there may be multiple liable municipal O/Os and the Region may determine that it is appropriate to settle for less than the presumption for an individual O/O. A group or coalition of two or more municipalities with the same nexus to a site, at the same time or during continuous operations under municipal control, should be considered a single O/O for purposes of developing a cost share (e.g., two cities operated together in joint operations or in cost sharing agreements). In cases where a municipal O/O is also liable as an MSW G/T, EPA would offer to resolve such liability for an additional payment amount developed pursuant to the MSW G/T settlement methodology.

EPA proposes the 20% baseline settlement contribution on the basis of several considerations. EPA examined the data from past settlements of CERCLA cost recovery and contribution cases with municipal O/Os at co-disposal sites where there were also PRPs who were potentially liable for the disposal of non-MSW, such as industrial waste. In examining that data, EPA considered that such historical settlements also typically reflected resolution of the municipality's liability not only as an owner/operator, but also as a generator or transporter of MSW. Under the final policy, such liability will be resolved through payment of an additional amount, calculated pursuant to the MSW G/T methodology. The 20% baseline does not reflect this separate basis for liability and the respective additional payment.

The 20% baseline figure also reflects the requirement that municipal O/Os that settle under the final policy will be required to waive all contribution rights against other parties as a condition of settlement. By contrast, in many historical settlements, municipal O/Os retained their contribution rights and hence were potentially able to seek recovery of part of the cost of their settlements from other parties.

In addition, the 20% baseline figure reflects EPA's evaluation of public interest considerations relating to municipalities. For example, Section 122(e)(3) of CERCLA authorizes the President to perform ''nonbinding preliminary allocations of responsibility'' for the purpose of promoting settlements and to include ''public interest considerations'' in developing such allocations. EPA believes it is in the public interest to consider collectively: the unique public health obligation of municipalities to provide waste disposal services to their citizens; the municipalities' non-profit status; and the unique fiscal planning considerations for municipalities that require multi-year planning.

Under this proposal, the Regions may adjust the settlement in a particular case upward from the presumptive percentage, not to exceed a 35% share, based on consideration of the following factors:

(1) Whether the municipality performed specific activities that exacerbated environmental contamination or exposure (e.g., the municipality permitted the installation of drinking wells in known areas of contamination);

(2) Whether the O/O received operating revenues net of waste system operating costs during ownership or operation of the site that are substantially higher than the O/O's presumptive settlement amount pursuant to this policy; and

(3) Whether an officer or employee of the municipality has been convicted of performing a criminal activity relating to the specific site during the time in which the municipality owned or operated the site.

The Regions may adjust the presumptive percentage down based on whether the municipality, on its own volition, made specific efforts to mitigate environmental harm once that harm was evident (e.g., the municipality installed environmental control systems, such as gas control and leachate collection systems, where appropriate; whether the municipality discontinued accepting hazardous waste once groundwater contamination was discovered; etc.). The Regions may also consider other equitable factors at the site.

Financial Considerations in Settlement

In all cases under this proposal, the U.S. will consider municipal claims of limited ability to pay. Municipalities making such claims are required to provide Regions all necessary documentation relating to the claim. Recognizing that municipal O/Os may be uniquely situated to perform in-kind services at a site (e.g., mowing, road maintenance, structural maintenance), EPA will carefully consider any forms of in-kind services that a municipal O/O may offer as partial settlement of its cost share.

Steven A. Herman,

Assistant Administrator, Office of Enforcement and Compliance Assurance.

[FR Doc. 97–18247 Filed 7–10–97; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–748; FRL–5728–7]

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–748, must be received on or before August 11, 1997.

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

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

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

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

Product Manager	Office location/telephone number	Address
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail:miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
George LaRocca (PM 13).	Rm. 204, CM #2, 703–305–6100, e-mail: larocca.george@epamail.epa.gov.	Do.
James Tompkins (PM 25).	Rm. 229, CM #2, 703–305–7830, e-mail: tompkins.james@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–748] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–748] 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: July 1, 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. DowElanco

PP 6G3306

EPA has received a pesticide petition (PP) 6G3306 from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268 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 renewing a temporary tolerance for the combined residues of herbicide triclopyr (3,5,6-trichloro-2-pyridinyl)oxyacetic acid and its metabolites 3,5,6-trichloro-2-pyridinol and 2-methoxy-3,5,6-trichloropyridine in or on the raw agricultural commodities fish and shellfish at 0.2 part per million (ppm). An allowable residue level of 0.5 ppm in potable water is also being renewed. The proposed analytical method is gas chromatography. 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. Triclopyr Uses

Triclopyr as the triethylamine salt solution is currently registered for use on rights-of-way, industrial sites, non-crop areas, forest sites, rangeland, permanent grass pastures, roadsides, fence rows, ornamental turf, non-irrigation ditchbanks, and rice. It is recommended for the selective control of unwanted woody plants and annual and perennial broadleaf weeds on these sites.

Triclopyr is to be experimentally used for the selective control of aquatic weeds such as alligatorweed, Eurasian watermilfoil, parrot's feather, pickerelweed, purple loosestrife, and water hyacinth growing in lakes, ponds, reservoirs, and wetlands. It will also be tested for the control of woody brush and herbaceous weeds growing in wetlands and the banks and shores of aquatic sites. Application timing will coincide with the seasons of the year when the target species are actively growing. The maximum rates for triclopyr are 2 gallons per acre for the treatment of floating or emerged weeds, 3 gallons per acre for treatment of woody plants, and 2.5 ppm in water for treatment of submersed weeds.

B. Residue Chemistry

1. *Analytical method.* Adequate methodology is available for the enforcement of tolerances for triclopyr residues of concern. Gas chromatography methods are available for the determination of triclopyr residues of concern. Residues of triclopyr, 3,5,6-trichloro-2-pyridinol, and 2-methoxy-3,5,6-trichloropyridine can be separately determined. The detection limits range from 0.01 to 10 ppm depending on the compound being analyzed.

2. *Magnitude of residues.* In field studies, triclopyr in water has a half-life of 0.5 - 3.5 days. Triclopyr residues were below 0.5 ppm after 3 days. The metabolite, 3,5,6-trichloro-2-pyridinol was not detected within the treatment area. Within the treatment area, triclopyr was detected at <0.01 - 0.03 ppm in water collected 21 days after application. The average concentration did not exceed 0.5 ppm at 600 ft from the border of the treated area. Residues of triclopyr and its metabolites 3,5,6-trichloro-2-pyridinol and 2-methoxy-3,5,6-trichloropyridine were detectable only at the limit of detection, 0.01 ppm and non-detectable after day eight in fish flesh. Shellfish residues were

greater, with less than 0.1 ppm remaining in the edible portion after two weeks of treatment.

C. Toxicology Profile

1. *Acute toxicity.* Acute toxicity studies conducted with the triethylamine salt of triclopyr indicate low toxicity with the exception of eye irritation. The acute oral LD_{50}, in rats with the triethylamine salt of triclopyr is 2,574 mg/kg (males) or 1,847 mg/kg (females) (Toxicity Category III.) The acute dermal LD, in rabbits using the triethylamine salt of triclopyr was > 2,000 mg/kg (Toxicity Category III.) The acute inhalation LD_{50}, in rats was > 2.6 mg/L (maximum attainable concentration) with a Toxicity Category of III. In a primary eye irritation study in rabbits the triethylamine salt of triclopyr was found to be corrosive, with corneal involvement present through day 21 post-dose. The triethylamine salt of triclopyr was found to be non-irritating to the skin of white rabbits. In dermal sensitization studies in guinea pigs, sensitization was observed with the triethylamine salt of triclopyr.

2. *Genotoxicity.* The genotoxic potential of triclopyr has been evaluated in a range of assays *in vivo* and *in vitro*. These assays demonstrate triclopyr is non-mutagenic *in vivo* and *in vitro*. Mutagenicity data included gene mutation assays with E. coli and S. typhimurium (negative); DNA damage assays with B. subtillis (negative); an unscheduled DNA synthesis with rat hepatocytes (negative), a chromosomal aberration test in Chinese hamster cells and rat cells (negative) and dominant lethal assays in rats and mice (negative).

