rats. The NOEL was 80 ppm, and the LOEL was 400 ppm. There were no ophthalmologic, biochemical changes, or gross pathological treatment-related effects except for increased liver and thyroid weights in the 400 ppm and above level. An 80 week dietary oncogenicity study on rats with dose levels of 120, 600, 3,000 and 6,000 ppm showed that the principal effect of ETOC® was increased liver weights in those rats given the 3,000 to 6,000 ppm diet. There was no indication of any treatment related effect on the incidence of neoplastic findings.

6. *Animal metabolism*. Solutions of (4S), (1R)-*trans*- and (4S), (1R)- *cis*-S-4068SF (ETOC®) labeled with ^{14}C were given to rats by single oral dose or subcutaneous administration at 2 mg/kg. Both isomers were rapidly absorbed, widely distributed to various tissues, and then readily metabolized and excreted. Neither isomer was retained or accumulated in any tissues. There was no marked difference in metabolic fate between sexes and administration routes. The absorption and disposition of ^{14}C-S-4068SF *cis* and *trans* isomers in rats was determined after oral administration of the compounds at 2 and 100 mg/kg and at 2mg/kg after 14 daily doses of the non-labeled compounds at the same dose level. The results indicated that the dose was rapidly eliminated at all dose levels. A greater proportion was excreted in the urine of rats receiving the *trans*-compound compared to the *cis*-compound, indicating a greater ester cleavage of the *trans*-isomer. Concentrations of compound in tissues were not significantly affected by repeat doses of unlabelled compound and concentrations at the higher dose level were in proportion to the increase in dose. The greatest concentrations were detected in the organs responsible for excretion and metabolism (liver and kidneys). Concentrations in these tissues were greater in females.

7. *Endocrine effects*. The standard battery of required toxicity studies is generally considered to be sufficient to detect any endocrine effects, and is complete for ETOC®. No developmental or reproductive effects were noted. The potential for ETOC® to produce any significant endocrine effects is considered minimal

8. *Metabolite toxicology*. There is no evidence that prallethrin contains metabolites of toxicological concern.

C. Aggregate Exposure

1. *Dietary exposure*. A chronic dietary exposure analysis was conducted for exposure to potential prallethrin residues in all food commodities that can be exposed to prallethrin by indoor ULV fogging treatment, crack and crevice, and hard surface applications in food-handling establishments. Residue amounts from MGK field trials in a simulated warehouse situation were used in the analysis. Chronic dietary exposure to prallethrin has been conservatively estimated to be less than 1% of the RfD for all population groups.

2. *Drinking water*. ETOC® is presently registered only for indoor, non-food uses. No agricultural uses are planned for ETOC®, so residues in drinking water are not likely to be present.

3. *Non-dietary exposure*. Acute and short-term non-dietary exposure assessments were conducted to determine the non-dietary exposure risk of prallethrin from both registered and pending, occupational and residential uses. These assessments considered oral, dermal, and inhalation exposure to prallethrin during application and post-application of total release aerosols, crack and crevice sprays, broadcast carpet/hard surface sprays, pet dipping, and indoor ULV fogging concentrate/contact spray. Incidental ingestion of ETOC® residues by children's hand-to-mouth behavior was included in the assessment. All of the MOE's for the occupational setting were greater than 5,200, the residential MOE's were greater than 4,900, and the aggregate residential assessment was greater than 1,400. These MOE values allow a reasonable certainty that no harm will occur from exposure to residues of prallethrin.

D. Cumulative Effects

The EPA guidelines for product safety testing address noticeable toxic effects rather than the underlying mode of toxicity. There is very little information or data available to determine whether or not the toxic mode of action of prallethrin is sufficiently similar to other Type I pyrethroids to be cumulative.

E. Safety Determination

1. *U.S. population*. Based on the conservative aggregate exposure estimates noted above and the complete and reliable toxicology database for prallethrin, it is safe to conclude that the aggregate exposure of the whole U.S. population to prallethrin will be 0.2% or less of the RfD of 0.05 mg/kg bw/day. Children from 1 to 6 years old may be exposed to a slightly higher amount of prallethrin; 0.3% of the RfD.

Generally speaking, EPA has no concerns about exposures which are less than 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. It is therefore concluded that there is a reasonable certainty that no harm will result from aggregate exposure to prallethrin residues.

2. *Infants and children*. Developmental toxicity studies of prallethrin orally administered to rats and rabbits did not demonstrate any pre-natal sensitivies for developing fetuses. The maternal NOEL for rats was 10 mg/kg/day, and the maternal NOEL for rabbits was 100 mg/kg/day.

A two-generation reproduction study of rats administered prallethrin in their feed did not reveal any treatment-related reproductive or developmental effects in either generation. The NOEL for adult rats was found to be 120 ppm while the LEL was 600 ppm. The NOEL for fetotoxicity was found to be 600 ppm and the LEL was 3,000 ppm.

Since no special sensitivities to offspring were noted in these studies, there is no need for an additional fold safety factor to be applied to risk assessments.

F. International Tolerances

There are no international maximum residue limits established for prallethrin; therefore, incompatibility is not an issue. (Adam Heyward)

8. Valent U. S. A. Corporation

PP 2F4144, 3F4186, 4F4327

EPA has received a request from Valent U. S. A. Corporation, 1333 North California Blvd., Walnut Creek, CA 94596–8025 pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.466 to remove the time limitations on tolerances for residues of the pyrethroid insecticide chemical fenpropathrin, *alpha*-cyano-3-phenoxybenzyl 2,2,3,3-tetramethylcyclo-propanecarboxylate, in or on the raw agricultural commodities cottonseed at 1.0 parts per million

(ppm), peanut nutmeat at 0.01 ppm, peanut vine hay at 20 ppm, strawberry at 2.0 ppm, tomato at 0.6 ppm, meat and meat by-products of cattle, goats, hogs, horses and sheep at 0.1 ppm, fat of cattle, goats, hogs, horses and sheep at 1.0 ppm, milk fat (reflecting 0.08 ppm in whole milk) at 2.0 ppm, and poultry meat, fat, meat by-products and eggs at 0.05 ppm, and in the processed products cottonseed oil at 3.0 ppm and cottonseed soapstock at 2.0 ppm. The tolerances were first established in response to pesticide petitions PP 2F4144, 3F4186, and 4F4327 and were only made time limited because of concerns associated with toxicity to aquatic arthropods. EPA has determined that the request contains data or information consistent with 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 request. Additional data may be needed before EPA rules on the request.

A. Residue Chemistry

Summary. An extensive plant and animal metabolism data base demonstrates that the appropriate definition of aged fenpropathrin residue is parent. Ruminant and poultry feeding studies have shown that feed to residue ratios are very low in most commodities, with higher (but still relatively low) ratios in fat and milk fat. This section will describe residue data supporting the establishment of tolerances for residues of fenpropathrin in or on the raw agricultural commodities cottonseed at 1.0 parts per million (ppm), peanut nutmeat at 0.01 ppm, peanut vine hay at 20 ppm, strawberry at 2.0 ppm, tomato at 0.6 ppm, meat and meat by-products of cattle, goats, hogs, horses and sheep at 0.1 ppm, fat of cattle, goats, hogs, horses and sheep at 1.0 ppm, milk fat (reflecting 0.08 ppm in whole milk) at 2.0 ppm, and poultry meat, fat, meat by-products and eggs at 0.05 ppm, and in the processed products cottonseed oil at 3.0 ppm and cottonseed soapstock at 2.0 ppm. The approved analytical method is capillary gas-liquid chromatography with flame ionization detection.

1. *Plant metabolism*. The plant metabolism of fenpropathrin has been studied in five different crop plant species: cotton, apple, tomato, cabbage, and bean. Radiocarbon labeling has been in the cyclopropyl ring of the acid, in the aryl rings of the alcohol, and in the nitrile of fenpropathrin, a cyanohydrin ester. The permutations of radiocarbon label position and plant species yield a total of 17 separate, reviewed studies. Each of the studies involved foliar treatment of the plants under either greenhouse or field conditions and, while the actual treatment conditions and times to harvest varied from study to study, the results of the many studies are remarkably consistent. The total toxic residue is best defined as parent, fenpropathrin.

Fenpropathrin remains associated with the site of application and only traces are found in seeds (e.g., bean or cotton) or in other parts of the plant not directly exposed to the application. Much of the parent residue can be removed from the plant material with a mild hexane/acetone or hexane rinse, demonstrating that the residue is located on or near the outside surface of the plant material. The primary metabolic pathway for fenpropathrin in plants is similar to that in mammals. There are no qualitatively unique plant metabolites; the primary aglycones are identical in both plants and animals.

