a common mechanism of toxicity with any other registered pesticides. Therefore, only exposure from buprofezin is being addressed at this time.

E. Safety Determination

The toxicity and residue databases for buprofezin are considered to be valid, reliable and essentially complete. The standard margin of safety approach is considered appropriate to assess the risk of adverse effects from exposure to buprofezin for both acute and chronic effects. EPA has adopted a temporary RfD for buprofezin at 0.002 mg/kg/day. This RfD was based on the systemic lowest effect level (LEL) of 2.0 mg/kg/day (LDT) from a 2–year dog study and using a 1,000-fold uncertainty factor. An extra factor of 10 was added to the standard 100-fold safety factor since the RfD was based on a LEL (rather than a NOEL) and the database lacked an acceptable reproductive study. Additional data have been submitted to upgrade the reproduction study and to support the lowest dose in the 2–year dog study as a NOAEL. With the upgrading of these studies, the critical study for the establishment of a permanent RfD would be the rat chronic/oncogenicity study. The NOEL for this study is 1 mg/kg/day. Applying a standard safety factor of 100 for this study, to account for interspecies extrapolation and intraspecies variation, would result in a RfD of 0.01 mg/kg/day. It is this proposed RfD which was used to assess risk to the public.

1. *U.S. population*. —i. *Acute risk*. EPA has previously selected, in their approval of the section 18 emergency exemption use, a developmental NOEL of 200 mg/kg/day from a rat developmental study for the acute dietary endpoint. However, it appears that this is an inappropriate acute endpoint since the clinical effects noted at the higher dose (800 mg/kg/day) occurred only after at least 5 days of dosing and the fetal effects (reduced fetal body weight and delayed ossification) are not likely to be due to an acute (1 day) exposure. Based on this assessment, AgrEvo has not evaluated the risk from acute exposure to any subgroup of the population. Previously, EPA has assessed the acute risk from use of buprofezin on citrus and cotton to the population subgroup of females 13+ years of age. Using the developmental NOEL of 200 mg/kg/day, the Margin of Exposure (MOE), according to EPA calculations, was 5,000 for this subgroup.

ii. *Chronic risk*. Chronic dietary exposures for the US population as a whole utilize 65% of the buprofezin RfD in the worst case scenario of 100% of crop treated and all residues at the proposed tolerance level (lettuce, cucurbits) and temporary tolerance level (cotton, citrus, meat/milk commodities from the section 18s). In the more realistic scenario, adjusting for the percent crop treated, the U.S. population chronic dietary exposure utilizes only 1.75% of the RfD. There is generally no concern for exposures below 100% of the RfD since it represents the level at or below which noappreciable risks to human health is posed. Therefore, there is reasonable certainty that no harm would result to the U.S. population from exposure to buprofezin.

2. *Infants and children*. Data from rat and rabbit developmental toxicity studies and rat multigeneration reproduction studies are generally used to assess the potential for increased sensitivity to 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 prenatal and postnatal exposure to the pesticide.

No indication of increased sensitivity to infants and children was noted in either of the developmental studies. However, in the reproduction studies, the NOEL for pups (100 ppm) was lower than for adults (1,000 ppm). Based on the intake of buprofezin in pups up to 8 weeks of age, the RfD for children, using a 1,000 fold safety factor, would be 0.01 mg/kg/day. This is the same RfD that is calculated for chronic exposure utilizing the rat chronic/oncogenicity study.

Evaluation of the dietary exposure to infants and children was conducted utilizing the same assumptions as for the U.S. population as a whole. Adjustment for the percent crop treated resulted in dietary exposures that were 2.5% and 3.4% of the RfD for non-nursing infants less than 1 year old and children (1–6 years), respectively. This scenario still assumes that all residues in the crops that are treated are at the tolerance level.

There is generally no concern for exposures below 100% of the RfD since it represents the level at or below which no appreciable risks to human health is posed. Thus, there is a reasonable certainty that no harm will result to the most highly exposed population subgroups, non-nursing infants, less than 1 year old, and children between 1 and 6 years of age, from exposure to buprofezin.

F. International Tolerances

Buprofezin was reviewed by the Joint Meeting of the Food and Agriculture Organization Panel of Experts on Pesticide Residues in Food and the Environment and the World Health Organization Expert Group on Pesticide Residues (JMPR) to establish Codex MRLs in 1991, 1995 and 1997. Permanent MRLs were granted for cucumbers and tomatoes, and a temporary MRL was granted for oranges, as described below. Additional residue trial data on oranges will be available for the 1999 JMPR meeting to determine if this MRL should also be made permanent.

Commodity	MRL
Cucumber	0.3 ppm
Tomato	0.5 ppm
Oranges, Sweet, Sour	0.3 ppm (temporary).

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ENVIRONMENTAL PROTECTION AGENCY

[PF–826; FRL–6023–5]

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–826, must be received on or before September 25, 1998.