3. *Reproductive and developmental toxicity.* A developmental toxicity study in rats fed dosage levels of 0, 30, 100, and 300 mg/kg/day, with a maternal lowest observed effect level (LOEL) = 300 mg/kg based on the increased incidence of salivation and mortality and a maternal no-observed effect level (NOEL) = 100 mg/kg. Developmental toxicity was evident in this study at the 300 mg/kg dose level, and included decreased mean fetal body weight, increased fetal and litter incidence of skeletal anomalies and an increase in the number of fetuses with unossified sternebrae. The developmental LOEL = 300 mg/kg-based on decreased mean fetal weight, increased fetal and fitter incidence of skeletal anomalies, and increased fetal incidence of unossified sternebrae. The developmental NOEL = 100 mg/kg.

A developmental toxicity study in rabbits fed dosage levels of 0, 10, 30, and 100 mg/kg/day with a maternal LOEL = 100 mg/kg based on the decreased body weight gain, decreased food efficiency, and increased liver and kidney weight. The maternal NOEL = 30 mg/kg. Developmental toxicity was evident at the 100 mg/kg dose level in the form of reduced number of litters, reduced number of corpora lutea, reduced number of total implants, reduced total live fetuses, increased embryonic deaths and deaths/dam, and increased pre-implantation loss. The developmental LOEL =100 mg/kg based an the decreased number of live implants, decreased live fetuses, and increased embryonic deaths. The developmental NOEL = 30 mg/kg.

A 2–generation reproduction study in rats fed dosages of 0, 5, 25, and 250 mg/kg/day with a Parental Systemic Toxicity NOEL = 5 mg/kg/day (males and females); the parental Systemic Toxicity LOEL = 25 mg/kg/day, based on increased incidence of proximal tubular degeneration in male and female P_1 and P_2 rats. The Reproductive/Systemic Toxicity NOEL = 25 mg/kg/day; the Reproductive/Systemic Toxicity LOEL = 250 mg/kg/day, based on decreased litter size, decreased body weight and weight gain, and decreased survival in the F_1 and F_2 litters.

4. *Subchronic toxicity.* A subchronic oral toxicity study in rats receiving dietary concentrations of triclopyr at doses of 0, 5, 20, 50, or 250 mg/kg/day for 13 weeks with a systemic NOEL was 5 mg/kg/day, and the systemic LOEL of 20 mg/kg/day, based on histopathological changes in the kidneys of both sexes.

A 183-day toxicity study in dogs receiving dietary doses of triclopyr technical at 0, 0.1, 0.5, or 2.5 mg/kg/day with decreased rate of phenolsulfanthalein (PSP) excretion was observed in dogs receiving 2.5 mg/kg/day triclopyr. This effect is a result of competition between triclopyr and PSP for renal excretion, and is not toxicologically relevant. The systemic NOEL is 2.5 mg/kg/day and the systemic LOEL is > 2.5 mg/kg/day in both sexes.

5. *Chronic toxicity.* In a 1–year dietary toxicity study, triclopyr was administered to dogs at doses of 0, 0.5, 2.5, or 5.0 mg/kg/day. There were no significant effects of treatment on mortality, clinical signs, body weight, or food consumption in male and female dogs at any dose level treated. Increases in urea nitrogen and creatinine were observed at 2.5 and 5.0 mg/kg/day; these changes in clinical chemistry values do not represent a toxic response to the test chemical, but a physiologic response of the dog, based on the limited ability of the dog to excrete organic acids at higher plasma concentrations. The lack of histopathologic alterations in the kidneys of both sexes is supportive of this conclusion. The systemic NOEL is $\geqq$ 5.0 mg/kg/day for both sexes; the systemic LOEL is >5.0 mg/kg/day.

In a chronic toxicity/carcinogenicity study, triclopyr was administered in the diet to mice at dose levels of 0, 50 ppm, 250 ppm, or 1,250 ppm. There were no compound-related tumors observed in mice. The LOEL was considered to be 143 mg/kg/day in male mice and 135 mg/kg/day in female mice, based on the decreased body weight gain. The NOEL is considered to be 28.6 mg/kg/day in male mice, and 26.5 mg/kg/day in female mice.

In a chronic toxicity/carcinogenicity study, triclopyr was administered to Fischer 344 rats for 2 years at dose levels of 0, 3, 12, or 36 mg/kg/day. Mortality in treated groups of male rats was lower than that in the control group. Cumulative mortality was 50%, 32%, 26%, and 36% for control, low, mid, and high dose level male rats. Red cell count, hemoglobin, and hematocrit in male rats was numerically decreased at the high dose at 6, 12, and 24 months. Statistical significance was achieved for the decrease in red cells at 12 months, for hemoglobin at 6 months, and for hematocrit at 6 and 22 months. Absolute and relative kidney weight was significantly increased (10-13%) at the high dose in male rats, with an apparent dose-related trend at 12 months. Female rats showed an increased incidence of pigmentation of the proximal descending tubule at all dose levels compared to control, while male rats in the 6-month satellite group showed increased incidence of proximal tubule degeneration at the 12 and 36 mg/kg/day dose levels compared to control. There were no significant increasing trends in tumor incidence for rats.

As a result of the August 9, 1995 meeting of the Health Effects Division Carcinogenicity Peer Review Committee, triclopyr was classified as a Group D chemical (not classifiable as to human carcinogenicity).

6. *Animal metabolism*. Disposition and metabolism of ^{14}C-triclopyr was investigated in rats at a low oral dose (3 mg/kg), repeated low oral doses (3 mg/kg x 14 days), and a high dose (60 mg/kg.) Comparison of disposition data in intravenously dosed and orally dosed rats demonstrated that triclopyr was well absorbed after oral administration. Excretion was relatively rapid at the low dose, with a majority of radioactivity eliminated in the urine by 24 hours. At 60 mg/kg, urinary elimination of ^{14}C-triclopyr derived radioactivity was decreased in rats from 0-12 hours, due to apparent saturation of renal

elimination mechanisms. Fecal elimination of ^{14}C-Triclopyr derived radioactivity was a minor route of excretion, as was elimination via exhaled air. No significant effect was observed on metabolism or disposition of ^{14}C-triclopyr from repeated low oral dosing.

Urinary metabolites of ^{14}C-triclopyr were isolated and identified by HPLC and GC/MS. Unmetabolized parent chemical represented >90% of urinary radioactivity, with the remainder accounted for by the metabolite 3,5,6-trichloro-2-pyridinol (3,5,6-TCP), and possible glucuranide and/or sulfate conjugates of 3,5,6-TCP. Plasma elimination following intravenous administration of ^{14}C-triclopyr was consistent with a one-compartment model with an elimination half-life of 3.6 hr and zero-order kinetics from 0-12 hours at the 60 mg/kg dose.

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

D. Aggregate Exposure

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

A chronic dietary exposure analysis was performed using tolerance level residues and 100% crop treated information to estimate the Theoretical Maximum Residue Contribution (TMRC) for the general population and 22 subgroups. Existing tolerances result in a TMRC which represents 0.81% of the RfD for the U.S. general population. The highest subgroup, Non-Nursing Infants (<1 year old) occupies 2.65% of the RfD. The chronic analysis for triclopyr is a worse case estimate of dietary exposure with all residues at tolerance level and 100% of the commodities assumed to be treated with triclopyr. Based on the risk estimates calculated in this analysis, it appears that chronic dietary risk from the uses currently registered, is not of concern.

Since the toxicological endpoint to which exposure is being compared in the acute dietary risk analysis is a developmental NOEL (30 mg/kg/day), females (13* years) is the sub population of particular interest. The Margin of Exposure (MOE) is a measure of how close the high end exposure comes to the NOEL (the highest dose at which no effects were observed in the laboratory test), and is calculated as the ratio of the NOEL to the exposure (NOEL/exposure = MOE.) Generally, acute dietary margins of exposure greater than 100 tend to cause no dietary concern. The high end MOE value of 2,500 is above the acceptable level and demonstrates no acute dietary concern.