2. *Analytical method*. Adequate analytical methodology is available to detect and quantify fenpropathrin (and its metabolites) at residue levels in numerous matrices. The methods use solvent extraction and partition and/or column chromatography clean-up steps, followed by separation and quantitation using capillary column gas-liquid chromatography with flame ionization detection. The extraction efficiency has been validated using radiocarbon samples from the plant and animal metabolism studies. The enforcement methods have been validated at independent laboratories, and by EPA. The limit of quantitation for fenpropathrin in raw agricultural commodity samples is 0.01 ppm.

3. *Magnitude of residues— Cotton*. The time limited section 408 tolerance for fenpropathrin in/on cottonseed is 1.0 ppm. The use pattern allows a maximum single application rate of 0.3 lb ai/acre, a total maximum seasonal use of 0.8 lb ai/acre, and a 21-day phi. The field residue experiments were performed in six years at thirty-three sites in nine states. There were 38 separate treatments yielding 101 separate, treated samples for analysis. The existing time limited tolerance of 1.0 ppm is based on all of the field residue data, including treatments at exaggerated rates. For the subset of the field residue samples that most closely match the present, labeled use pattern, 0.3 lb ai/acre, 5 applications, and a 21-day phi, the average residue was 0.069 ppm ($n = 14$, $\sigma_{n-1} = 0.091$). The highest average residue (HAR) found in these crop field trials for fenpropathrin in/on cottonseed was 0.28 ppm.

There are existing time limited section 408 tolerances for fenpropathrin in the processed products cottonseed oil (3.0 ppm) and cottonseed soapstock (2.0 ppm). Three processing studies yielding hulls, extracted meal, crude cottonseed oil, refined cottonseed oil, and cottonseed soapstock were performed. These studies demonstrated that fenpropathrin residues were reduced in extracted meal but did concentrate in refined cottonseed oil (average concentration factor = 2.77) and soapstock. Tolerances for the processed products cottonseed oil and cottonseed soapstock were needed because the concentration factors were greater than unity. Soapstocks are no longer considered significant feed commodities. The HAR times the average concentration factor for cottonseed oil (0.28 ppm x 2.77 = 0.78 ppm) is less than the tolerance of 1.0 ppm. Under present residue chemistry guidelines, tolerances for cottonseed oil and soapstock would no longer be required.

The calculated mean residue value for cottonseed of 0.07 ppm was used in both the chronic and acute dietary exposure and risk assessments since cottonseed is a blended commodity. Processing factors used in the assessments were refined cottonseed oil (2.77), cottonseed meal (0.48), and cottonseed hulls (0.90).

Peanut. The time limited section 408 tolerances for fenpropathrin in/on peanut nutmeat is 0.01 ppm and in/on peanut vine hay is 20.0 ppm. The use pattern allows a maximum single application rate of 0.3 lb ai/acre, a total maximum seasonal use of 0.8 lb ai/acre, and a 14-day interval before digging the peanuts or feeding the vines or hay. The field residue experiments were performed in two years at seven sites in five states. There were 9 separate treatments yielding 22 separate, treated samples for analysis for nutmeats, green vines, and dried vine hay. Data from the subset of the field residue samples that most closely match the present, labeled use pattern, 0.3 lb ai/acre, 2 or more applications, and a 14-day phi were used to support the tolerances.

Peanut nutmeats. No finite residues were detected (< 0.01 ppm) in 17 of 18 samples. In a single sample a finite residue of .01 ppm was detected.

Peanut vine hay. Field dried vines, peanuts removed, were sampled at 14-days plus 2- to 9-days field drying time following the last application. The average residue found in/on peanut vine hay was 8.31 ppm ($n = 16$, $\sigma_{n-1} = 4.64$ ppm). The HAR for peanut vine hay was 16 ppm. A peanut processing study using a very highly exaggerated field

application rate showed positive concentration in peanut oil and other processed products. However, Agency guidance has indicated that no additional tolerances are needed.

Except for a single sample of peanut nutmeat (0.01 ppm) all appropriate field trial data were non-detects. Therefore, 0.005, or half the limit of detection (LOD), was used for the chronic dietary risk assessment, and 0.01 ppm (full LOD) was used for the acute assessment. Calculated mean residue values were used for peanut commodities in both the chronic and acute assessments because peanuts is a blended commodity. The processing factor for deodorized bleached refined oil (1.33) was used in the risk assessments since this is the grade of peanut oil available for human consumption. For feed, the processing value for expeller presscake (1.33) was used for peanut meal.

Strawberry. The time limited section 408 tolerance for fenpropathrin in/on strawberries is 2.0 ppm. The use pattern allows a maximum single application rate of 0.4 lb ai/acre, a minimum 30-day interval between treatments, a total maximum seasonal use of 0.8 lb ai/acre, and a 2-day phi. The field residue experiments were performed in three years at twelve sites in six states. There were 47 separate treatments yielding 128 separate, treated samples for analysis. For the subset of the field residue samples that most closely match the present, labeled use pattern, 0.4 lb ai/acre, 1 or 2 applications with a (approximately) 30-day interval between treatments, and a 2-day phi, the average residue was 0.65 ppm ($n = 34$, $\sigma_{n-1} = 0.44$). The HAR found in these crop field trials in/on strawberries was 1.45 ppm.

For chronic dietary exposure and risk assessment, the mean residue value (0.65 ppm) was used. For acute assessment, the complete distribution of the appropriate field trial data was used.

Tomato. The time limited section 408 tolerance for fenpropathrin in/on tomato is 0.6 ppm. The use pattern allows a maximum single application rate of 0.2 lb ai/acre, a total maximum seasonal use of 0.8 lb ai/acre, and a 3-day phi. The field residue experiments were performed in four years ateighteen sites in eight states. There were 27 separate treatments yielding 118 separate, treated samples for analysis. For the subset of the field residue samples that most closely match the present, labeled use pattern, 0.2 lb ai/acre, 4 (or more) applications, and a 3-day phi, the average residue was 0.166 ppm ($n = 54$, $\sigma_{n-1} = 0.132$). The highest average residue (HAR) found in these crop field trials for fenpropathrin in/on tomatoes was 0.55 ppm.

A tomato processing study using an exaggerated field application rate showed positive concentration in wet and dried tomato pomace. However, Agency guidance has indicated that no additional tolerances are needed.

The mean residue value of 0.17 ppm was used for all tomatoes in the chronic dietary assessment, and for the blended commodities in the acute assessment (paste, puree, juice, and catsup). In the acute assessments, a complete distribution of the appropriate field trial data was used for whole and dried tomatoes. Appropriate concentration factors were used for processed commodities: tomato juice (0.05), canned tomatoes (0.08), tomato paste (0.3).

Secondary residues. Residues in animal feed may transfer to animal products, meat, milk, and eggs, used in human food. The existing time limited tolerances are meat and meat by-products of cattle, goats, hogs, horses and sheep at 0.1 ppm, fat of cattle, goats, hogs, horses and sheep at 1.0 ppm, milk fat (reflecting 0.08 ppm in whole milk) at 2.0 ppm, and poultry meat, fat, meat by-products and eggs at 0.05 ppm. The feed items that are associated with the existing registered uses for beef and dairy cattle are peanut hay, cottonseed, cotton gin by-products (feeding restriction), cottonseed hulls, cottonseed meal and peanut meal in descending order of the magnitude of the anticipated residues. For poultry and swine only cottonseed and peanut meals are significant feed items. Tissue to feed residue ratios vary from a high of 0.0139 in fat to 0.001625 in milk, to a low of 0.00004 in liver in cattle. In poultry, tissue to feed ratios vary from a high of 0.0069 in fat to a low of 0.0002 in muscle. Both chronic and acute dietary assessments show very low residue contribution from secondary residues in animal products to all population subgroups.

B. Toxicological profile

Summary. The existing registrations and tolerances of fenpropathrin are supported at EPA by a complete toxicology data base. Toxicity endpoints of concern have been identified by the Agency's Health Effects Division, Hazard Identification Assessment Review Committee. The identified endpoints are an Acute Dietary of 6.0 mg/kg/day (systemic) and a Chronic Dietary of 2.5 mg/kg/day (RfD = 0.025 mg/kg/day, UF = 100). No endpoints of concern were identified by the Committee for occupational or residential, dermal or inhalation exposures of any duration.

1. *Acute toxicity*. The following acute toxicity studies using fenpropathrin technical as the test material have been reviewed and accepted by EPA to support registration.

Acute oral, rat. The rat oral LD_{50} values were determined to be 54.0 and 48.5 milligrams per kilogram body weight (mg/kg) for male and female rats, respectively. Toxicity Category I.

Acute dermal, rat. The rat dermal LD_{50} values were determined to be 1600 and 870 mg/kg for male and female rats, respectively. Toxicity Category II.