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

Comments and data may also be submitted electronically 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: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Beth Edwards (PM 3)	Rm. 206, CM #2, 703–305–5400, e-mail:edwards.beth@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Sidney Jackson (PM 22)	Rm. 233, CM #2, 703–305–7610, e-mail: jackson.sidney@epamail.epa.gov.	Do.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–826] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

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

List of Subjects

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

Dated: August 13, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. AgrEvo USA Company (acting as registered US agent for Hoechst Schering AgrEvo, S.A.)

PP 7F4909

EPA has received a pesticide petition (PP 7F4909) from AgrEvo USA Company (acting as registered U.S. agent for Hoechst Schering AgrEvo, S.A.), 2711 Centerville Road, Wilmington, DE 19808 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of deltamethrin in or on various food and feed commodities. Tolerances are currently established at 40 CFR 180.435 in or on the following commodities for residues of deltamethrin [(1R, 3R)-3(2,2-dibromovinyl)-2,2-dimethylcyclopropanecarboxylic acid (S)-alpha-cyano-3-phenoxybenzyl ester] and relevant metabolites: cottonseed at 0.04 parts per million (ppm), cottonseed oil at 0.2 ppm, tomatoes at 0.2 ppm, and tomato products (concentrated) at 1.0 ppm.

EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

Based on the fact that tralomethrin, another synthetic pyrethroid insecticide, is rapidly metabolized in plants and animals to deltamethrin, and the toxicological profile of the two compounds is similar, it is appropriate to consider a combined exposure assessment for tralomethrin and deltamethrin.

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 various commodities for which registrations have been approved, or are being sought, have been submitted to the Agency. These methods, based on

gas chromatography (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 proposed tolerance levels as a result of the use of these active ingredients on target crops, or at target sites.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral LD_{50} values for deltamethrin in the rat are 66.7 milligram/kilograms (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. *Genotoxicty*. 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*—i. *Deltamethrin*. A rat developmental toxicity study conducted with deltamethrin indicated a maternal no observed effect level (NOEL) of 3.3 mg/kg/day based on clinical observations, decreased weight gain and mortality. The developmental NOEL was 11 mg/kg/day higest 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 3-generation rat reproduction study and a more recent, 2-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.

ii. *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 2-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*— i. *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's 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.

ii. *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 and oncogenicity*— i. *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.

ii. *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 14 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 carcinogenicity 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. *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.

In an acute neurotoxicity study with deltamethrin in rats, effects were noted after a single oral administration of a dose of 50 mg/kg. In addition, potential effects (limited to a single male and female) were observed at a dose level of 15 mg/kg. Therefore, the no observed adversed effect level (NOAEL) for neurotoxicity in this study was 5 mg/kg.

In a subchronic neurotoxicity study with deltamethrin in rats, effects were noted after daily dietary administration for 13 consecutive weeks at 800 ppm. The NOAEL for systemic toxicity and neurotoxicity in this study was found to be 200 ppm (14 and 16 mg/kg/day for males and females, respectively).

7. *Animal metabolism*— i. *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.

ii. *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.

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 and direct application to livestock), commercial, and industrial food use areas. Thus, aggregate non-occupational exposure would include exposures resulting from non-food use 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—Food*. Food tolerances have been established 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, tolerances in support of registrations currently exist for deltamethrin on cottonseed and cottonseed oil. Additionally, tolerances 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. Additional tolerances are being proposed for deltamethrin in the subject pesticide tolerance petition. Potential acute exposures from these relevant 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, and the tolerances proposed in this petition for deltamethrin, were estimated using NOVIGEN's dietary exposure evaluation mode (DEEM). This chronic risk assessment was conduced using anticipated residues based on field trial or monitoring data, percent crop treated, and percent food handling establishments treated.

2. *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 <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.

3. *Non-dietary exposure*. As noted above, deltamethrin and tralomethrin are broad spectrum insecticides registered for use on a variety of food and feed 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.

D. Cumulative Effects

When considering a tolerance, 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''. AgrEvo USA Company, acting as registered U.S. 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.

E. 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. In accordance with EPA's ''Toxicology Endpoint Selection Process'' Guidance Document for acute exposures, the toxicology endpoint from the deltamethrin rat acute neurotoxicity study, 5.0 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 1,406; the MOE for the 99th percentile is 3,500; and at the 95th percentile the MOE is 8,613. For the overall U.S. population, chronic dietary exposure results in a utilization of 1.4% 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 31,100 for adults. There is generally no concern for MOE's greater than 100. For chronic exposure, there is generally no concern for exposure 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.