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

Population Subgroup Exposure	(mg/kgBW/day)	MOE
U.S. Population	0.00230	13050
Females	0.00184	16277

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

ii. *Drinking water*. The use of triclopyr in the proposed EUP does not add any additional exposure of triclopyr to humans. The only additional source that needs to be considered is drinking water. The proposed EUP labeling requires that the product not be applied within one mile of a potable water intake, and treated water is not to be used for domestic purposes for 21 days after application. The basis for these restrictions is a study conducted at Lake Seminole, GA. In this study, triclopyr was not detected one mile downstream from the treated area for up to 42 days after treatment. Within the treatment area, triclopyr was detected at <0.01 - 0.03 ppm in water collected 21 days after application. At 1 hour after application, water from the treated area contained 2.6 ppm of triclopyr, and was below the temporary tolerance level of 0.5 ppm at 3 days after treatment.

If the proposed labeling is followed precisely, that is, potable water is not collected within one mile of a treated area, triclopyr residues will not be detected (<0.01 ppm), and there will be no contribution from water to the ''risk cup'' for triclopyr. If water is collected from the treated area 21 days after treatment and used in drinking water supplies, the maximum residue of 0.03 ppm in the Lake Seminole study would increase the amount of the RfD used for non-nursing infants (<1 yr old) from 2.6 % to 7.0 % for chronic exposure.

For a worst case estimate of potential drinking water exposure, the water residue data from the treated area in the Lake Seminole study was utilized. It was assumed that potable water was collected from the treatment area during the 21 days following the application. The data were integrated over the time period to find an ''average'' value, which calculated to be 0.2 ppm. When this residue level is considered, the following analysis demonstrates the risk is minimal.

Acute NOEL (Pregnant females) = 30 mg/kg/day; Acute NOEL (Children 1-6 years); Chronic NOEL (all population subgroups) = 5 mg/kg/day Time weighted concentration during the mitigation period = 0.2 ppm = 2.0 X 10^{-1} mg/L

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

(2.0 X 10^{-1} mg/L X 1 L/day) / 10 kg = 2.0 X 10^{-2} mg/kg/day MOE = NOEL/Exposure = 5 mg/kg/day / 2.0 X 10^{-2} mg/kg/day MOE = 250

For a 10 kg child consuming 1 Liter a day (Chronic):

Percent of RfD = (2.0 X 10^{-2} mg/kg/day / 0.05) X 100 = 40%

For a 60 Kg pregnant female consuming 2 Liters a day (Acute):

(2.0 x 10^{-1} mg/L X 2 L/day) / 60 kg = 6.7 X 10^{-3} mg/kg/day

MOE = 30 mg/kg/day / 6.7 X 10^{-3} mg/kg/day = 4478

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

Percent of RfD = (6.7 X 10^{-3} mg/kg/day / 0.05) x 100 = 13.4%

2. *Non-dietary exposure*. There are potential exposures to homeowners during usual use-patterns associated with triclopyr. These involve application of triclopyr-containing products by means of aerosol cans, pump spray bottles, squeeze bottles, ''weed sticks,'' hose-end sprayers, power sprayers, paint brush, rotary and

drop spreaders. It is unlikely that power sprayers will be used by homeowners; this is an application method requiring special applicator equipment more apt to be used by agricultural or commercial applicator.

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

E. Cumulative Effects

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

F. Safety Determination

1. *U.S. population*. Because of the toxicological characteristics of triclopyr (no dermal endpoint of concern), post-application exposure assessment was not necessary. Residential exposure is considered to be negligible. Therefore, residential exposure was not considered in the aggregate risk calculation. The water exposure value used the time weighted concentration during the mitigation period = 0.2 ppm = 2.0 X 10^{-1} mg/L in the calculations below for drinking water exposure. The high end (99.5 percentile) exposure from the acute dietary analysis is used for the populations below.

13+ *pregnant females Dietary + Drinking water*

0.0018 mg/kg/day + 6.7 X 10^{-3} mg/kg/day = 8.5 X 10^{-3} mg/kg/day

Acute MOE = 30 mg/kg/day / 8.5 X 10^{-3} mg/kg/day = 3529

Non-nursing infants Dietary + Drinking water

0.006 mg/kg/day + 0.02 mg/kg/day = 2.6 X 10^{-2} mg/kg/day

Acute MOE = 5 mg/kg/day / 2.6 X 10^{-2} mg/kg/day = 192

Children (1-6 years), Dietary + Drinking Water

0.0035 mg/kg/day + 0.02 mg/kg/day = 2.35 X 10^{-2} mg/kg/day

Acute MOE = 5 mg/kg/day/2.35 X 10^{-2} mg/kg/day = 213

Determination of Safety for U.S. Population

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

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

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

The effects noted for the RfD NOEL are parental effects, not developmental. Even using the time weighted concentration during the mitigation period for drinking water risk is minimal.

G. International Tolerances

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

H. Endocrine Effects

An evaluation of the potential effects on the endocrine systems of mammals has not been determined; However, no evidence of such effects were reported in the chronic or reproductive toxicology studies described above. There was no observed pathology of the endocrine organs in these studies. There is no evidence at this time that triclopyr causes endocrine effects. (James Tompkins)

2. DowElanco

PP 4F4379, 8F3600, and 8H5551

EPA has received pesticide petitions (PP) 4F4379 (sweet corn and popcorn) and 8F3600 and 8H5551 (sugar beets) from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268-1054, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR Part 180 by establishing a tolerance for residues of the herbicide clopyralid in or on the raw agricultural commodities (RACs) sweet corn, fodder at 10.0 ppm; sweet corn, forage and cannery waste at 3.0 ppm; sweet corn, grain at 1.0 ppm and kernel plus cob with husks removed (K + CWHR) at 0.5 ppm; and pop corn, fodder at 10.0 ppm, and pop corn, grain at 1.0 ppm; and revising the tolerance for residues of the herbicide clopyralid in or on the raw agricultural commodities sugar beet, roots at 1.0 ppm and sugar beet, tops at 1.0 ppm and on the processed agricultural commodity (PAC) sugar beet, molasses at 8.0 ppm. The proposed analytical method is available for enforcement purposes.

Pursuant to the section 408(d)(2)(A)(i) of the FFDCA, as amended, DowElanco has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by DowElanco and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioners and not necessarily EPAs and to remove certain extraneous material.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism in plants is adequately understood. No metabolites of significance were detected in plant metabolism studies.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of clopyralid in or on food with a limit of quantitation (LOQ) of 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. The method is available to anyone who is interested in pesticide residue enforcement.

3. *Magnitude of residues*— i. *Sugar beets.* Tolerances for residues of the herbicide clopyralid in or on the following raw agricultural commodities, sugar beet roots and tops and the processed agricultural commodity molasses, were established on August 12, 1988 (53 FR 33488, 33489) at 0.5, 0.5, and 7.0 parts per million (ppm), respectively, based upon residue data generated by Craven Laboratories. The validity of these data were in question and DowElanco repeated the residue studies. The last of the required residue data were submitted to the Agency in June 1994. The range of the residues

found for sugar beet, roots was to no detected residues above the LOQ of the method - 0.7 ppm; sugar beet, tops; was to no detected residues above the LOQ of the method - 0.9 ppm; and the residues in the processed agricultural commodities when clopyralid was applied at the maximum labeled rate were 0.5, 0.09, and 6.3 ppm for pulp, sugar and molasses respectively. The proposed revised tolerances would adequately cover these anticipated residues.

ii. *Sweet corn*. Clopyralid was applied at the maximum label rate and residues were detected at the following ppm ranges: Grain, 0.087 - 0.12; Forage, 0.34 - 2.0; Ears (K + CWHR), 0.029 - 0.23 and Cannery Waste; no residues were detected above the LOQ of the method. The proposed tolerances would adequately cover these anticipated residues.

iii. *Pop corn*. Clopyralid was applied at the maximum label rate and residues were detected at the following ppm ranges; Grain: 0.03 - 0.91, Fodder: No detectable residues above the LOQ of the method - 0.60, and Forage 0.14 - 1.2, The proposed tolerances would adequately cover these anticipated residues.

B. Toxicological Profile

1. *Acute toxicity*. Clopyralid has low acute toxicity. The rat oral LD_{50} is 5,000 mg/kg or greater for males and females. The rabbit dermal LD_{50} is greater than 2,000 mg/kg and the rat inhalation LC_{50} is greater than 1.0 mg/L air (the highest attainable concentration). In addition, clopyralid is not a skin sensitizer in guinea pigs and is not a dermal irritant. Technical clopyralid is an ocular irritant but ocular exposure to the technical material would not normally be expected to occur to infants or children or the general public. End use formulations of clopyralid have similar low acute toxicity profiles and most have low ocular toxicity as well. Therefore based on the available acute toxicity data, clopyralid does not pose any acute dietary risks.