Acute inhalation, rat. A high dosage inhalation study is technically not possible because of the low vapor pressure and thick, viscous nature of fenpropathrin technical. The study has been waived by the Agency. Toxicity Category IV.

Primary eye irritation, rabbit. No corneal involvement; mild iris and conjunctival irritation. Toxicity Category III.

Primary dermal irritation, rabbit. No irritation. Toxicity Category IV.

Dermal sensitization, guinea pig. Not a sensitizer.

Acute oral and acute dermal toxicity studies have also been submitted on the mouse and rabbit. In the acute oral and dermal studies, clinical signs of toxicity included tremors, hyperexcitability, muscular fibrillation, ataxia of the hind limbs, urinary incontinence, diarrhea, and salivation. The intoxicated animals from the oral studies showed no major changes in tissues or organs at necropsy. Where there were sexual differences in toxicity, females were consistently slightly more sensitive than males. Surviving animals recovered in two days in the case of rats and mice and within 4 days in the case of rabbits. In surviving animals, all clinical signs were completely reversible.

2. *Genotoxicty*. Fenpropathrin does not present a genetic hazard. The Agency has reviewed, accepted, and classed as negative the following genotoxicity tests: A gene mutation assay (Ames), a chromosomal aberration study in rodents, an in vitro cytogenics assay, a sister chromatide exchange on CHO-K1 cells, and DNA damage/repair in *Bacillus subtilis*.

3. *Reproductive and developmental toxicity*. There is no evidence from reproduction or developmental toxicity studies that the developing fetus, young growing and developing animals, or adult reproducing animals are any more sensitive to fenpropathrin effects than mature adult animals. In addition, reproductive parameters were

unaffected at dosages higher than those that caused overt adult toxicity.

Three-generation reproduction study, rats. Dietary concentrations of 0, 40, 120, and 360 ppm were fed continuously to rats for three generations to assess the effect of fenpropathrin on reproductive function. (Parent) Systemic no effect level (NOEL) of 40 ppm (M/F 3.0/3.4 mg/kg/day). Systemic lowest effect level (LEL) of 120 (M/F 8.9/10.1 mg/kg/day)—body tremors with spasmodic muscle twitches, increased sensitivity, and maternal lethality. Reproductive NOEL 120 ppm (M/F 8.9/10.1 mg/kg/day). Reproductive LEL 360 ppm (M/F 26.9/32.0 mg/kg/day)—Decreased mean F_2 loss. (Pups) Developmental NOEL 40 ppm (M/F 3.0/3.4 mg/kg/day). Developmental LEL 120 ppm (M/F 8.9/10.1 mg/kg/day)—body tremors, increased mortality.

Developmental toxicity, rabbits. Female rabbits were treated by gavage on days 7 through 19 of pregnancy with 0, 4, 12, and 36 mg/kg/day in corn oil to assess the maternal and developmental toxicity of fenpropathrin. Maternal NOEL 4 mg/kg/day, maternal LEL 12 mg/kg/day (grooming, anorexia, flicking of the forepaws). Developmental NOEL > 36 mg/kg/day, there were no compound-related effects on development. Clinical signs included grooming, anorexia, flicking of the forepaws and hindfeet, shaky movements, trembling, stamping of the hindfeet, and lethargy.

Developmental toxicity, rats. Female rats were treated by gavage on days 6 through 15 of pregnancy with 0, 0.4, 1.5, 2.0, 3.0, 6.0 and 10 mg/kg/day in corn oil to assess the maternal and developmental toxicity of fenpropathrin. Maternal NOEL 6 mg/kg/day, maternal LEL of 10 mg/kg/day (death, moribundity, ataxia, sensitivity to external stimuli, spastic jumping, tremors, prostration, convulsion, hunched posture, squinted eyes, chromodacryorrhea, and lacrimation). Developmental NOEL > 10 mg/day. No developmental effects were observed at a dose that was lethally neurotoxic to 7 of 30 dams.

4. *Subchronic toxicity- Subchronic feeding, rat 3-month*. Fenpropathrin was fed to rats at dietary concentrations of 0. 3, 30, 100, 300 and 600 ppm. The NOEL was determined to be 300 ppm (15 mg/kg/day). The LEL was 600 ppm (30 mg/kg/day)—body weight reduction (F), body tremors, reduced kaolin-cephalin clotting time (F), increased alkaline phosphatase and potassium (M), increased brain (F) and kidney (M) weights.

Subchronic feeding, dog 3-month. Groups of six male and six female beagle dogs were fed diets containing 250, 500. and 750 ppm fenpropathrin for 13 weeks. The NOEL was not determined and is less than 250 ppm (7.25 mg/kg/day). At this dosage there were signs of GI tract disturbance (note dog chronic, below). At higher feeding levels the following effects were observed: 500 ppm (15 mg/kg/day) produced tremors and body weight loss in females, 750 ppm (22.25 mg/kg/day) produced tremors, ataxia and blood changes (reduced RBC, HCT, HGB).

Dermal, rabbit 21-day. Ten rabbits of each sex at each dose, half with intact skin and half with abraded skin, were treated dermally with 500, 1200 and 3000 mg/kg/day. The experimental animals were treated 5 days per week for three weeks. There was localized dermal irritation but there were no systemic effects. The systemic NOEL was determined to be greater than 3000 mg/kg/day.

5. *Chronic toxicity*. A complete chronic data base supported by appropriate subchronic studies for fenpropathrin is available to the Agency. A chronic RfD has been identified, and a safety factor of 100 is appropriate. Fenpropathrin shows no evidence of oncogenicity at maximum tolerated dosages. Clinical signs of chronic toxicity were observed as body tremors, at high dosages with little other effects noted.

Oral toxicity study, dogs 12-month. Groups of male and female beagle dogs were fed diets containing 0, 100, 250. and 750 ppm fenpropathrin for 52 weeks. Systemic NOEL of 100 ppm (2.5 milligram (mg)/kilogram (kg)/day) and a systemic LEL of 250 ppm (6.25 mg/kg/day).

Chronic/carcinogenicity feeding, rat 24-month. Groups of male and female Charles River CD rats were fed diets containing 0, 50, 150, 450, and 600 ppm fenpropathrin for 104 weeks. Systemic NOEL's of 450 ppm in males, 150 ppm in females (17.06 mg/kg/day and 7.23 mg/kg/day, respectively). Systemic LEL of 600 ppm [(HDT): 22.80 mg/kg/day] in males (increased mortality, body tremors, increased pituitary, kidney, and adrenal weights), and systemic LEL of 450 ppm (19.45 mg/kg/day) in females (increased mortality and body tremors). There were no oncogenic effects observed at any dose level.

Chronic/carcinogenicity feeding study, mouse 24-month. Groups of male and female Charles River (UK) CD-1 mice were fed diets containing 0, 40, 150, and 600 ppm fenpropathrin for 104 weeks. Systemic NOEL greater than 600 ppm HDT (males and females; 56.0 and 65.2 mg/kg/day, respectively). There were no indications of toxicity or carcinogenicity other than marginally increased hyperactivity in females dosed at 600 ppm.

Carcinogenicity. Fenpropathrin has been classified in EPA Weight-of-the Evidence Category ''Group E—Evidence of Non-Carcinogenicity for Humans'' for carcinogenicity by the EPA/RFD/PR committee reviewed 1/29/93 and EPA verified 3/18/93. Studies in two species with adequate dosing show no evidence of oncogenicity.

6. *Animal metabolism*. Acceptable rat metabolism studies have been performed using single high (25 mg/kg), and single and multiple low (2.5 mg/kg) doses using both sexes. Elimination was similar in both sexes. The urine: feces ratio of elimination was 1:2 following the high or low single dose, and 1:1 following the 15 daily doses. The half life was 11-16 hours in the urine, and 7-9 hours in the feces. After 7 days, greater than 99% of the administered dose was excreted. A small percentage of radiolabel was found in the tissues (primarily in the fat). The major biotransformations included cleavage of the ester, oxidation at the methyl group of the acid moiety, and hydroxylation at the 4'-position of the alcohol moiety. Ester cleavage products, 2,2,3,3-tetramethylcyclopropanecarboxylic acid and (after oxidation) 3-phenoxybenzoic acid, were excreted either directly or conjugated as sulfates or glucuronides. Parent was detected in the feces, but not in the urine. Eight urinary metabolites and 4 fecal metabolites were identified.

There are no qualitatively unique plant metabolites . The primary aglycones are identical in both plants and animals; the only difference is in the nature of the conjugating moieties employed.

7. *Metabolite toxicology*. The metabolism and potential toxicity of the small amounts of terminal plant metabolites have been tested on mammals. Glucoside conjugates of 3-phenoxy-benzyl alcohol and 3-phenoxybenzoic acid, administered orally to rats, were absorbed as the corresponding aglycones following cleavage of the glycoside linkage in the gut. The free or reconjugated aglycones were rapidly and completely eliminated by normal metabolic pathways. The glucose conjugates of 3-phenoxybenzyl alcohol and 3-phenoxy-benzoic acid are less toxic to mice than the corresponding aglycones.