In conclusion, there is reasonable certainty that no harm will result to the U.S. population, in general, from dietary or aggregate exposure to 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 5.0 mg/kg/day from the rat acute neurotoxicity 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 666; at the 99th percentile the MOE is 1,491; and at the 95th percentile the MOE is 8,755. For the population subgroup described as children 1-6 years old, the MOE for acute dietary exposure is 871 for the 99.9th percentile; at the 99th percentile the MOE is 1,527; and at the 95th percentile the MOE is 3,167. For non-nursing infants, chronic dietary exposure results in a utilization of 1.9% of the RfD, and

for children 1-6 years old 3.7% 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 6,775 for infants less than 1 year old, and an MOE of 5,700 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% 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.

F. International Tolerances

Deltamethrin is a broad spectrum insecticide used throughout the world to control pests of livestock, crops, ornamentals plants and turf, and household, commercial, and industrial food use areas. A reevaluation of the maximum residue limits (MRL's) was conducted in 1994, in accordance with the EC Directive (91/414/EEC) Registration Requirements for Plant Protection Products. A comparison of the proposed/current CODEX MRL's and proposed/established tolerances for deltamethrin is presented below:

Commodity	Proposed Tolerance (USEPA) (PPM)	Proposed/Current MRL (CODEX) (PPM)
Barley, grain	0.50	1.0
Broccoli	0.50	0.2
Cattle, fat	0.15	---
Cattle, mbyp	0.05	---
Cattle, meat	0.05	---
Cereal grain dust	65.0	---
Corn, field, grain	1.0	1.0
Corn, pop, grain	0.5	1.0
Corn, sweet, grain	0.5	1.0
Corn, forage (field)	0.7	---
Corn, fodder (field)	7.0	0.5
Cucurbits vegetables	0.05	0.2
Eggs	0.02	---
Goats, fat	0.15	---
Goats, mbyp	0.05	---
Goats, meat	0.05	---
Hogs, fat	0.15	---
Hogs, mbyp	0.05	---
Hogs, meat	0.05	---
Horses, fat	0.15	---
Horses, mbyp	0.05	---
Horses, meat	0.05	---
Lettuce, head	1.0	0.2
Lettuce, leaf	3.0	0.5
Milk, Fat (reflecting 0.07 ppm in whole milk)	0.6	0.01 (milk)
Oats, grain	0.5	1.0
Poultry, fat	0.3	---
Poultry, mbyp	0.02	---
Poultry, meat	0.02	---
Rice, grain	0.5	1.0
Rye, grain	0.5	1.0
Sheep, fat	0.15	---
Sheep, mbyp	0.05	---
Sheep, meat	0.05	---
Sorghum, grain	1.0	1.0
Sorghum, forage	0.5	---
Sorghum, fodder	2.0	0.5
Soybeans	0.05	0.1
Sunflower seed	0.05	0.1
Tomatoes	0.3	0.2
Triticale, grain	0.5	1.0
Wheat, forage	8.0	---
Wheat, grain	1.0	1.0
Wheat, hay	8.0	0.5
Wheat, straw	8.0	0.5
Corn, refined oil	10.0	---
Corn, flour	3.0	---
Corn, meal	2.0	---
Tomato products (concentrated)	1.5	---
Wheat bran	4.0	5.0
Wheat germ	8.0	---
Soybean hulls	0.25	0.5
Cereal bran	2.0	---
Rice hulls	6.0	---

Commodity	Proposed Tolerance (USEPA) (PPM)	Proposed/Current MRL (CODEX) (PPM)
Corn, milled byproducts	3.0	---

As far as can be determined, no CODEX MRL's are established or proposed for tralomethrin.

G. Proposed Tolerances

This pesticide petition proposes to amend 40 CFR 180.435 for the insecticide deltamethrin as it relates to the following raw agricultural, food, or feed commodities:

Commodity	Parts per million
Barley, grain	0.5
Broccoli	0.5
Cattle, fat	0.15
Cattle, mbyp	0.05
Cattle, meat	0.05
Cereal bran	2.0
Cereal grain dust	65.0
Corn, field, grain	1.0
Corn, pop, grain	0.5
Corn, sweet, grain	0.5
Corn, forage (field)	0.7
Corn, fodder (field)	7.0
Corn, refined oil	10.0
Corn, flour	3.0
Corn, meal	2.0
Corn, milled byproducts	3.0
Cottonseed	0.04
Cottonseed oil	0.2
Cucurbits vegetables	0.05
Eggs	0.02
Goats, fat	0.15
Goats, mbyp	0.05
Goats, meat	0.05
Hogs, fat	0.15
Hogs, mbyp	0.05
Hogs, meat	0.05
Horses, fat	0.15
Horses, mbyp	0.05
Horses, meat	0.05
Lettuce, head	1.0
Lettuce, leaf	3.0
Milk, Fat (reflecting 0.07 ppm in whole milk)	0.6
Oats, grain	0.5
Poultry, fat	0.3
Poultry, mbyp	0.02
Poultry, meat	0.02
Rice, grain	0.5
Rice, hulls	6.0
Rye, grain	0.5
Sheep, fat	0.15
Sheep, mbyp	0.05
Sheep, meat	0.05
Sorghum, grain	1.0
Sorghum, forage	0.5
Sorghum, fodder	2.0
Soybeans	0.05
Soybean hulls	0.25
Sunflower seed	0.05
Tomatoes	0.3
Tomato products (concentrated)	1.5
Triticale, grain	0.5
Wheat, bran	4.0
Wheat, forage	8.0
Wheat, germ	8.0
Wheat, grain	1.0
Wheat, hay	8.0
Wheat, straw	8.0