2. *Genotoxicity*. Clopyralid is not genotoxic. The following studies have been conducted and all were negative for genotoxic responses. Ames bacterial mutagenicity assay (with and without exogenous metabolic activation); Host-Mediated assay *In vivo* cytogenetic test, rat; *In vivo* cytogenetic test, mouse; *In vivo* dominant lethal test, rat; *In vitro* unscheduled DNA synthesis assay in primary rat hepatocyte cultures; *In vitro* mammalian cell gene mutations assay in Chinese hamster ovary cell cultures (with and without exogenous metabolic activation).

3. *Reproductive and developmental toxicity*. Developmental toxicity was studied using rats and rabbits. The developmental study in rats resulted in a developmental NOEL of >250 mg/kg/day (a maternally toxic dose) and a maternal toxicity NOEL of 75 mg/kg/day. A 1974 study in rabbits revealed no evidence of developmental or maternal toxicity at 250 mg/kg/day; thus the developmental and maternal NOEL was >250 mg/kg/day. A more recent study in rabbits (1990) resulted in developmental and maternal NOELs of 110 mg/kg/day based on maternal toxicity at 250 mg/kg/day. Based on all of the data for clopyralid, there is no evidence of developmental toxicity at dose levels that do not result in maternal toxicity. In a 2–generation reproduction study in rats, pups from the high dose group which were fed diets containing clopyralid had a slight reduction in body weight during lactation and an increase in liver weights in F1a and F1b weanlings. The NOEL for parental systemic toxicity was 500 mg/kg/day. There was no effect on reproductive parameters at >1,500 mg/kg/day nor was there an adverse effect on the morphology, growth or viability of the offspring; thus, the reproductive NOEL is >1500 mg/kg/day.

4. *Subchronic toxicity*. The following studies have been conducted using clopyralid. In a rat 90–day feeding study, Fischer 344 rats were fed diets containing clopyralid at doses of 5, 15, 50, or 150 mg/kg/day with no adverse effects attributed to treatment. In a second study, Fischer 344 rats were fed diets containing clopyralid at doses of 300, 1,500, and 2,500 mg/kg/day. Effects at the highest doses were decreased food consumption accompanied by decreased body weights and weight gains in both males and females. Slightly increased mean relative liver and kidney weights were noted in males of all doses and in females at the top 2 doses. Because there were no other effects, the kidney and liver weight effects were judged as being adaptive rather than directly toxic. The no-observed-adverse-effect level (NOAEL) was 1,500 mg/kg/day for males and females. The no-observed-effect level (NOEL) was 300 mg/kg/day for females. In a mouse 90–day feeding study, B6C3F1 mice were fed diets containing clopyralid at doses of 200, 750, 2,000 or 5,000 mg/kg/day. A slight decrease in body weight occurred at the top dose in both sexes. The liver was identified as the target organ based on slight increases in liver weights and minimal microscopic alterations at the higher dose levels. The liver changes were considered to be reversible and adaptive. The NOEL for males was 2,000 mg/kg/day and for females was 750 mg/kg/day. In a 180–day feeding study, beagle dogs were fed diets containing clopyralid at doses of 15, 50, or 150 mg/kg/day; there were no adverse effects. In a second dietary study, dogs also were fed diets containing clopyralid at doses of 15, 50, or 150 mg/kg/day; the only effect was an increase in the mean relative liver weight in females at the 150 mg/kg/day. In a 21–day dermal study, clopyralid was applied by repeated dermal application to New Zealand White rabbits at dose levels up to 1,000 mg/kg/day. Treatment produced no systemic effects.

5. *Chronic toxicity*. In a chronic toxicity and oncogenicity study, Sprague-Dawley rats were fed diets containing clopyralid at doses of 5, 15, 50 or 150 mg/kg/day. The only effect was a trend toward a decreased body weight of female rats receiving the 150 mg/kg/day dose with a NOEL of 50 mg/kg/day. In a second study clopyralid was fed to Fischer 344 rats in the diet at doses of 15, 150, or 1,500 mg/kg/day. The effects were confined almost entirely to the 1,500 mg/kg/day dose groups and included slightly decreased food consumption and body weights, slightly increased liver and kidney weights and macroscopic and microscopic changes in the stomach. No tumorigenic response was present. The NOEL for this study was 150 mg/kg/day. B6C3F1 mice were maintained for 2 years on diets formulated to provide targeted dose levels of 10, 500, or 2,000 mg/kg/day. The only evidence of toxicity was body weight depression in males dosed at 2,000 mg/kg/day. There was no evidence of tumorigenic response at any dose level. Based on the chronic toxicity data, EPA has established the RfD for clopyralid at 0.5 milligrams (mg)/kilogram (kg)/day. The RfD for clopyralid is based on a 2–year chronic oncogenicity study in rats with a no-observed-effect level (NOEL) of 50 mg/kg/day and an uncertainty (or safety) factor of 100. Thus, it would not be necessary to require the application of an additional uncertainty factor above the hundredfold factor already applied to the NOEL.

6. *Carcinogenicity*. Using its Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), clopyralid would be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of the carcinogenicity studies. There was no evidence of carcinogenicity in 2–year feeding studies in mice and rats at the dosage levels tested. The doses tested are adequate for identifying a cancer

risk. Thus, a cancer risk assessment would not be appropriate.

7. *Animal metabolism*. Disposition and metabolism of clopyralid were tested in male and female rats at a dose of 5 mg/kg (oral). The majority of a radioactive dose was excreted in 24 hours of all dose groups. Fecal elimination was minor. Detectable levels of residual radioactivity were observed in the carcass and stomach at 72 hours post-dose. HPLC and TLC analysis of urine and fecal extracts showed no apparent metabolism of clopyralid.

8. *Metabolite toxicity*. There are no clopyralid metabolites of toxicological significance.

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

C. Aggregate Exposure

1. *From food and feed uses*. For purposes of assessing the potential dietary exposure under these tolerances, exposure is estimated based on the TMRC from the existing and pending tolerances for clopyralid on 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. Exposure of humans to residues could also result if such residues are transferred to meat, milk, poultry or eggs. The following assumptions were used in conducting this exposure assessment: 100% of the crops were treated, the RAC residues would be at the level of the tolerance, certain processed food residues would be at anticipated (average) levels based on processing studies and all current and pending tolerances were included. This results in an overestimate of human exposure and a conservative assessment of risk. Based on a NOEL of 50 mg/kg/day in a 2–year chronic feeding/oncogenicity study in the rat and a hundredfold safety factor, the reference dose (RfD) would be 0.5 mg/kg/day. Consequently, all existing and pending tolerances have a theoretical maximum residue contribution of 0.005135 mg/kgBW/day and would utilize less than 2.3% of the RfD.

2. *From potable water*. Another potential source of dietary exposure to residues of pesticides are residues in drinking water. There is no established Maximum Concentration Level for residues of clopyralid in drinking water. Although there has been limited detections at ppb levels in some of the specially designed studies under highly vulnerable test conditions, no ongoing monitoring studies (U.S. Geological Survey, Selected Water Resources Abstracts, and Pesticides in Ground Water Database - A Compilation of Monitoring Studies: 1971-1991 National Summary; U.S. Department of Agriculture, AGRICOLA database; and, U.S. Department of Commerce, National Technical Information Service) have reported residues of clopyralid in ground or surface waters.