8. *Endocrine disruption*. No special studies to investigate the potential for estrogenic or other endocrine effects of fenpropathrin have been performed. However, as summarized above, a large

and detailed toxicology data base exists for the compound including studies acceptable to the Agency in all required categories. These studies include evaluations of reproduction and reproductive toxicity and detailed pathology and histology of endocrine organs following repeated or long term exposure. These studies are considered capable of revealing endocrine effects and no such effects were observed.

C. Aggregate Exposure

1. *Dietary exposure*. Toxicity endpoints of concern have been identified by the Agency's Health Effects Division, Hazard Identification Assessment Review Committee (July 17 and 24, 1997). The identified endpoints are a Chronic Dietary of 2.5 mg/kg/day (RfD = 0.025 mg/kg/day, UF = 100) and an Acute Dietary of 6.0 mg/kg/day (systemic). Thus, both chronic and acute exposure and risk analyses are necessary.

2. *Food*. Chronic and acute dietary exposure analyses were performed for fenpropathrin using anticipated residues and accounting for proportion of the crop treated. The crops included in the analyses are cottonseed, currants, peanuts, strawberries, tomatoes, and the secondary residues in meat, milk, and eggs. These exposure/risk analyses have been submitted to the Agency along with a detailed description of the methodology and assumptions used.

Chronic dietary exposure was calculated for the U.S. population and 26 population subgroups. The results from several representative subgroups are listed below. In all cases, chronic dietary exposure was at or below 0.2 % of the reference dose and strawberries was the commodity contributing the most exposure.

Population subgroup	Exposure (mg/kg bw/day)	Percent of RfD
Total U.S. Population	0.000020	008
Females (13+/Nursing)	0.000036	0.14
Non-Hispanic other than B/W	0.000053	0.21
Children (1-6 Years)	0.000035	0.14
All Infants (<1 Year Old)	0.000002	0.008
Non-Nursing Infants (<1 Year Old)	0.000003	0.012

Acute dietary exposure was calculated for the U.S. population and five children subgroups. The calculated exposures and margins of exposure (MOE) for the higher exposed proportions of the subgroups are listed below. It should be noted that the population sizes are small at the lower probability exposures (e.g. 99th and 99.9th percentiles) oftentimes leading to unrealistically high calculated exposures. In all cases, margins of exposure exceed one-hundred.

Calculated Acute Dietary Exposures to Fenpropathrin Residues in Food

Population Subgroup	99th Percentile		99.9th Percentile	
	Exposure (mg/kg bw/day)	MOE	Exposure (mg/kg bw/day)	MOE
U.S. Population	.000682	8,804	.002800	2,143
Children 1-6	.000916	6,547	.007465	804
Children 7-12	.000619	9,687	.003012	1,992
All Infants	.001084	5,533	.001510	3,974
Nursing Infants (<1)	.000297	20,230	.000416	14,412
Non-Nursing Infants (<1)	.001237	4,851	.001572	3,816

3. *Drinking water*. Since fenpropathrin is applied outdoors to growing agricultural crops, the potential exists for fenpropathrin or its metabolites to reach ground or surface water that may be used for drinking water. Fenpropathrin is extremely insoluble in water (14 ppb), with a high octanol/water partitioning coefficient (K_{ow} 1.19 x 10^5) and a relatively short soil half-life for parent and environmental metabolites. The Agency has determined that it is unlikely that fenpropathrin or its metabolites can leach to potable groundwater. The residence time of fenpropathrin in surface water is short because of its very low water solubility and high affinity to bind to soil. In pond studies, fenpropathrin half-lives in the water column were less than 1.5 days.

To quantify the potential small exposure from drinking water, screening evaluations of leaching potential of a typical pyrethroid, cypermethrin, were conducted using EPA's Pesticide Root Zone Model (PRIZM3). Based on this assessment, the potential concentrations of the pyrethroid in groundwater at depths of 1 to 2 meters are essentially zero (<< 0.001 parts per billion). Potential surface water concentrations for the pyrethroid were estimated using PRIZM3 coupled with EPA's Exposure Analysis Modeling System (EXAMS) using standard EPA cotton runoff and Mississippi farm pond scenarios. The maximum concentration predicted in the simulated pond water was 0.052 ppb. Using standard assumptions about body weight and water consumption, the chronic exposure from this drinking water would be 1.5 x 10^{-6} and 5.2 x 10^{-6} mg/kg bw/day for adults and children, respectively; less than 0.02 percent of the RfD for children. Concentrations in actual drinking water would be much lower than the levels predicted in the hypothetical small stagnant farm pond modeled, since drinking water from surface sources receives treatment prior to consumption. Based on these analyses, the contribution of water to any the dietary risk analyses is negligible.

4. *Non-dietary exposure*. Fenpropathrin, as the product TAME

2.4 EC Spray, is registered for professional non-food use both indoors and outdoors on ornamentals and non-bearing nursery fruit trees. Fenpropathrin has no animal health, homeowner, turf, termite, or industrial uses. Quantitative information concerning human exposure from this ornamental use is not available, but exposure to the general public from this use of fenpropathrin is expected to be minimal. It is important to note that no endpoints of concern were identified by the Health Effects Division, Hazard Identification Assessment Review Committee for occupational or residential, dermal or inhalation exposures of any duration. Thus, no risk assessment is needed.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that the Agency must consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' ''Available information'' in this context 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.

There are numerous other pesticidal compounds, pyrethroids and natural pyrethrins, that are structurally related to fenpropathrin and may have similar effects on animals. In consideration of potential cumulative effects of fenpropathrin and other substances that may have a common mechanism of toxicity, there are currently no available data or other reliable information indicating that any toxic effects produced by fenpropathrin would be cumulative with those of other chemical compounds. Thus, only the potential risks of fenpropathrin have been considered in this assessment of aggregate exposure and effects.

Valent will submit information for EPA to consider concerning potential cumulative effects of fenpropathrin consistent with the schedule established by EPA at 62 FR 42020 (August 4, 1997) and other EPA publications pursuant to the Food Quality Protection Act.

E. Safety Determination

The Food Quality Protection Act introduces a new standard of safety, a reasonable certainty of no harm. To make this determination, at this time the Agency should consider only the incremental risk of fenpropathrin in its exposure assessment. Since the potential chronic and acute exposures to fenpropathrin are small (<< 100% of RfD, MOE >> 100) the provisions of the FQPA of 1996 will not be violated.

1. *U.S. population— Chronic*. Using the dietary exposure assessment procedures described above for fenpropathrin, chronic dietary exposure is minimal with all population subgroups at or below 0.2 percent of the RfD. Addition of the small potential chronic exposure from drinking water (calculated above) increases the occupancy of the RfD by only 0.006 percent. Generally, the Agency has no cause for concern if total residue contribution is less than 100 percent of the RfD.

Acute. The potential acute exposure from food to the U.S. population (shown above) provides an MOE greatly exceeding 100. In a conservative policy, the Agency has no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger.

2. *Infants and children— Safety factor for infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of fenpropathrin, FFDCA section 408 provides that EPA shall apply an additional margin of safety, up to ten-fold, for added protection for infants and children in the case of threshold effects unless EPA determines that a different margin of safety will be safe for infants and children.

The toxicological data base for evaluating pre- and post-natal toxicity for fenpropathrin is complete with respect to current data requirements. There are no special pre- or post-natal toxicity concerns for infants and children, based on the results of the rat and rabbit developmental toxicity studies or the three-generation reproductive toxicity study in rats. EPA HED Hazard ID Committee has concluded that reliable data support use of the standard 100-fold uncertainty factor and that an additional uncertainty factor is not needed to be further protective of infants and children.

Chronic risk. Using the conservative exposure assumptions described above, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of fenpropathrin ranges from 0.14 % for children (1–6 years old) and 0.012% for non-nursing infants (< 1 year old). Addition of the small potential chronic exposure from drinking water (calculated above) increases the occupancy of the RfD by only 0.02 percent. Generally, the Agency has no cause for concern if total residue contribution is less than 100 percent of the RfD.

Acute. The potential acute exposure from food to populations of infants and children (shown above) provide MOE values greatly exceeding 100 In a conservative policy, the Agency has no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger.

Aggregate acute or chronic dietary exposure to various sub-populations of children and adults demonstrate acceptable risk. Chronic exposures to fenpropathrin occupy considerably less than 100% of the RfD, and all acute MOE values exceed 100. Chronic and acute dietary risk to children from fenpropathrin should not be of concern. Further, fenpropathrin has no other uses, such as indoor pest control, homeowner or turf, that could lead to unique, enhanced exposures to vulnerable sub-groups of the population. It can be concluded that there is a reasonable certainty that no harm will result to any sub-group of the U.S. population, including infants and children, from aggregate chronic or acute exposure to fenpropathrin residues.