H. Conclusions

The proposed establishment of food and food/feed additive tolerances for deltamethrin resulting from application to growing crops, stored grain, and direct application to livestock would not pose a significant risk to human health, including that of children, and is in compliance with the requirements of the Food Quality Protection Act of 1996. Thus, the tolerances proposed for residues of deltamethrin can be established.

2. Gowan Company

PP 8F4985

EPA has received a pesticide petition (PP 8F4985) from Gowan Company, P.O. Box 5569, Yuma, AZ 85366-5569 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the acaricide hexythiazox in or on strawberries, apples, wet apple pomace, cottonseed and cotton gin byproducts. The chemical name of hexythiazox is trans-5-(4-chlorophenyl)-N-cyclohexyl-4-methyl-2-oxothiazolidine-3-carboxamide. Metabolites containing the (4-chlorophenyl)-4-methyl-2-oxo-3-thiazolidine moiety are included in the tolerance expression. Time-limited tolerances for strawberries, cotton seed and cotton gin byproducts are currently in effect. Gowan Company has proposed that the tolerances for cotton seed and cotton gin byproducts be geographically limited to California only. A permanent tolerance exists for apples, but Gowan Company proposes to increase the tolerance level in connection with a proposed change in the use pattern. A tolerance for residues in wet apple pomace has not been proposed previously.

EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition. The proposed analytical method is high performance liquid chromatography with an ultraviolet detector. As required by section 408(d) of the FFDCA, as recently amended by the Food Quality Protection Act (FQPA) Pub. L. 104-170, Gowan Company included in the petition a summary of the petition and authorization for the summary to be published in the **Federal Register** in a notice of receipt of the petition. The summary represents the views of Gowan Company; EPA, as mentioned above, is in the process of evaluating the petition. As required by section 408(d)(3) of the FFDCA, EPA is including the summary as a part of this notice of filing.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of hexythiazox in apples, pears, grapes, and citrus has been studied. The major portion of the residue is parent compound. The metabolites are hydroxycyclohexyl and ketocyclohexyl analogs of hexythiazox and the amide formed by loss of the cyclohexyl ring.

2. *Animal metabolism*. The metabolism of hexythiazox in goats, hens and rats has been studied. Metabolic pathways in animals are similar to those in plants.

3. *Analytical method*. An adequate analytical method (HPLC with UV detection) is available for enforcement purposes. Parent compound and all of its metabolites are converted to a common moiety before analysis.

4. *Magnitude of residues*—i. *Strawberries*. Seventy samples of treated strawberries were analyzed. The maximum residue observed (MRO) at a preharvest interval of 3-days was 2.06 ppm and the average residue was 0.67 ppm. A tolerance of 3 ppm was proposed.

ii. *Cotton*. Twenty residue studies were conducted in the U.S., Brazil, and Spain. Four additional studies, including a processing study, were conducted in California. The MRO in cotton seed was 0.097 ppm and the average residue was 0.065 ppm. A tolerance of 0.2 ppm was proposed. The maximum residue observed in cotton gin byproducts was 2.29 ppm and the average residue was 1.07 ppm. A tolerance of 3 ppm was proposed. The proposed tolerances are geographically limited to California only. A field crop rotation study indicated that residues would not be present in crops planted 4-months after application of hexythiazox.

iii. *Apples—a total of 20 trials were conducted*. The maximum residue in apples having a preharvest interval of 1-month was 0.38 ppm and the average residue was 0.14 ppm. A tolerance of 0.4 ppm was proposed. Processing studies indicated that hexythiazox residues concentrate by a factor of 1.7 in wet apple pomace, and a tolerance of 0.7 ppm was proposed.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral and dermal LD_{50} of technical hexythiazox is > 5,000 mg/kg, and the 4-hour acute inhalation LC_{50} is > 2 mg/L. It is not a dermal irritant or sensitizer and is a mild eye irritant.

2. *Genotoxicity*. The following genotoxicity tests were all negative: Ames gene mutation, CHO gene mutation, CHO chromosome aberration, mouse micronucleus and rat hepatocyte unscheduled DNA synthesis.