Based on the physical and chemical characteristics of clopyralid, such as water solubility and its stability under hydrolysis and photolysis, it has potential for downward movement through the soil profile. However, the behavior of the compound under field conditions demonstrates fairly rapid degradation and limited downward movement. Degradation based on 20 field dissipation sites indicated an average half-life of 34 days. Degradation is driven primarily by microbial processes. Downward movement through the soil profile was generally confined to the upper 18 inches of the soil profile. Validated computer modeling also predicted the maximum depth of residues to be 18-inches, with no detections predicted at 6 months after application. Because the laboratory derived physical/chemical properties of clopyralid indicate a potential for downward movement, lysimeter studies were conducted. In a U.S. study, undisturbed soil columns (lysimeters), 8 inches in diameter, and 3 feet deep, were treated with 950 g ae/ha (about 5 X labeled use rates) in actual field conditions. Residues of clopyralid in soil as well as soil-solution (leachate) were collected in the closed system. The average depth of movement for the majority of clopyralid (center of mass) was 11 inches, and no detectable residues were observed in the leachate. In a European study, lysimeters 1 - 3 ft. diameter, and 3 ft. deep, were treated with 120 and 240 g ae/ha in actual field conditions. The average center of mass was 12 inches. No detectable residues were observed in the lysimeters. The amount of 14_C in leachate accumulated over 2 years in the degraded loess and silty sand lysimeters, was only 0.6% and 0.3% of applied, respectively. The leachate concentrations of 14_C-labeled clopyralid in degraded loess and silty sand throughout the first 10-16 months of the study ranged from 0.002-0.14 µg/l (ppb) and 0.003-0.02 ppb, respectively. A second European lysimeter study with silty sand lysimeters treated with 120 g ae/ha revealed a 2–year cumulative clopyralid leachate of only 0.1% of applied (0.04 ppb). These studies demonstrate that in lysimeter test systems, under field environmental conditions, clopyralid rapidly dissipates through mineralization to carbon dioxide. Also the very low levels observed in leachate demonstrate that there is very little potential for clopyralid to leach through soil and to contaminate ground water.

In summary, these data on potential water exposure indicate insignificant additional dietary intake of clopyralid and any exposure is more than offset for in the conservative dietary risk evaluation. Therefore, it is concluded that there is a reasonable certainty of no harm even at potential upper limit exposures to clopyralid from drinking water.

3. *From non-dietary uses*. There is only one non-dietary use registered under the Federal Insecticide, Fungicide and Rodenticide Act. The use is for weed control in residential turf. Potential exposures for children from non-occupational uses is therefore limited to turf re-entry and this exposure is low.

4. *Short-term or intermediate-term*. The data for clopyralid does not indicate any evidence of significant toxicity by the dermal and inhalation routes. Consequently, there is no concern for short-term or intermediate-term residential risk. Therefore, a short-term or intermediate-term residential risk assessment would not be required.

5. *Chronic*. As part of a hazard assessment process an endpoint of concern is determined for the chronic occupational or residential risk assessment. However, as indicated, the exposures that would result from the use of clopyralid are of an intermittent nature. The frequency and duration of these exposures do not exhibit a chronic exposure pattern. The exposure does not occur often enough to be considered a chronic exposure; i.e., a continuous exposure that occurs for a least several months. Therefore, it would not be appropriate to aggregate exposure from the residential use with exposure from food and drinking water.

6. *Acute*. No concern would exist for an acute dietary assessment for clopyralid because the available data indicates no evidence of significant toxicity from a one day or single event exposure by the oral route. Therefore, an acute dietary risk assessment would not be required.

D. Cumulative Exposure to Substances with Common Mechanism of Toxicity

The potential for cumulative effects of clopyralid and other substances that have a common mechanism of toxicity was considered. The mammalian toxicity of clopyralid is well defined. However, no reliable information exists to indicate that toxic effects produced

by clopyralid would be cumulative with those of any other chemical compound. Additionally, clopyralid does not appear to produce a toxic metabolite produced by other substances. Therefore, consideration of a common mechanism of toxicity with other compounds is not appropriate at this time. Thus only the potential exposures to clopyralid were considered in the aggregate exposure assessment.

E. Determination of Safety

1. *U.S. population in general.* Based on a NOEL of 50.80 mg/kg/bwt/day from a 2–year rat feeding study with a decreased mean body weight gain effect, and using an uncertainty factor of 100 to account for the interspecies extrapolation and intraspecies variability, a Reference Dose (RfD) of 0.5 mg/kg bwt/day was used for this assessment of chronic risk. As indicated, there is no endpoint of concern identified with acute and short- or intermediate-term exposures. Based on the known toxicity and exposure data, the proposed and existing tolerances would utilize approximately 2% of the RfD for the U.S. population. And, as indicated previously, whatever upper limit might be used for drinking water exposure, the exposure estimate for clopyralid would not exceed the RfD. Generally, exposures below 100% of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to clopyralid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of clopyralid, data from developmental toxicity studies in the rat and rabbit and a 2–generation reproduction study in the rat were considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism during prenatal development resulting from pesticide exposure to one or both parents. Reproduction studies provide (1) information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and (2) data on systemic toxicity.

Developmental toxicity was studied using rats and rabbits. The developmental study in rats resulted in a developmental NOEL of >250 mg/kg/day (a maternally toxic dose) and a maternal toxicity NOEL of 75 mg/kg/day. A 1974 study in rabbits revealed no evidence of developmental or maternal toxicity at 250 mg/kg/day; thus the developmental and maternal NOEL was >250 mg/kg/day. A more recent study in rabbits (1990) resulted in developmental and maternal NOEL's of 110 mg/kg/day based on severe maternal toxicity at 250 mg/kg/day. Based on all of the data for clopyralid, there is no evidence of developmental toxicity at dose levels that do not result in maternal toxicity.

In a 2–generation reproduction study in rats, pups from the high dose group which were fed diets containing clopyralid had a slight reduction in body weight during lactation and an increase in liver weights in F1a and F1b weanlings. The NOEL for parental systemic toxicity was 500 mg/kg/day. There was no effect on reproductive parameters at >1500 mg/kg/day nor was there an adverse effect on the morphology, growth or viability of the offspring; thus, the reproductive NOEL is >1,500 mg/kg/day.

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 for children is complete. These data suggest minimal concern for developmental or reproductive toxicity and do not indicate any increased pre- or post-natal sensitivity. Therefore, an additional uncertainty factor is not necessary to protect the safety of infants and children and that the RfD at 0.5 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

The percent of the RfD that will be utilized by the aggregate exposure from all tolerances to clopyralid will be much less than 10% for non-nursing infants and for children (1 - 6 years of age). Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to clopyralid residues.

F. International Tolerances

There are no Codex maximum residue levels established for clopyralid. (Joanne Miller)

3. E.I. DuPont Company

PP 4F4391

In the **Federal Register** of October 25, 1995, (60 FR 54607), EPA established a time-limited tolerance pursuant to the Federal Food Drug and Cosmetic Act (FFDCA) for residues of the herbicide pyrithiobac sodium salt (sodium 2-chloro-6-[(4,6-dimethoxypyrimidin-2-yl)thio]benzoate) in or on the raw agricultural commodity cottonseed at 0.02 part per million (ppm). The time-limited tolerance expires September 30, 1997. The tolerance was requested in pesticide petition (PP) 4F4391 by E. I. DuPont de Nemours and Co., Inc. (DuPont), Barley Mill Plaza, P.O. Box 80083, Wilmington, DE 19880-0038. The tolerance was issued as a time-limited tolerance because EPA required additional residue data on the commodity of cotton gin byproducts. The petitioner proposes to renew the time-limited tolerance for a 2–year period and retain the pesticide labeling previously accepted under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA), as amended, which bears a restriction against feeding cotton gin byproducts from treated fields to livestock. DuPont has requested this tolerance extension pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, as amended, 21 U.S.C. 346a(d), by the Food Quality Protection Act of 1996 (Pub. L. 104-170, 110 Stat. 1489). The request addresses the requirements of the new FFDCA Section 408(d)(2). The time-limited tolerance would expire on September 30, 1998. An adequately validated analytical method is available for enforcement purposes. Pursuant to section 408(d)(2)(A)(i) of the FFDCA, as amended, DuPont has submitted the following summary of information, data and arguments in support of its pesticide petition. This summary was proposed by DuPont and EPA has not yet fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments presented are those of the petitioner and not necessarily EPA's and to remove certain extraneous material.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residues of pyrithiobac sodium in cotton is adequately understood. Metabolism studies with pyrithiobac sodium indicate the major metabolic pathway being o-dealkylation of the parent compound resulting in o-desmethyl pyrithiobac sodium (O-DPS). O-DPS, both free and conjugated, was the major metabolite identified in cotton foliage. The results of a confined crop rotation study with pyrithiobac sodium revealed the presence of a metabolite 2-chloro-6-sulfobenzoic acid (CSBA) not seen in the cotton metabolism study. This metabolite appeared to originate from soil metabolism of pyrithiobac sodium. Since preemergence applications of pyrithiobac sodium are allowed, crop residues of CSBA were

considered a possibility. In consideration of PP 4F4391 CBTS, in consultation with the HED Metabolism Committee has previously concluded that for the proposed use on cotton, none of the pyrithiobac sodium metabolites including O-DPS and CSBA warrant inclusion in the tolerance regulation, and that the only residue of concern is the parent, pyrithiobac sodium.