F. International Tolerances

Codex Maximum Residue Limits

186	Fenpropathrin
Main uses	8 Insecticide/acaracide
JMPR	83
ADI	0.03 mg/kg body weight (1993)

Codex Maximum Residue Limits—Continued

Residue	Fenpropathrin (fat soluble)

Commodity		MRL (mg/kg)	Step	JMPR	CCPR
Code	Name				
MM 0812	Cattle meat	0.5 (fat)	6	93	
ML 0812	Cattle milk	0.1 F	6st	93	
MO 0812	Cattle, Edible offal of	0.05	CXL		(1995)
SO 0691	Cotton seed	1	CXL		(1995)
OC 0691	Cotton seed oil, Crude	3	CXL		(1995)
VO 0440	Egg plant	0.2	6	93	
PE 0112	Eggs	0.01 (*)	CXL		(1995)
VC 0425	Gherkin	0.2	CXL		(1995)
FB 0269	Grapes	5	6	93	
VO 0445	Peppers, Sweet	1	CXL		(1995)
FP 0009	Pome fruits	5	CXL		(1995)
PM 0110	Poultry meat	0.02 (fat)	CXL		(1995)
PO 0111	Poultry, Edible offal of	0.01 (*)	CXL		(1995)
VO 0448	Tomato	1	CXL		(1995)

There are small differences between the section 408 tolerances and the Codex MRL values for secondary residues in animal products. These minor differences are mainly caused by differences in the methods used to calculate animal feed dietary exposure. The only substantial difference between the US tolerance and the Codex MRL value is for tomatoes. The JMPR reviewer required that the MRL exceed the highest field residue, and rounded to unity. The EPA reviewer agreed with Valent that one set of field residue samples was possibly compromised by the presence of a high rate processing treatment nearby. High outliers were ignored, and the tolerance was set at 0.6 ppm. (Adam Heyward)

9. Zeneca Ag Products

PP 7G3518, 7F3521, 4F4406

EPA has received a request from Zeneca Ag Products, P. O. Box 15458, Wilmington, DE, 19850–5458 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by removing the time limitation for tolerances established for residues of the insecticide and pyrethriod Tefluthrin in or on the raw agricultural commodities corn, grain, field and pop; corn, forage and fodder, field, pop and sweet; and corn, fresh (including sweet K and corn with huskremoved (CWHR)) at 0.06 ppm. The International Union of Pure and Applied Chemist (IUPAC) name for tefluthrin is (2,3,5,6-tetrafluro-4-methylphenyl)methyl-(1 alpha, 3 alpha)-(Z)-(+/−)-3(2-chloro-3,3,3-trifluoro-1-propenyl)-2,2-dimethylcyclopropanecarboxylate) and its metabolite (Z)-3-(2-chloro-3,3,3-trifluroro-1-propenyl)-2,2-dimethylcyclopropanecarboxylic acid. The tolerances were originally requested in Pesticide Petition Numbers 7G3518, 7F3521, and 4F4406. 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 tefluthrin residues in plants and animals for corn use is adequately understood. The residue of concern is tefluthrin and its metabolite. There is no reasonable expectation of secondary residues in animal tissues and milk from the use as delineated in 40 CFR 180.6(a)(3).

2. *Analytical method*. An adequate analytical method, gas liquid chromatography with an electron capture detector, is available for enforcement purposes. The enforcement methodology has been submitted to the Food and Drug Administration, and is published in the Pesticide Analytical Manual Vol. II (PAM II).

3. *Magnitude of residues*. Tefluthrin (also know as FORCE® Insecticide) is an effective granular soil insecticide registered for use against a number of soil corn pest; the most economically significant being soil dwelling pest, such as corn rootworm, wireworm, cutworm, and white grubs. Residue data covering all the uses associated with the permanent tolerances requested by this petition have been previously submitted to EPA for review and have been found by EPA to support the requested tolerances. See February 1, 1989 (54 FR 5080); and May 3, 1996 (61 FR 19852) (FRL–5358–5).

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies with the technical grade of the active ingredient tefluthrin: Oral LD_{50} in the rat is 22 mg/kg) for (males) and 35 mg/kg for (females); dermal LD_{50} in the rat is 316 mg/kg in (males) and 177 mg/kg in (females); acute inhalation LC_{50} in the rat is 0.04 milligram/liter (mg/l) and 0.05 mg/l in female and male rats, respectively; primary eye irritation in the rabbit study showed slight irritation; primary dermal irritation in the rabbit study showed none to slight irritation, and the dermal sensitization in the guinea pig study showed no skin sensitization.

2. *Genotoxicty*. The following genotoxicity test were all negative: A gene mutation assay (Ames), dominant lethal (mouse *in vivo*), mouse micronucleus (*in vivo*), acute cytogenetic study in the rat, unscheduled DNA synthesis and a mouse lymphoma cells test.

3. *Reproductive and developmental toxicity*. In a rat developmental study,

delayed ossification was noted in the highest dose group (5 mg/kg/day), along with significant maternal toxicity (decreased body weight (bwt)). The developmental no observed effect level (NOEL) for this study was established at 3 mg/kg/day. However, the effects observed were most likely a secondary effect resulting from maternal stress.

In a developmental toxicity study in rabbits given gavage doses of 0, 3, 6, and 12 mg/kg/day, the maternal NOEL is 3 mg/kg/day and the developmental NOEL is > 12 mg/kg/day. No developmental effects were observed under the conditions of the study.

In a rat multi-generation reproduction study, conducted at 0, 15, 50, and 250 ppm with tefluthrin in the diet, a reproductive NOEL was established at 50 ppm (3.4 mg/kg/day) based on reduced pup weight and litter size observed at 250 ppm (12.5 mg/kg/day). Parental toxicity (in the form of abnormal, sprayed, or high-stepping gait) was also observed at 250 ppm. Thus, the effects observed in offspring at 250 ppm is considered to be secondary to maternal toxicity.

4. *Subchronic toxicity*. A 90–day feeding study in which rats were fed doses of 0, 50, 150, and 350 ppm with a NOEL of 50 ppm and a lowest observed effect level (LOEL) of 150 ppm based on mild dose changes in hemoglobin, cholesterol, and liver weight.

A 90–day feeding study in which dogs were fed doses of 0, 0.1, 0.5, and 1.5 mg/kg with a NOEL of 0.5 mg/kg and a LOEL of 1.5 mg/kg based on increased triglycerides and AST.

A 21–day dermal study in which rats were exposed dermally to doses of 1, 5, and 50 mg/kg/day, 6 hours/day with a toxicological NOEL of 1mg/kg.

5. *Chronic toxicity*. A 12–month feeding study in dogs was conducted with a NOEL of 0.5 mg/kg/day. The LOEL for this study is established at 2 mg/kg/day based upon ataxia.

A 24–month rat and mouse chronic feeding/oncogenicity studies were conducted with systemic NOEL's of 1.1mg/kg/day and 3.4 mg/kg/day with no oncogenic effects observed at dose levels up to and including 18.2 mg/kg/day and 54.4 mg/kg/day, the highest dose levels tested for rats and mice, respectively.

6. *Animal metabolism*. A metabolism study in the rat demonstrated that distribution patterns and excretion rates in multiple oral dosing periods are similar to single-dose studies. The metabolism of tefluthrin in livestock has been studied in the goat and chicken. The nature of tefluthrin residue in animals for corn use is adequately understood. The residue of concern is tefluthrin and its metabolite. There is no reasonable expectation of secondary residues in animal tissues and milk from the use as delineated in 40 CFR 180.6(a)(3).

7. *Metabolite toxicology*. The nature of tefluthrin residue in plants and animals for corn use is adequately understood. The residue of concern is tefluthrin and its metabolite. There is no reasonable expectation of secondary residues in animal tissues and milk from the use as delineated in 40 CFR 180.6(a)(3). An adequate analytical method, gas liquid chromatography with an electron capture detector, is available for enforcement purposes. The enforcement methodology has been submitted to the Food and Drug Administration, and is published in the Pesticide Analytical Manual Vol. II (PAM II).

8. *Endocrine disruption*. EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect produced by a naturally occurring estrogen, or such other endocrine 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 passage of the Food Quality Protection Act (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.

C. Aggregate Exposure

The primary source of human exposure to tefluthrin will be from ingestion of raw and processed food commodities which have been treated with tefluthrin. These commodities include corn, grain, field and pop; corn, forage and fodder, field, pop and sweet; and corn, fresh (including sweet K and CWHR) as listed in 40 CFR 180.440. There is no reasonable expectation of secondary residues in animal tissues, milk, or eggs from use as delineated in 40 CFR 180.6(a)(3).