3. *Reproductive and developmental toxicity*. Hexythiazox has not been observed to induce developmental or reproductive effects. The lowest reproductive or developmental no-observed effected level (NOEL) was 200 milligram/kilogram/day (mg/kg/day), the highest dose tested (HDT), in a 2-generation rat reproduction study.

4. *Chronic toxicity*. The Office of Pesticide Programs has established the Reference Dose (RfD) for hexythiazox at 0.025 mg/kg/day. The RfD for hexythiazox is based on a 1-year dog feeding study with a NOEL of 2.5 mg/kg/day and an uncertainty factor of 100. The endpoint effect of concern was hypertrophy of the adrenal cortex in both sexes, decreased red blood cell counts, hemoglobin content and hematocrit in males.

5. *Carcinogenicity*. The Agency has classified hexythiazox as a category C (possible human) carcinogen based on an increased incidence of hepatocellular carcinomas ($p = 0.028$) and combined adenomas/carcinomas ($p = 0.024$) in female mice at the HDT (1,500 ppm) when compared to the controls as well as a significantly increased ($p < 0.001$) incidence of pre-neoplastic hepatic nodules in both males and females at the HDT. The decision supporting a category C classification was based primarily on the fact that only one species was affected and mutagenicity studies were negative. In classifying hexythiazox as a category C carcinogen, the Agency concluded that a quantitative estimate of the carcinogenic potential for humans should be calculated because of the increased incidence of liver tumors in the female mouse. A Q1* of 0.039 (mg/kg/day)-1 in human equivalents was calculated.

C. Aggregate Exposure

Tolerances have been established (40 CFR 180.448) for combined residues of hexythiazox [trans-5-(4-chlorophenyl)-N-cyclohexyl-4-methyl-2-oxothiazolidine-3-carboxamide] and its metabolites containing the (4-chlorophenyl)-4-methyl-2-oxo-3-thiazolidine moiety in or on apples at 0.02 ppm and pears at 0.3 ppm. Use on several other crops had been previously proposed [PP 6F4738], and an aggregate exposure analysis has taken into consideration all current and proposed uses. The nature and metabolism of

hexythiazox in plants and animals is adequately understood.

Hexythiazox is also registered for use on outdoor ornamental plants by commercial applicators only. It is believed that non-occupational exposure from this use is very low. Hexythiazox is not registered for greenhouse, lawn, garden, or residential use. The environmental fate of hexythiazox has been evaluated, and the compound is not expected to contaminate groundwater or surface water to any measurable extent.

1. *Chronic exposure*. A chronic dietary exposure analysis was conducted for the general U.S. population and 26 population subgroups. In this analysis it was assumed that 100% of crops were treated. A chronic exposure of 0.000172 mg/kg/day was calculated for the average U.S. population. Non-nursing infants, the most heavily exposed subgroup, had a calculated exposure of 0.000972 mg/kg/day. Actual exposure would be much lower, however, because far less than 100% of crops would be treated.

The Agency has not conducted a detailed analysis of potential exposure to hexythiazox via drinking water or outdoor ornamental plants. However, it is believed that chronic exposure from these sources is very small.

2. *Acute exposure*. No developmental, reproductive or mutagenic effects have been observed with hexythiazox. Therefore, an analysis of acute exposure has not been conducted.

D. Cumulative Effects

At this time the Agency has not reviewed available information concerning the potentially cumulative effects of hexythiazox and other substances that may have a common mechanism of toxicity. For purposes of this petition only, the Agency is considering only the potential risks of hexythiazox in its aggregate exposure.

E. Safety Determination

1. *U.S. population*—i. *Chronic risk*. Chronic risk was calculated using anticipated residue concentrations from all current and proposed uses of hexythiazox and assuming that 100% of each crop is treated. Dietary exposure of the general U.S. population was equivalent to 0.7% of the RfD. Exposure of the most heavily exposed subgroup, non-nursing infants, was equivalent to 3.9% of the RfD.

ii. *Oncogenic risk*. Oncogenic risk was evaluated using anticipated residue concentrations and taking into account the percent of crop known or expected to be treated. Lifetime oncogenic risk for the U.S. population was calculated to be 4.5 x 10-7.

iii. *Acute risk*. An estimate of acute risk with this compound has not been conducted since no acute reproductive or developmental effects have been observed.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of hexythiazox, EPA considered data from developmental toxicity studies in the rat and rabbit and a 2-generation 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.

No developmental or reproductive effects have been observed in any study with hexythiazox. The lowest acute NOEL was 2,400 ppm in the diet (200 mg/kg/day), the HDT, in the 2-generation rat reproduction study. In the rat developmental study, the maternal and fetotoxic NOEL was 240 mg/kg/day and the developmental NOEL was 2,160 mg/kg/day, the HDT. In the rabbit developmental study, the maternal and developmental NOEL was 1,080 mg/kg/day, the HDT.