2. *Analytical method*. There is a adequately validated practical analytical method available using HPLC-UV with column switching, to measure levels of pyrithiobac sodium in or on cotton with a limit of quantitation that allows monitoring of cottonseed at or above tolerance levels. EPA has provided information on this method to FDA for future publication in PAM II.

3. *Magnitude of residues*. Crop field trial residue data from a 60 day PHI study shows that the established pyrithiobac sodium time-limited tolerance on cottonseed of 0.02 ppm will not be exceeded when DuPont Staple Herbicide is used as directed. An adequate cottonseed processing study shows that pyrithiobac sodium does not concentrate in cottonseed processed commodities; thus no tolerances on these commodities are required.

B. Toxicological Profile

1. *Acute toxicity*. Pyrithiobac sodium technical has been placed in EPA Toxicity Category II for acute eye irritation based on the test article inducing irritation in the form of corneal opacity, iritis and conjunctival redness, and discharge in the eyes of rabbits after receiving ocular doses of 36 mg (0.1 ml). Signs of irritation were clear within 14 days of treatment. Pyrithiobac sodium has been placed in Toxicity Category III for acute dermal toxicity based on the test article being nonlethal and nonirritating at the limit dose of 2,000 mg/kg (highest dose tested). Pyrithiobac sodium has been placed in Toxicity Category III for acute oral toxicity based on acute oral LD_{50}s of 3,200 mg/kg for both male and female rats. Pyrithiobac sodium has been placed in Category IV for the remaining acute toxicity tests based on the following: a rat acute inhalation study with an LC_{50} of >6.9 mg/l; and a primary dermal irritation test that did not induce a dermal irritation response. A dermal sensitization test with pyrithiobac sodium technical in guinea pigs demonstrated no significant effects. Based on these results, pyrithiobac sodium does not pose an acute dietary or exposure risk.

2. *Genotoxicty*. Pyrithiobac sodium technical was negative (non-mutagenic and non-genotoxic) in the following tests: Ames microbial mutation assay; the hypoxanthine-guanine phosphoribosyl transferase gene mutation assay using Chinese hamster ovary cells; and induction of unscheduled DNA synthesis (UDS) in primary rat hepatocytes. Pyrithiobac sodium was positive in an *in vitro* assay for chromosome aberrations in human lymphocytes. It was negative for the induction of micronuclei in the bone marrow cells of male and female CD-1 mice administered the test article by oral gavage at 500, 1,000 or 2,000 mg/kg. Based on the weight of these data, pyrithiobac sodium is neither genotoxic nor mutagenic.

3. *Reproductive and developmental toxicity*. A 2–generation, 4 litter reproduction study with CD rats treated at dietary levels of 0, 25, 1,500, 7,500 or 20,000 ppm of pyrithiobac sodium demonstrated a maternal NOEL of 1,500 ppm (103 mg/kg/day) and a maternal LOEL of 7,500 ppm (508 mg/kg/day), based on decreased body weight gain and food efficacy. An offspring NOEL of 7,500 ppm (508 mg/kg/day) and LOEL of 20,000 ppm (1,551 mg/kg/day) were also demonstrated based on decreased offspring body weight. Pyrithiobac sodium was not teratogenic when administered to rats or rabbits. A developmental toxicity study with pyrithiobac sodium in rats demonstrated a maternal NOEL of 200 mg/kg and LOEL of 600 mg/kg due to increased incidence of salivation. A developmental NOEL of 600 mg/kg and LOEL of 1,800 mg/kg were demonstrated based on an increased incidence of skeletal variations. A developmental toxicity study with pyrithiobac sodium in rabbits demonstrated maternal and developmental NOELs of 300 mg/kg and a maternal LOEL of 1,000 mg/kg based on mortality, decreased body weight gain and feed consumption, increased incidence of clinical signs, and an increase in early resorptions. A developmental LOEL of 1,000 mg/kg was based on decreased fetal body weight gain. Based on the weight of these data, pyrithiobac sodium is not considered a reproductive or developmental hazard. In addition, there were no effects observed in offspring in the absence of maternal toxicity; therefore, the offspring were not uniquely susceptible to the effects of compound administration.

4. *Subchronic toxicity*. In a 90-day feeding study in rats conducted with pyrithiobac sodium at dietary levels of 0, 10, 50, 500, 7,000 and 20,000 ppm, the NOEL was 500 ppm (31.8 and 40.5 mg/kg/day, M/F) and the LOEL was 7,000 ppm (466 and 588 mg/kg/day, M/F) based on decreased body weight gains and increased rate of hepatic B-oxidation in males. In a 90-day feeding study in mice conducted with pyrithiobac sodium at dietary levels of 0, 10, 50, 500, 1,500 and 7,000 ppm, the NOEL was 500 ppm (83.1 and 112 mg/kg/day, M/F) and the L0EL was 1,500 ppm (263 and 384 mg/kg/day, M/F) based on increased liver weight and increased incidence of hepatocellular hypertrophy in males and decreased neutrophil count in females. In a 90-day feeding study in dogs conducted with pyrithiobac sodium at dietary levels of 0, 50, 5,000, or 20,000 ppm, the NOEL was 5,000 ppm (165 mg/kg/day) and the LOEL was 20,000 ppm (626 mg/kg/day) based on decreased red blood cell count, hemoglobin, and hematocrit in females and increased liver weight in both sexes. In a 21-day dermal study with rats conducted with pyrithiobac sodium at exposure levels of 0, 50, 500, or 1,200 mg/kg/day, the dermal irritation NOEL was 500 mg/kg/day and the dermal irritation LOEL was 1,200 mg/kg/day. There were no systemic effects observed at this high dose; therefore, the systemic NOEL is considered to be 1,200 mg/kg/day.

5. *Chronic toxicity*. A 1-year feeding study in dogs conducted with pyrithiobac sodium at dietary levels of 0, 100, 5,000, and 20,000 ppm resulted in a NOEL of 5,000 ppm (143 and 166 mg/kg/day, M/F) and a LOEL of 20,000 ppm (580 and 647 mg/kg/day, M/F) based on decreases in body weight gain and increased liver weight. A 78-week oncogenicity study in mice was conducted with pyrithiobac sodium at dietary levels of 0, 10, 150, 1,500 and 5,000 ppm. The systemic NOEL is 1,500 ppm (217 and 319 mg/kg/day, M/F) and the LEL is 5,000 ppm (745 and 1,101 mg/kg/day, M/F), based on decreased body weight gain and liver lesions. Kidney effects were also observed at 5,000 ppm; however, these were present at low incidence and were of minimal severity and were considered to be of only minimal biological significance. Increased incidence of foci/focus of hepatocellular alteration was observed in males fed 5,000 ppm diets. Increased incidences of hepatocellular neoplasms (adenomas or adenomas plus carcinomas) were observed only in 150 and 1,500 ppm males. The incidence of these liver tumors was not significantly increased in the 5,000 ppm males or in females at any dose level; the 5,000 ppm male tumor incidence was within the historical control range. A 2-year study in rats was conducted at dietary pyrithiobac sodium levels of 0, 5, 25,

1,500 or 5,000 ppm for males and 0, 5, 25, 5,000 or 15,000 ppm for females. The NOEL for systemic effects was 1,500 ppm (58.7 mg/kg/day) for males and 5,000 ppm (278 mg/kg/day) for females. The LEL was 5,000 ppm (200 mg/kg/day for males)/15,000 ppm (918 mg/kg/day) for females. The LEL was based on the following: decreased body weight, body weight gain and food efficiency (for females); mild changes in hematology and urinalysis, clinical signs indicative of urinary tract dysfunction (both sexes); increased incidence of focal cystic degereration in the liver and increased rate of hepatic peroxisome beta-oxidation (males); and an increased incidence of inflammatory and degenerative microscopic lesions in the kidney (females). There was evidence of oncogenicity based on an increased trend for kidney tubular combined adenoma/ carcinoma in male rats and an increased trend for kidney tubular adenomas in female rats. Although the incidences were low, they were statistically significant. The highest dose level tested in male rats (5,000 ppm) was considered adequate for assessment of oncogenic potential, that in female rats (15,000 ppm) exceeded the Maximum Tolerated Dose (MTD).