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure under these tolerances, aggregate exposure is estimated based on the Theoretical Maximum Residue Contribution (TMRC) from the existing tolerances for tefluthrin in food crops. The TMRC is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of those food products eaten by various population subgroups. The following assumptions were used in conducting this exposure assessment: 100 percent of the crops were treated, and the raw agricultural commodities (RAC) residues would be at the level of the tolerance. This results in an overestimate of human exposure and a conservative assessment of risk.

2. *Food*. The acute dietary risk assessment used tolerance level residues and assumed that 100 percent of all crops were treated. Thus, this acute dietary exposure estimate is considered ''worst-case'' and severely overestimates potential exposure. The acute dietary Margin of Exposure (MOE) for the most highly exposed population subgroup was children ages one to six. The MOE's were 2,436 at the 95th percentile, 1,342 at the 99th percentile, and 738 at the 99.9th percentile. EPA concludes that there is a reasonable certainty of no harm for MOE of 100 or greater. Therefore, the acute dietary risk assessment for tefluthrin clearly indicates a reasonable certainty of no harm.

For the chronic dietary assessment Zeneca used the standard EPA conservative exposure assumptions (i.e. tolerance level residues and 100 percent market share), and based on the completeness and reliability of the toxicity data Zeneca has concluded that the aggregate exposure to this chemical will utilize less than one percent (0.40 percent) of the reference dose (RfD) for the U. S. population. The most highly exposed population subgroup was children ages one to six with a total dietary exposure of 0.000049 mg/kg bwt/day (1.0 percent of the RfD). Since EPA generally has no concern for exposures below 100 percent of the RfD, there is a reasonable certainty that no harm will result from aggregate exposure to residues.

3. *Drinking water*. Tefluthrin is immobile in soil and, therefore, will not leach into ground water. Additionally, due to the insolubility and lipophilic nature of tefluthrin, 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 synthetic pyrethroid was conducted using EPA's Pesticide Root Zone Model (PRZM3). Based on this screening assessment, potential concentrations of a pyrethroid in ground water at depths of 1 to 2 meters are essentially zero (<0.001 ppb). Surface water concentrations for pyrethroids were estimated using PRZM3 and Exposure Analysis

Modeling Systems (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.

4. *Non-dietary exposure*. The potential for non-occupational exposure to the general population is expected to be essentially zero. Tefluthrin is not registered for aquatic and/or domestic outdoor or indoor uses. The major use (corn) is applied only once per year at planting as a granular formulation. The other use is limited to commercial seed treatment of field corn, popcorn, seedcorn, and sweet corn seed. There is a commercial use in liquid slurry seed treaters and seed coating equipment, which is not for use on agricultural establishments in hopper-box, planter-box, slurry-box, or other seed treatment applications. The other minor use is for the treatment of fire ants for containerized and balled nursery stock under the USDA/APHIS Imported Fire Ant Quarantine Program (Department of Agriculture-Animal and Plant Health Inspection Service-7 CFR part 301).

D. Cumulative Effects

Zeneca will submit information for EPA to consider concerning potential cumulative effects of tefluthrin consistent with the schedule established by EPA on August 4, 1997 (62 FR 42020) (FRL–5734–6) and other EPA publications pursuant to the FQPA. At this time, Zeneca cannot make a determination, based on available and reliable information, that tefluthrin and other substances that may have a common mechanism of toxicity would have cumulative effects. Therefore, for purposes of this request it is appropriate only to consider the potential risks of tefluthrin in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. EPA recently reviewed all of the toxicity end points for the synthetic pyrethroids. Based on this review EPA concluded that the chronic RfD is 0.005 mg/kg/day. This RfD is based on a 1–year dog feeding study with a NOEL of 0.5 mg/kg/day for ataxia, and a 100–fold uncertainty factor. In addition, EPA derived an acute NOEL of 0.5 mg/kg/day for use in acute dietary risk assessment. This NOEL is based on the 1–year dog feeding study in which increased incidence of tremors in both sexes of dogs was observed on the first day of dosing.

Using these RfD's and EPA's standard default assumptions (i.e. tolerance level residues and 100 percent market share), Zeneca assessed the potential acute and chronic dietary risk to the general U.S. population and 22 subpopulations. These analyses are considered ''worst-case'', and the results concluded that for the U.S. population, uses were 0.000021 mg/kg/day (0.4 of the RfD). The acute MOE's at the 95th, 99th, and 99.9th percentile were 5.195, 2,449, and 1,091 respectively. The most highly exposed population subgroup (children ages one to six), utilizes 1.0 percent of the chronic RfD, and the acute dietary MOE's at the 95th, 99th, and 99.9th percentiles were 2,436, 1,342, and 738, respectively. These assessments indicate a reasonable certainty that no harm will result from aggregate exposure to residues.

2. *Infants and children*. Section 408 of the FFDCA provides that EPA shall apply an additional 10–fold 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. EPA generally defines the level of appreciable risk as exposure that is greater than 1/100 of the NOEL in the animal study appropriate to the particular risk assessment. This 100–fold uncertainty (safety) factor/margin of exposure is designed to account for combined inter- and intra-species variability. EPA believes that reliable data support using the standard 100–fold margin/factor, not the additional 10–fold margin/factor, when EPA has a complete database under existing guidelines and when the severity of the effect in infants and children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard margin/factor.

In assessing the potential for additional sensitivity of infants and children to residues of tefluthrin, EPA considered the data from oral developmental toxicity studies in the rat and rabbit, as well as data from a multi-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects in the developing organism resulting from pesticide exposure during prenatal development in the mothers. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

3. *Pre-natal effects*. In a rat developmental study delayed ossification was noted in the highest dose group (5 mg/kg/day), along with significant maternal toxicity (decreased bwt). The developmental NOEL for this study was established at 3 mg/kg/day. However, the effects observed were most likely a secondary effect resulting from maternal stress.

In a developmental toxicity study in rabbits given gavage doses of 0, 3, 6, and 12 mg/kg/day the maternal NOEL is 3 mg/kg/day, and the developmental NOEL is > 12 mg/kg/day. No developmental effects were observed under the conditions of the study.

4. *Post-natal effects*. In a rat multi-generation reproduction study conducted at 0, 15, 50, and 250 ppm with tefluthrin in the diet, a reproductive NOEL was established at 50 ppm (3.4 mg/kg/day), based on reduced pup weight and litter size observed at 250 ppm (12.5 mg/kg/day). Parental toxicity (in the form of abnormal, sprayed, or high-stepping gait) was also observed at 250 ppm. Thus, the effects observed in offspring at 250 ppm is considered to be secondary to maternal toxicity.

In EPA's review of the toxicity endpoints for tefluthrin they concluded that the data on developmental and reproductive toxicity tests do not indicate any increased pre- or post-natal sensitivity. Therefore, EPA concluded that reliable data support use of a 100–fold safety factor, and additional 10–fold safety factor is not needed. This aggregate assessment of tefluthrin clearly demonstrates that there is no harm for all population groups.

F. International Tolerances

There are no Codex Maximum Residue Levels (MRL's) established for tefluthrin. (John Hebert)

10. Zeneca Ag Products

PPs 7F3560, 7H5543, 7F3488, 1F3952, 1H5607, 1F3992, 2F4109, 2F4100, 2F4114, 1F3985, and 6F4769