Taking into account current toxicological data requirements, the database for hexythiazox relative to prenatal and postnatal effects is complete. In the rat developmental study, the NOELs for maternal toxicity and fetotoxicity were the same, which suggests that there is no special prenatal sensitivity in the absence of maternal toxicity. Furthermore, the lowest developmental or reproductive NOEL is two orders of magnitude higher than the chronic NOEL on which the RfD is based. It is concluded that there is a reasonable certainty of no harm to infants and children from aggregate exposure to hexythiazox residues.

F. International Tolerances

Codex MRLs of 0.5 mg/kg for residues of hexythiazox in strawberries and apples have been established. The U.S. tolerance proposals are somewhat at variance with the Codex MRLs because they are based upon different preharvest intervals. Also, it is believed that the U.S. proposed tolerance levels allow for a greater margin of safety than the Codex MRLs. There are no Codex MRLs for the other commodities in this petition. There are no Canadian or Mexican MRLs for hexythiazox. (Beth Edwards).

3. Interregional Research Project

PP 7E4833

EPA has received a pesticide petition (PP 7E4833) from the Interregional Research Project Number 4 (IR-4), proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of the herbicide glyphosate [N-phosphonomethyl)glycine] in or on the raw agricultural commodities (RACs) durian at 0.2 ppm, mangosteen at 0.2 ppm, and rambutan at 0.2 ppm. Durian, mangosteen, and rambutan are tree fruits which are grown commercially in Hawaii and Puerto Rico.

EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition. This notice includes a summary of the petition prepared by Monsanto Agricultural Group (MAG), the registrant.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in plants and animals is adequately understood. The residue to be regulated is the parent glyphosate.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of glyphosate in or on food with a limits of detection (0.05 ppm) that allows monitoring of food with residues at or above the levels set in these tolerances. EPA has provided information on this method to FDA.

3. *Magnitude of residues*. The proposed use for glyphosate is for orchard floor treatment. The registrant referenced extensive experience and data with glyphosate in/on tree fruit and nuts crops which show that when orchard floor applications are made, no detectable residues of the herbicide are recovered in the harvested fruit. Based on these data Monsanto expects no detectable residues of glyphosate in durian, mangosteen or rambutan when glyphosate is applied in a similar manner.

Tolerances for the combined residues of glyphosate and its metabolite, aminomethylphosphonic acid (AMPA), have been established at 0.2 ppm on a number of tree fruit and nuts, as well as a variety of tropical fruit: acerola, atemoya, avocado, banana, breadfruit, canistel, carambola, cherimoya cocoa beans, coconuts, dates, figs, genip, jaboticaba, jackfruit, longan, lychee,

mango, mayhaw, passion fruit, persimmon, pomegranate, sapodilla, sapote, soursop, sugar apple and tamarind. Any secondary residues occurring in milk, eggs, meat, fat, liver and kidney of cattle, goats, horses, hogs, poultry and sheep are covered by existing tolerances.

The Agency's Health Effects Division - Metabolism Committee has determined that AMPA should be dropped from the tolerance expression. Tolerances that are the subject of this notice are based solely on residues of glyphosate.

B. Toxicological Profile

1. *Acute toxicity*. Results from an acute oral study in rats show a combined lethal dose $(LD)_{50}$ for glyphosate of >5,000 milligram/kilogram (mg/kg).

An acute dermal study in rabbit resulted in a LD_{50} of > 5,000 mg/kg.

The results of a primary eye irritation study in the rabbit showed severe irritation for glyphosate acid. However, glyphosate is normally formulated as one of several salts and eye irritation studies on the salts showed essentially no irritation.

A primary dermal irritation study showed essentially no irritation.

A primary dermal sensitization study showed no sensitization. Based on these data, Monsanto concludes that the acute toxicity and irritation potential of glyphosate is low.

2. *Genotoxicity*. A number of mutagenicity studies were conducted and were all negative. These studies included: chromosomal aberration *in vitro* (no aberrations in Chinese hamster ovary cells were caused with or without S9 activation); deoxyribonucleic acid(DNA) repair in rat hepatocyte; *in vivo* bone marrow cytogenic test in rats; rec-assay with B. subtilis; reverse mutation test with *S. typhimurium*; Ames test with *S. typhimurium*; and dominant-lethal mutagenicity test in mice.

Negative results were obtained when glyphosate was tested in a dominant-lethal mutation assay. While this assay was designed as a genetic toxicity test, agents that can affect male reproduction function will also cause effects in this assay. More importantly, the multi-generation reproduction study in rodents is a complex study design which measures a broad range of endpoints in the reproductive system and in developing offspring that are sensitive to alterations by chemical agents. Glyphosate has been tested in two separate multi-generation studies and each time the results demonstrated that glyphosate is not a reproductive toxin.