6. *Carcinogenicity*. In consideration of PP 4F4391 the HED Carcinogenicity Peer Review Committee has previously concluded that the available data provide limited evidence of the carcinogenicity of pyrithiobac sodium in mice and rats and has classified pryithiobac sodium as a Group C (possible human carcinogen with limited evidence of carcinogenicity in animals) in accordance with Agency guidelines published in the **Federal Register** in 1986 (51 FR 33992, September 24, 1986) and recommend that for the purpose of risk characterization a low-dose extrapolation model should be applied to the experimental animal tumor data for quantification for human risk (Q1*). This decision was based on liver adenomas, carcinomas and combined adenoma/carcinomas in the male mouse and kidney tubular adenomas, carcinomas and combined adenoma/ carcinomas in the male rat. The unit risk, Q1* (mg/kg/day)-1, of pyrithiobac sodium is 1.05 x 10-3 (mg/kg/day)-1 in human equivalents based on male kidney tumors.

7. *Animal metabolism*. Disposition and metabolism of pyrithiobac sodium were tested in male and female rats using two radiolabeled forms of pyrithiobac sodium. Either phenyl-labeled or pryimidine-labeled compounds were administered orally at 5 or 250 mg/kg. In addition, i.v. administration was evaluated at 5 mg/kg. Essentially all of the dose was excreted in the urine and feces, with greater than 90% being excreted within 48 hours. No label was detected in the expired air. Only minute quantities of radioactivity (at or near the limit of detection) were detected in the major organs of metabolism and excretion. This study indicates that pyrithiobac sodium has low toxicity and does not accumulate within the body. The major compound eliminated in urine and feces was O-DPS (desmethyl metabolite), formed by demethylation of the pyrimidine ring. There was evidence that conjugation with glucuronic acid and 5-hydroxylation of the pyrimidine ring of pyrithiobac sodium were additional minor routes of metabolism in the rat.

8. *Metabolite toxicology*. There is no evidence that the metabolites of pyrithiobac sodium as identified in either the plant metabolism, confined crop rotation, or animal metabolism studies are of any toxicological significance.

9. *Neurotoxicity*. A 90–day rat neurotoxicity screen battery conducted with pyrithiobac sodium resulted in a systemic NOEL of 7,000 ppm (466 and 588 mg/kg/day, M/F) and a systemic LOEL of 20,000 ppm (1,376 and 1,609 mg/kg/day, M/F) based on reduced body weight gain and food efficiency and increased liver weight. Slight reductions in hind-leg grip strength and slightly increased foot splay in males were observed in 20,000 ppm males. However, because these were of small magnitude, lacked statistical significance and corresponding histopathology, pyrithiobac sodium was not considered a neurotoxin. The NOEL for neurotoxicity was 20,000 ppm [highest dose tested (HDT)].

10. *Endocrine effects.* No special studies investigating potential estrogenic or other endocrine effects of pyrithiobac sodium have been conducted. However, the standard battery of required toxicology studies has been completed and found acceptable. These 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 to doses that far exceed likely human exposures. Based on these studies there is no evidence to suggest that pyrithiobac sodium has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure under this tolerance, an estimate of aggregate exposure is made using the tolerance on cottonseed at 0.02 ppm. The potential exposure is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of cottonseed products translated as cottonseed meal and cottonseed oil eaten by various population subgroups. Cottonseed is fed to animals, thus exposure of humans to residues of cottonseed might result if such residues are transferred to meat, milk, poultry, or eggs. However, in consideration of PP 4F4391 CBTS has previously concluded that secondary residues in meat, milk, poultry and eggs are not expected from the use of cottonseed (undelinted) as an animal feed. There are no other established tolerances or registered uses for pyrithiobac sodium in the United States. Based on a NOEL of 58.7 mg/kg/day, from the chronic rat toxicity study and a hundredfold safety factor, the reference dose (RfD) is 0.58 mg/kg/day. Assuming residues at tolerance levels and that 100% of the crop is being treated, a theoretical maximum residue contribution (TMRC) of <0.000001 mg/kg/day is calculated. With the above assumptions which clearly overestimate potential human exposure and are a most conservative assessment of risk, dietary (food) exposure to pyrithiobac sodium will utilize significantly less than 1% of the RfD for the overall US population. For the most highly exposed subgroup, children aged 1 to 6 years, the TMRC is 0.000001 mg/kg/day, which is still less than 1% of the RfD. The unit risk, $Q1^*$ (mg/kg/day)-1, of pyrithiobac sodium is 1.05×10^{-3} (mg/kg/day)-1 in human equivalents based on male kidney tumors. Based on this upper bound potency factor ($Q1^*$), a 70-year life-span, and the assumption that 100% of the crop is treated with pyrithiobac sodium, the upper-bound limit of a dietary carcinogenic risk is calculated in the range of 1 incidence in a billion (1.0×10^{-9}).

ii. *Drinking water*. Other potential dietary sources of exposure of the general population to pesticides are residues in drinking water. There is no Maximum Contaminant Level established for residues of pyrithiobac sodium. The petitioner has reported to the Environmental Fate and Groundwater Branch of EPA (EFGWB) the interim results of a prospective groundwater monitoring study conducted at a highly vulnerable site. In consideration of this information in support of PP 4F4391 EFGWB has previously concluded by preliminary evaluation, that pyrithiobac sodium may

not be stable enough to leach to groundwater at most use sites, even in sandy soils. All other environmental fate data requirements for pyrithiobac sodium have been satisfied and based on these studies and the conditions of use, the potential for finding pyrithiobac sodium residues in drinking water is minimal.

2. *Non-dietary exposure*. Pyrithiobac sodium is not registered for any use which could result in non-occupational, non-dietary exposure to the general population.

D. Cumulative Effects

Pyrithiobac sodium is based on a new chemical class; there are no known registered herbicides with similar structure. Therefore, EPA should consider only the potential risks of pyrithiobac sodium in its exposure assessment. The herbicidal activity of pyrithiobac sodium is due to the inhibition of acetolactate synthase (ALS), an enzyme only found in plants. ALS is part of the biosynthetic pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the low toxicity of pyrithiobac sodium in animals. There is no evidence to indicate or suggest that pyrithiobac sodium has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population*. Based on a complete and reliable toxicity database, the EPA has adopted an RfD value of 0.58 mg/kg/day using the NOEL of 58.7 mg/kg/day, from the 2-year chronic toxicity study in rats and a hundredfold safety factor. Using crop tolerance levels and assuming 100% of the crop being treated a Theoretical Maximum Residue Contribution (TMRC) was calculated for the overall US population and 22 population subgroups. This analysis concluded that aggregate exposure to pyrithiobac sodium will utilize significantly less that 1 percent of the RfD for either the entire U.S. population or any subgroup population. The TMRC for the most highly exposed subgroup identified as children aged 1 thru 6 years was 0.000001 mg/kg/day. EPA generally has 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 risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to pyrithiobac sodium residues. The unit risk, $Q1^*$ (mg/kg/day)-1, of pyrithiobac sodium is 1.05×10^{-3} (mg/kg/day)-1 in human equivalents based on male kidney tumors. Based on this upper bound potency factor ($Q1^*$) and assuming a 70 year lifetime exposure an upper-bound limit of a dietary carcinogenic risk is calculated in the range of 1 incidence in a billion (1.0×10^{-9}). This indicates a negligible cancer risk.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of pyrithiobac sodium, data from the previously discussed developmental and reproduction toxicity studies were considered. Developmental studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during pre-natal 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. Based on the weight of these data, pyrithiobac sodium was not a reproductive toxicant. Maternal and developmental effects (NOEL's, LOEL's) were comparable indicating no increase in susceptibility of developing organisms. No evidence of endocrine effects were noted in any study. 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 current toxicological data requirements, the database for pyrithiobac sodium relative to pre- and post-natal effects for children is complete. The NOEL of 58.7 mg/kg/day from the 2-year rat study with pyrithiobac sodium, which was used to calculate the RfD, is lower than any of the NOEL's defined in the developmental and reproductive toxicity studies with pyrithiobac sodium. When the weight of these facts is considered an additional safety factor is not warranted for developmental effects. As stated above, aggregate exposure assessments utilized significantly less than 1% of the RfD for either the entire U.S. population or any of 22 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 pyrithiobac sodium residues.