EPA has received a request from Zeneca Ag Products, 1800 Concord Pike, P.O. Box 15458, Wilmington, Delaware 19850–5458, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR 180.438 by removing the time limitation for tolerances established for residues of lambda-cyhalothrin and its epimer in or on the following crops and commodities: broccoli at 0.4 ppm;

cabbage at 0.4 ppm; cattle, fat at 3.0 ppm; cattle, meat at 0.2 ppm; cattle, meat and meat by-products (mbyp) at 0.2 ppm; corn, grain (field and pop) at 0.05 ppm; corn, fodder at 1.0 ppm; corn, forage at 6.0 ppm; corn, sweet (k+kwhr) at 0.05 ppm; cottonseed at 0.05 ppm; dry bulb onion at 0.1 ppm; eggs at 0.01 ppm; garlic at 0.1 ppm; goats, fat at 3.0 ppm; goats, meat at 0.2 ppm; goats, mbyp at 0.2 ppm, hogs, fat at 3.0 ppm; hogs, meat at 0.2 ppm; hogs, mbyp at 0.2 ppm; horses, fat at 3.0 ppm; horses, meat at 0.2 ppm; horses, mbyp at 0.2 ppm; lettuce, head at 2.0 ppm; milk, fat (reflecting 0.2 ppm in whole milk) at 5.0 ppm; peanuts at 0.05 ppm; peanuts, hulls at 0.05 ppm; poultry, fat at 0.01 ppm; poultry, meat at 0.01 ppm; poultry, mbyp at 0.01 ppm; rice, grain at 1.0 ppm; rice, hulls at 5.0 ppm; rice, straw at 1.8 ppm; sheep, fat at 3.0 ppm; sheep, meat at 0.2 ppm; sheep, mbyp at 0.2 ppm; soybeans at 0.01 ppm; sorghum, grain at 0.02 ppm; sorghum, grain dust at 1.5 ppm; sunflower, seeds at 0.2 ppm; sunflower, forage at 0.2 ppm; tomatoes at 0.1 ppm; wheat, grain at 0.05 ppm; wheat, forage at 2.0 ppm; wheat, hay at 2.0 ppm; wheat, straw at 2.0 ppm; wheat, grain dust at 2.0 ppm; corn, grain flour at 0.15 ppm; sunflower, oil at 0.30 ppm; sunflower, hulls at 0.50 ppm; tomato pomace (dry or wet) at 6.0 ppm; and wheat, bran at 0.2 ppm. The IUPAC name for lambda-cyhalothrin is a 1:1 mixture of (*S*)-alpha-cyano-3-phenoxybenzyl-(*Z*)-(1R,3R)-3-(2-chloro-3,3,3-trifluoroprop-1-enyl)-2,2-dimethylcyclopropanecarboxylate and (*R*)-alpha-cyano-3-phenoxybenzyl-(*Z*)-(1S,3S)-3-(2-chloro-3,3,3-trifluoroprop-1-enyl)-2,2-dimethylcyclopropanecarboxylate and the epimer of lambda-cyhalothrin is a 1:1 mixture of (*S*)-alpha-cyano-3-phenoxybenzyl-(*Z*)-(1S,3S)-3-(2-chloro-3,3,3-trifluoroprop-1-enyl)-2,2-dimethylcyclopropanecarboxylate and (*R*)-alpha-cyano-3-phenoxybenzyl-(*Z*)-(1R,3R)-3-(2-chloro-3,3,3-trifluoroprop-1-enyl)-2,2-dimethylcyclopropanecarboxylate. These tolerances were originally requested in Pesticide Petition Numbers 7F3560, 7H5543, 7F3488, 1F3952, 1H5607, 1F3992, 2F4109, 2F4100, 2F4114, 1F3985, and 6F4769. EPA has determined that the petitions 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 the request. Additional data may be needed before EPA rules on the request.

A. Residue Chemistry

1. *Plant metabolism*.The metabolism of lambda-cyhalothrin has been studied in cotton, soybean, cabbage and wheat plants. The studies show that the metabolism generally follows that of other pyrethroid insecticides. The ester linkage is cleaved to form cyclopropanecarboxylic acids and the corresponding phenoxybenzyl alcohol. Overall the studies show that unchanged lambda-cyhalothrin is the principal constituent of the residue on edible portions of these crops.

2. *Analytical method*. An adequate analytical method (gas liquid chromatography with an electron capture detector) is available for enforcement purposes.

3. *Magnitude of residues*. Field residue trials, meeting EPA study requirements, have been conducted for each crop in this petition. These data have previously been reviewed and classified by the Agency as supportive of these tolerances.

B. Toxicological Profile

The following toxicity studies have been conducted to support this request.

1. *Acute toxicity*. Acute toxicity studies with the technical grade of the active ingredient lambda-cyahothrin: oral LD_{50} in the rat of 79 millgrams/kilogram (mg/kg) (males) and 56 mg/kg (females), dermal LD_{50} in the rat of 632 mg/kg (males) and 696 mg/kg females, primary eye irritation study showed mild irritation and primary dermal irritation study showed no irritation.

2. *Genotoxicty*. The following genotoxicity tests were all negative: a gene mutation assay (Ames), a mouse micronucleus assay, an *in-vitro* cytogenetics assay, and a gene mutation study in mouse lymphoma cells.

3. *Reproductive and developmental toxicity*. A three-generation reproduction study in rats fed diets containing 0, 10, 30, and 100 ppm with no developmental toxicity observed at 100 ppm, the highest dose tested. The maternal NOEL and LOEL for the study are established at 30 (1.5 mg/kg/day) and 100 ppm (5 mg/kg/day), respectively, based upon decreased parental body weight gain. The reproductive NOEL and LOEL are established at 30 (1.5 mg/kg/day) and 100 ppm (5 mg/kg/day), respectively, based on decreased pup weight gain during weaning.

A developmental toxicity study was conducted in rats given gavage doses of 0, 5, 10, and 15 mg/kg/day with no developmental toxicity observed under the conditions of the study. The developmental NOEL is greater than 15 mg/kg/day, the highest dose tested. The maternal NOEL and LOEL are established at 10 and 15 mg/kg/day, respectively, based on reduced body weight gain.

A developmental toxicity study was conducted in rabbits given gavage doses of 0, 3, 10, and 30 mg/kg/day with no developmental toxicity observed under the conditions of the study. The maternal NOEL and LOEL are established at 10 and 30 mg/kg/day, respectively based on decreased body weight gain. The developmental NOEL is greater than 30 mg/kg/day, the highest dose tested.

4. *Subchronic toxicity*. A 90–day feeding study in rats fed doses of 0, 10, 50 and 250 ppm with a NOEL of 50 ppm and a LOEL of 250 ppm based on body weight gain reduction.

A study where lambda-cyhalothrin in olive oil was applied to the skin of rats for 21 successive days at dose rates of 1, 10, or 100 (reduced to 50 after 2–3 applications) mg/kg/day. A NOEL of 10 mg/kg/day is based on clinical signs of slight general toxicity at 50 mg/kg/day.

5. *Chronic toxicity*. A 12–month feeding study was conducted in dogs fed dose (by capsule) levels of 0, 0.1, 0.5, 3.5 mg/kg/day with a NOEL of 0.1 mg/kg/day. The LOEL for this study is established at 0.5 mg/kg/day based upon clinical signs of neurotoxicity.

A 24–month chronic feeding/carcinogenicity study was conducted with rats fed diets containing 0, 10, 50, and 250 ppm. The NOEL was established at 50 ppm and LOEL at 250 ppm based on reduced body weight gain. There were no carcinogenic effects observed under the conditions of the study.

A carcinogenicity study was conducted in mice fed dose levels of 0, 20, 100, or 500 ppm (0, 3, 15, or 75 mg/kg/day) in the diet for 2 years. A systemic NOEL was established at 100 ppm and systemic LOEL at 500 ppm based on decreased body weight gain in males throughout the study at 500 ppm. The Agency has classified lambda-cyhalothrin as a Group D carcinogen (not classifiable due to an equivocal finding in this study). It is Zeneca's position that no treatment-related carcinogenic effects were observed under the conditions of the study.

6. *Animal metabolism*. Metabolism studies in rats demonstrated that distribution patterns and excretion rates in multiple oral dose studies are similar to single-dose studies. Accumulation of unchanged compound in fat upon chronic administration shows slow elimination. Otherwise, lambda-cyhalothrin was rapidly metabolized and excreted. The metabolism of

lambda-cyhalothrin in livestock has been studied in the goat, chicken, and cow. Unchanged lambda-cyhalothrin is the major residue component of toxicological concern in meat and milk.

Human metabolism of lambda-cyhalothrin was assessed by administering 5 mg lambda-cyhalothrin orally to six male volunteers (average dose was 0.06 mg/kg) and dermally at 20 mg/800 cm^2 to five volunteers. No adverse effects were noted in the individuals given an oral dose, and only mild signs of parasthesia were noted in individuals receiving a dermal dose. Absorption by these two routes of exposure were determined by analysis of urinary metabolites. An average amount of 59% of the oral dose was absorbed. Dermal absorption was extremely low, and estimated to be 0.12% (range 0.04–0.19%).

7. *Metabolite toxicology*. The Agency has previously determined that the metabolites of lambda-cyhalothrin are not of toxicological concern and need not be included in the tolerance expression. Given this determination, it is concluded that there is no need to discuss metabolite toxicity.

8. *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 endocrine 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.

C. Aggregate Exposure

Zeneca has conducted an aggregate exposure assessment for lambda-cyhalothrin. This assessment included exposures resulting from agricultural crop use and non-dietary residential use.

1. *Dietary exposure*. For the purposes of assessing the potential chronic dietary exposure for all existing and pending tolerances for lambda-cyhalothrin, Zeneca has utilized available information on anticipated residues (FDA monitoring data, average field trial residues and processing data) and percent crop treated. For the acute dietary assessment, a Monte Carlo modeling was used to estimate exposure.