3. *Reproductive and developmental toxicity*. An oral developmental toxicity study with rats given doses of 0, 300, 1,000 and 3,500 milligram/kilogram/day (mg/kg/day) with a maternal no-observed-effect level (NOEL) of 1,000 mg/kg/day based on clinical signs of toxicity, body weight effects and mortality, and a fetal NOEL of 1,000 mg/kg/day based on reduced body weights and delayed sternebrae maturation at the highest dose tested (HDT) of 3,500 mg/kg/day.

An oral developmental toxicity study with rabbits given doses of 0,75, 175 and 350 mg/kg/day with a maternal of NOEL of 175 mg/kg/day based on clinical signs of toxicity and mortality, and a fetal NOEL of 350 mg/kg/day based on no developmental toxicity at any dose tested.

A 3-generation reproduction study with rats fed dosage levels of 0, 3, 10 and 30 mg/kg/day with a NOEL for systemic and reproductive/developmental parameters of 30 mg/kg/day based on no adverse effects noted at any dose level.

A 2-generation reproduction study with rats fed dosage levels of 0, 100, 500 and 1,500 mg/kg/day with a NOEL for systemic and developmental parameters of 500 mg/kg/day based on body weight effects, clinical signs of toxicity in adult animals and decreased pup body weights, and a reproductive NOEL of 1,500 mg/kg/day.

4. *Subchronic toxicity*. A 90-day feeding study in mice fed dosage levels of 0, 5,000, 10,000 and 50,000 with a NOEL of 10,000 ppm based on body weight effects at the high dose.

A 90-day feeding study in rats fed dosage levels of 0, 1,000, 5,000 and 20,000 ppm with a NOEL of 20,000 ppm based on no effects even at the HDT.

A 90-day feeding study in dogs given glyphosate, via capsule, at doses of 0, 200, 600 and 2,000 mg/kg/day with a NOEL of 2,000 mg/kg/day based on no effects even at the HDT.

5. *Chronic toxicity*. The reference dose (RfD) for glyphosate based on maternal effects in a developmental study with rabbits (NOEL of 175 milligram/killogram/body weight day (mg/kg/bwt/day)) and using a hundred-fold safety factor is calculated to be 2.0 mg/kg/bwt/day.

The EPA Carcinogenicity Peer Review Committee has classified glyphosate in Group E (evidence of non-carcinogenicity for humans), based upon lack of convincing carcinogenicity evidence in adequate studies in two animal species. There was no evidence of carcinogenicity in an 18-month feeding study in mice and a 2 year feeding study in rats at the dosage levels tested (DLT). The doses tested were adequate for identifying a cancer risk.

A mouse carcinogenicity study with mice fed dosage levels of 0, 150,750 and 4,500 mg/kg/day with a NOEL of 750 mg/kg/day based on body weight effects and microscopic liver changes at the high dose. There was no carcinogenic effect at the HDT of 4,500 mg/kg/day.

A 12-month oral study in dogs given glyphosate, via capsule, at doses of 0, 20, 100 and 500 mg/kg/day with a NOEL of 500 mg/kg/day based on no adverse effects at any dose level.

A 24-month chronic/feeding carcinogenicity study with rats fed dosage levels of 0, 89, 362 and 940 mg/kg/day (males) and 0, 113, 457 and 1,183 mg/kg/day (females) with a systemic NOEL of 362 mg/kg/day based on body weight effects in the female and eye effects in males. There was no carcinogenic response at any dose level.

A 26-month chronic/feeding carcinogenicity study with rats fed dosage levels of 0, 3, 10 and 31 mg/kg/day (males) and 0, 3, 11 and 34 mg/kg/day (females) with a systemic NOEL of 31 mg/kg/day (males) and 34 mg/kg/day (females) based on no carcinogenic or other adverse effects at any dose level.

Monsanto believes that these data support their conclusion that glyphosate does not produce adverse reproductive effects and is not a developmental toxin, mutagen, carcinogen or a neurotoxin.

6. *Animal metabolism*. Animal metabolism data were not submitted with this petition. However, Monsanto believes that the treated commodities are not fed to animals, therefore, there will be no residues transferred to meat, milk, poultry, or eggs.

C. Aggregate Exposure

1. *Dietary exposure—Food*. For purposes of assessing the potential dietary exposure, Monsanto has estimated aggregate exposure based on the tolerances for glyphosate on jackfruit, sugar apple and lychee, all with established 0.2 ppm tolerances. As the consumption of durian, mangosteen and rambutan is so limited, the theoretical maximum residue contribution (TMRC) calculations were based on similar or related tropical fruit: durian and jackfruit are similar in size, with thick rinds and similar growth habit; mangosteen and sugar apple fruit are also similar in size and growth habit: and rambutan and lychee are from the same botanical family, the Sapindaceae. The fruit are not fed to animals, therefore, there will be no exposure of humans to residues transferred to meat, milk, poultry, or eggs. Other potential sources of exposure of the general population to residues of pesticides are

residues in drinking water and exposure from non-occupational sources.