F. International Tolerances

There are no established Codex MRLs for pyrithiobac sodium on cottonseed. An established Mexican tolerance for pyrithiobac sodium on cottonseed is identical to the U.S. tolerance. Compatibility is not a problem at this time. (James Tompkins)

4. Zeneca AG

PP 5F4588

EPA has received a pesticide petition (PP 5F4588) 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, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for combined residues of the insecticide lambda-cyhalothrin and its epimer in or on the raw agricultural commodities (RACs) alfalfa forage at 5.0 parts per million (ppm), alfalfa hay at 6.0 ppm, leaf lettuce at 2.0 ppm, head and stem Brassica crop subgroup at 0.4 ppm, aspirated grain fractions at 2.0 ppm and increasing the existing time-limited tolerance for poultry fat to 0.03 ppm. The proposed analytical method is gas liquid chromatography with an electron capture detector.

Pursuant to section 408 (d) (2) (A) (i) of the FFDCA, as amended, Zeneca Ag Products has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by Zeneca and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioner's and not necessarily EPA's.

A. Residue Chemistry

1. *Plant Metabolism*. The metabolism of lambda-cyhalothrin in plants is adequately understood for this use. Any secondary residues occurring in meat and meat by-products will be covered by the existing tolerances with the exception of the fat of poultry, which is discussed under Magnitude of Residues.

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

3. *Magnitude of residues*—i. *Alfalfa*. Sixteen field trials were carried out on alfalfa forage and hay in twelve states during 1990 in the USA. The trials were conducted in the states of Arizona, California, Iowa, Idaho, Kansas, Michigan, Minnesota, Montana, Nebraska, New York, South Dakota, and Wisconsin. The number and geographical distribution of the trials agrees with the recommendation given in the ''EPA Residue Chemistry Guidance'' (1994).

In these trials, the maximum combined residues of lambda-cyhalothrin and epimer in or on alfalfa

forage is 5.0 ppm and alfalfa hay is 6.0 ppm.

ii. *Leaf lettuce*. Eight field trials were carried out on leaf lettuce in eight states during 1990 in the USA. The trials were conducted in Arizona, California, Colorado, Florida, Michigan, New York, Texas, and Washington. The number and geographical distribution of the trials agrees with the recommendation given in the ''EPA Residue Chemistry Guidance'' (1994).

In these trials, the maximum combined residues of lambda-cyhalothrin and epimer in or on leaf lettuce is 1.8 ppm.

iii. *Head and stem Brassica crop subgroup*. No additional residue crop field data were conducted for the head and stem Brassica crop subgroup. The tolerance request is based on existing data and the existing time-limited tolerances for combined residues of lambda-cyhalothrin and epimer in or the Brassica crops, cabbage, and broccoli at 0.4 ppm.

iv. *Aspirated grain fractions*. The existing tolerance for wheat grain dust at 2.0 ppm is being revised to read ''aspirated grain fractions'' at the same tolerance level. This change reflects Agency policy to establish grain dust tolerances in terms of aspirated grain fractions which include a mixture of all aspirated grains for which the pesticide has a tolerance and is established at the highest current tolerance for any grain dust.

v. *Poultry fat*. Alfalfa forage, hay, meal and silage are animal feed items for beef and dairy cattle. Alfalfa meal is a feed item for poutry and swine. No feed items are involved with the proposed uses on leaf lettuce and the head and stem Brassica crop subgroup. Based on calculated realistic worst case secondary dietary burdens for animal commodities, the maximum calculated residues expected for the fat of poultry is 0.0225 ppm compared to the existing tolerance of 0.01 ppm.

B. Toxicological Profile

The following toxicity studies have been conducted to support the request for a regulation for residues of lambda-cyhalothrin in or on rice.

1. *Acute toxicity*. Acute toxicity studies with the technical grade of the active ingredient lambda-cyahothrin: oral LD_{50} in the rat of 79 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. *Genotoxicity*. 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 3-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 (no-observed effect level) and LOEL (lowest observed effect level) 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 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.

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 21-day study in rabbits exposed dermally to doses of 0, 10, 100, and 1,000 mg/kg/day, 6 hours/day, 5 days/week with a systemic NOEL >1,000 mg/kg/kg. There were no clinical signs of systemic toxicity at any dose level tested.

5. *Chronic toxicity*. A 12-month feeding study 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 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 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 with slow elimination. Otherwise, lambda-cyhalothrin was rapidly metabolized and excreted. The metabolism of lambda-cyhalothrin in livestock is also adequately understood for the proposed use on alfalfa.

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.

C. Aggregate Exposure

1. *Dietary exposure*—i *Food*. For the purposes of assessing the potential dietary exposure for all existing and pending tolerances for lambda-cyhalothrin, Zeneca has utilized available information on anticipated residues and percent crop treated. For all existing and pending tolerances the Anticipated Residue Contribution (ARC) is estimated at 0.000310 mg/kg/bwt/day.

ii. *Drinking water*. Laboratory and field data have demonstrated that lambda-cyhalothrin and its degradates are immobile in soil and will not leach into groundwater. 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.

2. *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. Zeneca has no data upon which to estimate exposure from these

uses. However, given the extremely low vapor pressure of lambda-cyhalothrin (1.5×10^{-9} millimeters of Hg) and the low use rates, it is anticipated that inhalation and dermal exposure from these uses will be insignificant.

D. Cumulative Effects

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 these tolerances it is appropriate only to consider the potential risks of lambda-cyhalothrin in an aggregate exposure assessment.

E. Safety Determination

The acceptable Reference Dose (RfD) based on a NOEL of 0.1 mg/kg/body weight/day from the chronic dog study and a safety factor of 100 is 0.001 mg/kg/body weight/day. A chronic dietary exposure/risk assessment has been performed for lambda-cyhalothrin using the above RfD. Available information on anticipated residues and percent crop treated was incorporated into the analysis to estimate the Anticipated Residue Contribution (ARC) for all existing and the proposed tolerances. The ARC is generally considered a more realistic estimate than an estimate based on tolerance level residues.

1. *US population.* The ARC from established tolerances and the current and pending actions are estimated to be 0.000310 mg/kg/bwt/day and utilize 31.04 per cent of the RfD for the U.S. population.

2. *Infants and children.* The ARC for children, aged 1 to 6 years old, and nonnursing infants (subgroups most highly exposed) utilizes 60 and 67% 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.

F. International Tolerances

There are no Codex maximum residue levels [MRL] established for residues of lambda-cyhalothrin in or on alfalfa hay, forage, leaf lettuce, or Brassica crop subgroup. (George LaRocca)

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BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–741; FRL–5723–1]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–741, must be received on or before August 11, 1997.

ADDRESSES: By mail submit written comments to: Public Response and Program Resources Branch, Field Operations Division (7505C), 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 by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

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

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

Product Manager	Office location/telephone number	Address
George LaRocca (PM 13).	Rm. 204, CM #2, 703–305–6100, e-mail:.@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Mary Waller (PM 21)	Rm. 265, CM #2, 703–308–9354, e-mail:waller.mary@epamail.epa.gov.	Do.
Cynthia Giles-Parker (PM 22).	Rm. 229, CM #2, 703–305–5540, e-mail: giles-parker.cynthia@epamail.epa.gov.	Do.

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

The official record for this notice, as well as the public version, has been established for this notice of filing under docket control number PF–741 (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''.

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 control number PF–741 and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

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

List of Subjects

Environmental protection, Agricultural commodities, Food