2. *Food*. The Agency has stated that the acute dietary risk assessment for lambda-cyhalothrin should be based on a toxicological NOEL from a 1–year dog study. Zeneca disagrees with EPA's selection of a multiple-dose toxicological endpoint (0.5 mg/kg) for the acute dietary risk assessment, and have requested the Agency to base the acute dietary NOEL on single-dose effects. Acute risk, by EPA definition, results from 1–day consumption of food and water, and reflects toxicity which could be expressed following a single oral exposure to pesticide residues. Therefore, an appropriate NOEL must be based on effects noted after a single dose, even if the endpoint is selected from a repeat dose study, such as a 1–year dog. Nonetheless, sufficient margins of exposure are achieved at percentiles of exposure up to and including the 99.9th percentile based on the Agency's NOEL of 0.5 mg/kg.

Based on the Agency's selected acute toxicity endpoint of 0.5 mg/kg bw day, the acute dietary MOE for the most highly exposed population subgroup was children 1–6 years old. The MOEs were 658 at the 95th percentile, 248 at the 99th percentile, and 132 at the 99.9th percentile. EPA concludes that there is a reasonable certainty of no harm for a MOE of 100 or greater. Therefore, the acute dietary risk assessment for lambda-cyhalothrin clearly indicates a reasonable certainty of no harm. The assessment of chronic dietary exposure was estimated to be 5.0% of the chronic reference dose (RfD) for the overall U.S. population. The RfD for lambda-cyhalothrin, 0.001 mg/kg bw /day, is based on the NOEL of 0.1 mg/kg from the 1–year dog study and an Uncertainty Factor of 100. For the most exposed subgroup, children 1–6 years old, the exposure was estimated to be 0.000159 mg/kg bw/day, or 15.9% of the RfD. Since EPA generally has no concern for exposures below 100 percent of the RfD, there is a reasonable certainty that no harm will result from chronic dietary exposure to lambda-cyhalothrin residues.

3. *Drinking water*. Laboratory and field data have demonstrated that lambda-cyhalothrin and its degradates are immobile in soil and will not leach into ground water. Other data show that lambda-cyhalothrin is virtually insoluble in water and extremely lipophilic. As a result, residues reaching surface waters from field runoff will quickly adsorb to sediment particles and be partitioned from the water column. Together these data indicate that residues are not expected in drinking water.

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 pyrethorid in ground water at depths of 1 and 2 meters are essentially zero (< 0.001 parts 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.

4. *Non-dietary exposure*. Other potential sources of exposure are from non-occupational sources such as structural pest control and ornamental plant and lawn use of lambda-cyhalothrin. In its review of toxicity endpoints for assessing risks for lambda-cyhalothrin, the Agency concluded that the most appropriate endpoint for non-dietary risk assessment is 10 mg/kg bw/day based on the NOEL from the 21–day dermal toxicity study. Exposure was estimated using available market use information and surrogate indoor exposure data. The resulting MOEs were 15,000 for the U.S. population, 7,000 for non-nursing infants and 7,200 for children 1–6 years old. The aggregate risk assessment of combined exposures from chronic dietary, drinking water and non-dietary residential sources has been conducted. The resulting MOEs are 14,000 for the U.S. Population, 6,500 for non-nursing infants and 6,500 for children 1–6 years old. EPA concludes that there is a reasonable certainty of no harm for MOE of 100 or greater. Therefore, the non-dietary and overall aggregate risk assessments for lambda-cyhalothrin clearly indicates a reasonable certainty of no harm.

D. Cumulative Effects

Zeneca Ag Products will submit information for EPA to consider concerning potential cumulative effects of lambda-cyhalothrin consistent with the schedule established by EPA at 62 FR 42020 (August 4, 1997)(FRL–5734–6) and other EPA publications pursuant to the FQPA. At this time, Zeneca cannot make a determination based on

available and reliable information that lambda-cyhalothrin and other substances that may have a common mechanism of toxicity would have cumulative effects. Therefore for purposes of this request it is appropriate only to consider the potential risks of lambda-cyhalothrin in an aggregate exposure assessment.

E. Safety Determination

The acceptable RfD based on a NOEL of 0.1 mg/kg bw/day from the chronic dog study and a safety factor of 100 is 0.001 mg/kg bw/day. A chronic dietary exposure/risk assessment has been performed for lambda-cyhalothrin 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.

1. *U.S. population*. The ARC from established tolerances and the current and pending actions are estimated to be 0.00005 mg/kg bw/day and utilize 5.0 per cent of the RfD for the U.S. population. For the acute dietary assessment the MOEs at the 95th, 99th, and 99.9th percentiles are 2074, 742, and 237, respectively.

2. *Infants and children*. 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. EPA generally defines the level of appreciable risk as exposure that is greater than 1/100 of the NOEL in the animal study appropriate to the particular risk assessment. This hundredfold uncertainty (safety) factor/margin of exposure is designed to account for combined inter and intraspecies variability. EPA believes that reliable data support using the standard hundredfold margin/factor and not the additional tenfold margin/factor when EPA has a complete database under existing guidelines and when the severity of the effect in infants and children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard margin/factor.

In assessing the potential for additional sensitivity of infants and children to residues of lambda-cyhalothrin, EPA considered the data from oral developmental toxicity studies in the rat and rabbit, as well as data from a multi-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects in the developing organism resulting from pesticide exposure during prenatal development in the mothers. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

i. *Pre-natal effects.* A developmental toxicity study in rats given gavage doses of 0, 5, 10, and 15 mg/kg/day with no developmental toxicity observed under the conditions of the study. The developmental NOEL is greater than 15 mg/kg/day, the highest dose tested. The maternal NOEL and LOEL are established at 10 and 15 mg/kg/day, respectively, based on reduced body weight gain.

A developmental toxicity study in rabbits given gavage doses of 0, 3, 10, and 30 mg/kg/day with no developmental toxicity observed under the conditions of the study. The maternal NOEL and LOEL are established at 10 and 30 mg/kg/day, respectively based on decreased body weight gain. The developmental NOEL is greater than 30 mg/kg/day, the highest dose tested.

ii. *Post-natal effects.* A three-generation reproduction study in rats fed diets containing 0, 10, 30, and 100 ppm with no developmental toxicity observed at 100 ppm, the highest dose tested. The maternal NOEL and LOEL for the study are established at 30 (1.5 mg/kg/day) and 100 ppm (5 mg/kg/day), respectively, based upon decreased parental body weight gain. The reproductive NOEL and LOEL are established at 30 (1.5 mg/kg/day) and 100 ppm (5 mg/kg/day), respectively, based on decreased pup weight gain during weaning.

In EPA's review of the toxicity endpoints for lambda-cyhalothrin they concluded that the data on developmental and reproductive toxicity tests do not indicate any increased pre- or post-natal sensitivity. Therefore, EPA concluded that reliable data support use of a hundredfold safety factor and that an additional tenfold safety factor is not needed.

Based on this information the ARC for children 1-6 years old, and non-nursing infants (subgroups most highly exposed) utilizes 0.000159 mg/kg bw/day (15.9% of the RfD) and 0.000101 mg/kg bw/day (10.1% of the RfD), respectively. Generally speaking, the Agency has no cause for concern if anticipated residues contribution for all published and proposed tolerances is less than the RfD.

For the acute dietary assessment the MOEs at the 95th, 99th, and 99.9th percentiles are 658, 248, and 132, respectively for children 1–6 years old. For non-nursing infants the MOEs at the 95th, 99th and 99.9th percentiles are 710, 316, and 152, respectively.

F. International Tolerances

There are Codex maximum residue levels established for residues of cyhalothrin, as the sum of all isomers, in or on the following crops and commodities: pome fruits at 0.2 ppm; cabbage, head at 0.2 ppm; potatoes at 0.02 ppm; cotton seed at 0.02 ppm; cotton seed oil, crude at 0.02 ppm; and cotton seed oil, edible at 0.02 ppm. (Adam Heyward)

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FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) Submitted to OMB for Review and Approval

September 19, 1997.

SUMMARY: The Federal Communications Commission, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection(s), as required by the Paperwork Reduction Act of 1995, Pub. L. 104–13. An agency may not conduct or sponsor a collection of information unless it displays a currently valid control number. No person shall be subject to any penalty for failing to comply with a collection of information subject to the Paperwork Reduction Act (PRA) that does not display a valid control number. Comments are requested concerning (a) whether the proposed collection of information is necessary for the proper performance of the functions of the Commission, including whether the information shall have practical utility; (b) the accuracy of the Commission's burden estimate; (c) ways to enhance the quality, utility, and clarity of the information collected; and (d) ways to minimize the burden of the collection of information on the respondents, including the use of automated collection techniques or other forms of information technology.

DATES: Written comments should be submitted on or before October 27, 1997. If you anticipate that you will be submitting comments, but find it difficult to do so within the period of