Based on the available acute toxicity data, Monsanto believes that glyphosate does not pose any acute dietary risks.

2. *Drinking water*. A Maximum Concentration Level (MCL) has been established for residues of glyphosate in drinking water at 0.7 mg/l since glyphosate is approved for direct application to water. The MCL represents the level at which no known or anticipated adverse health effects occur, allowing for an adequate margin of safety (MOE), and is based on the RfD.

Monsanto reports that glyphosate adsorbs strongly to soil and is not expected to move vertically below the 6-inch soil layer; residues are expected to be immobile in soil. Glyphosate is readily degraded by soil microbes to AMPA, which is degraded to carbon dioxide. Monsanto believes that glyphosate and AMPA are not likely to move to ground water due to their strong adsorptive characteristics. However, due to its aquatic use patterns and through erosion, glyphosate does have the potential to enter surface waters, where, according to Monsanto, it will adsorb to sediment and undergo microbial degradation.

3. *Non-dietary exposure*. Exposure (non-occupational) of the general population to glyphosate is expected based on the currently-registered uses; however, due to the low acute toxicity and lack of other toxicological concerns, Monsanto believes that the risk posed by non-occupational exposure (NOE) to glyphosate is minimal.

D. Cumulative Effects

Because the existing data base is insufficient to fully assess cumulative toxic effects that may be caused by glyphosate along with other chemical compound(s) that may share a common mechanism of toxicity, Monsanto believes that any consideration of such an analysis of toxicity is inappropriate at this time.

E. Safety Determination

1. *U.S. population*. The TMRC for existing, published tolerances for glyphosate is 0.021460 mg/kg/bwt/day or 1.0% of the RfD for the overall U.S. population. Even using conservative exposure assumptions and substituting the more widely consumed jackfruit, sugar apple and lychee, there is not enough exposure to calculate a significant contribution to the TMRC. As the exposure from durian, mangosteen and rambutan would be even less, the aggregate exposure of these three fruits will not add to the RfD for the overall U.S. population. EPA generally has no concern for exposures below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Monsanto concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of glyphosate, including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of glyphosate, data were considered from developmental toxicity studies in the rat and rabbit and multi-generation reproduction studies in rats.

No birth defects were observed in the offspring of rats given glyphosate by gavage at dose levels of 0, 300, 1,000, and 3,500 mg/kg/day on days 6 through 19 of gestation. The NOEL for this study was 1,000 mg/kg/day based on maternal and developmental toxicity observed at the HDT, 3,500 mg/kg/day. The high-dose in this study was 3.5 times higher than the limit dose that is currently required by the guidelines.

No birth defects were observed in the offspring of rabbits given glyphosate by gavage at dose levels of 0, 75, 175, and 350 mg/kg/day on days 6 through 27 of gestation. The NOEL for this study is considered to be 175 mg/kg/day based on maternal toxicity at the high-dose of 350 mg/kg/day. Because no developmental toxicity was observed at any dose level, the developmental NOEL is considered to be 350 mg/kg/day.

Male and female rats were fed glyphosate at dose levels of 0, 3, 10, and 30 mg/kg/day every day throughout the production of three successive generations. No adverse treatment-related effects on reproduction were observed. Because no toxicity was noted even at the HDT, a second reproduction study at higher dose levels (HDLs) was performed and is described below.

Male and female rats were fed glyphosate at dose levels of 0, 100, 500, and 1,500 mg/kg/day every day throughout the production of two successive generations. Reduced body weights and soft stools occurred at 1,500 mg/kg/day (3% of the diet); therefore, the systemic NOEL is considered to be 500 mg/kg/day. Glyphosate did not affect the ability of rats to mate, conceive, carry or deliver normal offspring at any dose level.

3. *Reference dose*. The TMRC for existing, published and pending tolerances (including durian, mangosteen, and rambutan) for glyphosate range from 0.015 for nursing infants to 0.049 for non-nursing infants (0.8 to 2.5% of the RfD). EPA generally has no concern for exposures below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Monsanto concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of glyphosate, including all anticipated dietary exposure and all other non-occupational exposures.

4. *Endocrine effects*. No known factors were identified in sub-chronic, chronic or developmental toxicity studies to indicate any endocrine-modulating activity by glyphosate.

F. International Tolerances

Codex maximum residue levels (MRLs) have not been established for residues of glyphosate on durian, mangosteen and rambutan. (Sidney Jackson).

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ENVIRONMENTAL PROTECTION AGENCY

[PF–825; FRL–6023–4]

Notice of Filing of Pesticide Tolerance 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–825, must be received on or before September 25, 1998.

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

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No Confidential Business Information (CBI) should be submitted through e-mail.

Information submitted as a comment concerning this document may be